RECENT DEVELOPMENTS IN

ALCOHOLISM

VOLUME 1

RECENT DEVELOPMENTS IN

Edited by

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ALCOHOLISM

VOLUME 1

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Current Concepts in Diagnosis

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Preface

The purpose of this series is to provide an overview of recent research developments in the field of alcoholism so that interested professionals and researchers may keep abreast of this complex, multidisciplinary work. These annual volumes will present a scholarly review and analysis of selected research topics prepared by leading figures in the field. Where appropriate, the attempt is made to present contrasting perspectives and views, particularly on issues where there is ongoing controversy.

The American Medical Society on Alcoholism and the Research Society on Alcoholism have undertaken this collaborative venture because of the perceived need for such a comprehensive resource. These groups are both component organizations of the National Council on Alcoholism, a broad-based coalition which supports alcoholism treatment, training, and research on a national and international level. This professional network has enabled us to draw on a panel of Associate Editors and on authors of international prominence. The series should reflect a sophistication that will allow it to serve as a standard reference for the field.

Each volume will include four sections, each addressing an important area of recent research developments. In this first volume, we have elected to focus on the following topics. *The Role of Genetics in the Expression of Alcoholism* is much better understood in recent years because of a sizable body of research on the nature of genetic markers, twin and adoption studies, and pharmacogenetics. Under section editor Henri Begleiter, these findings are also contrasted with a perspective on the etiologic role of environmental issues. *The Behavioral Treatment of Alcoholism* has evolved considerably over the past two decades. A section on this topic edited by Edward Gottheil defines the theoretical base for this work and the behavioral techniques applied. Research on treatment outcome is then reviewed. Because there are contrasting views on this approach, four experts from diverse backgrounds have been invited to place this work into a multidisciplinary perspective.

Prevention strategies are of central importance to the alcoholism field. Because of this, we elected to consider the role of certain *Social Mediators of Alcohol Problems,* which are now subject to government controls. This section, edited by Alfonso Paredes, reviews techniques for ascertaining the prevalence of alcohol problems. Prevalence levels are then considered in light of alcohol availability and cost, and restraints on its purchase by youth. Our final section deals with *Current Concepts in the Diagnosis of Alcoholism.* To update the work in this area, we have focused on recent developments in standardized tests for alcoholism, the development of typologies of alcoholism, and the neuropsychological assessment of the alcoholic.

This volume addresses issues drawn from the full breadth of the alcoholism field, from pharmacogenetics to diagnosis and epidemiology. It should therefore serve as a valuable introduction to the sophisticated clinician or investigator who has a limited acquaintance with current work in the field. On the other hand, for the alcoholism expert, it provides valuable and diverse material to put in focus the most recent research in a number of areas vital to progress in the field. In this respect, it helps promote the roles of the Medical and Research Societies on Alcoholism, which sponsor this undertaking, to advance our understanding of the causes of alcoholism and to promote effective treatment.

The editors wish to express appreciation to the American Medical Society on Alcoholism and the Research Society on Alcoholism and their officers for providing assistance and leadership in the inception of this book series. In addition, the Commonwealth Fund has given invaluable and thoughtful support for this work as part of its book award program.

Marc Galanter, M.D.

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I

The Role of Genetics in the Expression of Alcoholism

Henri Begleiter, Section Editor

Overview

Donald W. Goodwin

In 1944, as that year's session of the Yale Summer School of Alcohol Studies was coming to its conclusion, E. M. Jellinek was asked the following question:

"Sir, you referred to a sample of over 4300 inebriates of whom 52% came from alcoholic parents. What was the extent of the alcoholism of the parents?"

Replied Jellinek: "The alcoholism in those parents was real honest-to-goodness inebrity, . . . but this does not mean the alcoholism was transmitted biologically. It was transmitted socially."[1]

How did this least dogmatic of alcohologists come to such a dogmatic conlusion? He never said. However, from his other comments that day, one can suggest several possibilities.

Jellinek recognized that alcoholism ran in families but was equally impressed by how many families it did not run in. Somehow this suggested to him that "if a hereditary constitutional factor is present, it does not become operative without intercurrent social factors."

Second, Jellinek indeed disliked dogmas. One of the dogmas of the day, held by almost everybody, was that alcoholism was hereditary. Since there was almost no evidence for this except for the familialness of the condition, Jellinek rebelled, although the rebellion might have gone a little far.

Third, Jellinek fell back on a non sequitur that one still hears on the alcoholism lecture circuit: If alcoholism is hereditary, it cannot be treated. If it cannot be treated, it must not be hereditary. Jellinek surely perceived the illogic of this but still believed that therapists should not be thwarted by "implacable fate," i.e., heredity.*

* That Jellinek had mixed feelings about alcoholism and heredity was evident when he came up with a second non sequitur of even greater beauty: "The only permissible conclusion is that not a disposition toward alcoholism is inherited but rather a constitution involving such instability as does not offer sufficient resistance to the social risk of inebriety."[1] No better example of having one's cake and eating it can be found in the alcoholism literature.

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This is nonsense, of course, since many hereditary illnesses are treatable, and treatability is not relevant to the issue of etiology in the first place.

Still, Jellinek knew what questions to ask. If anything is inherited, what is it? Does it involve tolerance? Are there internal "musts" about alcoholism that must be present for the illness to appear? Is a predisposition to alcoholism, a "readiness to acquire the disease," reflective of a specific proneness to alcohol abuse or a manifestation of general psychological proclivities that favor the development of alcoholism if no other "escape" is available?[1]

The chapters in this section deal in one way or another with these questions.

Since Jellinek's talk, even more evidence that alcoholism is a family disorder has accumulated. One reviewer found some 140 studies reporting on the prevalence of alcoholism in families of alcoholics; all showed a greatly increased risk of alcoholism in both male and female relatives of alcoholics compared to relatives of nonalcoholics.[2]

Not everything that "runs in families," however, is hereditary. Speaking French runs in families but is not hereditary. There are several ways "nature" and "nurture" can be separated; all have now been applied to alcoholism. One is the genetic marker approach, another involves studying twins, and a third uses the "experiment of nature" of adoption to separate genetic or at least congenital influences from those apparently caused by upbringing.

When Jellinek discussed the subject in 1944, there existed only one nature–nurture study, and it was not published until a year later. Roe[3] studied children of heavy drinkers who had been raised by foster parents, by then in their early 30s. None was an excessive drinker. Roe concluded that heredity was not a factor in alcoholism. Jellinek no doubt knew about the Roe study—both were at Yale—but did not refer to it in his 1944 Summer School talk. Murray, in his excellent review of twin and adoption studies, ventures some reasons why Roe's findings differed from those in later studies.

There have been many genetic marker studies, reviewed comprehensively here by Swinson. A genetic marker is a trait known to be inherited, usually in a straightforward Mendelian fashion, such as blood groups, color blindness, and certain diseases. Should more alcoholics have the "marker" than would be expected by chance, this would indicate that alcoholism is influenced by heredity.

Many studies show an association of alcoholism with some marker. However, for almost every study showing an association, another study shows none. Color blindness studies have been most consistent. A group in Chile first reported that alcoholics often were color-blind. Other groups observed the same thing, but found that the color blindness disappeared in time. In short, the color blindness apparently was of nutritional or other nongenetic origin.

There have been a dozen twin studies. In perhaps the most ambitious, identical twins were found to be more concordant for alcoholism than fraternal

twins.[4] The other studies showed some difference in drinking habits between identical and fraternal twins, implying a genetic factor.

Three adoption studies have been published in the last few years. The first was conducted in Denmark.[5] It involved interviewing alcoholics' children raised in nonalcoholic foster families. The boys, by their late 20s, were four times more likely to be alcoholic than adopted boys of nonalcoholic parentage. The girls, by their mid-30s, were four times more likely to be alcoholic than nonadopted controls. Adopted controls, however, also had a high rate of alcoholism (for women), raising the possibility that adoption itself may favor the development of alcoholism. (There is no other supporting evidence for this.)

Alcoholism was defined by operational criteria of the DSM-III type. There were also categories labeled "heavy drinking" and "problem drinking." There was no difference between probands and controls in either of these groups. Nor were probands (sons of alcoholics) likely to have a psychiatric illness other than alcoholism. Alcoholism in these individuals had emerged full-blown when they were in their teens and 20s and was severe enough to require treatment. Drug abuse did not distinguish probands from controls, a finding relevant to Jellinek's question about substance abuse specificity.

Subsequently two other adoption papers were published. Bohman[6] found that Swedish adoptees with a biological parent with many alcohol problems were much more likely to have alcohol problems than were adoptees of non-alcoholic parentage. If their biological parents were criminal, they were no more likely to be criminal or alcoholic. In other words, transmission appeared to be specific for alcoholism only and not for criminality.

Cadoret and Gath[7] studied a group of Iowa adoptees and found that those with a biological parent who was alcoholic tended to be alcoholic themselves and lacked any other diagnosable psychiatric disorder.

Based on these studies, all conducted in different countries with different methodologies, the following tentative conclusions can be drawn:

1. Children of alcoholics are about four times more likely to become alcoholic than are children of nonalcoholics, whether raised by their alcoholic biological parents or by nonalcoholic foster parents.
2. Their alcoholism develops at a rather early age, almost explosively in some cases.
3. The alcoholism is particularly severe.
4. They are no more prone to other psychiatric disorders, including drug abuse, than are sons of nonalcoholics.

These conclusions apply only to men, since the studies of women have produced ambiguous results.

From these studies, it appears that "familial alcoholism" might be a useful subtype of alcoholism—a distinction perhaps more useful for research pur-

poses than clinical treatment. Such a subtype would have these distinguishing characteristics:

1. There would be a positive family history of alcoholism.
2. The alcoholism would commence early in life and assume a florid course.
3. The likelihood of there being an "underlying" or second diagnosable psychiatric disorder would be no greater than would exist in nonalcoholics.

Separating alcoholics into familial and nonfamilial types has advantages for research. To begin with, most alcoholism wards break almost evenly into patients with a positive family history of alcoholism and those without one. This provides equal-size subsets, which is useful for statistical analysis. There is usually no question about the family history. Almost always, when an alcoholic patient has one family member who is alcoholic, he has two or more who are alcoholic. In comparing familial and nonfamilial alcoholics, one could look for distinguishing variables such as age of onset, severity, and a wide variety of social, psychological, and biological factors.

Such studies have indeed been conducted in the past 2 or 3 years. Significantly, all indicate that alcoholics with a family history of alcoholism have a particularly extreme form of alcoholism and are younger than alcoholics without a positive family history.

Jellinek asked: "If anything is inherited, what is it? Does it involve tolerance?"[1]

We can now say, unequivocally, that it involves tolerance—or at least lack of tolerance. It has only recently been appreciated how many millions of people possess a profound intolerance for alcohol. Among Orientals, the intolerance afflicts (if that is the word) more than half of the population. In response to very little alcohol, Orientals develop a cutaneous flush and a strong disinclination to continue drinking.

There is a low rate of alcoholism in the Orient, usually attributed to cultural sanctions against drunkenness and a preference for derivatives of the poppy plant. It now appears that a physiological intolerance for alcohol may be just as important a deterrent. Unquestionably, this intolerance is inherited; it is genetic. One can give tiny amounts of alcohol to Oriental infants, and they immediately flush.

Tolerance to alcohol can be increased in animals by selective breeding, as Peterson notes in his excellent review.

There is a tendency today to view alcoholism as a product of many forces: biological, sociological, psychological. To some extent, this may be a device to make all the students of alcoholism feel useful—biologists, sociologists, psychologists. There is a kind of unspoken gentlemen's agreement that since experts from diverse backgrounds study alcoholism, alcoholism must have diverse origins.

It may be so. In certain ways, it clearly is so. Genes give us enzymes to

metabolize alcohol; society gives us alcohol to metabolize; and our psyches respond in wondrous ways to these combined gifts. Nevertheless, beyond this obvious level, the evidence for multiple causes of alcoholism is not better or no worse than the evidence for a single cause. When one does not know, it is best to say one does not know.

This point needs amplification, particularly since almost everybody still talks about the "multifactorial" nature of alcoholism. I close by quoting Lewis Thomas, President of the Sloan–Kettering Cancer Institute and the Montaigne of medicine:

> The record of the past half century has established, I think, two general principles about human disease. First, it is necessary to know a great deal about underlying mechanisms before one can really act effectively. . . .
>
> Second, for every disease there is a single key mechanism that dominates all others. If one can find it, and then think one's way around it, one can control the disorder. This generalization is harder to prove, and arguable—it is more like a strong hunch than a scientific assertion—but I believe that the record thus far tends to support it. The most complicated, multicell, multitissue, and multiorgan diseases I know of are tertiary syphilis, chronic tuberculosis, and pernicious anemia. In each, there are at least five major organs and tissues involved, and each appears to be affected by a variety of environmental influences. Before they came under scientific appraisal, each was thought to be what we now call a "multifactorial" disease, far too complex to allow for any single causative mechanism. And yet, when all the necessary facts were in, it was clear that by simply switching off one thing—the spirochete, the tubercle bacillus, or a single vitamin deficiency—the deficiency—the whole array of disordered and seemingly unrelated pathologic mechanisms could be switched off, at once.
>
> I believe that a prospect something like this is the likelihood for the future of medicine. I have no doubt that there will turn out to be dozens of separate influences that can launch cancer, including all sorts of environmental carcinogens and very likely many sorts of virus, but I think there will turn out to be a single switch at the center of things, there for the finding. I think that schizophrenia will turn out to be a neurochemial disorder, with some central, single chemical event gone wrong. I think there is a single causative agent responsible for rheumatoid arthritis, which has not yet been found. I think that the central vascular abnormalities that launch coronary occlusion and stroke have not yet been glimpsed, but they are there, waiting to be switched on or off.[8]

Who knows? Maybe alcoholism also has a single switch. Maybe it too could be "turned off" if we knew how.

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Genetic Markers and Alcoholism

Richard P. Swinson

Abstract. Genetic markers are genetically determined characteristics that have been found to occur in association with some common disorders. In alcoholism, four types of genetic markers have been studied to determine their frequency compared with the general population frequency. The markers studied are (1) blood groups and serum proteins, (2) secretion of ABH blood group substance, (3) phenylthiourea taste sensitivity, and (4) color vision defects.

There have been found to be significant associations between alcoholism and all the above markers apart from blood groups and serum proteins. However, the associations appear to result from acquired rather than inherited factors.

1. Introduction

In 1966, Lester wrote[1] that "an association of alcoholism with other characteristics known to be inherited would appear to afford some support for a biological factor in the etiology of alcoholism." This cautiously worded statement summarizes the aims of research efforts in the area of genetic marker studies in alcoholism.

There are many genetically determined characteristics that have been identified in human subjects. Some of these have been found to be consistently associated with the occurrence of common diseases. Carcinoma of the stomach is associated with the presence of ABO blood group A, and prepyloric peptic ulceration is associated with blood group O.[2] The ABO blood groups are one example of genetically determined characteristics that have been used as markers in the investigation of the etiology of alcoholism.

In the study of genetic markers in alcoholism four characteristics have been examined. These are:

1. Blood groups and serum proteins.
2. The secretion or nonsecretion of ABH blood group substance in the saliva.
3. The taste sensitivity of subjects for solutions of phenylthioureas.
4. Color vision defects.

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These four characteristics are examples of genetic polymorphisms. Clarke[3] defined genetic polymorphisms as "variations in which individuals with clearly distinct qualities exist together in a freely interbreeding single population." Ford[4] emphasized the "occurrence together in the same habitat of two or more discontinuous forms, or phases, or a species, the rarest of which cannot be maintained by recurrent mutation."

The ABO blood groups are an example of a genetic polymorphism with four phases, the proportions of which vary from population to population. There are no intermediate phases between the phenotypical expressions of blood groups A, O, B, and AB. Similarly, with the secretion of ABH blood group substance, there is a two-phase polymorphism: people either secrete or do not secrete ABH substance. There are no intermediate phases. Phenylthiocarbamide tasting is a two-phase polymorphism, taste and nontaste, with taste being dominant. Color vision defects occur in a number of different forms but are generally two-phase polymorphisms, with normal and defective color vision as the phases.

The basic research strategy in studying genetic markers in alcoholism has been to determine the frequency of the occurrence of each phase of a polymorphism in a group of alcoholics and to compare these observed frequencies with those found in the normal population in the same area of the world. Although this strategy is very straightforward, many methodological problems have been encountered. A basic problem is the determination of what is meant in each study by the term "alcoholic." Frequently, there has been no definition of the term in the reports published. On other occasions, attendance at an Alcoholics Anonymous meeting or attendance at an addiction outpatient clinic has been sufficient to allow the subjects involved to be included as alcoholic. Very occasionally, independent diagnosis by two physicians has been taken as the criterion.

The fitness of the subjects to engage in complicated tests, particularly of color vision, has at times been ignored. Color vision tests in particular have been administered and interpreted in arbitrary ways. These, and other problems in specific studies, have led to more confusion in this area than would seem likely given the initial simple question: Is there a significant association between the occurrence of alcoholism in a person or group of people and the presence of a specific genetically determined characteristic? The four areas of investigation will be considered separately.

An area of investigation distinct from the study of genetic markers in alcoholism is the study of nongenetic markers of alcoholism. Nongenetic markers are chemical abnormalities found in association with excessive alcohol consumption. Morgan[5] has described the ideal marker for detecting and monitoring alcoholics as being "sensitive and highly specific for alcohol abuse; its value should be affected by changes in alcohol intake over relatively short periods of time" (p. 1). She concludes that a combination of measurements of serum aspartate transaminase, serum γ-glutamyltranspepsidase, and mean

corpuscular volume can detect the majority of alcoholics. Other markers, such as serum transferrin, have been investigated.[6]

These biochemical markers are transient, changing variables that fluctuate with the recent drinking habits of an alcoholic. The ideal genetic marker would be stable over time and unaffected by alcohol intake. This chapter deals solely with genetic markers, and where the word marker is used, it is the genetic variety that is implied.

2. Blood Groups and Serum Proteins

2.1. The ABO Blood Groups

As noted above, a number of associations have been demonstrated between the presence of certain physical illnesses and changes in the proportions in the ABO blood group distribution from those proportions found in the general population. Similar investigations have been carried out in subjects with psychiatric disorders including alcoholism.

Separate studies in North America,[7] Australia,[8] and Austria[9] have reported significant associations between the diagnosis of alcoholism and an increase in the proportion of subjects of blood group A. Nordmo investigated the blood group distribution of 5637 patients in the Colorado State Hospital. Of this large group, 939 patients were diagnosed as being alcoholic. Among these subjects, the proportion of blood group A was found to be significantly increased ($P < 0.004$). The finding was confirmed equally for the two sexes.

Blood group distribution is affected by racial origin. Nordmo reported[7] that in the Mexican population of the area there is a predominance of blood group O. Of the 939 subjects studied, 25% had Mexican surnames, compared with 13.6% of the total hospital population who acted as controls for the alcoholic group. Given that there was a bias toward a greater proportion of blood group O subjects in the alcoholic group, the finding of a significantly increased proportion of group A is all the more noteworthy.

The two other studies[8,9] were concerned with the investigation of blood group proportions in subjects with portal cirrhosis who were not necessarily alcoholic. In both of these studies, it was found that the proportion of blood group A was significantly increased.

In contrast to the above findings, however, nine studies have failed to confirm the association between alcoholism and ABO blood group distribution. These studies will be considered in the chronological order of their publication.

Achté, in 1958, investigated the blood group distribution of 212 alcoholics from the Helsinki area; 1383 healthy subjects from the same area acted as controls.[10] The distribution of ABO blood groups in the two sets of subjects was almost identical, and no statistical differences were demonstrated.

In 1964, the Iowa Blood Type Disease Research Project[11] reported on the findings of research into the blood group distribution on almost 50,000 controls and over 14,000 patients with various diseases. Of these patients, 272 had portal cirrhosis, and 62 had other types of cirrhosis. No difference was found between the blood group distribution of the controls and the cirrhotics.

Camps and his colleagues reported on two series of British alcoholic subjects in 1967 and 1969.[12,13] The main objective of the two overlapping studies was to determine the frequency of the secretion or nonsecretion of ABH substance in the saliva of alcoholics; this aspect of the research is considered in the next section. It is necessary to determine ABO blood grouping in order to carry out the comparison of secretor–nonsecretor status, and the data on 1000 alcoholics and 1000 controls can be derived from the reports. The earlier study reported the findings in 218 subjects. Further data were collected until information had been gathered on the 1000 subjects in the second report. A comparison of the ABO blood group distributions of the alcoholics and the controls revealed no significant differences between the two groups.

Swinson[14] also failed to find any change in ABO blood group proportions from those expected in a study of 222 alcoholics in the Northwest of England. Buckwalter *et al.*[11] commented on the difficulties posed by the statistical analysis of blood group proportions. In areas such as the United Kingdom where there is mixing of groups of different ethnic origin, it is possible to obtain statistically significant results in blood group distribution comparisons as a result of the heterogeneity of the distributions within the ethnic groups making up the subject population. Thus, in Swinson's study, using the statistical method of comparison described by Aird *et al.*[15] it was found that there was a statistically significant decrease in alcoholic subjects of blood group A and an excess of subjects of groups B and AB. However, using the correction for heterogeneity as proposed by Woolf,[16] these significant findings disappeared, although the trends were still in the same directions.

Blood group data were collected in a further 226 subjects, and the findings in the total group of 448 alcoholics were reported by Swinson and Madden.[17] These data were compared with the ABO blood group distribution found in 7757 blood donor controls from the same geographical area of Northwest England and North Wales. The trends found in the first study were confirmed: that is, blood group A was found to be less frequent than expected in the alcoholic group, but the degree of heterogeneity was significantly increased, and this factor accounted for the statistically significant results obtained. Thus, no definite evidence for a change in blood group proportions in alcoholics could be demonstrated. The data in this study and those data from Colorado and Helsinki were combined according to Woolf's method,[16] and again, the statistically significant result obtained was found to result from the heterogeneity of the samples.

Hill and her colleagues[18] from St. Louis obtained data on 11 serological markers, including ABO blood groups, from 48 alcoholics and 46 of their

nonalcoholic first-degree relatives. No differences were found in the distribution of ABO blood groups in the two subject groups, and it was not possible to demonstrate significant linkage between alcoholism and any genetic marker.

The last study in this series is that by Winokur and his co-workers[19] in Iowa who examined a number of serological markers in 110 members of 16 families, each containing at least one alcoholic and one depressive. A total of 31 alcoholics were investigated. No significant differences were found in the ABO blood group distributions.

Data from the above nine studies from widely varying parts of the world indicate that there is no significant difference between the ABO blood group distributions of alcoholics and the controls used. Although these data appear to be conclusive, it would not have been surprising to find significant results. In several studies, blood group A has been found to be in excess in subjects with cirrhosis of the liver.[2] Conversely duodenal ulceration, which is common in alcoholics,[20] is associated with group O.[2] It would perhaps be of value to control for the presence of these two disorders in any further investigations.

A further problem in blood group research is that of racial stratification which can produce considerable problems in obtaining appropriate controls. Nordmo[7] commented on the bias in his study, which was against the direction of the trend that he found in his subjects. In Swinson's study,[17] one of the difficulties encountered was that of dealing with a transient population. At the time of the study, 40% of the subjects were not in contact with any family members, and many of them had recently arrived in the area in which the study was performed. A transient population that is largely of, say, Scottish, Irish, or Mexican descent can provide great difficulties in the search for appropriate control subjects.

The use of family members as controls as reported above by Hill *et al.*[18] and Winokur *et al.*[19] is a way to avoid the difficulty of racial stratification. However, it then leaves open the question of whether relatively stable alcoholics are typical of the total alcoholic population in terms of their genetic endowment.

At the present time, it can be concluded that there is no evidence to support the contention that there is an association between ABO blood groups and alcoholism.

2.2. Other Blood Groups

Hill and her colleagues[18] investigated the possibility of an association or linkage between alcoholism and 11 serological markers. In addition to the ABO blood groups reported above, they examined the distribution of the MNSs, rhesus, Kell, Duffy, Xg, and Gm(a) γ-globulin systems together with haptoglobins, group-specific component (Gc), complement C3, and α-antitrypsin. The same 48 alcoholics and 46 nonalcoholic first-degree relatives included in the ABO investigations were the subjects for the investigation.

There were three significant findings. In the MNSs system, the S antigen

frequency among the alcoholics was close to the normal population frequency. Among the nonalcoholic relatives, the homozygous recessive ss condition was found to be significantly increased ($P < 0.01$). It was suggested that this condition may be a relative protector against the development of alcoholism.

In the rhesus system, evidence was found to suggest linkage in repulsion between the D gene and alcoholism. The D gene pair determines whether a person is rhesus positive or rhesus negative. It was found within the families that sib pairs unlike for the presence of alcoholism had a higher incidence of like Rh types than pairs who were alike for alcoholism.

The complement C3 system is not a blood group system but is a serum protein. Approximately 54% of Caucasians are of the phenotype SS; in Hill's study,[18] all the alcoholics and their relatives were of the SS phenotype ($P < 0.01$).

Winokur *et al.*[19] replicated the above study but were not able to confirm the findings. They found no significant correlations between alcoholism and the MNSs or C3 systems. They found the expected rate of approximately 50% of phenotype SS in the C3 system as opposed to the 100% reported by Hill *et al.*[18]

At present, there is no conclusive evidence of an association between any blood group system and alcoholism.

3. ABH Substance Secretion

The secretion of ABO blood group substances in the saliva occurs in approximately 77% of people of European extraction. Secretion of the antigens A, B, and H of the ABO system is controlled by a single pair of allelomorphic genes Se and se, the homozygous recessive sese giving rise to the nonsecretor condition.

Camps and his colleagues, in the series quoted above in the blood group discussion,[12,13] observed a total of 1000 alcoholics from whom they collected blood and saliva samples. The ABH content of the saliva was determined by the accepted method.

It was found that 32.7% of the alcoholics were nonsecretors which compared with an expected incidence of 22.7%. Disregarding 15 subjects whose blood group was not determined, the highest percentage of nonsecretors was found to be among those alcoholics of group A, of whom 37.9% were nonsecretors. Control groups were used for the subjects of different ethnic origins within the British Isles, and the increase in nonsecretors in group A was found to be highly statistically significant ($P < 0.0001$). For group O, the results were nonsignificant. As noted earlier, the proportion of alcoholics of group A was not changed from the expected proportion, but the decrease in secretors was confined almost exclusively to the group A subjects. It was also found that there was a higher percentage of male nonsecretors than female nonsecretors, but the number of women ($N = 194$) was fairly small.

The findings were confirmed by Swinson and Madden[17] in their series

of 222 alcoholics: 36% of the subjects were found to be nonsecretors, whereas 680 controls from the same area were found to be nonsecretors in 24.3% of cases.[21] The difference between the two groups was statistically significant ($P < 0.001$). When the ABO groups were examined separately, the difference between alcoholics and controls was restricted to the proportion of nonsecretors in group A as reported by Camps *et al.*[13]

Reid *et al.*[22] examined the secretor statis of 100 cirrhotic patients and found no difference in the secretor and nonsecretor proportions between their subjects and controls.

Camps *et al.*[13] concluded that the best available explanation for their findings was that chronic alcohol intake produced an effect on the phenotypic expression of secretion in genetically constituted group A secretors. In order for this explanation to be acceptable, it would also be necessary to accept that alcohol has a similar action on the uptake of Lewis antigens by the red cells, since the nonsecretors in the series were found to have the expected red cell Lewis type that is consonant with salivary nonsecretion. In a large series, it should also be possible to show an effect in blood group B subjects. An alternative explanation is that there is a genetically determined association between group A nonsecretion and alcoholism.

It is necessary to study a large sample of alcoholics together with carefully selected controls, preferably nonalcoholic family members, in order to clarify these findings.

4. Phenylthiourea Taste Sensitivity

Phenylthiocarbamide (PTC) is one example of a phenylthiourea, and this compound has been used in the majority of the studies of taste sensitivity in alcoholics. Phenylthiocarbamide taste sensitivity is a two-phase genetic polymorphism, with approximately 30% of normal subjects being classified as nontasters. Changes in PTC taste sensitivity proportions have been shown to occur in a number of illness states. Kitchin *et al.*[23] and Harris *et al.*[24] have shown that the proportion of nontasters is increased in subjects with adenomatous thyroid goiter. Friere-Maia *et al.*[25] found that nontasters were increased in a group of 55 white male schizophrenics but not in a group of female schizophrenics.

Phenylthiocarbamide forms a bitter-tasting colorless aqueous solution which has a slight odor. In order to determine a subjects taste sensitivity accurately, it is necessary to use the method described by Harris and Kalmus.[26] In this method, 14 solutions of PTC are used. Solution 1, the most concentrated, contains 1.3 g/liter PTC in distilled water. Solution 2 is half as concentrated as solution 1, and subsequent solutions are made up with half the concentration of the preceding one. In Harris and Kalmus's method, eight glasses are placed in front of the subject, four containing distilled water at room temperature and four a PTC solution at the same temperature. Starting at the most dilute solution, subjects are asked to discriminate between the

two tastes when the glasses are distributed randomly. Those subjects who can discriminate between water and solutions 5 to 14 are classified as tasters, and the rest as nontasters in the alcoholic group.

Peeples[27] tested 52 alcoholics and 72 controls by means of the above method and found a significant increase in nontasters. Reid *et al.*[22] included PTC taste testing in their examination of 100 cirrhotics. Unfortunately, they chose an alternative, highly unreliable method of testing. They asked their subjects to taste filter paper impregnated with PTC solution containing 0.65 g/liter PTC. This concentration is equivalent to solution 2 and not the accepted antimode solution 4. They found no differences between their cirrhotic subjects and controls, but the method was too unreliable to allow any conclusions to be drawn.

Swinson[28] tested 146 alcoholics using the Harris and Kalmus method. All of the subjects had been independently diagnosed as meeting WHO criteria for alcoholism by two psychiatrists. The subjects were all abstinent inpatients who were receiving no medication. The controls were subjects from the same geographical area as the subjects.[23]

Using the accepted cut-off point for tasters and nontasters, 26.04% of alcoholics were found to be nontasters. This was not significantly different from the control group. However, an examination of the taste sensitivity distribution across all the concentrations of PTC by means of the Kolmogorov–Smirnov statistic[29] revealed a significant difference between the two groups ($P < 0.001$). This difference was accounted for by a loss of taste sensitivity to the most dilute solutions among the alcoholics. This was probably because of the age of the alcoholics, who were considerably older than the controls (means 43.2 and 21.2 years, respectively), or the fact that the alcoholics drank a lot of alcohol and smoked heavily.

One of the problems in using PTC is that it has an odor, and it might be possible for subjects to detect the odor rather than the taste of the solution. Spiegel[30] tested 30 alcoholics and 30 controls by means of solutions of 6-*n*-propyl-2-thiouracil (Prop) administered by the eight-glass method. It was found that there was a significant excess of nontasters among the alcoholics.

Two studies have reported an excess of nontasters, and one has found no increase in nontasters in alcoholic populations. It is not clear whether the loss of taste sensitivity is a result of alcohol intake or another acquired factor or is caused by genetic mechanisms. It would be worthwhile to replicate these studies in a large group of alcoholics with family controls and to follow a series of alcoholics who become abstinent to see if their taste sensitivities change over time.

5. Color Vision

The final group of genetic polymorphisms to have been investigated in association with alcoholism is that of color vision defects. Color vision defects

Table I. Classification of Color Blindness

Major defects
Total color blindness
Red–green color blindness
Lacking in red
Protanopia
Extreme protanomaly
Simple protanomaly
Lacking in green
Deuteranopia
Extreme deuteranomaly
Simple deuteranomaly
Yellow–blue blindness
Tritanopia
Tritanomaly
Tetartanopia
Minor defects
Deviant
Color weak

occur as major or minor defects. The classification shown in Table I, after Pickford,[31] is commonly accepted, although Kalmus[32] warns that any division of color vision defects is artificial and composite.

The investigations of color vision defects in alcoholism have been concerned with the major defects of red–green or yellow–blue defects. It is accepted that red–green defects are inherited by sex-linked mechanisms, so that affected males suffer from red–green color blindness and affected females are heterozygous carriers of the X-linked gene. Yellow–blue defects are caused by autosomal dominant genes, and the sexes are thus equally affected.

In addition to the types of color vision defects described above, there are other defects that are not the result of genetic causes but are acquired defects as a consequence of disease affecting the eye or the central nervous system. These conditions may be transient or progressive.[33–37]

There have been many reports of investigations into color vision defects in alcoholism. Unfortunately, many of the studies have been seriously affected by methodological faults, and others by the drawing of doubtful conclusions.[38,39] Four basic methodological problems have occurred in the studies reported: these have concerned the target populations, the vision test, interpretation of test results, and the fitness of the subjects.

5.1. Target Population

Frequently, the target population has not been defined, and a number of studies have been performed on groups diagnosed as cirrhotic who are not necessarily alcoholic. It is not clear how comparable the groups of subjects have been in the different studies and thus how comparable are the findings.

5.2. Type of Color Vision Test Used

Unfortunately, little attention has been paid to the choice of color vision test employed in many of the studies. Color vision testing is possible in a multitude of ways that vary in their applicability, accuracy, and diagnostic ability.

Kalmus[32] pointed out that no single test of color vision or single method of testing is infallible in detecting abnormal color vision. Eight of the published reports relied on the use of a single test[22,40–46]; others have used two tests[47–49]; and two studies have made use of three tests.[38,50]

The most commonly used type of test of color vision defects is the pseudoisochromatic plate test. In this type of test, cards printed with multiple colored dots are shown to the subject one at a time under a real North light or its artificial equivalent. Normal subjects report seeing a number or pattern that color-defective subjects cannot discriminate from the background. There are a number of different pseudoisochromatic plate tests, the commonest of which is the Ishihara plate series.

Color-matching tests can be used, of which the most accurate and complex is the Farnsworth–Munsell 100-Hue test that Cruz-Coke and his group used in many of their studies. The most reliable method of determining if a subject is color normal or defective is to use an anomaloscope.[32] In an anomaloscope, red light and green light can be mixed in varying proportions to produce a match with a fixed yellow-colored filter. The proportions of red and green light and the range of matches accepted by the subject allow color vision defects to be accurately discriminated from normal color vision and also allow for the defects to be accurately classified. Similarly, blue and yellow light may be matched against neutral light. By these means, major and minor defects can be detected, whereas the pseudoisochromatic plates tests miss the minor defects.

5.3. Interpretation of Test Results

Gorrell,[43] in his study of 55 male alcoholics whose "diagnoses were based on hospital records," made use of the 1964 version of the Ishihara plates. He classified his results into major red–green defects and minor defects that are not usually accepted as being detectable by the test. Varela *et al.*,[57] in a study of 65 male alcoholics and their first-degree relatives, also used a single test, the Farnsworth–Munsell 100-Hue test. This test identifies reduced hue discrimination and is not a diagnostic test[33]; however, it was used by Varela and his colleagues to make a diagnostic classification. The results of these studies cannot be taken as conclusive whatever the findings because of the use of an unreliable method of assessment.

5.4. Fitness of the Subjects

Some color vision tests require that the subject be able to understand complex instructions and also be able to cooperate in a complex task. This is

particularly true of the 100-hue test and anomaloscope testing. It is therefore important to know that alcoholic subjects are not intoxicated at the time of testing.

In one of the studies by Cruz-Coke and Varela,[42] the subjects were 100 alcoholics selected randomly from an outpatient clinic population. They were tested on a single occasion by means of a single test, the Hardy–Rand–Rittler (HRR) pseudoisochromatic plates. Eighteen of the subjects were found to be color defective, which was highly significant when compared with a normal population of 633 male students ($P < 0.0001$).

It is possible, and in fact likely, that at least some of the alcoholic subjects had been drinking shortly before the time of color vision testing. It is further possible that color vision defects found in drinking alcoholics would disappear after a period of abstinence. In two studies,[45,48] it was found that a return to normal color vision did occur within a few days of abstinence.

Fialkow and his colleagues[48] tested 24 male and 22 female patients shortly after hospital admission. They used the HRR plates and the Ishihara plates. Forty percent of the males and 41% of the females were found to be color vision defective. Of the original 19 color-defective subjects, nine were available for retesting later in their hospital stay; seven of the nine had normal color vision on the second test, and one had changed from showing a severe deutan defect to showing a mild unclassifiable defect.

Smith[45] used the Ishihara plates to test 205 alcoholic patients. Initially 38% of the men and 39% of the women were classified as color vision defective. On retesting 10 days later, 10% of the males and 15% of the females were found to be color defective. It was concluded that the color vision defects were caused by a biochemical or metabolic effect consequent on either alcoholism or cirrhosis of the liver.

It should be noted that the proportion of color-defective subjects in the two studies was remarkably similar and that there were as many color-defective women as color-defective men. This latter finding argues against a genetic etiology, since in any sex-linked condition, many more men than women are affected.

The overall findings of the color vision studies fall into three groups:

1. Those studies showing no association between alcoholism or cirrhosis and color vision defects.[22,43,49] Gorrell[43] tested 55 alcoholics by means of the Ishihara plates. Only three defectives were found in contrast to 4.2 expected. In a group of 26 cirrhotics, two defectives were found, and three were expected. In Thuline's study of 172 alcoholics, 5.8% were found to be color defective by means of the HRR and Ishihara plates, compared with a normal control frequency of 7.3%. Reid's group also found no increase in color vision defects in their study of 100 alcoholics using the Ishihara plates.
2. Those studies showing a transient association between alcoholism or cirrhosis and color vision defects.[45,48] These studies are discussed above.
3. Those studies showing a persistent association between alcoholism or cirrhosis and color vision defects.[38–42,44,46,47,50–53,57]

It has been from this group of studies that the possibility of using color vision defects as genetic markers for alcoholism has arisen. Initial interest was aroused by a report by Cruz-Coke in 1964[40] who found a statistically significantly increased frequency of color vision defects among 58 men and 12 women with cirrhosis; 27.5% of the men and 33% of the women had color vision defects as detected by HRR plates. Cruz-Coke reported that a pedigree analysis showed a classical sex-linked pattern, and he hypothesized that an X-linked chromosome might be implicated in mediating a pathway for a "common disease" of cirrhosis and color blindness.

Further reports from Santiago have produced results along the same lines. In 1965, Cruz-Coke reported an incidence of color vision defects of 31.2% in 77 cirrhotic males, 85% of whom were alcoholic.[41] The outpatient study quoted above found 18% of alcoholics to be color vision defective on a single test.

Cruz-Coke and Varela[50] extended their studies to the families of alcoholics by means of the 100-hue test which had been shown by Krill and Schneidermann[54] to be able to detect heterozygous carriers of color vision defects. In 20 families, it was found that there was a high correlation between the mean errors of alcoholic fathers and their daughters but a significant difference between the mean errors of the fathers and their sons. This finding is consistent with an X-linked hypothesis. It was suggested that alcoholism might be considered to be a genetic polymorphism. One of the features of genetic polymorphisms is that there is an advantage to the heterozygote carrier. It was hypothesized that the advantage for the female carriers of the "alcoholic gene" might lie in the areas of "fertility or viability."

The color vision defects discovered by the Chilean group using the 100-hue test have mainly been along the blue–yellow axis. Ugarte *et al.*[46] found that 39% of 149 male alcoholics showed defects in the blue–yellow zones of the 100-hue test. Saraux *et al.*[53] also used the 100-hue test but found that the majority of defects were in the red–green range. Carta *et al.*,[51] in a very small sample of 16 male cirrhotics, found a large proportion of blue–yellow defects. Dittrich and Nebauer[52] also reported blue–yellow (tritan) defects in 120 cirrhotic subjects.

Sassoon *et al.*[44] tested two groups of alcoholics by means of the Farnsworth D-15 panel; 22% of the alcoholics in the first group and 11% of controls showed blue–yellow defects, and 4% of each group red–green defects. In the second group, family members of alcoholics were themselves diagnosed as alcoholic in 29% of cases. The criteria for the diagnosis are unclear. Of those family members, 15% had definite blue–yellow defects, 22.5% questionable blue–yellow defects, and 4% red–green defects. In 44% of the families, at least one member showed blue–yellow deficiency, but there was no evidence to support an X linkage.

This author[38,39] used three methods of color vision testing in a study of hospitalized abstinent alcoholics. The tests were the Ishihara plates, the Dvorine plates, and the Crawford anomaloscope.[55,56] A total of 149 patients were tested, although only 96 were tested on all three tests. The results are presented in Table II.

Table II. Percentages of Color Vision Defects on the Three Tests Administered

Test	Percentage of defective males	Percentage of defective females
Ishihara	6.61	Nil
Dvorine	14.81	14.28
Anomaloscope	26.92	33.33

It can be seen that the percentage of defects found depended on the test used and that there were as many color-defective women as color-defective men. In those studies that have reported finding an increase in color vision defects, the proportion of color-defective subjects has been consistently about 30%.

The types of color vision defects were compared with Pickford's[31] breakdown of the proportions of the various types of color vision defects found in the normal population (Table III).

The two distributions are significantly different ($P < 0.025$), and it appears that defects found in Swinson's study were not typical of the genetically determined defects found in the normal population.

The simplest explanation for the finding of very high rates of color vision defects that occur with equal frequency in both sexes and have been shown in some studies to be transient is that the defects are acquired and do not have a genetic etiology. François and Verriest[36,37] and Cox[33–35] have shown that acquired color vision defects are most commonly of the blue–yellow type.

There is a need to repeat the family studies performed in Santiago in a well-defined group of alcoholics and by means of a number of tests including an anomaloscope.

6. Summary

There have been found to be significant associations between three of the genetic polymorphisms investigated and alcoholism. In the light of nine negative studies as against three positive ones, it appears safe to conclude that there is no evidence of an association between blood groups and alcoholism.

Table III. Color Vision Defects Distribution

	Protanopes (%)	Deuteranopes (%)	Protanomalous (%)	Deuteranomalous (%)
Pickford[31]	27.7	39.36	9.1	25.13
Swinson[38]	19.05	14.29	52.38	14.29

The evidence with the other three groups of markers is in the direction of an association. No one has so far reported a negative finding with changes in ABH secretor status, and these studies are worth replicating. The evidence regarding taste sensitivity is mixed, and again it would be valuable to replicate these studies. The evidence regarding the color vision findings is confusing, but close attention to the methodology of any further studies in this area should help to clear up the confusion quite quickly.

The question remains with respect to these three findings of whether the changes are caused by alcohol intake or are present before excessive alcohol intake begins. Family studies involving the nonalcoholic sibs of alcoholics and prospective assessments in the children of alcoholic parents would answer this question. At the present, there is no hard evidence to support an association between a genetic marker and alcoholism.

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2

Twin and Adoption Studies
How Good Is the Evidence for a Genetic Role?

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Abstract. Research into the possibility that heredity may influence drinking habits is still in its infancy, and the conclusions that can be reached from a number of the available twin and adoption studies are limited by their methodological deficiencies. Nevertheless, the balance of evidence suggests a modest genetic effect on both normal drinking and alcoholism in men, though similar evidence for women is so far lacking. Further studies are required to assess the significance of the genetic contribution, to elucidate exactly what is inherited, and to examine the nature of gene–environment interactions.

1. Introduction

Students of alcoholism must continually beware lest they fall victim to the extravagant swings of intellectual fashion that so bedevil the field, and nowhere is such vigilance more necessary than in considering the possible etiological role of heredity. Thus, in 1945, Jellinek[1] complained that "looking at the literature on inebriety one gets the impression that all excessive drinking is due to an inherited constitution." However, 25 years later, one could search the literature without finding more than the occasional disparaging mention of heredity.

But now the pendulum has swung again, and there has been an upsurge of interest in the possibility of some genetic predisposition to alcoholism. Many reviews of the relevant studies have appeared, but, unfortunately, these have mainly been of a descriptive nature and have not critically examined the evidence in the light of the standards of modern behavior genetics. The available evidence comes from family, twin, and adoptive studies, but the first is beyond the scope of our present chapter. We propose, therefore, to examine dispassionately how strong the twin and adoptive evidence for a

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genetic role is, to point to where it is deficient, and also, we hope, to indicate how further progress might be made.

2. Twin Studies of Normal Drinking

Twin studies have addressed two main issues. First, does heredity influence normal drinking, and second, does it contribute to the occurrence of abnormal drinking? In this chapter we have greater interest in addressing the second question, but we briefly review the evidence for the first for three reasons, (1) the question is interesting for its own sake, (2) much recent research suggests that the factors that influence normal drinking also influence abnormal drinking (see Bruun *et al.*[2]; Royal College of Psychiatrists[3]), and (3) since alcoholic twins are difficult to come by, the use of normal twins provides a means of collecting sufficient data on which to carry out the powerful statistical analyses developed by biometric geneticists.

The twin method relies on the fact that identical or monozygotic (MZ) twins share 100% of their genes, whereas nonidentical or dizygotic (DZ) twins share, on average, only 50%. If MZ twins are found to be more similar (i.e., to have higher concordance rates) for a given characteristic than DZ twins, then the excess concordance of the MZ twins is assumed to be caused by their greater genetic similarity. Thus, the MZ/DZ concordance ratio indicates the extent of the genetic contribution. An alternative measure is heritability (h^2), which may be defined as the proportion of variance of a characteristic that can be attributed to genetic factors; the greater its value, the greater the contribution of heredity.

2.1. The Finnish Study

The most thorough and detailed of the normal twin studies was carried out by Partanen *et al.*[4] who interviewed 902 Finnish male twins aged between 28 and 37 years. Factor analysis of the data produced three main factors. The first, *density*, was a measure of the frequency and regularity of drinking. The second, *amount*, concerned the volume consumed at a session. Inability to control and cease drinking constituted the third factor, which was termed *loss of control*. Heritability was 0.39 for density and 0.36 for amount, suggesting that normal drinking habits are influenced to a significant extent by heredity. However, drunkenness arrests, addictive symptoms, and social complications, which one might have thought closely related to alcoholism, had heritabilities of only 0.06, 0.06, and 0.16, respectively. Loss of control was subject to greater genetic influence in younger twins—heritability was −0.07 for older pairs but 0.54 for younger twins. The greatest genetic influence was seen when subjects were dichotomized into drinkers and abstainers.

Cloninger *et al.*[5] have drawn attention to the fact that an individual has to be a very heavy drinker before he can show any features suggestive of

alcoholism, and, unfortunately, Partanen's sample contained very few heavy drinkers. These authors have reinterpreted Partanen's data in light of canonical correlation analysis which suggests a greater genetic contribution, but whether this reinterpretation is legitimate is itself open to question.

2.2. Incidental and Small Studies

Several studies have asked a few questions about drinking in the course of larger inquiries. Thus, Cederlof *et al.*,[6] who examined a huge sample of 13,000 pairs of Swedish twins, concluded that normal drinking was not greatly influenced by heritable factors. Similarly, in his National Merit Twin Study, Loehlin[7] reported that items dealing with specific alcohol-related problems such as drinking before breakfast (0.36) showed only moderate heritability, but that for hangovers was much higher (0.62). Some genetic control over such aversive consequences of alcohol might explain why both Partanen[4] and Jonsson and Nilsson,[8] who studied 750 male pairs, found that whether an individual abstained from alcohol or not was under some degree of genetic influence. Jonsson and Nilsson's MZ and DZ twins showed very similar concordance rates for volume drunk, frequency of drinking, and occurrence of intoxication.

Conterio and Chiarelli[9] reported concordance frequencies for a sample of 34 MZ and 43 DZ pairs of male Italian twins. They found no increased similarity of MZ twins over DZ twins for drinking/nondrinking categories. Since wine was the most frequently used beverage, amounts of wine consumed were also examined; these did not show increased similarity of MZ twins over DZ twins. In another small study, Perry[10] investigated attitudes to various drugs and reported heritabilities of 0.51 for alcohol as against 0.19 and 0.12 for coffee and cigarettes, respectively. Neither this Texan nor the previous Italian study is extensive enough to be taken seriously.

2.3. Recent Studies

Pederson[11] examined alcohol use in 137 pairs of Swedish twins. There was no evidence for a genetic contribution to beer or wine consumption, but the broad heritability for spirits was 0.28 and 0.71 for heavy drinking. Analysis was also carried out on other family members. These generally confirmed the familial nature of drinking practices, but the exact roles of genetic and environmental influences remained essentially unclear.

A second important study has now emerged from Finland by Kapprio *et al.*[12] Beer, wine, and spirit consumption were examined in several thousand pairs of twins. Among males, the overall heritability for total alcohol consumption was 0.37. However, genetic influence seemed to lessen with age. The highest heritability (0.53) was for the age range 20–24 years, but by age 70–74, heritability had declined to -0.04. Overall heritability for female consumption was rather lower at 0.25.

We[13] have reported similar findings on 494 pairs of normal twins from the Institute of Psychiatry register. Among males, genetic factors were of considerable importance in determining overall consumption levels and also in "escape" and "social" drinking. Common family environment had its greatest influence on problem drinking, whereas specific environmental factors acting uniquely on individuals from outside the family had the greater influence on the psychological effects of alcohol. This latter finding is compatible with the observation that the immediate effects of alcohol are to a large extent dependent on the drinking context, e.g., whether the drinker is alone or in a bar. Generally speaking, genetic factors appeared to be of less importance among females.

3. Twin Studies of Alcohol Abuse

3.1. Kaij's Study

The first twin study specifically concerned with alcohol abuse was that of Kaij[14] who studied all male twin pairs born in southern Sweden and in whom one or both twins had been reported to the local Temperance Board. Kaij identified 174 such pairs, and with astounding persistence succeeded in personally interviewing 292 individual twins.

Kaij classified the drinking habits of his twins in two ways. First, he used the information in the official registers of the Temperance Board, the state alcohol companies, and the local psychiatric clinics to allot the twins to one of five categories of increasingly severe drinking problems (Table I). Second, he combined this record data with information he gathered by interview and produced a "compound" classification, again with five categories, this time ranging from abstainers and below-average consumers to chronic alcoholics (Table I). Kaij then compared the extent to which the co-twins in monozygotic (MZ) and dizygotic (DZ) pairs fell into the same drinking category as the probands.

According to the classification by official records, MZ co-twins were more likely to be concordant than DZ co-twins, although this difference was only just significant at the $P < 0.05$ level; 25.4% of MZ co-twins and 15.8% of DZ co-twins fell into exactly the same drinking category as the probands. Categorization by "compound" classification produced a more significant difference: 53.5% of the co-twins of MZ probands were allocated to the same drinking category, compared with only 28.3% of co-twins of DZ probands. When only probands with chronic alcoholism were considered, 71.4% of the 14 co-twins of MZ probands were also chronic alcoholics, compared with 32.3% of co-twins of DZ probands.

On the face of it, therefore, Kaij's study suggests a considerable genetic contribution to alcohol abuse and particularly to chronic alcoholism, and his findings have been much quoted in an uncritical fashion. We believe that

Table I. Drinking Classifications Employed by Kaij

	Official records	Compound classification
0	Entirely blank records	Abstainers and below average consumers
1	One recent or two old convictions	Average consumers
2	Two or more recent convictions	Weekend drinkers and above average consumers
3	Reported by relatives, under supervision, or treated >10 years before	Heavy abusers
4	Compulsory treatment <10 years before, alcoholic psychosis and cirrhosis	Chronic alcoholics

Kaij's work is of such importance that his methodology requires examination in considerable detail.

3.1.1. Twin Methods. The essence of a twin study is to obtain a sample of twins whose ascertainment is unbiased by the zygosity of the twins. One strange fact, commented on by Kaij himself, concerns the low proportion of MZ twins in his sample. There appear three possibilities. First, MZ twins may constitute a smaller proportion of same-sexed twins in southern Sweden, a possibility not borne out by other more recent studies. Second, there may have been some bias in methods of ascertainment employed. Third, some pairs may have been misclassified. Kaij himself was responsible for establishing both zygosity and drinking habits and points out that since it was not possible to obtain blood groups on all pairs, "a few MZ pairs may be concealed in the DZ group." This raises the question of whether MZ twins discordant for drinking, who often look very different, may have been wrongly considered DZ.

3.1.2. The Nature of the Sample. Perhaps the key question concerning the extent to which one can generalize from Kaij's findings concerns the source of his sample. Kaij chose as his probands male twins registered with two County Temperance Boards. It is difficult for non-Swedes to fully understand the functions of these Boards, which are legally constituted to be responsible for the social care of alcoholics. However, some idea of their nature can be obtained from Kaij's writings:

> To this board the police, the courts of law, a person's relatives, and others report alcohol abuse, be it temporary or continuous. Doctors usually report their alcoholic patients only when they do not manage to help them.[14]

Such descriptions raise the question of to what extent can those registered with the Temperance Board be considered representative of alcohol abusers in general? Kaij himself was aware of the possibility of sample bias and stated:

> It does not seem very probable that the police make arrests at random, and people in general do not go out into the street and attract attention when drunk. On the contrary, it seems more probable that the majority of those who do are, e.g., psychopaths.

To examine the possibility of sample bias, Kaij[15] reinvestigated men whom Essen-Moller had previously identified as alcohol abusers in the course of an epidemiological study in a rural Swedish community: 41 men from this sample were registered with the Temperance Board, whereas another 112 men were considered by Essen-Moller to be alcohol abusers but were not so registered. Furthermore, 60–75% of those known to the Temperance Board were psychopathic, though surprisingly, the difference between the two groups was not significant. Unfortunately, Kaij did not compare the conviction rate in the two groups. This was a pity, because his monograph shows that in 44 of the 45 pairs of twins described in detail, the proband had been convicted of one or more alcohol-related criminal acts. This is obviously a much higher rate than that reported for most samples of alcoholics.

Kaij's monograph was written before it became clear that there is a considerable genetic contribution to both psychopathy and criminality.[16–18] But, if his sample included an excess of psychopathic and criminally inclined individuals, would this have influenced the findings? Could those antisocial aspects of an alcoholic abuser's personality that were responsible for him being convicted and thereby in the sample have influenced the drinking categorization given to him? The MZ and DZ concordances for being on the register (54.2% and 31.5%) are remarkably similar to those for the "compound" drinking classification (53.5% and 28.3%, respectively). Furthermore, five of the six MZ pairs concordant for chronic alcoholism were also concordant for having convictions.

3.1.3. Investigational Methods. There was a considerable degree of agreement between the two classification symptoms, but this is not evidence that either was valid, since the second incorporated information used in the first. Furthermore, the ratio of the concordance rates for MZ over DZ twins was greater when the categorization of the latter was made on the basis of the "compound" rather than "official record" classification. This could be because the former was a more accurate reflection of drinking habits or because this system of classification was more subject to bias. This latter possibility is raised by the fact that whereas most of the DZ twins were interviewed only once for 30–60 min, 32 of the MZ pairs were persuaded to attend the Department of Psychiatry in Lund for the purposes of detailed psychometric examination. In the course of persuading the MZ twins and in this attendance, further information regarding their drinking habits may have been ascertained.

If more information were available about the drinking habits of MZ than DZ twins, could this have differentially increased their concordance rates? The probands were, by definition, pathological drinkers. Since most alcohol abusers are reticent about their drinking habits, co-twins less adequately in-

vestigated might have appeared wrongly discordant, thus lowering the DZ corcordance rates.

3.1.4. Diagnostic Criteria. Kaij was well ahead of his time in recognizing the difficulties inherent in using social criteria to make the diagnosis of alcoholism.[19,20] Consequently, he employed a "medical definition" that emphasised (1) pathological desire for alcohol, (2) regular blackouts, and (3) physical dependence. He categorized as chronic alcoholics all those who admitted two of these symptoms, those who were continually intoxicated, and those who had had cirrhosis or psychosis.

But would other psychiatrists agree with his diagnoses? For this reason we submitted the case summaries of all 10 MZ pairs that included one or more chronic alcoholics (so called by Kaij) to Dr. D. L. Davies and Professor G. Edwards, two of the foremost British authorities on alcoholism. Kaij's diagnoses and information as to zygosity were witheld, and the two raters were asked whether they considered each twin (1) a normal drinker, (2) an alcohol abuser, or (3) an alcoholic.

The diagnoses of the two raters were almost identical with each other and with Kaij. All three considered the same six pairs concordant and the same four pairs discordant for alcoholism. The two independent raters were also asked whether they considered each twin alcohol dependent or not; Davies rated five pairs concordant and Edwards four pairs concordant for dependence. Edwards also spontaneously rated severity of dependence—he regarded six out of the 16 twins regarded as alcoholic by Kaij to be severely dependent. Thus, Kaij's criteria for alcoholism appear very similar to that of British clinicians, but the majority of his alcoholic twins would not be regarded as severely dependent.

3.2. Recent Studies

We are currently attempting to replicate Kaij's findings in a study based on the Maudsley Hospital twin register which has provided the data base for several important twin studies of other psychiatric conditions.[21,22] We have used this register to identify a consecutive series of same-sexed alcoholic probands. Information regarding the probands and their co-twins has been gathered by a number of means including extensive record searches, a standardized psychiatric interview, the SADS-L,[23] and a standardized alcohol interview schedule.

As present, the identical twin probands and their co-twins have been investigated more thoroughly than their nonidentical counterparts. So far, reliable information has been obtained on 56 pairs of twins at interview, and zygosity has been firmly established. We have deliberately chosen to present our findings in terms of dependence on alcohol rather than alcoholism. The reason for this is that many definitions of alcoholism include social criteria (e.g., arrests for drunken driving) that reflect public attitudes to excessive drinking rather than the drinking behavior itself.

Our preliminary findings[24] suggest pairwise concordance rates for the alcohol dependence syndrome[25] of 21% for MZ twins and 25% for the DZ twins. Analysis of the age distribution of these twins shows that 38% were still below the age of 40 years when last interviewed, and so alcohol dependence may yet develop in a proportion of co-twins. Nevertheless, it is clear that our preliminary findings do not show the same tendency for alcohol dependence to be heritable as Kaij found in Sweden.

It is, of course, possible that our eventual findings will show slightly higher rates for MZ pairs, but this seems unlikely as investigation of the identical pairs is nearly complete. There are a number of possible reasons for the discrepancy between Kaij's findings and ours. First, he studied only men, whereas we included women. Second, patterns of alcohol consumption in the general population in Sweden in the immediate postwar period differ from those in the United Kingdom a generation later. Third, different diagnostic criteria were used. Fourth, whereas his sample may have been biased towards the antisocial abuser, ours, based on a psychiatric hospital, may have been contaminated by other psychopathology. We are currently examining these possibilities.

Recently Hrubec and Omenn[26] have reviewed the computerized medical records of 15,924 male twins aged between 51 and 61 years. As all the twins had served in the U.S. Armed Forces, the investigators were able to ascertain whether any had been listed in the files of the Veterans Administration as suffering from an alcohol-related disorder.

It can be seen from Table II that MZ twins were significantly more often recorded as concordant for alcoholism than DZ twins ($P < 0.05$). They were also more frequently recorded as concordant for alcoholic psychosis and for liver cirrhosis than DZ twins; indeed the MZ/DZ ratios for these latter conditions were higher than for simple alcoholism. Hrubec and Omenn interpret their findings as suggesting a genetic predisposition, particularly to organ-specific complications of alcoholism.

This study obviously refers to a much larger number of twins than either of the two previous studies—271 MZ pairs with at least one member alcoholic compared to ten such pairs in Kaij's study and 28 such pairs in the study of Gurling and his colleagues. But Hrubec and Omenn never personally saw any of their twins, and, indeed, in nine cases out of ten, the only information they had was the diagnosis from the Veterans Administration computer register. The authors acknowledge that their ascertainment of cases was far from complete—they assume that only 50% of cases of alcohol-related disorder were identified. This is borne out by their prevalence figures for alcoholism which, at 2.6–3.1%, are far below what one would expect in a population of veterans.

This low detection rate would not matter if ascertainment of MZ and DZ twins were equally poor. However, the higher concordance for MZ over DZ twins could be explained if alcoholic co-twins of MZ alcoholic probands were more likely to be identified than the alcoholic co-twins of DZ probands. Such

Table II. U.S. Veterans Twin Study

Recorded diagnosis	Monozygotic		Dizygotic		Ratio of MZ/DZ concordance rates
	No. of pairs with at least one affected	Casewise concordance (%)	No. of pairs with at least one affected	Casewise concordance (%)	
Alcoholism	271	26.3	444	11.9	2.21
Alcoholic psychosis	34	21.1	65	6.0	3.52
Liver cirrhosis	140	14.6	216	5.4	2.70

a bias might have occurred given that the relatedness of the MZ twins would be more noticeable than that of the DZ twins.

A second deficiency is that zygosity was determined by blood group in fewer than 10% of the twins, and, consequently, as in Kaij's study, some discordant MZ pairs may have been wrongly recorded as DZ. It is to be hoped that Hrubec and Omenn will now be able to proceed to personally follow up a sufficient number of their twins to clarify these points.

4. Adoption Studies

Adoption studies have been widely employed in psychiatric research as a means of disentangling genetic and environmental influences, since adoptees receive their genes from one set of parents and their family upbringing from another. Four different groups of researchers have used the adoption strategy to study alcoholism. Their methods are summarized in Tables III and IV, and their work is here reviewed in detail.

4.1. Roe's Study

The first study of the adopted-away offspring of abnormal drinkers was that of Roe and her colleagues,[27,28] which is remarkable not only in that it employed the adoptive strategy some 20 years before its general value for genetic research in psychiatry was realized but also for the detailed interviews and the large amount of data collected. Roe compared 36 fostered children whose biological fathers were "alcoholics" with 25 fostered controls whose biological parents were well adjusted without known history of psychiatric disorder or alcoholism. Two of the children with a heavy drinking father and one of the children of normal biological parentage had a drinking problem themselves in adolescence.

However, by adulthood, these small differences had disappeared. The overall personality, social, and occupational adjustment of those in the two groups was similar, and no alcoholics were detected in either group. Roe consequently concluded that "the reported high incidence of inebriety and psychosis in the offspring of alcoholics is not explicable in terms of any hereditary factor." But although the study design was far ahead of its time, the execution of that design left a lot to be desired, thus raising questions about how valid were Roe's conclusions.

4.1.1. Sample Selection. Although the children were selected from "thousands" referred to a child-placing society in New York, it is not clear what the selection procedure was. Certainly, children who met the unstated selection criteria were discarded because they could not be located or lived too far away. Could those not traced have included a disproportionate number of abnormal drinkers? This possible source of bias is compounded by the fact that reliable information about drinking was obtained from 88% of the children of normal parents but from only 75% of the index children.

Table III. Methodological Issues Concerning the Biological Parents of Adoptees

Investigators	Source of adoptees	Selection of biological parents of probands		Selection of control biological parents	Other comments
		Affected parent	Coparents		
Roe and Burks[28]	744 children placed by New York charity between 1898 and 1921	Fathers noted in adoption records to be "heavy drinkers with syndrome"	50% Mothers "sex deviants"; 44% mistreated children; 14% generally inadequate	25 neither of whom were heavy drinkers, psychotic, or epileptic	Method of selecting 36 out 136 alcoholic fathers uncertain
Goodwin *et al.* Male adoptees[29] Female adoptees[31]	5483 adoptees in Copenhagen area born between 1924 and 1947	67 parents (85% ♂) hospitalized for alcoholism	No information	70 of whom neither had psychiatric hospitalization and 37 where one had been hospitalized for a psychiatric condition other than alcoholism; 65 pairs with no psychiatric hospitalization	The combination of the two male control groups was unwise
Cadoret and Gath[33]	1646 adoptees placed by Iowa Childrens Agency between 1939 and 1965	Fathers shown in adoption records to be heavy drinkers	Two mothers mentally retarded, one "deviating personality"	78 pairs without evidence of heavy drinking	Small sample
Bohman[39]	2324 adoptees born illegitimately in Stockholm between 1930 and 1949	Fathers and mothers registered for alcohol abuse with Temperance Board	No information provided	723 fathers and 1029 mothers registered for neither alcohol abuse nor criminality	The illegitimacy of all the adoptees may have biased the sample towards deviancy

Table IV. Methodological Issues Concerning the Adoptees and Their Investigation

Investigators	No. of probands studied	Extent of matching with controls: Age separated from biological parents	Extent of matching with controls: Age, sex	Extent of matching with controls: Experience of fostering	Proportion studied	Source of information	Diagnostic criteria
Roe and Burks[28]	21 ♂ 15 ♀	Mean age of probands, 3.97 yrs; controls, 1.28 yrs	Probands younger and more female	Probands in less affectionate and poorer rural homes, less likely to be formally adopted or receive higher education	37% probands 56% controls	2- to 4-hr interviews with adoptees	Alcohol consumption levels
Goodwin *et al.*[29,31]	55 ♂ 49♀	Both < 6 weeks "First few weeks of life"	All ♂ very close All ♀ very close	Very similar but information only from adoptees	82% probands 73% controls 75% probands 72% controls	2- to 4-hr interviews with adoptees and hospital records	Threshold for heavy drinking very low; alcoholism definition includes social criteria
Cadoret and Gath[33]	4 ♂ 2 ♀	"Separated at birth"	Uncertain	Uncertain	49% of total sample	From adoptive parents and in 54% also via telephone call to adoptee	Feighner criteria
Bohman[39]	131 ♂ 197 ♀	Before 3 years, but probands 3 months later than controls	Very close	Little information	100%	From Temperance Board registers	Registration for alcohol abuse with the Temperance Board

Some[29] have doubted whether the biological fathers of Roe's index cases were, indeed, alcoholic. Certainly, they were not personally interviewed by the investigators, but all save one were regarded as "heavy drinkers with syndrome." By this, Roe and her colleagues meant that their records contained phrases such as "heavy drunkard" and "dipsomaniac" and also showed "a picture of overaggressiveness, disorganized or escape behavior such as repeated loss of job or disorderly conduct." Roe states that the fathers of index cases showed inebriety "in extreme degree."

4.1.2. Control Group. Other questions arise concerning the adequacy of the comparison group, which differed from the index group in a number of important ways (Table IV). The index offspring were older at the time of examination, included a higher proportion of males, and had been, on the average, twice as old as those in the normal parentage group when they were first placed in foster homes. This latter difference arose because many of the index children were removed from unsatisfactory homes by court action. Furthermore, the experience of fostering of the index children was less fortunate, with more of them being placed with less affectionate foster fathers in poor farm homes where they were less likely to be formally adopted or receive further education than the control children. Thus, the index group had a less favorable environment in that they spent more time with their unsatisfactory biological families and then had a less satisfactory experience of fostering. It certainly seems unlikely that such factors would diminish the likelihood of the index offspring developing alcoholism unless some of them associated their biological fathers' drinking with their unhappy early years and contrasted this with happier days with foster parents and consequently turned against alcohol.

4.2. The Iowa Study

Cadoret and Gath[33] compared six adult adoptees who had a biological parent with a drinking problem with 78 adult adoptees whose biological parents had no such history. Two of the former but only one of the latter were blindly diagnosed as "definite" primary alcoholics. When the criteria were broadened to encompass "probable" primary alcoholism, the figures were three out of six and one out of 78. The difference between the two groups for definite alcoholism was significant at about the 3% level, and that for "probable" alcoholism at the 1% level.

However, the picture changes considerably when one considers secondary alcoholism. Seven adoptees received a secondary diagnosis of definite or probable alcoholism, but all of them came from the group without a biological parent with a drinking problem. Cadoret and Gath interpret their findings as supporting an important genetic contribution to primary but not to secondary alcoholism.

In an extension of the Iowa study, Cadoret *et al.*[34] examined the frequency of alcoholism in the relatives of the biological parents of male adoptees to see

if there was a stronger family history of alcoholism in alcoholic than nonalcoholic adoptees. They found that alcoholic adoptees did indeed show a greater frequency of alcoholism in both their first- and second-degree family members than controls, and, in addition, the alcoholic adoptees were more often diagnosed as having had a childhood conduct disorder.

4.2.1. The Sample. Adoptees were identified from an Iowa adoption society, but the procedure was far from simple, possibly because the alcoholism study was just one part of a much larger investigation. Some 1646 adoptive records were searched, and 190 adoptees chosen on the basis that they had been separated at birth and had had no further contact with their biological parents, one or both of whom had "a psychiatric condition or showed behavior consistent with a psychiatric condition." The 194 control adoptees were also selected without a biological family history of psychiatric disturbance and matched the index cases for age, sex, age of biological mother at the time of birth, and time spent in foster care.

Thus, in comparison with Roe's study, the adoptees had much less contact with their biological parents, and, at first sight, the matching process appears to have been much more thorough. However, the matching was not carried out with respect to whether the adoptees were alcoholic or not, and we do not know whether the six adoptees with alcoholism were atypical in ways other than their psychiatric diagnosis.

The 384 total adoptees were than narrowed down to those of 18 years and over, but, unfortunately, 10% of these 173 adult adoptees could not be located, and in a further 40%, the adoptive parents refused to cooperate, so that only 84 adoptive parents eventually were interviewed. The authors claim that those adoptees who had to be discarded for the above reasons did not differ from those who were finally investigated in demographic characteristics, but they could not, of course, show that they were similar in terms of psychopathology or drinking habits. Consequently, this study has been criticized on the basis of the high subject attrition rate and the possible resultant bias.[35]

4.2.2. Information Gathering and Diagnosis. Information concerning the biological parents came from the adoption agency and was inevitably of variable quality. Consequently, criteria for diagnosing alcoholism in the biological parents were rather loose. Perhaps for this reason, the major analyses took as the index group children whose biological parents were heavy drinkers rather than necessarily alcoholic. This is of considerable importance, as it implies a close and possibly genetically transmitted relationship between heavy drinking and alcoholism which some would doubt (see discussion of Goodwin's work Section 4.3).

Information regarding the adoptees themselves was obtained first by a research assistant who interviewed one or more of the adoptive parents using a structured questionnaire with some 150 items, only a minority of which dealt with alcohol use. In only 45 out of the 84 cases was any contact made with the adoptee, and in these cases, contact was limited to a telephone interview of a structured type. Given the proclivity of alcohol abusers to conceal their excessive drinking not only from their parents but also from

strangers calling them on the telephone, there must remain some doubt as to the validity of this data.

The actual criteria used for diagnosing definite and probable alcoholism in the adoptees were the Feighner criteria and are obviously acceptable. What is more controversial is their division of the alcoholics into primary and secondary alcoholics. This distinction, which was based on the work of Robins and Guze,[36] is not universally accepted and is often extremely difficult to make in patients very well known to the investigator. How validly one can make such a distinction on the basis of information from a relative or from the subject himself over the telephone must remain open to question.

4.3. The Copenhagen Study

It was, of course, Goodwin and his colleagues[30–33] who reinvigorated the whole debate about a possible genetic contribution to alcoholism. In their initial investigation,[30] these authors reported on 55 male adoptees with an alcoholic biological parent and on 78 control adoptees without such a history. The former were nearly four times more likely themselves to become alcoholic in adult life (Table V). As a further refinement, Goodwin *et al.*[31] then compared adopted-away sons of alcoholics with their own brothers who had been raised by the alcoholic biological parent; alcoholism rates were similar in the two groups (Table V). Length of exposure to the alcoholic parent was not associated with the development of alcoholism, but the severity of parental alcoholism was positively related to alcoholism in the offspring.

Forty-nine adopted-away daughters of alcoholics were then similarly compared to 47 control adoptees.[32,33] This time, 2% of the index adoptees were diagnosed as alcoholic as opposed to 4%, a difference that, although in the opposite direction to that expected, did not reach significance; 3% of nonadopted daughters of alcoholics became alcoholic in later life.

This series of studies obviously points strongly towards a major genetic component towards alcoholism in males, though not in females. Indeed, Goodwin *et al.*[31] go so far as to state "environmental factors contributed little, if anything, to the development of alcoholism in the sons of severe alcoholics in this sample." This statement is, of course, somewhat implausible given that the availability of alcohol is itself an environmental factor. Goodwin and his colleagues also examined the frequency of other psychiatric disorder in their various groups. There were no differences in the occurrence of sociopathy in either sex (Table V). However, those daughters of alcoholics who were raised by the alcoholic parent were more at risk in later life of both depression and drug abuse than either their adopted-away sisters or control adoptees. These findings point to the damaging consequences for girls of being raised in homes disrupted by the presence of an alcoholic parent.

4.3.1. The Sample. This was drawn from a pool of 5483 nonfamilial adoptees established for the famous adoption studies of schizophrenia by Kety and his colleagues.[37] One of the biological parents of each of the index cases had been hospitalized for alcoholism; 85% of these affected parents

Table V. Copenhagen Adoptee Study

	Sons			Daughters		
	Adoptees without a biological parent with alcoholism (n = 78) (%)	Biological parent hospitalized for alcoholism		Adoptees without a biological parent with alcoholism (n = 47) (%)	Biological parent hospitalized for alcoholism	
		Adopted (n = 55) (%)	Not adopted (n = 30) (%)		Adopted (n = 49) (%)	Not adopted (n = 81) (%)
Heavy drinker	36	22	3	0	2	1
Problem drinker	14	9	3	0	2	2
Alcoholic	5	18	17	4	2	3
Sociopathy	6	5	6	0	2	3
Depression	20	15	20	15	14	27
Drug abuse	2	9	6	0	2	12

were, in fact, the fathers. This is in contrast to the adoption studies of schizophrenia in which the affected parent generally was the mother, and it raises the question of how sure the investigators could be that these were the real fathers. Among the general population, it is estimated that one in ten of supposed fathers is not the real father, and the proportion is likely to be higher among women who cohabit with or have sexual relations with an alcoholic, and put their children up for adoption.

So it seems unlikely that all of the index cases were, in fact, the certain children of alcoholic fathers. This does not diminish the significance of the findings; rather, it enhances them, since the index group was likely contaminated by control cases.

In the remaining 15%, the mother was the alcoholic. Here, one cannot rule out the theoretical possibility that her alcohol consumption during pregnancy could have biased the unborn child towards heavy alcohol consumption in later life. Goodwin and his colleagues were writing before the fetal alcohol syndrome became well known, and, therefore, they did not ascertain whether those adoptees who became alcoholic in later life had had an alcoholic biological father or mother. This they need now to do, bearing in mind that the spouses of some of the alcoholic fathers may also have been heavy drinkers. For the adoption strategy as employed by Goodwin and his colleagues does not take into account the fact that the adoptees had two and not just one biological parent. As Rosenthal[38] has said in another context, "such a strategy is rather like Gregor Mendel crossing one type of plant with other plants whose characteristics he knew nothing about and attempting to relate all the characteristics of the second generation to the parent plants he was familiar with."

4.3.2. Controls. Perhaps a more serious objection concerns the control group used in the first study of adopted-away sons. Originally, two control groups were employed—70 adoptees whose biological parents had had no psychiatric hospitalizations and 37 adoptees whose biological parents had been hospitalized for a psychiatric condition other than alcoholism. One would have thought that the first group would have provided a "pure" control, but for some unspecified reason, the two control groups were combined. Could this have been to increase the likelihood of finding significant differences? Since certain psychiatric conditions associated with diminished alcohol consumption are also partly heritable, e.g., obsessional neurosis, this combined control group cannot be regarded as satisfactory.

4.3.3. The Follow-up. Further difficulties arise with the follow-up in that 14 out of the 67 index male adoptees either could not be located or refused to cooperate (20.9%) compared with 27 out of 107 male controls (25.3%). Since those who were not included are likely to have preferentially included alcoholics, the larger proportion of controls discarded may have resulted in an artificially low rate of alcoholism in them.

4.3.4. Diagnosis. Goodwin *et al.*[29,30] clearly describe their criteria for alcoholism; to qualify, an individual had to be a heavy drinker and also have

had alcohol problems in three out of four groups. However, the consumption levels stipulated were by no means large, and two of the four problem groups concerned society's response to drinking rather than the actual drinking itself. Thus, it would have been possible for an adoptee to have been diagnosed as alcoholic on the basis of (1) having drinks daily for 1 year and having had 6 or more drinks at least two or three times a month, (2) having had a parent who disapproved of his drinking, (3) having had a traffic arrest for drinking, or (4) having experienced loss of control of drinking.

These criteria do not appear very strict but nevertheless produce a curious anomaly. If the cut-off point for abnormality is widened to include not just alcoholism but also problem drinking, then evidence for any genetic predisposition vanishes. Indeed, the control adoptees were more frequently categorized as heavy or problem drinkers than the index adoptees (Table V). This finding contradicts the evidence of Kaij's twin study and Cloninger's adoption study *(vide infra)* that not only alcoholism but also milder alcoholic abuse is under some degree of genetic influence. Furthermore, this finding runs counter to all the evidence that heavy drinking and alcoholism are closely related.[2,3] Could it be that Goodwin's findings are simply an artifact produced by the threshold for alcoholism accidentally dividing heavy drinkers in the index and control groups unevenly?

4.4. Swedish Adoption Study

In 1978, Bohman[39] reported on a large adoption study of alcohol abuse that depended on data from the Swedish Temperence Board. The sample consisted of all 2324 illegitimate children born in Stockholm between 1930 and 1949 who were later placed in adoptive homes before the age of 3 years. Records reviewed showed that male adoptees whose mothers or fathers had been registered as alcohol abusers were more likely themselves to be similarly registered.

Then Bohman and his colleagues selected 50 male adoptees whose fathers had been repeatedly registered for alcohol abuse—i.e., they were probably alcoholics—and very carefully matched them with adoptees whose parents had no such history. Twenty percent of the former but only 6% of the latter were similarly registered.

Thus, this study suggests a genetic contribution to alcoholism in men, but, as in Goodwin's study when similar analyses were made for female adoptees, no evidence in favor of a genetic transmission was apparent. Of course, since alcohol abuse was uncommon among women at the time this study was carried out in Scandinavia, it may have been that the sample was too small to show any significant effect.

Very recently, Cloninger and his colleagues[40] have reexamined and enlarged Bohman's data on male adoptees. This study incorporates detailed and exhaustive statistical procedures including discriminant function analyses of data concerning both the biological and adoptive parents. Cloninger *et al.*[40]

conclude that alcohol abuse in males is a manifestation of several partly overlapping processes with distinct genetic and environmental backgrounds. They distinguish between a less common but highly heritable type of abuse associated with extensive treatment for both alcohol abuse and criminality in the biological father and a more common "milieu-limited" type in which either parent may or may not be a mild abuser though not a criminal and in which environmental factors are of crucial importance.

4.4.1. Comments. Taken together, the findings of Bohman and Cloninger are quite remarkable. The sophisticated design of the studies enables them to examine environmental as well as genetic variables and allows for interaction as well as additive effects. It is very likely that their methodological approach will be employed in further studies not only of alcohol abuse but also of other behavioral disorders.

Of course, like Kaij, these workers relied on the Swedish Temperance Board to identify alcohol abuse, and consequently, many of the criticisms made of Kaij's study on this account also apply to their data. Like Kaij, too, they subdivided alcohol abuse into types of varying severity largely on the basis of how many registrations individuals had with the Temperance Board. They proceeded to apply very powerful statistical procedures to these data, but one has doubts about the validity of the original distinctions.

Cloninger *et al.*[40] did address one of the criticisms often made of adoption studies, i.e., that the adoptees are abnormal because of their initial contact with their biological parents. They found that adoptees who lived with their biological mother for more than 6 months had 1.5 times more risk of later alcohol abuse than others, but this did not account for the differences between the biological children of alcohol abusers and nonabusers.

4.5. Half-Siblings

Half-siblings offer similar opportunities for separating possible genetic and environmental effects on drinking behavior. In 1972, Schuckit *et al.*[41] reported on the frequency of alcoholism in the 164 half-siblings of 69 alcoholics. Such half-siblings are of particular interest because, depending on their biological parentage and on by whom they were raised, they share a varying degree of genetic and environmental relatedness with an alcoholic. Thus, 46 of the half-sibs had at least one alcoholic biological parent, and 118 had no alcoholic biological parent: 14 of the former and 73 of the latter were raised with the (later alcoholic) proband.

Schuckit *et al.* demonstrated that it seemed to matter little whether the half-sibs were raised by an alcoholic parental figure or not, since the same proportion of both groups became alcoholic. Having an alcoholic biological parent appeared to be much more important. Thus, only 14% of those without an alcoholic biological parent themselves became alcoholic, whereas 50% of those with an alcoholic biological parent but raised by a nonalcoholic pair of parents became alcoholic.

4.5.1. Comment. This appears an excellent methodological strategy, but the results may be affected by the rather disrupted lives that many of these children had. For instance, 81% of the alcoholic half-sibs lived in a broken home. Another strange finding is the fact that having been raised with the proband who later became alcoholic appears to diminish the chance of a half-sib also becoming alcoholic.

This study has also been criticized on the basis of the predominance of black subjects,[35] but many other studies could be criticized because all their subjects were white.

A much more important criticism is on the basis of the very wide concept of alcoholism employed. Schuckit and his colleagues state that "alcoholism is defined as drinking in a manner that interferes with ones life." This wide definition may explain the very high rates of "alcoholism" that the authors found, and it may be more correct to regard the study as being concerned with alcohol abuse or problem drinking rather than alcoholism.

5. Conclusions

Many of the studies of normal twins have methodological deficiencies concerning their size, the nature of their samples, and the validity of their data concerning alcohol consumption. For instance, only in the study of Clifford *et al.*[13] has any attempt been made to check self-report with serum-glutamyl transpeptidase levels. Nevertheless, the most detailed of these studies[4,12,13] all suggest a modest but significant genetic contribution to overall consumption patterns in men. The latter two studies concur in finding that female drinking is under less genetic influence, whereas the first and third both suggest that heredity plays a smaller role in problem drinking than in amount consumed. These studies, however, are not ideally suited to examining problem drinking because of their predominantly normal samples.

The two twin studies specifically confined to abnormal drinking in men have both suggested a genetic contribution with the greatest effect on the most severely affected—chronic alcoholics in Kaij's[14] study and those with alcoholic psychosis or cirrhosis in the study of Hrubec and Omenn.[26] Of course, Kaij's findings, strictly speaking, refer to a population biased towards the antisocial, whereas Hrubec and Omenn's study is open to criticism on account of its poor and possibly biased ascertainment of abnormal drinkers. Perhaps, therefore, one should not too lightly dismiss the third study, that of Gurling *et al.*,[24] which failed to find any difference in MZ and DZ concordance rates for alcoholism in a psychiatric hospital-based population.

There is also some question over how appropriate the classical twin method, on which these three studies depend, is for studying alcoholism. This method assumes that any excess in concordance for MZ over DZ twins can be attributed to heredity. Criticisms of the method[42] have been largely rebutted for

disorders such as schizophrenia and manic–depressive psychosis where proximity to an affected person does not play a major etiological role. However, S. Torgersen (personal communication) has suggested that greater similarity of rearing patterns may increase concordance for neurosis in MZ twins, and peer pressure to drink is known to increase risks of alcoholism.[43] Since MZ twins are more likely to live together in adult life than DZ twins, could this proximity increase their concordance for alcoholism? An examination of the six MZ pairs in Kaij's study who were concordant for chronic alcoholism reveals that only one pair were reared apart from infancy, and another pair remained single and living together throughout life. Seven twins out of the remaining four pairs did marry, but at a mean age of 30 years. Thus, at least some of the pairs concordant for chronic alcoholism may well have been living together through the critical early years in which drinking habits are often established.

Adoption studies, of course, avoid such problems but raise others instead. As Gottesman and Shields[22] point out, "adoption studies are very useful, but like all strategies, limited in their resolving power and not necessarily the best for genetic research once the field of enquiry has been delimited. . . . " One particular limitation is the fact that parents who give up their children for adoption are not typical of the general population or probably of the general run of alcoholic parents. Neither are adoptees typical of the population in general. Hutchings and Mednick,[18] who studied the same adoption pool as that used by Goodwin *et al.*, found that the adoptees were twice as likely to have a criminal record. Thus, the samples of the adoption studies, like those of Kaij, may be biased towards the antisocial.

If we turn to the individual adoption studies, it is clear that the work of Roe and her colleagues was extremely careful, with a great deal of attention given to questions of method such as objectifying the interviews employed. But in certain crucial areas (e.g., sampling), the execution of the study design was imperfect, and in others (e.g., the inadequacy of the controls), the material itself let the investigators down. The investigators were well aware of the deficiencies, and their supervising committee[44] concluded that "little could come out of the study that would meet the standards of scientific genetics."

The results of the Iowa studies clearly contradict those of Roe and her colleagues, but can we put any more credence on these positive findings than Roe's negative ones? The authors are sophisticated investigators, but their initial work suffers from being a by-product of a larger study. Their high attrition rate, use of second-hand information, and their division of alcoholism into primary and secondary types are all open to criticism. Further investigations currently under way in Iowa are likely to produce more durable results.

There can be no doubt that Goodwin's series of studies is most impressive in both its design and execution, as one would expect considering that it was modeled on the famous adoption study of schizophrenia by Kety *et al.*[37] There

are two major specific criticisms concerning (1) the merging of two control samples and (2) the diagnostic criteria for alcoholism given the paradoxical distribution of heavy but nonalcoholic drinking.

It ought to be within the powers of the investigators to clarify these points. First, they could compare the data concerning the adopted-away sons of alcoholics against only those control adoptees without any history of psychiatric disturbance in biological parents. Second, by making available the actual case histories of the index and control adoptees, the authors should enable other researchers to apply their own independent diagnostic criteria.

The question of whether similar etiological factors operate on alcohol abuse as on alcoholism is a crucial one. Several studies suggest that there is little or no genetic contribution to problem drinking that is not a result of primary alcoholism,[4,13,30,34] whereas others suggest that there is indeed such a contribution.[14,39] Studies of the type carried out by Cloninger *et al.*[40] offer the prospect of resolving these contradictions, as their analyses take into account not only the possibility of heterogeneity but also of the differential interactions of specific combinations of genetic and environmental variables. It is perhaps unfortunate that the original information concerning the adoptees' drinking habits may not have been sufficiently accurate to merit such elegant analyses.

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3

Pharmacogenetic Approaches to the Neuropharmacology of Ethanol

Dennis R. Petersen

Abstract. The literature cited in this review clearly demonstrates that many of the behavioral and pharmacological responses to either acute or chronic actions of alcohol are indeed heritable. This conclusion is supported by data derived from several different animal models that have been genetically manipulated to display a wide variety of alcohol-related responses. It is doubtful if any one specific animal model will be developed that will serve as a prototype for human alcoholism. When one considers the amount of knowledge resulting from the pharmacogenetic studies reviewed here, it is more likely that major advances in our understanding of alcohol's complex actions will be derived from several different animal models.

1. Introduction and Basic Considerations

Pharmacological responses elicited by alcohol are numerous and complex. The factors that control an individual's pharmacological response to ethanol can be grossly divided into two major categories, environmental and genetic. Examples of environmental factors are the dose, frequency, and duration of ethanol administration. These ethanol-related environmental factors could in turn influence a number of other environmental factors such as nutritional status or selected organ toxicities that could have a profound effect on the pharmacodynamics of ethanol. There is no doubt that genetic factors also play a major role in an individual's pharmacological and biochemical responses to acute or chronic alcohol consumption. Realistically, most alcohol-related phenotypes are a combination of environmental and genotypic components that can potentially interact to complicate further the assessment of a given behavior or pharmacological response. The number and complexity of the environmental and genetic influences on response to ethanol make it very difficult to utilize human volunteers in studying the acute or chronic actions of alcohol. In addition, there are also ethical considerations and rigid guidelines that must be taken into account in conducting such human studies.

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In view of the difficulty of conducting human research in this area, it is not surprising that nonhuman primates and laboratory rodents have been employed extensively in studying both the behavioral and the biochemical effects of alcohol. There is little doubt that the use of animal models permits better control of the environmental factors discussed above. Likewise, by utilizing laboratory rodents of known genotype, more accurate assessments of alcohol-related phenotypes are possible. Given these considerations, properly chosen animal models will continue to play a very important role in the study of the neuropharmacology and toxicology of alcohol ingestion.

2. General Utility of Pharmacogenetic Approaches in Studying the Neuropharmacology of Ethanol

As noted above, a major advantage of employing animal models in alcohol research is the investigator's ability to decrease the environmental sources of variation that may affect the phenotype of interest. This is advantageous since drastic changes in environmental conditions may be only temporary, and their effects may be difficult to assess, especially by means of behavioral or pharmacological measures. It is obvious that more accurate evaluations of responses to alcohol can be obtained when environmental factors such as temperature and nutrition are controlled. The same holds true with respect to the genetic components that contribute to phenotypic variation. The ability to identify and control both environmental and genotypic variation will result in a more predictable and reproducible response.

It is important to note that genotypic variation is undesirable only if the researcher is not aware that it is contributing to the phenotype under investigation. As will be obvious from the following discussion, the proficiency of recognizing and manipulating genetic variance associated with many alcohol-induced behavioral or biochemical responses can be very beneficial. Clearly, attempts to control environmental influences and recognize genotypic parameters of a particular animal model will result in greater understanding of behavioral and pharmacological responses. The assumption that there will be genotypic variation between animals of the same species, unless records of pedigree indicate otherwise, is the most fundamental concept of pharmacogenetics.

The use of pharmacogenetic concepts in developing animal models for alcohol research has yielded important information and will continue to do so. Typically, investigators have attempted to identify specific individuals within a given species that differ greatly from each other in a behavioral or pharmacological response to alcohol. Once these individuals or subpopulations have been identified, attempts are made to establish a relationship between the observed response and some biochemical or neurochemical parameter that may explain why the response differs from one individual or subpopulation to another. This approach exemplifies a basic utility of phar-

macogenetics in delineation of the biochemical or neurochemical basis for individual responses to alcohol.

The use of genetically different subpopulations or individual animals of the same species exemplifies another basic pharmacogenetic approach: the utilization of genetic variation to test certain hypotheses concerning acute or chronic actions of alcohol. Without a doubt, the recognition and manipulation of the genetic variation existing in a given animal model represents one of the most valuable tools of pharmacogenetics. It will become apparent in the following sections of this chapter that implementation of these basic concepts concerning genotypic variation has been successful in generating a number of animal models that are highly divergent in their behavioral or biochemical responses to alcohol.

3. Feasibility of Utilizing Pharmacogenetics in Animal Model Development

A number of investigators have conducted pharmacogenetic studies to generate animal models for alcohol preference as well as for the behavioral responses resulting from acute or chronic alcohol exposure. Regardless of the phenotype under investigation, animal models in alcohol research have been developed with the hope that they will somehow help to explain why some people abuse alcohol. If this question can be answered by one or more animal models, the cost–benefit ratio is indisputable. Available data indicate that at least partial answers to this question can be obtained by employing various pharmacogenetic animal models. To believe that one animal model will encompass all of the parameters necessary to provide a true template of human alcoholism is naive. However, it is quite possible that the use of several different animal models may enable us to determine the roles that such factors as innate or acquired tolerance play in human alcoholism.

4. Principles of Pharmacogenetic Models Used to Study the Neuropharmacology of Ethanol

4.1. Inbred Strains

Inbred strains of mice and rats are commonly used as pharmacogenetic animal models to explore behavioral and pharmacological responses to alcohol. Twenty consecutive generations of strict brother/sister matings are necessary to generate what is customarily accepted to be an inbred strain. Because this extensive inbreeding forces homozygosity at all loci, genetic variability within inbred strains is reduced to near zero. Thus, for all practical purposes, all individuals within a given inbred strain are genetically identical.

Given the mating system used to generate inbred strains, it is not sur-

prising that the specific genetic architecture of an inbred strain is dependent on that existing in the original brother/sister parents and becomes fixed in that configuration as a matter of chance. This fact accounts for the very limited genetic variation within inbred strains as well as for the extensive genetic variation that exists among strains. The among-strain genetic variation has served as the basis for utilizing inbred strains of mice or rats as a source of subpopulations, within a given species, that are very divergent in their responses to alcohol. The fact that inbred strains represent genotypes that are stable over time makes them extremely useful in alcohol research. They do, however, suffer from the disadvantage that their genotype is fixed purely by chance. Therefore, when one observes a correlated response between two traits in an inbred animal, it should not be inferred as being indicative of a cause–effect relationship.

4.2. F_1 and F_2 Populations

The offspring that result from mating two different inbred strains are designated the F_1 generation. These offspring are heterozygous at each locus at which the parental strains differed. Being the product of two inbred strains, the F_1 offspring are genetically identical. Pharmacogenetic studies utilizing F_1 offspring are usually conducted to determine if a given F_1 phenotype has a greater resemblance to one or the other parental strain. At this level of analysis, no conclusion can be drawn about the number of genes or the genetic mechanisms controlling the response.

When two individuals of an F_1 generation are mated, the resulting offspring are termed the F_2 generation. Because the F_1 individuals are heterozygous at each locus at which the parental strains differed, the F_2 offspring represent a genetically segregating population. As a result, each F_2 animal is genetically unique. An associated feature of the F_2 generation is that genetic segregation and recombination have been forced, thereby breaking up spurious associations of genes or gene complexes. Thus, more meaningful interpretations can be made concerning the cause–effect relationships of correlated biochemical or behavioral responses. It is generally believed that the genetic variation in an F_2 generation is limited by that originally existing in the parental strains used to generate the F_1 offspring. This may or may not be true, depending on the phenotype being studied. McClearn[1,2] has provided detailed discussions of the use of inbred strains and F_1 or F_2 populations in alcohol research.

4.3. Heterogeneous Stocks

The utility of genetically segregating populations of laboratory animals was pointed out in reference to the F_2 offspring described above. It was also pointed out that the genetic variation in these offspring could be limited by

the between-strain differences existing in the parental stocks used to produce the respective F_1 offspring. The utilization of heterogeneous animal populations takes advantage of an animal model that is genetically segregating and displays an extremely high degree of genetic variation.

Several heterogeneous mouse stocks are available. These stocks are usually established by intermating a number of inbred strains and mating the resulting offspring in a systematic rotational scheme that maximizes outbreeding (see McClearn *et al.*[3] for a detailed example of such a scheme). The result is a population of animals that display a high degree of heterozygosity, with each animal being genetically unique.

Because heterogeneous stock animals are genetically segregating, they are of great value in performing studies designed to determine the potential association or cause–effect relationship between two traits. Thus, these animals provide the best model for investigating the relationship between a given neurochemical or biochemical mechanism and a specific alcohol-related behavior. The validity of such studies is based on the fact that use of a genetically segregating population significantly decreases the possibility of spurious association or correlation of the phenotypes under investigation. If a consistent pattern of association of two or more traits is observed in a number of heterogeneous stock animals, it is likely that these traits may share a common mechanism or component of variance.

As noted previously, genetically heterogeneous animals typically display extensive genetic variation and, if maintained properly, very little inbreeding. For these reasons, they are usually the preferred foundation population for selective breeding experiments.

4.4. Selectively Bred Lines

There are a number of different strategies that can be used to generate animal models for alcohol research by selective breeding. Regardless of the strategy implemented, the ideal outcome would be the generation of selectively bred lines that are highly divergent with respect to the selected phenotype. Such divergent populations are extremely valuable since they can be used for correlational analyses to determine what traits share common mechanisms with the selected phenotype.

The animals serving as foundation stock for selection studies should represent a genetically segregating population. Thus, selected lines could be derived either from a heterogeneous stock or from genetically segregating F_2 or F_3 generations described above. The major advantages of such animals are that they are genetically defined, exhibit a high degree of genetic variation, and should be randomly segregating at all loci. In initiating a selection study, all animals in the foundation population are tested for the phenotype in question. Females displaying low values for the trait are mated with males showing low values, whereas females having high values are mated with males exhibiting high scores. The lines begun by these matings are maintained

by selectively mating males and females with high scores to generate the "high" line and males and females with low scores for the "low" line. This selection procedure represents a bidirectional scheme that, if successful, maximizes the divergent phenotypic values. As selection is continued, an increase in homozygosity within lines for all genes influencing the trait will occur, whereas genes at all loci not influencing the trait should continue to segregate randomly.

The optimally designed selection study employs the basic design described above except that replicate high and low lines are maintained in order to strengthen interpretation concerning correlated responses of traits in either line. In addition, duplicate control lines (populations of animals contemporaneously mated) should be maintained to monitor between-generation environmental sources of variation that are only temporary and could affect expression of the selected trait. Another important mechanistic consideration of selective breeding experiments is the importance of maintaining a large enough number of male–female mating pairs within each line to avoid significant inbreeding, which could result in spurious correlated responses of various traits with the selected phenotype.

A classic selection study designed with these optimal guidelines in mind was that performed by DeFries *et al.*[4] to generate lines of mice that are highly divergent with respect to open-field activity. The results of this selection study (see Fig. 1) illustrate that there was approximately a 20-fold difference in mean open-field activity between the replicate low and replicate high lines by the 30th selected generation. Figure 1 also illustrates how temporary environmental effects can be detected in the randomly mated replicate control lines. For a detailed explanation of the theory and design of selection studies, the reader is referred to Falconer[5] and DeFries.[6]

5. Pharmacogenetic Studies of Ethanol Preference or Voluntary Ethanol Consumption

5.1. Inbred Strain Comparisons

Whatever the physiological basis for alcohol preference is, we must assume that it is somehow related to the acute actions of alcohol. These acute actions may include its taste, its olfactory properties, or perhaps its caloric value. At present, it is difficult to justify that alcohol preference in laboratory animals has any relevance to the reasons why humans consume alcohol for its drug-related properties. Given the number of alcohol preference studies that have been conducted, very little has been learned about the biochemical and neurochemical mechanisms controlling this phenotype.

In the 1960s, alcohol preference was one of the most extensively studied alcohol-related phenotypes in laboratory animals. Alcohol preference is measured by offering individual animals free choice of either tap water or tap

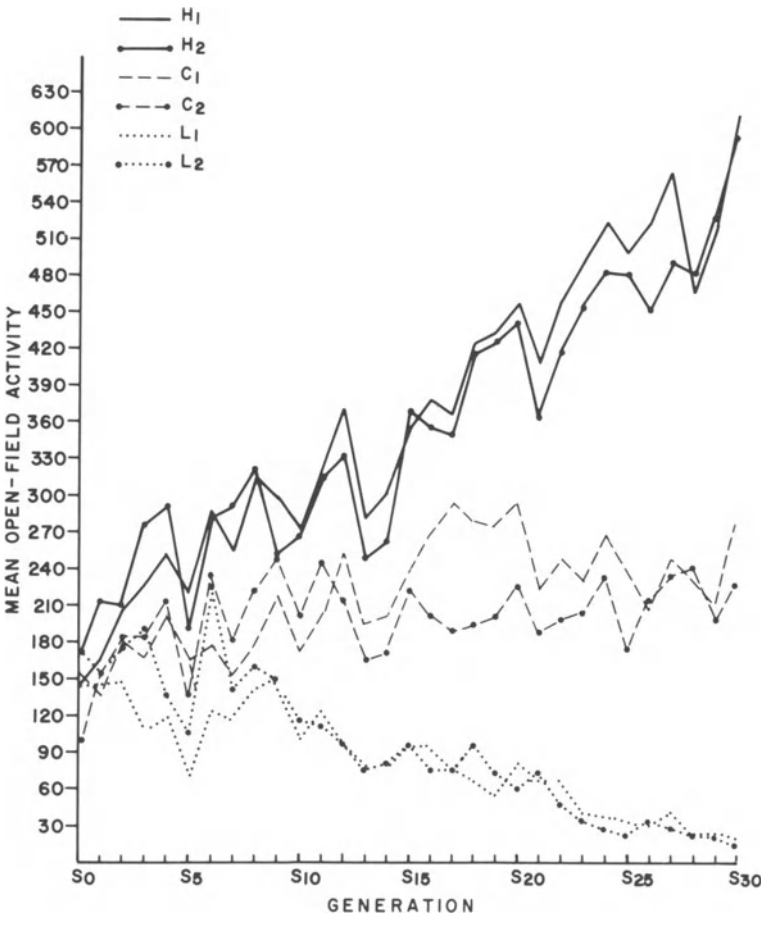

Figure 1. Mean open-field activity scores. Average activity scores for six lines of mice selected for high open-field activity (H_1 and H_2), low open-field activity (L_1 and L_2), and two randomly mated within-line controls (C_1 and C_2). The animals used as the foundation populations for this selection study were F_3 generation mice derived from BALB/cJ and C57BL/6J crosses. Within-litter selection was practiced within each closed line. (Reprinted, with permission, from DeFries *et al.*[4])

water containing 10% v/v ethanol. Alcohol preference is expressed in terms of a ratio calculated by:

$$\frac{\text{Alcohol solution consumed}}{\text{Water consumed} + \text{Alcohol solution consumed}}$$

Obviously, high values indicate a greater preference for alcohol, and low values indicate water preference. Typically, alcohol preference ratios have

been based on mean consumption data collected over a period of 14 consecutive test days.

The vast majority of alcohol preference data has been obtained utilizing inbred strains of mice. A number of investigators[7–9] have demonstrated that inbred mouse strains exhibit a wide range of ethanol preference ratios. A consistent feature of these studies is the fact that C57BL mice display mean preference ratios (0.6–0.95) that are significantly greater than those observed for DBA mice (0.0–0.04). Other inbred mouse strains have been found to have preference ratios ranging between 0.1 and 0.2, indicating that, aside from C57BL mice, laboratory mice generally prefer water to a 10% solution of alcohol. Some investigators[8,9] have attempted to determine the genetic mechanism by which preference might be inherited by evaluating alcohol preference scores in F_1, F_2, or back-cross generations of the C57BL and DBA inbred strains. In general, it was concluded that the presence of a higher proportion of C57BL genes in a given genetically defined population results in a higher mean preference ratio. However, the distribution of phenotypic variances obtained in these studies indicates that inheritance of alcohol preference is most likely dependent on many different genes. Likewise, the influences of various environmental factors are of importance.

Other investigators,[7,10] using C57BL and DBA mice and their F_1 or F_2 generations, generally replicated the alcohol preference studies described above. Collectively, the data concerning the inheritance of alcohol preference in inbred strains of mice clearly indicate polygenic inheritance.

It is of obvious interest to quantitate what proportion of the observed phenotypic variances in alcohol preference can be designated as genetic in nature. Heritability estimates represent such a quantitative term and reflect the ratio of additive genetic variance to total phenotypic variance. Estimates of heritability are useful in that they generally describe genetic variance that is additive and predictable. Heritability estimates range from 0 to 1, with values approaching 1 indicative of a trait whose phenotypic variance is largely genetic in origin.

Conservative heritability estimates based on reanalyzed data from the above studies[11] have resulted in a maximal estimate of 0.15. These estimates indicate that the additive genetic variance associated with the alcohol preference behavior is rather low. For example, assume that an individual mouse is found to have a mean alcohol preference score of 0.20 and that the mean score of the population of mice tested was 0.10. By subtracting the mouse's individual value from the population mean and multiplying that difference by the heritability estimate, $(0.20 - 0.10) \times 0.15 = 0.015$, we get a rough estimate of the proportion of the observed preference score that is genetic in nature. Thus, in this hypothetical example, 0.015 units (15%) of the preference score could be considered to have an additive genetic basis, whereas the remaining 0.085 units (85%) was caused by temporary environmental and genetic effects that are not predictable with respect to their contributions to future generations.

Based on these calculations, it is apparent that environmental factors play a large role in the phenotypic expression of alcohol preference. In addition, the use of inbred strains in these studies has not allowed the identification of behavioral or biochemical mechanisms that might account for the difference in alcohol preference displayed by the highly divergent C57BL and DBA mice. This is a major disadvantage that is encountered when inbred strain comparisons are used to answer mechanistic questions concerning virtually any phenotype, whether it be behavioral or biochemical.

5.2. Selection Studies

5.2.1. Alcohol Preference. Another pharmacogenetic approach that has been taken to study alcohol preference in laboratory rodents is the genetic selection studies for high and low alcohol preference in rats initiated at the Research Laboratories of the State Alcohol Monopoly (ALKO) of Finland.[12] The initial goals of this selection study were to produce a genetic stock of rats that voluntarily drink physiologically effective amounts of alcohol in relation to a line or stock of rats that voluntarily consume very small amounts of alcohol.

The animals used in this selection study originated from a population of rats derived by cross breeding Wistar and Sprague–Dawley albino rats. The cross-bred offspring were then systematically mated in a manner that minimized inbreeding for an additional 20 generations. At this point, the animals were obviously randomly segregating and displayed extreme variation in voluntary alcohol preference.[13] At the age of 3 months, a 10% ethanol solution was provided as their only source of fluid for a period of 10 days. The rats were then offered a choice between 10% v/v ethanol solution or tap water for a 3-week test period. Preference scores were determined by the amount of ethanol consumed in relation to the total fluid consumption during these 3 weeks. From this base population, families displaying either high or low preference values were chosen for breeding purposes for formation of the high and low preferences lines, respectively. In subsequent generations, breeding animals were chosen from 10–15 families. Typically, about 20–25% of the individual animals making up these families were chosen for breeding purposes. Sib matings were intentionally avoided.

This selection study has resulted in two lines of rats that are highly divergent in their alcohol consumption during the self-selection period described above. These selected lines have been designated as AA (ALKO, Alcohol) and ANA (ALKO, Nonalcohol) for the high and low alcohol-preferring lines, respectively. Data reported for the F_{32} generation indicate that the preference ratio of AA rats averages 0.62, whereas that of the ANA animals is 0.14.[14] Thus, it appears that selection on the basis of preference scores has been very successful in generating two lines of rats that differ markedly. Throughout the development of AA and ANA lines, a number of behavioral, biochemical, and neurochemical studies have been performed, which are the

subject of a recent review article.[15] Despite these extensive studies, the exact biochemical or neurochemical mechanisms responsible for the difference in voluntary alcohol consumption by AA and ANA rats have yet to be identified.

Nevertheless, this selection study has contributed a great deal to our knowledge concerning different methods of assessing alcohol preference. For instance, up to the 19th generation of selection, AA and ANA lines were selected on the basis of preference scores as well as the quantity of alcohol consumed daily per kilogram body weight, with the later measurement receiving most consideration. The result of this approach is apparent when the average body weights of AA and ANA male and female rats in the F_{29} generation are examined. Male rats of the AA line are 30% heavier than the ANA males, and female AA rats are 15% heavier than ANA females.

This difference surfaced early in the selection study and prompted establishment of a new criterion for selection in generation 19 which was based on the preference for alcohol calories expressed as the energy derived from alcohol as a percentage of total energy intake. The results of this decision are apparent in the F_{32} generation: significant line differences in body weight have disappeared, although AA males have an alcohol intake (g/kg per day) fivefold greater than ANA males. Alcohol intake, measured on the same basis, is eightfold greater in AA females than in ANA females. The AA males derive 16.0% of their total energy from alcohol, whereas ANA males derive only 4.0%. Female AA rats obtain 10.0% of their gross energy from alcohol, as opposed to 3.0% for ANA females. Since the F_{29} generation, however, selection criteria have been formulated to consider alcohol preference ratio, alcohol intake (g/kg per day), and caloric intake. These phenotypes have been considered with equal weight during selection of subsequent generations.

The alcohol preference measures obtained during the time course of this selection study are interesting in several respects. First, the results of this study indicate that there is enough additive genetic variation associated with the preference phenotype to successfully select for and generate lines of rats that differ in alcohol preference when given a choice between 10% v/v ethanol and tap water. Second, the methods of selection employed in this study suggest that alcohol preference can be assessed not only by the standard calculations of alcohol consumed in relation to total fluid consumption but also by measures of alcohol consumption per unit body weight or the amount of alcohol-derived calories in relation to total caloric intake. If these lines had been generated with the goal of investigating the pharmacological or biochemical basis of human alcohol abuse, the latter measures, which take into account metabolic factors that could affect the concentration of alcohol in the body at any one time, might have been the most desirable selection criteria.

5.2.2. Selection for Free-Choice Alcohol Consumption. Very divergent populations of alcohol-preferring and alcohol-nonpreferring rats have been generated through selective breeding studies at the University of Indiana.[16] The foundation population used for this selection study originated from a closed colony of Wistar rats (Wrm:WRC[WI]BR) housed at the Walter Reed

Army Institute of Research. A number of male and female rats of this randomly mated population were given a 10% ethanol solution as their only source of fluid for 4 days immediately prior to a 3-week preference test during which each animal had access to both 10% ethanol and tap water. As is the case with most preference studies, food was provided on an *ad libitum* basis. Preference was designated as the average volume of 10% ethanol consumed daily during the last 2 weeks of the preference test period. Under these conditions, rats in this base population were found to have consumption scores ranging from 0 to 35 ml of 10% ethanol per day.

From this base population, the male and female with the highest ethanol consumption were mated to initiate the preferring strain (P), and a single pair with very low consumption were mated to begin the nonpreferring strain (NP). This type of testing for voluntary ethanol consumption and subsequent matings have continued, with the most recent data reported for the 13th selected generation. Based on the average amount of alcohol consumed per day per unit body weight during the last 2 weeks of the testing period, there has been an amazing response to selection. Voluntary ethanol consumption of the F_1 generation was in the range of 3 g/kg per day. By the tenth selected generation, ethanol consumption of the P and NP rats was 6.3 and 1.0 g/kg per day, respectively. It is important to note that a significant number of the P animals will consume ethanol at a rate that nearly exceeds the rate at which P and NP rats metabolize ethanol (5–7 g/kg per day). Thus, it is not surprising that blood ethanol levels in P rats 10.5 hr into their dark cycle range from 19 to 105 mg%.[17] Should the P animals continue to consume alcohol at this rate for a long enough period of time, it is entirely possible that they may represent an animal model that will voluntarily consume alcohol to the point of dependence. In fact, when the body weight of P rats was reduced by 20% prior to a 40-day, free-choice ethanol consumption period, the rats were found to consume ethanol at rates up to 10 g/kg per day. When ethanol was removed, a number of these animals displayed signs of withdrawal.

Like the AA and ANA rats, the P and NP rats have been used in a number of behavioral,[18] biochemical,[19] and neurochemical[20] studies. These animals display some very interesting differences in behavioral and neurochemical parameters. Given the high degree of inbreeding associated with the derivation of these lines, however, it is dangerous to assume that any of the observed behavioral or neurochemical differences are causally related to the observed divergence in voluntary ethanol consumption. Nevertheless, data obtained from the P and NP rats are extremely valuable for purposes of comparisons with data obtained from similar studies with inbred strains or with the AA and ANA selected lines. In addition, the P and NP rats could serve as valuable foundation stock for future genetic studies dealing with the neuropharmacology of ethanol.

Pharmacogenetic studies of alcohol preference and voluntary alcohol consumption leave little doubt that these are heritable traits. Genetic effects have been revealed by comparisons of inbred mouse strains and by successes in

generating selected lines of rats that either prefer or avoid alcohol in a free-choice situation. As noted previously, recalculated heritabilities for alcohol preference in a wide range of studies range from 0.10 to 0.15. Obviously, these low heritabilities do not restrict the degree of divergence attained but only influence the amount of time necessary to achieve maximum separation of the lines. The selection studies mentioned above represent successful attempts to generate animal models to study the pharmacogenetics of voluntary alcohol consumption. Most importantly, these animal models are now being carefully and systematically catalogued for a number of behavioral, biochemical, and neurochemical parameters.

6. Pharmacogenetic Studies Designed to Generate Animal Models for Studying the Acute Actions of Alcohol on the Central Nervous System

Humans most likely consume alcohol for its pharmacological effects. With this in mind, several investigators have attempted to generate animal models that display specific pharmacological responses to the acute administration of alcohol. These animal models were ostensibly developed to increase our understanding of the mechanisms involved in the acute actions of alcohol. In addition, it is likely that the models were developed with the idea in mind that the resulting alcohol-sensitive or alcohol-insensitive phenotypes might somehow be related to mechanisms directly or indirectly responsible for the chronic actions of alcohol.

6.1. Selection for Alcohol-Induced Sleep Time in Mice

One of the earliest and most successful selection studies for differential responses to the acute actions of alcohol involved the selective breeding of mice that differed in the duration of ethanol-induced sleep time following a hypnotic dose of ethanol.[21] The foundation animals for this selection study were from the heterogeneous stock (HS) mice described earlier. These mice represent a highly heterogeneous, randomly segregating population. A number of HS mice were given a 3.3 g/kg intraperitoneal injection of ethanol. Once an animal had succumbed to the hypnotic effects of this alcohol dose, it was placed on its back in a V-shaped trough in order to assess the duration of loss of the righting response. The test criterion for righting response was performance of the righting response three consecutive times in three consecutive 30-sec periods. Under these conditions, the duration of loss of the righting response ranged from 0 to approximately 7000 sec. From this foundation stock, 59 animals representing eight families were selected to serve as parents for the first generation of the "short-sleep" (SS) line, whereas 29

animals representing six families were chosen as parental stock for the "long-sleep" (LS) line.

Mass selection, which dictates that individuals are chosen solely on their own phenotypic merit, has been practiced throughout this selection study. Mates are assigned at random with a restriction against sib matings. The results of selection through the 25th generation are shown in Fig. 2. During this selection experiment, the number of family members has ranged from three to 13 in the SS line and one to 11 in the LS line. It can be seen in Fig. 2 that the duration of loss of the righting response in the fifth generation of selection was about 1500 sec for SS mice and about 4500 sec for LS mice. As indicated at the top of Fig. 2, a dose of 3.3 g/kg ethanol was used through the seventh generation. However, because a substantial number of SS mice did not lose the righting response at that dose, a dose of 3.5 g/kg was ad-

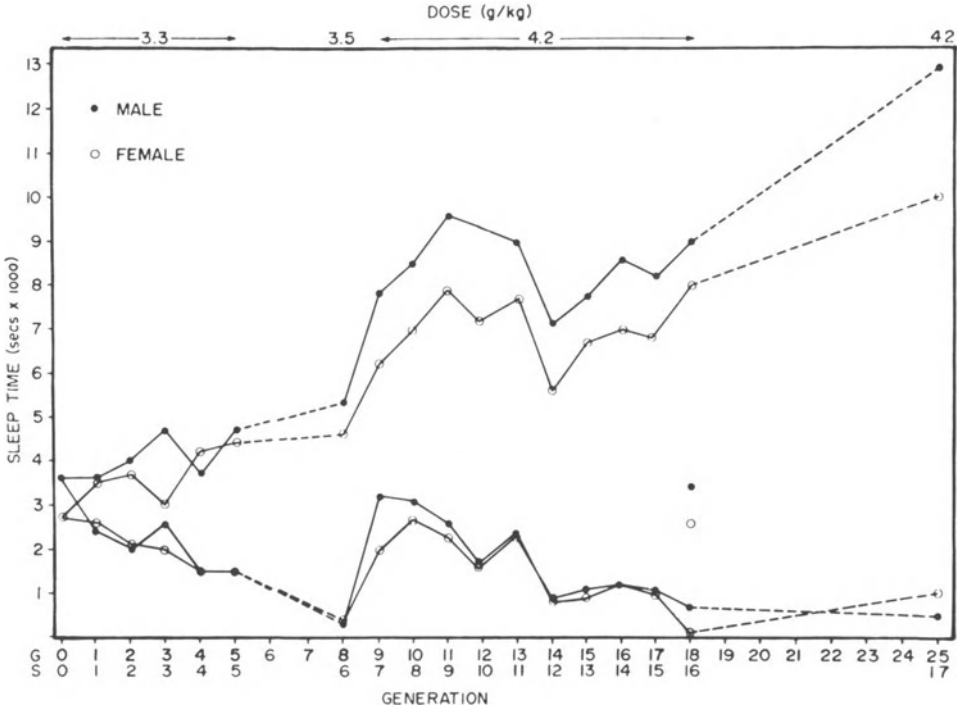

Figure 2. Mean times for loss of the righting response in long-sleep (LS) and short-sleep (SS) selected lines of mice. The data are plotted separately as a function of sex and line. The top two functions represent the LS line, and the bottom represent the SS line. The isolated data points at the S_{16} generation represent the mean values of male and female HS mice. As indicated in the text, the dashed lines indicate suspension of selection. (Modified from McClearn and Anderson.[31])

ministered in generation 8. The dose was again increased to 4.2 g/kg in generations 9 through 25. In the 25th generation, SS animals sleep an average of less than 1000 sec, whereas sleep time of the LS animals ranges from 10,000 to 13,000 sec. It should be noted from Fig. 2 that, even though this study has continued for 25 generations, selective breeding had actually been practiced for only 17 generations. This is because the lines were maintained by random mating within lines rather than by mass selection during generations 6, 7, and 19–24.[21] Thus, the divergence in duration of ethanol-induced loss of the righting response displayed in Fig. 2 is the result of only 17 generations of selective breeding.

The response to the first five generations of selection is for the most part linear and can be used to calculate a heritability estimate for this trait. Based on the first five generations, a heritability estimate of 0.18 has been obtained.[21] Thus, we have another example of a trait with a low heritability that under the appropriate selection pressure can be used to generate genetically distinct populations of animals. If the numbers of family members in each line in each generation are large enough, and inbreeding is kept at a minimum, these divergent populations should represent animals that are homozygous at those loci to which selection has been directed and randomly segregating at all nonpertinent loci.

Several behavioral, biochemical, pharmacological, and neurochemical studies that have been performed on these animals are the subject of a recent review.[22] These studies have found no definitive relationship between the phenotype selected for and other phenotypes such as innate tolerance to alcohol or the severity of withdrawal reactions following chronic alcohol administration. The fact that these selected lines do not differ in rate of ethanol disposition indicates that the genetic differences are mediated by the central nervous system.

A recent electrophysiological study[23] has very elegantly localized the neuroanatomic brain region that might partially explain the differential responses of these selected lines to acute hypnotic doses of ethanol. In this study, electrophysiological recordings were obtained from cerebellar Purkinje neurons in anesthesized LS and SS mice. These recordings were monitored following local acute ethanol administration by micropressure ejection. The dose of ethanol necessary to produce half-maximal depressions in spontaneous Purkinje cell firing was found to be 16-fold greater for SS mice. When effective doses of ethanol were repeatedly administered, LS neurons were reproducibly depressed, but SS neurons developed tolerance to the point that subsequent ethanol applications elicited no depression.

It appears that selective breeding for the duration of ethanol-induced sleep time has resulted in profound differences in electrophysiological activity of Purkinje neurons following the local application of ethanol. The exact mechanisms responsible for this difference in these excitable membranes remain to be determined. However, this electrophysiological study demon-

strates the potential value of selective breeding in generating animal models that can be used to test hypotheses concerning the actions of alcohol at macro- and micromolecular levels.

6.2. Selection Studies for Sensitivity to Subhypnotic Acute Ethanol Intoxication

6.2.1. Alcohol-Tolerant (AT) and Alcohol Nontolerant (ANT) Selected Lines of Rats. In 1973, the Physiology Department of the Research Laboratories of the State Alcohol Monopoly (ALKO) in Finland initiated a breeding program to produce a maximally heterogeneous colony of laboratory rats.[14,24] This breeding program represents a systematic cross-breeding scheme whereby various established stocks of laboratory rats (Wistar, Sprague–Dawley, Long–Evans, etc.) were mated with the previously described AA and ANA selected lines. The result of these systematic matings is a relatively genetically heterogeneous stock of rats which served as the foundation stock for selective breeding of the AT and ANT lines.

The purpose of this selection experiment was to produce lines of rats that are highly susceptible or resistent to acute subhypnotic doses of ethanol. The criteria used for assessment of sensitivity to acute doses of ethanol were alcohol impairment in open-field, tilting plane, and rotarod performance tests. Animals in the foundation population were subjected to each of these tests in the absence of ethanol for the establishment of base-line values. Thirty minutes following intraperitoneally administered ethanol, the animals were tested again to determine impairment on each of these behavioral tests. There was an interval of at least 7 days between tests. The ethanol doses varied according to test, with 1.2 g/kg used for open-field testing and 2.0 g/kg and 1.5 g/kg used for the tilting plane and rotarod, respectively. Blood alcohol determinations were made after each test. The selection of mating pairs was based mainly on the degree of impairment on the tilting plane test, with minor weight given to rotarod performance. Five to seven breeding pairs have been used each generation in the AT and ANT lines, and there is a restriction against sib matings. Approximately 20–30% of the animals tested each generation have been chosen for breeding purposes.

Data from the seventh generation of selection indicate that the AT rats are significantly more tolerant to the intoxicating effects of acute doses of ethanol as judged by performance on the tilting plane and rotarod tests. Blood ethanol levels in the two lines were not significantly different at the time the tests were performed, suggesting that observed differences in tolerance are mediated by the central nervous system and are not caused by differences in ethanol disposition. Another interesting feature of the AT and ANT rats is their voluntary ethanol intake. When naive AT and ANT rats are subjected to the typical two-bottle alcohol preference test, the AT rats will consume twice as much alcohol as the ANT animals.[25] Additional preference testing,

coupled with the appropriate correlational analyses, will be necessary to delineate the genetic association between innate tolerance and voluntary alcohol preference.

As the AT and ANT selection continues, it is likely that genetic stocks of rats will be generated that are even more highly divergent in their innate tolerance to subhypnotic doses of ethanol. As the selection proceeds, the genetic association between innate tolerance and such phenotypes as voluntary ethanol consumption and the development of tolerance to chronic ethanol ingestion will undoubtedly be vigorously tested.

6.2.2. Least Affected (LA) and Most Affected (MA) Selected Lines of Rats. This selection procedure[25] is similar to that employed to generate the AT and ANT rats in that the alcohol-related behavior was assessed following administration of subhypnotic doses of ethanol. The alcohol-related phenotype selected for in this study was the degree of locomotor impairment measured by means of an activity platform or stabilimeter following a 1.5 g/kg dose of ethanol.

The rather unique foundation stock used to generate the LA and MA selected lines consisted of Sprague–Dawley albino and Long–Evans hooded rats. After these animals were subjected to the locomotor test following a saline injection for establishment of base-line data, they were given a 1.5 g/kg intraperitoneal ethanol injection and evaluated for locomotor activity from 16 to 30 min after injection. The "least affected" Sprague–Dawley albino rats were mated with the "least affected" Long–Evans hooded rats, and the "most affected" rats from the albino and hooded stocks were mated. The offspring from these matings constituted the F_1 generation of the LA and MA selected lines. Four breeding pairs per line have generally been maintained, with the restriction that a male and female from each litter must contribute to the next generation. However, in the F_8 generation, this strategy was replaced by selecting the four most extreme rats of each line for mating in each generation but avoiding brother–sister matings.[25]

There was no significant divergence between the MA and LA lines up to the fifth selected generation. However, from the fifth generation to the 13th, a steady divergence has been observed, and the MA rats showed a sixfold greater decrement in locomotor activity by the 13th generation.[26] The data do indicate, however, that the response to selection has been asymmetrical, with a greater selection response apparent in the MA line. The apparent differential sensitivity of the LA and MA rats appears to be mediated through central nervous system mechanisms, since blood ethanol levels in the two lines are very similar at or shortly after time of testing.[27,28]

The LA and MA rats have been characterized with respect to a number of alcohol-related behaviors. The lines do not differ in consumption of unsweetened ethanol solutions in a free-choice situation or of saccharin-sweetened ethanol solutions when they are offered as a sole source of fluid.[28] However, MA rats are more sensitive than LA rats to the hypnotic effects of

ethanol in that their ethanol-induced sleep times following a 2.5–3.0 g/kg dose of ethanol range two- to threefold longer than LA rats.

Again, the role of innate alcohol tolerance in the development of tolerance and dependence during chronic alcohol ingestion remains to be determined. However, with the availability of the AT, ANT, LA, and MA animal models, such hypotheses can be further tested.

7. Selective Breeding for Intensity of Alcohol Withdrawal Reactions

7.1. General Considerations

Unlike many of the alcohol-influenced phenotypes discussed previously, very few attempts have been made to generate animal models that differ in their response to complex actions of chronic alcohol ingestion. The rarity of such selection studies is probably the result of two factors. First, there has been a great deal of difficulty associated with developing methods for chronically treating laboratory animals with alcohol. Second, problems involved in the assessment of phenotypes related to the development of tolerance and dependence associated with chronic alcohol ingestion in laboratory animals have been difficult to solve. During the past decade, however, several methods have been described for administering alcohol chronically to laboratory mice, and techniques for measuring withdrawal following the termination of chronic ethanol ingestion have been developed. In spite of these methodological advances, there have been only two attempts to breed animals selectively for intensity of withdrawal reactions following termination of chronic alcohol administration.

7.2. Selection for Intensity of Convulsions in Mice following Chronic Ethanol Administration

Goldstein[29] conducted a short-term selection study that demonstrated that the severity of seizures following termination of chronic alcohol exposure appears to have a heritable component. Male and female Swiss–Webster mice were chronically exposed to alcohol by inhalation of alcohol vapor for 3 days. Constant blood alcohol levels of 180 mg% were maintained by using pyrazole to inhibit alcohol metabolism. On withdrawal, the mice were repeatedly evaluated for severity of convulsions elicited by handling. Six weeks following withdrawal, the same mice were again subjected to chronic alcohol treatment for 3 days and scored for seizure severity in order to obtain replicate scores on each mouse. The observed seizure scores were found to be very consistent for any given animal. However, the population as a whole displayed a wide range of scores.

Two mating pairs of seizure-susceptible and seizure-resistant mice were retained and mated to form the high and low selected lines, respectively. When the F_1 animals resulting from these matings were subjected to the same testing procedures, the results indicated that the severity of seizures in the high line was nearly threefold greater than in the low line. Two pairs of the seizure-susceptible F_1 mice and two pairs of the seizure-resistant mice were mated to produce the F_2 generation, which was also evaluated for severity of alcohol withdrawal seizures. The seizures in the high line were found to be almost fourfold more severe than in the low line. An interesting feature of this study is that male mice consistently showed more severe seizures than did females. This was apparently because male mice maintained significantly higher blood alcohol levels during the chronic treatment period. Thus, the importance of standardizing blood alcohol levels during such a study is clearly indicated.

Although this selection study was short term in nature and was based on only a limited number of mating pairs and offspring, it clearly demonstrates the potential for a more extensive selection study based on phenotypes associated with chronic alcohol ingestion.

7.3. Selection for Multiple Phenotypes Indicative of Severe and Mild Ethanol Withdrawal Reactions in Mice

In most of the selection studies discussed up to this point, selection was based on only one phenotype. One of the most effective methods of generating an animal model for nearly any purpose is to select simultaneously for a number of phenotypes that are thought to be important components of the specific model. This type of selective breeding has been termed "index selection." The basis for this method of selective breeding involves measurement of predetermined phenotypes on each individual animal. Each phenotype is statistically weighted according to its predicted degree of importance, and these weighted phenotypic scores are inserted in a linear equation to generate a total score for each animal. Thus, the animals to be mated are chosen on the basis of a total score that represents the contribution of several different traits pertinent to the overall animal model.

This type of selection procedure is now being employed at the University of Colorado Alcohol Research Center to generate selected lines of mice that display either severe or mild withdrawal reactions following termination of chronic alcohol ingestion. The theory and statistical basis for this selection study are presented elsewhere.[30] The foundation stock for the study are the HS mice described above in Section 4.3. A large number of HS mice were treated chronically with alcohol for 9 days by means of the Lieber–DeCarli liquid diet. Six hours after termination of the 9-day chronic treatment period, the mice were subjected to a battery of behavioral and physiological measures indicative of intensity of withdrawal reactions. Since seizures on handling have been established as a valid measure of withdrawal severity, only those

traits that were significantly correlated with seizure score were chosen as phenotypes to be included in the selection index. The traits chosen on this basis included seizure score (a modification of Goldstein's method), rectal temperature during withdrawal, total alcohol consumption (g/kg per 9 days), and four behavioral activity measures during withdrawal (number of crossings, number of rearings, severity of seizures in a hole-in-wall apparatus, and number of squares crossed on a vertical screen). Because five of these seven measures showed a significant sex difference, scores of males and females were analyzed separately. Thus, seven weighted variables were included in male and female selection indices used to compute composite scores.

Another large population of male and female HS mice was subjected to the same procedure and evaluated for the calculation of composite scores. To begin within-family, bidirectional, replicate line selection, a male and a female were randomly selected from each of ten families to begin one control line (C-1). Then the highest and lowest scoring males and females remaining in each of the same ten families were mated to initiate "severe ethanol withdrawal" (SEW-1) and "mild ethanol withdrawal" (MEW-1) lines. Using a separate set of ten families, the same procedure was employed to form replicate control, SEW, and MEW lines. Thus, the study includes six lines: C-1, C-2, SEW-1, SEW-2, MEW-1, and MEW-2. Given this experimental design which utilizes within-family selection, we maintain ten families per line to minimize inbreeding.

This selection study is now in its sixth generation. The response has been bidirectional, with a clear divergence of lines that differ distinctly in the variables included in the selection index described above. Preliminary heritability estimates for the composite phenotype, that is, the basis for selection in this study range from 0.10 to 0.15.

The initial results of this selection study appear very promising. However, continued selection for several more generations will be necessary to achieve lines that are divergent enough to determine the behavioral, physiological, or neurochemical components that predispose these selected lines to their resistance or sensitivity to the withdrawal syndrome associated with chronic alcohol ingestion. Even though the full potential of this selection study remains to be determined, this selective breeding procedure illustrates the optimal approach for generating very robust animal models for nearly any phenotype of interest.

8. Conclusions

From the foregoing discussion, it is obvious that pharmacogenetics has been, and will continue to be, a very useful tool in studying the neuropharmacology of ethanol. When considered on an individual basis, none of the pharmacogenetic studies reviewed here has generated an animal model that can be considered a valid template for human alcoholism. Collectively, how-

ever, these studies have yielded data that clearly demonstrate that many of the behavioral and pharmacological responses to the acute or chronic actions of alcohol have an identifiable genetic component. Genetic influence is clearly indicated by the fact that nearly every attempt to breed selectively for an alcohol-related phenotype has been successful. It is important to note that the utility of animal models generated by these selection studies depends to a large extent on the manner in which they were derived. Such factors as the maintenance of replicate lines and unselected control lines in a manner that minimizes inbreeding are of extreme importance in the development of animal models that are useful for valid correlational analyses.

The purpose of this review has been to describe the various animal models that have been utilized in studying the pharmacogenetics of alcohol's action. This has, I hope, been done in a manner that brings to light the potential of pharmacogenetics in the successful development of valid animal models for studying the complex actions of alcohol or other drugs of abuse.

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II

The Behavioral Treatment of Alcoholism

Edward Gottheil, Section Editor

Overview

Edward Gottheil

Behavioral techniques and principles have been intimately involved in, if not central to, many of the controversies of the past decade and a half in the field of alcoholism. One need only mention nonabstinent outcome criteria, experimentally induced inebriation, controlled drinking, the medical model, or the Rand report to bring to mind the intensity, and sometimes the bitterness, of the arguments concerning these issues. In addition to generating heat, however, the controversies also stimulated many new ideas, research studies, and treatments.

Although the issues remain largely unresolved, the intensity of the arguments appears to have abated somewhat. It seems worthwhile at this time, therefore, to attempt to bring together a group of papers providing an overview of the theoretical rationale and current research support for the application of behavioral methods to the treatment of individuals with alcoholic problems, a description of the different treatment techniques that have been employed, an evaluation of the effectiveness of these techniques, and a sampling of perspectives regarding the current status of behavioral therapy in the treatment of alcoholism.

The first in this series of chapters, drawing on appropriate and relevant learning and conditioning principles and findings from animal and human laboratory studies, sets forth the theoretical background for the section. George and Marlatt trace what they consider to be an evolving behavioral perspective, which has progressed through the stages of classical conditioning, drive-reduction theory, operant conditioning, social learning theory, and cognitive–behavioral theory. Each stage or model and its relevant research findings are reviewed as are their explanatory powers and limitations. What is presented is a clear and organized distillate of a great deal of material that is consistently focused on therapeutic implications and on contributions to our understanding of the processes involved in the acquisition and maintenance of excessive drinking behavior.

Caddy and Block then describe the various behavioral treatment techniques and models that have been employed. The different methods are

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compared and contrasted, and the progression noted from earlier simple, narrow-band approaches to more and more complex broad-spectrum programs. Although the relevant research and rationale for the successive changes and developments are presented, there is a much greater "how to" focus in this chapter than in the one by George and Marlatt.

The effectiveness of the behavioral modification techniques is addressed by Litman and Topham who group the various techniques in the following four main categories: (1) aversion therapy, which subsumes electrical, chemical, and verbal (covert sensitization) techniques; (2) operant procedures, which include contingency management and contingency contracting; (3) broad–spectrum behavioral treatments; and (4) behavioral and self-control training and other cognitive therapies. For the most part, the studies selected for review were done subsequent to 1975 and used adequate methodological designs or at least adequate controls. Greater attention was given to those studies that compared behavioral techniques to more traditional programs and those that compared the efficacy of one behavioral technique with another. The authors conclude that we now have a respectable body of methodologically adequate studies on the use of behavioral programs for alcoholism, that behavioral programs have been shown to be at least as effective as conventional programs in many of the studies, and that in some studies the behavioral treatment outcome rates have been strikingly high. Despite these accomplishments, Litman and Topham note with concern that research into the application of behavioral techniques to the treatment of alcoholism has decreased in quality and quantity during the last 5 years and has not really affected the mainstream treatment of alcoholism. They attribute this mainly to the resistance of traditional regimes, which are based on the assumption that alcoholism is a disease, and to the political climate generated by the "perceived" association between behavioral approaches and controlled drinking.

The authors of the last four papers of the section were asked to comment about their views regarding the place of behavioral treatment in the current therapeutic armamentarium, whether it is likely to become more or less accepted, its impact on theory and understanding, and what clinical directions or developments might be expected in the future. The Sobells, as protagonists for the behavioral approaches, summarize the contributions that have been made with regard to understanding, prevention, treatment, and evaluation methodologies. They see, in contrast to Litman and Topham, an increasing acceptance of behavioral approaches into the mainstream of alcohol treatment research and practice and, at the same time, an increasing acceptance by behavioral researchers of the mainstream view that a comprehensive or biopsychosocial framework is necessary for the understanding and treatment of alcohol problems. It is their expectation that continuing maturation and rapprochement will occur.

Levinson, from an anthropological perspective, suggests that because

behavioral programs are especially broad in approach and goals, they are especially suitable for utilizing cultural factors in assigning individuals to appropriate treatments and in selecting appropriate treatment goals. He then describes the various categories of cultural factors that may be potentially useful to treatment in general and to behavior modification treatment in particular.

Lovibond focuses his remarks on what he expects will be the future directions of behavioral interventions. He suggests that since alcohol abuse is usually found in combination with other health-disrupting behaviors, treatment programs will increasingly be directed toward establishing health-promoting life styles and modifying a number of health-related behaviors simultaneously. The health-promoting programs will utilize a variety of behavioral techniques at appropriate stages to assist the development of behavioral self-management. From this point of view, behavioral management programs and methods are seen as assuming increasing importance.

The concluding paper, written by Moore, presents the viewpoint of a clinical psychiatrist. In general, he sees little that is incompatible in the methods employed by traditional counselors and psychotherapists, Alcoholics Anonymous, and behavioral therapists. All use a variety of behavioral techniques to modify behavior. Nevertheless, it is his impression that the formalized, specific behavioral techniques have not become part of the majority of treatment programs. He attributes this to the deprofessionalization that has occurred in the alcoholism treatment field and the concomitant resistance to new ideas, the reluctance of dynamically oriented professionals (often psychiatrists) to accept behavioral approaches, and the perception that behavior therapy has not yet been proven to be effective. Moore suggests that our treatment techniques have changed little in the past decade and that it is vital to keep the field open to new ideas and new data. If this is done, he believes, progress is most likely to emerge from the development and application of behavioral and/or biomedical interventions.

Taken together, the chapters seem to indicate that behavioral theory and methodology have developed and matured considerably over the past decade. The controversies are less prominent, and even the issue of controlled drinking is only touched on here and there. There is more evolution and less revolution. Biological and sociocultural influences are increasingly being accepted by behavioral therapists and researchers as important determinants in the etiology of alcoholism, although they are not yet fully incorporated into the theoretical framework. Although there are differences of opinion on the extent to which behavioral methodologies have entered the mainstream, further progress and acceptance are expected to occur.

The medical model continues to be, if not a "whipping boy," at least an irritant. This never ceases to puzzle me. Through medical school, internship, and residency, I was never presented with a definition of the medical model or instructed in its intricacies. Indeed, I never heard of it until much later

when I observed it variously defined by nonphysicians as "traditional," "bad," and "juxtaposed to whatever positive approaches" were espoused by the writer.

Actually, there is no medical model, nor is one likely to emerge that would be appropriate to measles, hemophilia, and heart failure. Similarly, no model is proposed by psychiatrists that purports to explain schizophrenia, hysteria, mental retardation, and alcoholism. Instead, there are many models and theories pertaining to the development of diverse symptoms, syndromes, or disease entities, the last of which, of course, has been noted to change markedly from time to time. I was never taught to focus on disease and resist prevention and health maintenance approaches, to eschew science, or to prescribe more medication than was good for my patients. I did hear about Koch, Pasteur, Salk, asepsis, water purification, and child development. I even heard about Wolpe and Sasz in addition to Freud and Meyer. I do not remember ever being told to be authoritarian. What I seem to recall are terms such as listening, nonverbal communication, nonjudgmental attitudes, empathy, ego support, sociocultural milieu, eclecticism, understanding, caring, and rapport. I must grant that some medical practitioners occasionally may overprescribe or display authoritarian attitudes, since I myself have witnessed an example or two. This does not represent *the* or *a* medical model, however, merely bad medical practice.

After reading the manuscripts prepared for this section and noting the differences in opinion about the extent to which behavioral approaches had impacted on the field, I decided to chat with a number of psychiatrists, psychologists, and counselors engaged in the treatment of alcoholics in private and clinic settings in an attempt to gain some information about the extent to which these practicing clinicians were aware of the controversies we have noted, the importance they attributed to them, whether their day-to-day treatment methods had been influenced by them, and how they accounted for the decreased frequency and intensity of debates about the suitability of behavioral techniques for the treatment of alcoholism. Clearly, the comments that follow were those selectively remembered from among those offered by my nonscientific sample of convenience.

Everyone seemed to be well aware of the controversies regarding controlled drinking, the Rand report, and *Clockwork Orange*, if not from the research literature then at least from the newspapers. Generally, these were not listed high as important practical issues concerning the treatment of alcoholism in the last decade. Most felt that abstinence was the desired goal of treatment if it could be attained, and, if not, attempts were directed at helping patients achieve decreased drinking and a better psychosocial adjustment. More important issues related to treatment were felt to be tensions between psychiatrists and psychologists and between degreed professionals and nondegreed therapists, the use of disulfiram (Antabuse®), whether alcohol and drug abusers could be treated together, the treatment of patients with com-

bined psychiatric disorders, the lack of reasonable insurance programs, and the uncertainty of program funding.

Initial responses to the question of whether behavioral views and studies had influenced their treatment practices were usually that they had not. They did not use conditioning procedures, and the more general controversial issues were seen as philosophical and not related to the nuts and bolts of actual practice. When asked whether and how treatment methods had changed over the last 10 years, however, most were quick to note that actual changes had occurred and that many of these charges reflected the influence of behavioral methodologies that they had somehow incorporated without a clear recognition of their source. Those in clinic settings pointed to the preparation of treatment plans, the setting of specific objectives, the reviewing of progress in terms of these objectives, and the setting of new short- and long-term objectives.

In addition to focusing on insights and on the therapeutic relationship, there was a much greater emphasis than in the past on demonstrable behavioral accomplishments. Much of this emphasis was attributed to governmental program regulations and the "system." The motivation for changes in the regulations was presumed to be the pressure for greater accountability and the need to demonstrate effectiveness in some objective fashion. Although there had been initial resistance to the regulatory demands, most were reasonably comfortable with the system and felt that it made sense. The use of behavioral principles by A.A., therapeutic communities, and methadone clinics was also noted.

The private practitioners, moreover, even though they were not concerned about keeping their records in order for governmental site visitors, were also thinking about treatment plans and objectives. In addition, they mentioned the use of contracts, relaxation techniques, patient self-monitoring logs, and other behavioral methods. In sum, nearly all felt that they had incorporated behavioral approaches into their style of treatment, that in doing so, they had changed how they thought about and related to patients, and that this had occurred gradually and almost imperceptibly. How and why had this come about? There were no clear explanations. It was not because they had accepted a new theoretical perspective or because they had seen research studies that had convinced them of the effectiveness of the methods. The system and climate seemed to have evolved and seemed to make sense.

Two main reasons were offered for the decreased intensity of the arguments about the applicability of behavioral techniques. The first was that the arguments had decreased because the principles and techniques had been incorporated. The second was that because of the current problems with respect to funding and the criticisms regarding accountability and effectiveness, individual positions have been abandoned in an effort to present a united front against a greater common threat and danger.

It would appear that behavioral methodologies have entered the field

and are likely to remain. A decade ago, in the midst of controversy and turmoil, many new ideas and treatments were generated. Whatever the reason for the apparent greater harmony in the field at present, the amount of research appears to have decreased. I hope that this is merely a period of consolidation setting the stage for new developments to emerge from the intermingling of ideas of cooperating disciplines.

* * *

Editors' Note:

Perspectives on Controlled Drinking

An article has appeared in *Science*[1] questioning the observations of two studies which were cited in this section.[2,3] These relate to the issue of controlled drinking, which at times has been considered in the context of behavioral treatment for alcoholism. The reader may wish to review that article to obtain an alternate perspective on this matter.

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4

How Environments and Persons Combine to Influence Problem Drinking
Current Research Issues

G. N. Braucht

Abstract. In this chapter, a brief review of existing empirical research on environmental correlates of problem drinking is presented. The review shows that environmental factors do relate to the prevalence of drinking problems and also to the way drinking problems are expressed. In the major section of the chapter, however, it is shown that our present knowledge of how environmental and personal factors combine to influence problem drinking is quite limited, perhaps because almost all of the existing empirical research has attempted to account for problem drinking by means of individual variables alone, environmental variables alone, or in terms of linear combinations of individual and environmental variables. It is shown that alternative approaches offer more promise for understanding how individual and environmental factors combine to influence problem drinking; these approaches are aimed at accounting for problem drinking in terms of the mutual interdependence between persons and their environments. Within two hypothetical sets of data, a number of conceptual and methodological issues, problems, and features of these kinds of interactional or transactional approaches are then illustrated. It is shown that although such approaches offer a promise of greater understanding, they also present a set of interrelated problems which run the gamut from measurement, statistical analysis, experimental design, and sampling issues to paradigm issues lying close to the realm of the philosophy of science.

1. Introduction

Other chapters in this section of the present volume present very thoughtful and careful reviews of existing empirical research bearing on the question of whether genetic factors have been shown to be associated with alcohol consumption behaviors. As a counterpoint to these chapters, the editors of this volume felt that a chapter representing an environmental perspective on the development of alcoholism would provide a valuable measure of balance to this section. In this chapter, I first try to meet this felt need by providing a brief review of some of the major environmental correlates of problem drink-

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ing. This review highlights existing empirical research bearing on the question, "What environmental factors are associated with problem drinking among adolescents and among adults?" However, I endeavor to make short work of this task for reasons that should become abundantly evident below.

Following the brief review, I attempt to show why maintaining separate individual (including genetic) and environmental perspectives is surely not the most useful route to understanding the development of real-life behaviors such as problem drinking or alcoholism. By "separate perspectives," I am referring here not only to attempts to account for problem drinking/alcoholism by means of either individual variables alone or environmental variables alone, but also to attempts to account for these kinds of behaviors in terms of additive linear combinations of individual and environmental variables. Such strategies are aimed at one or another of the following two questions. (1) Which type of factor, hereditary or environmental, is responsible for problem drinking? (2) How much of the variance in problem drinking is attributable to inherited factors and how much is attributable to environmental factors?

In this later section of the chapter, I identify and illustrate some of the desirable features—conceptual and methodological features—of cross-sectional and developmental research aimed at accounting for problem drinking/alcoholism in terms of the mutual interdependence between persons and their environments. It is seen that the prosecution of these kinds of interactional or transactional research strategies requires us to develop creative solutions for a number of interrelated issues. Although these issues present complex challenges, these approaches are aimed at providing answers to what I believe is a much more fruitful question: How do individual and environmental influences combine in the development of problem drinking?

2. Environmental Correlates of Adolescent Problem Drinking

One major national survey of adolescent drinking practices has furnished a basis for estimating the rate of adolescent problem drinking.[1,2] This survey of more than 13,000 junior and senior high school students' drinking practices, attitudes, and correlates was based on a probability sample of all students in grades 7 through 12 in the 48 contiguous states and the District of Columbia. The data included measures of drinking frequency, quantities drunk, negative consequences of drinking, indices of problem drinking, measures of other drug use, general deviant behavior, and a variety of psychosocial measures.[3,4] Rachal *et al.*[2] classified 27.8% of their sample as problem drinkers, based on a criterion of having been drunk four or more times during the prior year or reporting drinking-related negative consequences in two or more life situations (with friends, regarding school, with dates, with police, or while driving).

Donovan and Jessor[3] examined these data using three alternative definitions of problem drinking: (1) 19% were classified as problem drinkers based on a criterion of having been drunk at least six times during the prior year

or reporting negative consequences two or more times in the past year in at least three of the five life areas; (2) 9.4% were so classified using the criterion of having been drunk at least once a month during the prior year; and (3) 8.9% were classified as problem drinkers under a criterion of having experienced drinking-related negative consequences at least twice in any one of the five life areas, with at least one such experience in an additional life area.

All three of these alternative definitions of problem drinking which Donovan and Jessor[4] applied to the data of Rachal *et al.*[1,2] resulted in ratios of approximately three male drinkers for every two female problem drinkers in their total sample of seventh through 12th graders. Although quite different criteria for problem drinking were used, both Globetti[5] and Smart and Gray[6] have also found more high school males than females to be problem drinkers. Analyses of the data of Rachal *et al.* by grade in school have revealed that this gap in problem drinking between the sexes widens with increasing age. Whereas approximately equal percentages of both male and female seventh graders (5% and 4%) were classed as problem drinkers, approximately twice as many 12th-grade boys as girls (40% versus 21%) were classed as problem drinkers.[3] In addition to these age and sex differences, there is substantial agreement from recent national surveys that the South has the fewest adolescent heavy drinkers, whereas the Northeast and North Central regions' rates are highest.[1,7]

Thus, drinking is widespread among adolescents and, depending on the criteria one chooses, can be considered a significant problem for 10% to 20% of them. In this country, there are differential probabilities of being an adolescent problem drinker depending on age, sex, and region. With this basic epidemiologic perspective, we can now turn to the question of what environmental characteristics are associated with adolescent problem drinking.

In their extensive analyses of the national sample of 13,122 seventh through 12th graders reported by Rachal *et al.*, Donovan and Jessor found that perceived environment variables were consistently significant as discriminators between problem and nonproblem drinkers. These environmental differences between problem and nonproblem drinkers were consistently significant for males and females and manifested across all three of their alternative definitions of problem drinking: (1) problem drinkers perceived less compatibility between their friends' and parents' expectations for them than did nonproblem drinkers; (2) compared with nonproblem drinkers, problem drinkers attributed relatively more influence to their friends than to their parents; and (3) problem drinkers perceived more positive models of drinking and approval of drinking in their environment than did nonproblem drinkers.[3]

In this nationwide survey, a set of five perceived environmental variables accounted for approximately twice as much variance in predicting three indices of problem drinking than did a set of four personality variables: (1) in predicting problem drinking status (approximately 20% of the variance in this index of problem drinking was attributable to environmental variables versus 10% attributable to relatively enduring personal variables); (2) in predicting

the number of drunken occasions during the prior year (approximately 31% versus 15%); and (3) in predicting the total number of negative drinking-related consequences during the prior year (approximately 12–15% versus 6–7%). When both environmental and personality sets of predictors were combined, stepwise multiple regression analyses also indicated that the environmental variables had greater predictive power than did the personality variables. For example, in the regression predicting problem drinker status, friends' models for marijuana use and friends' models for drinking entered the equation before any of the three personality measures.

In a completely different investigation (a longitudinal study across the four high school years of a sample of youth from a small city in the Rocky Mountain region), Jessor and Jessor[8] found that several self-reported (perceived) environmental variables were associated with problem drinking in both male and female subsamples. Relative to nonproblem drinkers, problem drinkers viewed their environment as one in which there was (1) less compatibility between their parents' and their friends' expectations, (2) greater friends' as compared to parents' influence, (3) less parental disapproval of drinking, and (4) more peer models of problem behavior. In this carefully designed study, slightly over 20% of the variance in problem drinking status was attributable to a multiple linear combination of these perceived environmental variables.

Zucker and his colleagues[9–12] have examined the influences of the familial environment, parental characteristics, and parental behavior on adolescent problem drinking. Zucker and Barron[11] found that relative to boys who were not problem drinkers, problem-drinking boys had both mothers and fathers who were heavier drinkers themselves, who were more antisocial, and who utilized social isolation and deprivation as disciplinary techniques. Boys who drank more heavily viewed their mothers as less often present but in more neutral terms than they viewed their fathers; they were more negative in their perceptions of their fathers, seeing them as emotionally distant, unrewarding, and uncaring about their achievements.

In a subsequent report on adolescent girls who were heavy drinkers, Zucker and DeVoe[12] found their drinking to be related to several maternal characteristics. Mothers of more heavily drinking girls were heavier drinkers themselves, were characterized as having an aggressive sociability personality style, and utilized social isolation withdrawal of praise or affection to influence their daughters' behavior. Heavy drinking on the part of these adolescent girls was also related to the physical absence and to the drinking problems of their fathers. Thus, the studies of Zucker and his colleagues paint the family environments of problem drinkers as relatively harsh, negative in feeling tone, likely to feature tension-filled interactions, and likely to feature relatively low levels of parental involvement.

Zucker[9,10] has suggested that his findings and the earlier findings of Jessor, Graves, Hanson, and Jessor[13] point to major disturbances in three areas of those families that produce problem drinkers: (1) parental deviance

in personal behavior and heavier levels of drinking; (2) parental disinterest and lack of involvement with their problem-drinking children; and (3) lack of positive parent–child interaction, affection, and nurturance. Zucker considers these kinds of familial/parental environmental conditions to have an indirect influence on later adolescent drinking behavior, arguing that evidence from other research (e.g., 14) suggests that later drinking is more directly influenced by peer behavior.

In addition to these findings regarding environmental correlates of adolescent problem drinking *per se*, environmental factors related to the timing of onset to drinking status have been investigated rather extensively. These studies are of great interest here because longitudinal studies have shown that the precocity with which the abstainer–drinker transition is made is directly related to ultimate levels of problem drinking; problem drinking rates are highest among those who make this transition earliest, and rates are progressively lower among those who make the transition at successively older ages.[15] Therefore, studies focusing on the environmental factors associated with early onset of drinking are reviewed briefly below.

In examining predictions of the onset of drinking from antecedent perceived environmental variables in their 4-year longitudinal study, Jessor and Jessor[15] found that those who remained abstainers throughout their study perceived themselves to be in an environment that provided both the least parental and peer approval for drinking and the least opportunity for drinking. In contrast, the group of adolescents who made the transition from abstainer to drinker status earliest (the group whose members were more often problem drinkers) scored highest on these perceived environmental variables.

Jessor *et al.*[3,8,15] have interpreted their findings as consistent with a general developmental trend away from conventionality over the adolescent period. Their findings suggest that problem drinkers are more precocious in loosening ties with their parents and orienting toward their peers than are nonproblem drinkers. Problem drinkers perceive greater approval, models, and pressure for drinking than the nonproblem drinkers, thus experiencing more social support for drinking and probably more opportunity for drinking.

Findings consistent with this pattern were observed by Kandel *et al.*[16] who examined the relative influence of parents and peers in regard to initiation into the use of distilled liquor. They found that parents most strongly influenced initiation into hard liquor use by acting as role models. The importance of parental modeling is illustrated by their finding that 81% of families in which both parents drank hard liquor produced children who drank hard liquor, whereas, conversely, 72% of the families in which the parents abstained had children who were abstainers.

Kandel *et al.*[16] also found that peer influences were quite important in predicting use of hard liquor. In fact, the most important variable in predicting onset of hard liquor use was the degree of adolescent involvement in peer activities such as getting together with friends, dating, attending parties, or

driving around. In their analysis of the relative influence of peers and parents, Kandel *et al.* found that peer influences accounted for nearly twice as much variance as parental influences in their distilled spirits onset criterion. In another analysis of the onset of drinking in a sample that included both sexes, Margulies, Kessler, and Kandel[17] found that the influence of peers became increasingly powerful at successively older ages, whereas parental influences remained at essentially equivalent levels throughout the high school years.

Taken together, all of the above findings as well as others extensively reviewed by Braucht[18,19] tend to suggest that the acquisition of drinking behavior is a developmental phenomenon that starts in the home for the majority of youth and then progresses to a more peer-controlled context. However, as Zucker[9,10] has observed, there have been few conceptual efforts to outline the mechanisms through which parental and peer influences systematically produce differences in the drinking patterns of young people.

3. Environmental Correlates of Problem Drinking among Adults

Among adults in the United States (as among adolescents), the level of social support for drinking in the environment is clearly related to problem drinking. For example, in Cahalan's report[20] of a major national household survey, environmental support for heavy drinking had a higher correlation with problem drinking than did age, sex, socioeconomic status, or urbanization (city size) variables. These four demographic variables, however, were also significantly related to problem drinking. The gross findings were that problem drinking levels were greater (1) among men than women; (2) among persons under 50 than among older persons; (3) among persons of lower socioeconomic classes than among those of higher socioeconomic classes; and (4) among persons living in larger cities than among those living in smaller towns or rural areas.

In the case of the urbanization finding, it appeared that the underlying factor was environmental support for heavy drinking (there were also higher levels of social support for heavy drinking in larger cities than in smaller towns). Cahalan concluded that

> the inference drawn from findings of this study is that a lower level of social controls and a more permissive 'drinking climate' in the larger cities may be primarily responsible for much of the difference in problem drinking in larger and smaller cities or towns, rather than the often-presumed higher level of alienation and maladjustment among those living in larger cities (p. 141).

Further evidence of the association between social environmental factors and problem drinking was demonstrated in an analysis of the interactions of 16 variables in predicting problem drinking among men. This analysis showed that the subgroup with the highest problem drinking rate was a small group

distinguished by extremely favorable attitudes toward drinking, high scores in environmental support for heavy drinking, and having been high school dropouts.

This national survey also found that different kinds of problem drinking were related to membership in different ethnoreligious groups (e.g., Irish Catholics were high on social consequences problem drinking, whereas Jewish persons were quite low). Here again, levels of environmental support for heavy drinking among the various ethnoreligious groups tended to be associated with problem drinking in those groups (in this case, for example, Irish Catholics were high and Jewish persons low in environmental support for heavy drinking). Thus, not only did these ethnoreligious groups differ in levels of social support for heavy drinking, but the expression of those drinking-related problems was manifested in those groups in different and subculturally patterned ways.

Finally, this two-wave national study of adults showed that problem drinking levels were responsive to several kinds of changes over the 3-year span between survey waves. Drinking problems were found to increase or diminish in relation to environmental changes in (1) marital happiness, (2) frequency with which drinks were served when respondents were with their friends, (3) marital status, and (4) the death of a parent or child.

Thus, the findings of Cahalan's national study of U.S. adults replicated and extended a number of sex, age, socioeconomic status, level of urbanization, and ethnic–religious group differences in problem drinking. Although varying interpretations have been made of precisely which sociocultural constructs or mechanisms underlie these kinds of group differences, these differences themselves are ones consistently reported and consistently explained in sociocultural terms—in terms of group norms, group cohesiveness, social supports, social controls, etc. (see Cahalan,[20] pp. 75–78, for a succinct review of earlier findings and interpretations).

In another major stream of research, the relationship between the general level of alcohol availability in a given environment and the prevalence of problem drinking has been studied rather extensively. Both survey data and rates of mortality from liver cirrhosis have shown a link between the average per capita consumption of alcohol in a population and the prevalence of very heavy alcohol use in that population.[21–23] The precise relationship may not be that originally put forward in 1956 by Ledermann[24,25]; the explanation of what mechanisms of social interaction bring about the effect is uncertain,[26] and the controversy surrounding this link is charged with policy implications for prevention.[27,28]

Still, the balance of the evidence carefully reviewed by Makela[29] has led him to conclude that although there are important variations in problem drinking that are not related to average level of consumption, in those cases where overall consumption has been reduced, the decrease in average consumption has been accompanied by reduced consumption by heavy drinkers, and increases in average consumption levels are associated with higher prev-

alence of problem drinking (although the increases are probably less than proportionate to the increase in average consumption). This apparent relationship has spawned a number of studies examining the relationship between average consumption levels and various kinds of environmental variables plausibly bearing on availability, including legislation, regulation, spacing and density of retail outlets, advertising, and pricing of alcohol.[27,28,30]

Perhaps of greatest interest in the present context, however, are the large number of family studies that have found that problem drinking is associated with family histories in which poverty, heavy drinking, broken homes, and other circumstances are featured.[31,32] Of course, family studies almost always pose basic problems of interpretation because genetic and family environmental factors are usually confounded as well as a variety of other reasons.[33]

However, let us now turn to an examination of two recent studies reported by Cloninger and his colleagues.[34,35] Although these studies have come out of a research literature in which the primary focus is on genetic contributions to problem behaviors, it can be seen that their findings provide rather powerful evidence that the environment plays forceful roles in shaping the development of problem drinking.

First, Cloninger *et al.*[34] have reported an extensive analysis assessing the goodness of fit of three competing multifactorial models in accounting for observed sex differences in the prevalence of three problem behaviors. Sex differences in prevalence were observed in three samples: (1) in a sample of persons with antisocial personality diagnoses who were under probation or parole in St. Louis as a result of felony conviction; (2) in a sample of persons with interview diagnoses of alcoholism who had been admitted to one of two St. Louis psychiatric hospitals; and (3) in a sample of Danish twins who had criminal records. There were marked preponderences of men in all three samples.

In the three competing multifactorial models, specific and explicit assumptions were made regarding how much sex differences in prevalence could be caused by genetic factors, familial environmental factors, and nonfamilial environmental factors. Cloninger *et al.* found that sex differences in the prevalence of both antisocial personality and criminality appeared to be caused by familial factors. Sex differences in the prevalence of alcoholism, however, were found to be caused by nonfamilial (i.e., environmental) factors. Cloninger *et al.* also found that nonfamilial environmental factors appeared to be more important in influencing the development of alcohol abuse in women than in men and that the familial factors relevant to the development of alcohol abuse (an unknown mix of genetic and family environmental factors together) appeared to be the same for both sexes.

In the second paper, Cloninger and colleagues[35] reported a cross-fostering analysis of a sample of 862 Swedish men who had been adopted at an early age. In this investigation, Cloninger *et al.* began by recognizing two fundamental problems that have impeded research aimed at the question of how genetic and familial environmental influences combine to influence the de-

velopment of alcohol abuse: the clinical heterogeneity of alcohol abuse and the confounding of genetic and environmental influences within families. Therefore, they used a multivariate strategy based on a classification of the adoptees into one of four levels of alcohol abuse (no, mild, moderate, and severe abuse) and identification of (1) four different types of congenital predispositions to alcohol abuse (genetic predisposition to no abuse, to mild abuse, to moderate abuse, and to severe abuse) and (2) four types of familial environmental predispositions to alcohol abuse (again, environmental predispositions to no abuse, to mild abuse, to moderate abuse, and to severe abuse).

Having thus distinguished relatively homogeneous types of abusers and having a sample without confounded (but with known) types of genetic and family environmental backgrounds, they were able to identify two forms of alcohol abuse that were linked to different combinations of genetic and environmental factors. The occurrence of one type depended only on genetic background factors; the expression of moderate alcohol abuse did not depend on exposure to familial environments identified as ones predisposing to moderate abuse. The other type of abuse, however, was seldom expressed unless the adoptee had been reared in the type of postnatal family environment that predisposed to that type of abuse; whether this type of abuse occurred or not depended primarily on whether or not the adoptee had been exposed to the appropriate type of postnatal family environment. Cloninger *et al.*[35] were thus able to go quite far in showing that specific constellations of genetic and environmental factors combined in different ways to influence the development of alcohol abuse.

With regard to the importance of environmental factors, Cloninger *et al.*[35] concluded that

> the demonstration of the critical importance of sociocultural influences in most alcoholics suggests that major changes in social attitudes about drinking styles can change dramatically the prevalence of alcohol abuse regardless of genetic predisposition (p. 867).

In the context of "making the case for environmental factors," I would like nothing better than to leave this statement without comment here. However, it must be said that of the 40 abuse cases belonging to the milieu-limited type of alcohol abuse who did have the appropriate type of genetic predisposition, 25 did not have the appropriate type of environmental predisposition (calculated from Figs. 1 and 3, p. 865). Likewise, of the 36 abuse cases of the type Cloninger *et al.* referred to as "highly heritable" (p. 866), 19 did not have the appropriate type of genetic predisposition (calculated from Fig. 2, p. 865).

Three points are being made here. First, there were a great many sporadic cases of alcohol abuse in their study. Forty-five percent of all of the alcohol abuse cases (68 of the 151 abuse cases) had neither an appropriate genetic nor an appropriate environmental predisposition; for this sizeable group, no account at all could be given for their abuse. Second, the effects Cloninger *et*

al. found were based on very small numbers of cases relative to their large sample size. Third, all of the conceivable genetic and environmental factors that might predispose to alcohol abuse were obviously not available for their cases.

Therefore, although all of the interpretations by Cloninger *et al.* of their positive findings are valid, some of their statements regarding failures to find significant relationships are perhaps too sweeping. For example, their conclusion that "postnatal environment does not influence the risk of the male-limited form of alcohol abuse" (p. 866) could perhaps be better stated as "Postnatal environment *[as assessed in this study]* did not influence the risk of the male-limited form of alcohol abuse." This latter statement would correctly recognize that further research may well unearth environmental factors that do affect the development of persons with this type of genetic predisposition.

Despite these reservations, I believe that the research of Cloninger and his colleagues is of great importance and significance, especially as regards their strategy of isolating relatively homogeneous subsamples and examining how individual and environmental factors combine to influence the development of alcohol abuse in each subsample. And their work does show that environmental factors are quite potent, as indicated by their finding that "the 239 adoptees who stayed with their mothers beyond age 4 months had 1.5 greater risk for later alcohol abuse than the others" (p. 864). In fact, only one of the six genetic risk ratios that could be calculated from data in their paper was significantly greater than this single environmental risk ratio (for the presence of recurrent alcohol abuse in the biological father, the risk ratio calculated from data in Table 1, p. 863, was 1.7).

With this perspective on what is known regarding the environmental factors involved in problem drinking, let us now turn to a brief list of some things we do know and some things that we do not know about problem drinking. First, the results of a number of empirical studies have shown that neither the concept of "the adolescent problem drinker" nor "the (adult) problem drinker" are unitary, homogeneous concepts. In both age groups, there are multiple types of problem drinkers. The research of Braucht[36] and Donovan and Jessor[4] has shown that distinct constellations of personal and environmental factors are found in different types of adolescents with different levels of problem drinking. Of course, the recent research of Cloninger and his colleagues indicates that this is so for adult problem drinkers as well.[35] Their work also strongly indicates that there is more than one developmental pathway to (adult) problem drinking, as has been hypothesized in both the general field of human development by Block[37] and in the specific field of substance abuse by Braucht,[18,19] by Braucht and Braucht,[38] and by Gorsuch and Butler.[39]

However, we have as yet only begun to answer the question of how personal and environmental influences combine to influence the development (via multiple paths) of problem drinking. Neither do we know how those

who are at high risk of becoming involved in problem drinking (via various developmental pathways) could be influenced away from greater involvement in problem drinking. This knowledge would be of great interest and value to those who are attempting to implement effective primary or secondary prevention programs. Another way to state our lack of knowledge here that perhaps better conveys its applied significance is to say that we do not know whether different kinds of influences (e.g., various types of prevention programs) are differentially effective with different types of problem drinkers.[38]

Another question in this regard involves the issue of what becomes of various kinds of problem drinkers over time. For example, what types of adolescent problem drinkers "mature out" of their problematic involvement with alcohol, and what types go on to deeper and more chronic drinking in their adult years? And, what are the critical personal and environmental factors that mediate these later transitions? Of obvious importance to the applied field of tertiary prevention is the closely related question, "Are different treatment programs differentially effective with different types of problem drinkers (who, again, may have become problem drinkers via different developmental pathways)?"

Unfortunately, empirical research doing justice to questions such as those above encounters a number of interrelated conceptual and methodological problems—problems that precede empirical inquiry inasmuch as empirical work is necessarily based on assumptions about or solutions to them. The purpose of the following section is to identify some of these problems and to suggest promising features of future research in response to them. For the sake of clarity, these problems and features are illustrated below within two simple and hypothetical sets of data. I want to emphasize at the outset that these examples are for illustrative purposes only. Although the kinds of relationships portrayed in these hypothetical data sets are meant to be taken seriously as being the kinds of relationships and dynamics that cannot be ruled out in the real world, the substantive content portrayed in these hypothetical examples are most definitely fanciful ones that are not intended to be taken seriously.

4. A Hypothetical Example of a Cross-Sectional Study

That it is necessary to disregard the substantive content of these examples should become immediately and abundantly evident with the statement of premises for our hypothetical world. Let us assume that in this imaginary world: (1) a predisposition to problem drinking is genetically transmitted; (2) intellectual ability is also an inherited characteristic; (3) these two personal characteristics are immutable and fixed for life; and (4) they are perfectly correlated. Thus, our basic premises for this hypothetical world are that all intelligent people (and only intelligent people) are genetically predisposed to

alcoholism. Here, intelligence and predisposition to alcoholism are, in effect, one and the same variable inasmuch as to have one characteristic is to have the other, whereas not to have one is not to have the other.

Given these assumptions, assume that one is interested in understanding how individual differences in intellectual ability (and/or individual differences in genetic predisposition to alcoholism) interact with environmental press or demand for intellectual performance in their relationship to drinking behavior. Table I presents a set of hypothetical data on these variables for a representative sample of four persons from this imaginary world.

In examining Table I, readers should satisfy themselves that none of the three predictor variables alone, nor any linear combination of them, bears any relationship to the drinking variable. Despite this, the pattern of either one of the two (identical) personal variables together with the environmental variable enables a complete accounting of the variance in drinking events. If the personal variable and the environmental variable match, one level of drinking occurs. When they do not correspond, a different level of drinking occurs.

Using intellectual ability as the label for this personal variable for the moment, cases 1 and 4 in Table I could be viewed as "ecological matches" between the individuals' capacities for intellectual achievement (high and low) and environmental demand for intellectual performance (high and low). In both cases, the associated drinking events are of low magnitude (e.g., minimal or moderate drinking). In contrast, both cases 2 and 3 could be viewed as "ecological mismatches." Case 2 represents a person with low intellectual ability in an environment where there is a high level of press/demand for intellectual achievement. Case 3 is poorly suited to his/her situation in the opposite way, having a high level of intellectual capacity in an environment where there is a low level of demand for intellectual performance. In both case 2 and case 3, difficulties are plausible, case 2 being fraught with the potential for failure, feelings of frustration, etc., and case 3 being pregnant with possibilities for idleness, boredom, etc. In both cases, the associated

Table I. Hypothetical Data on Four Drinking Events to be Predicted from Individual and Environmental Data

	Predictor variables		
	Individual ability, intellectual ability	Environmental press, demand for intellectual performance	Criterion variable: drinking level
Case 1	2	2	1
Case 2	1	2	2
Case 3	2	1	2
Case 4	1	1	1

drinking behaviors are high-magnitude ones (e.g., alcoholic and/or problem drinking).

Of course, one could label the personal variable "genetic predisposition to alcoholism" instead of "genetically determined intellectual ability" (remember that they are the same variable here). Just as before, one could then account completely for the drinking criteria by using the pattern between this inherited personal variable and the level of environmental press/demand for intellectual performance. The point here is that whatever it is called and/or whatever it "is," this personal variable alone does not provide a means of understanding, predicting, or controlling the criterion behavior. Neither does the environmental variable alone or any linear combination of these variables. In contrast, a simple interactional rule involving the correspondence between the personal and environmental variables (i.e., a nonlinear combination of these two variables) allows a prediction of the drinking level of each and every case to be made. In addition, identifying this simple rule also leads to both plausible ways to understand the "why" of each case's drinking behavior and suggests tentative hypotheses about how to control the behavior in each case.

The point of this example is emphatically not that one or another label for this same genetically transmitted personal characteristic is preferable to the other. We can forget the labels for these variables altogether and simply call the personal variable *P* (it should be obvious that this variable may be genetically determined or not, so long as it is an enduring personal variable). We can go further and simply call the environmental factor *E* and the behavioral criterion *B*. We now have a general formulation within which the major point of this example may be framed. The larger point here is that in attempting to relate personal factors (*P*) and environmental factors (*E*) to behavior (*B*), one cannot rule out the possibility that the criterion behavior may be perfectly and reliably related in some way to a configural gestalt of personal and environmental variables, although at the same time, neither personal factors alone nor environmental factors alone, nor any linear combination of these factors may exhibit the slightest relationship to the criterion behavior.

5. Desirable Features of Research Suggested by This Example

5.1. Nonlinear Models of Scoring and/or Data Analysis

There is a formal identity between the hypothetical example shown in Table I and a seemingly paradoxical problem posed by Meehl more than 30 years ago.[40] Horst[41] and, in a more general way, Horn[42] have shown that the kind of quasiparadox represented in the data of Table I is really no paradox at all but that its solution does require a nonlinear mode of scoring or data analysis. Horn's formal analysis of this problem also reminds us that nonlinear combinations of variables contain at least as much information as linear ones

and potentially much more. This example implies that configural, typological, or other nonlinear modes of analysis may enable us to account for a substantially greater portion of the variance in drinking behavior than is the case within the more restrictive linear formulations now predominant in the field.

5.2. Representative Designs

This hypothetical (but theoretically extremely plausible) example also forcefully illustrates the necessity for truly representative sampling designs[43,44] reflecting the range of naturally occurring variation in both environmental and individual variables and their joint occurrences. In regard to this point, assume that the four cases in Table I were truly representative of individual–environmental variation in our hypothetical world but that sampling from only one kind of environment in Table I—say that characterized by high demand for intellectual performance—was done in a given empirical study. In this event, only cases 1 and 2 would be observed. From these (environmentally nonrepresentative) data, it is clear that one would be very likely to conclude that individual ability is negatively (and perfectly) related to level of drinking.

Note that the relationship observed here would be observed whether the nonrepresentative sampling of environments had been done by accident or design (the latter commonly referred to as "with the situation, stimulus, etc. 'held constant' ") and that the observed negative bivariate relationship is the exact opposite of the positive relationship that would be identified if the other kind of environment—that characterized by low demand for intellectual performance—were the only one sampled. Conversely, it should also be clear from inspection of Table I that nonrepresentative sampling of the other sort—in which individual differences were (in effect) "held constant"—would result in the observation of an equally fragmentary and contradictory apparent relationship between environmental press for intellectual performance and drinking (either a perfect negative or a perfect positive correlation).

5.3. Assessing Person–Environment Units of Analysis

Perhaps most fundamentally, Table I indicates that in designing empirical research for problems of this kind, an important consideration is choosing a basic unit of analysis. In Table I, neither the individual variable alone, the environmental variable alone, nor any linear combination of the two separate variables is related to drinking behavior. However, the pattern or relationship of one to the other is perfectly related to the drinking variable. To the extent that the hypothetical data illustrated in Table I are not implausible, this suggests the utility of some kind of person–environment unit of analysis. In this regard, there are an increasing number of provocative and persuasive arguments in the modern theoretical literature suggesting that one cannot mean-

ingfully separate (conceptually or analytically) the person from the environment in the interaction process and that therefore one's basic unit of analysis should be a person–environment unit.[44–46] In order to address questions like those posed above, this unit of analysis would ideally be a molar one suited to the task of reflecting whole persons and their environments. For examples of empirical studies of problem drinking that in effect use such units of analysis, see Braucht,[36] Donovan and Jessor,[4] and Cloninger *et al.*[35]

A very important related problem was not manifested in Table I, where both the individual variable and the environmental variable were conveniently represented on directly comparable metrics. In effect, both persons and environments were represented in Table I in a directly commensurate data language. It should be quite obvious that some way of directly relating persons to environments (or personal variables to environmental ones) will be a very desirable feature if not a requirement for research focusing on either linear or nonlinear combinations of personal and environmental factors in which the nature of the relationship between the two is the basic unit of analysis.

Of course, real world data is not likely to be quite so conveniently laid out. This promises to present quite a problem in measurement; it will not be possible to construct sensible comparisons of persons and environments based on separate normative measures in which measures of environments are given in units reflecting only the comparisons of environments to other environments and persons are represented in units that reflect only the standing of one person to other persons. This suggests that ways will have to be found to calibrate measures of theoretically related personal factors and environmental factors.

Sensible comparisons can sometimes be realized by resorting to ipsative types of measures (for example, see 37,48–50), but these have their own problems.[51] With difficulty, sensible comparisons can sometimes be achieved by transforming data from one universe of discourse into units that are then comparable to normative measures belonging to another universe of discourse. For an empirical example of this strategy in the field of suicidal behavior, see Braucht.[52] However, these strategies are neither universally applicable nor are without their own limitations. Thus, the pursuit of new ways of conceptualizing and realizing direct measurements of the interaction between persons and environments—a goal that Cattell[53] formally and rather wistfully contrasted with both normative and ipsative measurement—is of fundamental importance for the developing field of interactional and/or transactional research. For a recent analysis and proposed resolution of this problem, see Lamiell's paper proposing an "idiothetic" model.[54]

Finally, the example shown in Table I also suggests that routine or unthinking attempts to decompose this basic person–environment unit of analysis into separate additive linear components of variance are neither fruitful nor enlightening in understanding how individual differences and situations may interact in their relationship to behavior. Compelling arguments to this

effect have also been made in recent theoretical papers in personality research (e.g., see 55–57).

6. A Hypothetical Example of a Developmental Study

Although the first example (shown in Table I) conveys some of the conceptual and methodological problems involved in attempting to understand how personal and environmental factors relate to real-life behaviors, it does not illustrate all of them. In particular, because the example in Table I represents a static, cross-sectional "snapshot," it does not in any way illustrate the problems introduced by the variable of time. Because these problems are centrally involved in empirical work directed at assessing the process of development and change in problem drinking and/or alcoholism, a second hypothetical example, which does involve time, development, change (and ultimate stability), is presented in Tables II and III.

In Tables II and III, let us retain the same premises as were put forward above for the hypothetical example shown in Table I. In view of the earlier discussion in which it was shown that the main points to be gained do not depend on which label is used for this personal variable, I have developed an example using the construct of "inherited intellectual ability" for this variable and an intuitively related motivational variable. Readers may, of course, wish to develop a similar example using "genetic predisposition to alcoholism"

Table II. Initial State of Ten Hypothetical Cases of Drinking to be Predicted from Three Variables

	Initial levels of predictor variables			
	Individual intellectual ability (Variable 1)	Individual motivation to succeed (Variable 2)	Environmental press for intellectual performance (Variable 3)	Criterion variable: initial level of drinking behavior (Variable 4)
Case 1	2	2	2	1
Case 2	1	2	2	2
Case 3	2	2	1	2
Case 4	1	2	1	1
Case 5	1	2	1	1
Case 6	2	1	2	1
Case 7	2	1	2	1
Case 8	1	1	2	2
Case 9	2	1	1	2
Case 10	1	1	1	1

Table III. Final State of the Ten Hypothetical Drinking Cases

	Predictor variables			
	Final level individual intellectual ability (Variable 1)[a]	Final level individual motivation to succeed (Variable 2)[a]	Final level environmental press for intellectual performance (Variable 5)	Criterion variable: Final level of drinking behavior (Variable 6)
Case 1	2	2	3	2
Case 2	1	2	1	1
Case 3	2	2	1	2
Case 4	1	2	1	1
Case 5	1	2	1	1
Case 6	2	1	2	1
Case 7	2	1	2	1
Case 8	1	1	2	2
Case 9	2	1	2	1
Case 10	1	1	1	1

[a] Note: Neither individual variable undergoes any change in level during this simulation; thus, variable numbers are the same as they were in Table II for these two variables.

as a label for this inherited characteristic and their choice of a variable to replace the motivational variable.

Table II portrays the initial status of 10 hypothetical cases, each case having four attributes. The first, third, and fourth attributes represent the same three constructs as in Table I. In Table II, however, a second personal variable (a second personal attribute of each case) has been added: the personal level of motivation to succeed—in colloquial terms, the individual's "drive level," "desire," "indominability," or "Horatio Alger quality." Here at "time zero," even though the same *P–E* unit involving the match between (the initial values of) the person's level of intellectual ability and the environment's level of press/demand for intellectual performance relates perfectly to drinking behavior, none of the three predictor variables alone nor a linear combination of the three exhibit any relationship to the drinking variable.

A simulated dynamic process of development and change may be introduced by moving the initial state system in Table II through "time" according to the rules presented below. Once again, I want to emphasize that although I believe that we cannot rule out the kinds of relationships and dynamic(s) involved in this simulation, I would not want to be taken as implying that these particular simple simulation rules could reflect the actual process of development of drinking behaviors.

Simulation Rule 1: If the case is one in which the person has both a high intellectual ability and a high level of motivation to succeed, then the level of environmental press/demand for intellectual performance is increased by

1 unit for the next time period (until it reaches the maximum value of 3). Further, if the environmental press/demand variable has been at level 3 for three successive time periods, then, (1) if the case's initial environmental press level was 2, the case's environment remains at level 3 thereafter, but, (2) if the case's initial environmental press level was 1, then the case's environment drops back to that level for the next and all subsequent time periods.

Although this and the other governing rules are not put forward in an attempt to capture reality, they do correspond to an extremely plausible kind of dynamic. These persons who are both intelligent and motivated (and genetically predisposed to alcoholism) are initially distributed randomly with respect to level of environmental press/demand for intellectual performance. They seek out ever more challenging situations, until repeatedly frustrated by consistent overchallenge (accompanied by possible frustration and feelings of failure) for some significant stretch of time. They then finally react to this mismatch in different ways, depending on the nature of their early environments.

Simulation Rule 2: If the case is one in which the person has both a low level of intellectual ability and a low level of motivation to achieve success, then the level of environmental press/demand for intellectual performance for subsequent time periods remains unchanged.

This rule governing those who are neither intelligent nor motivated results in an inertia for these cases that is not implausible. Again, however, I do not intend the reader to take this simulation as any serious attempt to reflect reality.

Simulation Rule 3: If the case is one in which the two personal variables are not at the same level (either intellectual ability is high but motivational level is low or personal ability is low while motivation is high), then the level of environmental press/demand for the next time period is made more equal to the person's ability; it is brought more into line with the person's intellectual ability so that the difference for the next time period is half of what it is during the current time period.

Note that this rule entails "upward" shifts in environmental press/demand level for some cases, "downward" shifts for some, and no change in environmental press/demand level for still others. Here, a considerable degree of plausibility is maintained, as these cases neither overreach themselves (as do the intelligent and motivated people), nor do they fail to seek new environments (as do those who are neither intelligent nor motivated).

Applying these simulation rules (and retaining throughout our rule that drinking level is at all times and for all cases a simple function of the "fit" or match between the individual's level of intellectual ability and the environment's level of press/demand for intellectual performance), one finds that after a small number of simulated time periods, the status of each of these ten cases (on all variables) is asymptotically converging on a stable final state. The final state is portrayed in Table III, and Table IV presents the intercorrelations among all variables after stabilization.

Table IV. Intercorrelations of Initial and Final State Variables across the Ten Hypothetical Cases

Variables	V_1	V_2	V_3	V_4	V_5	V_6
Variable 1: Individual intellectual ability	1.0					
Variable 2: Individual motivation level	−0.20	1.0				
Variable 3: Initial level of environmental press for intellectual performance	0.20	−0.20	1.0			
Variable 4: Initial level of drinking behavior	0.00	0.00	0.00	1.0		
Variable 5: Final level of environmental press for intellectual performance	0.60	−0.30	0.60	−0.12	1.0	
Variable 6: Final level of drinking behavior	0.22	0.22	0.22	0.36	0.40	1.0

The multiple regression obtained by predicting the final level of drinking behavior from five variables (the other three final state variables and the initial levels of environmental press and drinking behavior) is 0.68 ($r^2 = 0.468$). The equation is as follows (variable numbers correspond to those in Table IV):

$$V_6 = -0.294 + (-0.088\ V_1) + (0.353\ V_2) + (-0.088\ V_3) + (0.412\ V_4) + (0.471\ V_5)$$

In this example, the multiple linear combination of all five available variables accounts for less than half of the variance in the final level of drinking variable, even though the final level of drinking behavior is perfectly related to the pattern or relationship existing between each individual's intellectual ability and (the final level in) his/her environment's press or demand for intellectual performance. In addition, the regression analysis fails to reveal anything of the principles that govern and direct the development of drinking behavior in this simulation. Neither does it reveal any understanding of how the developmental process unfolds; it affords no glimpse of how or why any of the cases change from one time period to the next. In fact, it actually provides some misleading results with regard to "what went on."

Specifically, these correlations and regression weights suggest only that the primary determinants of the final level of drinking are: (1) the initial level of drinking (". . . the best predictor of future behavior is past behavior . . .") and (2) the final state environmental press/demand for intellectual performance. Both of these variables do bear some linear relationship to the final level of drinking, and it is likely that they might be accepted as "the" determining relationships. All in all, the linear analyses of this simulation as a

whole look remarkably like a great many actual empirical results in the drinking field.

7. Additional Desirable Features Suggested by This Example

7.1. Provision for Potential Multiplicity of Developmental Pathways

This simulation involved cases that arrived at the same final status via different developmental pathways. The paths of development were truly different for different types of cases—not merely different in timing or in rate of progression, but fundamentally different in the sense of being different types of pathways going in different directions. It is important to notice here that not only did cases with the same final drinking status have different paths of development but also that cases with the same final status on all variables—personal, environmental, and behavioral—had different paths of development.

For example, cases 4 and 5 in the simulation above had identical "careers" in which neither case ever was in an environment for which he/she was ill suited. Neither case 4 nor case 5 had drinking problems at any time during his/her career. Case 2, however, began his/her simulated career as a problem drinker in an environment that demanded more in the way of intellectual performance than he/she could deliver. At the end of their careers, however, all three cases had identical status on all variables.

The "average" picture yielded by the usual linear combinations of variables gave no hint of this, however. In this regard, Block[37] has explicitly characterized the general field of developmental psychology as having clung too long to a key assumption:

> Across people, this presumption in its pure form asserts that all people develop in essentially the same way . . . [and] . . . across time, the hypothesis of uniformity suggests that relationships or qualities observed at one time [in life] may be expected to apply later as well [p. 10]. . . . The idea of different developmental paths—different in kind and direction rather than simply different in rate of traversal—is anathema to the nomothetic view that seeks universal laws applicable to one and all [p. 11].

This fourth point suggests that it will be desirable to develop ways to describe different types of problem drinkers not only in terms of their present status but also in developmental terms reflecting potential differences in "how they came to be that way." The importance of doing so lies in the realization that even though two individuals may look alike in terms of all available contemporary variables (both individual and environmental as well as behavioral), it is not implausible that the two may have arrived at this identical status via different developmental pathways and may thus be differentially responsive to various kinds of influences (either "naturally occurring" ones or planned interventions) toward or away from problem drinking.

7.2. Causal Models Capable of Reflecting Reciprocal Influences

Processes of development over time in which persons influenced environments that influenced persons (and so on) were modeled in the simulation above. To the extent that the kinds of developmental processes simulated above are not implausible, it may ultimately be desirable to develop conceptualizations and theories of problem drinking that do not neglect the possibility of multidirectional causation over time. Concepts such as "feedback," "cybernetics," "entropy," "self-organizing system," "open system," and others belonging to general systems theory[58,59] may prove useful in developing such models of problem drinking.

In the contemporary theoretical literature of personality research, "transactional" approaches have been distinguished from "interactional" ones by the former's emphasis on these sorts of models in which persons and environments are viewed as mutually and reciprocally influencing each other over time.[55,56,60,61] In transactional models, persons and environments truly lose their independent existence[57]; the meaning and force that either has for behavior is viewed as depending entirely on the nature of their relationship, the nature of their transactions. These models have much in common with the dialectical model of life-span development articulated by Klaus Riegel.[62,63]

8. Conclusion

We have seen that even the two extremely simple hypothetical data sets presented earlier involve a host of interrelated issues. In addition to the linear versus nonlinear/configural issue, there is also the problem of departures from representative design and what effect(s) these would have on the observed results. There is also the basic problem of how (or even whether) one can analytically separate persons from environments—this is the question of what the basic unit of analysis should be or can be in studying processes of development over time in which persons are affecting environments which are affecting persons (and so on). Thus, there is a whole congeries of conceptual and methodological issues involved here, running the gamut from measurement, statistical, experimental design, and sampling issues to paradigm issues close to the realm of philosophy of science.

It should be evident that these issues are germane to a very wide range of research areas in the alcohol research field, including (1) the area of research devoted to the study of how patterns of problem drinking develop and change over the life-span, (2) the emerging field of behavioral genetics as it is applied to the question of what roles are played by genetic and environmental factors in their influence on the development of problem drinking, and (3) the field devoted to developing and evaluating primary, secondary, and tertiary strategies for the prevention of problem drinking.

Although the issues articulated here were identified within hypothetical

sets of data, there should be no doubt that they are real issues that find expression in real life. A number of effects similar to those modeled in the present chapter's hypothetical examples have been observed in real-life data from the field of behavioral genetics (see Carter-Saltzman[64] for a thoughtful review of these effects). Of course, the recent empirical analyses of Cloninger and his colleagues provide a real-life example of similar phenomena in the field of alcohol research. All of these analyses indicate that, as opposed to the questions of *which* or *how much*, the question of *how* individual/genetic factors and environmental factors combine to influence the development of problem drinking is truly the real question of import to the alcohol research field.

To achieve more satisfying answers to this question, to move beyond our present knowledge of how individual and environmental factors combine to influence the development of problem drinking, I believe that we shall have to find ways of incorporating the desirable features of research that were identified above. Unless we can meet this challenge, we may find that we have reached a point at which gains in our knowledge will be derived with increasing difficulty and in increasingly smaller increments.

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5

Alcoholism
The Evolution of a Behavioral Perspective

William H. George and G. Alan Marlatt

Abstract. Behavioral approaches to understanding the etiology of alcoholism represent a divergence from traditional biomedical and psychodynamic models. The behavioral perspective subsumes a number of diverse conceptual models that share a common emphasis on the interplay between environmental forces and the response of the organism (S–R model). Yet these models—classical conditioning, drive reduction, and operant conditioning—offer different procedural paradigms and explanatory mechanisms. Through consistent application of the scientific method, certain behavioral principles from these models have survived empirical validation and continue to enrich our understanding of alcoholism. This chapter reviews these models and their attendant empirical findings, from both animal and human research, as they pertain to the etiology of alcoholism. The cognitive–behavioral approach, a recent product of this evolutionary development, focuses on cognitive processes that mediate stimulus–response relationships. The cognitive–behavioral model provides a comprehensive account of both the acquisition and maintenance of problem drinking and is discussed in some detail.

1. Introduction

A wide range of theoretical approaches has been advanced to explain the etiology of problem drinking and alcoholism. Historically, the more prominent views emphasized intraindividual determinants of alcohol abuse. One camp believed that alcoholism was an expression of a severe moral weakness. Advocates of this position sowed the seeds of the temperance movement and paved the way for moral and later legal proscriptions against alcohol use. Accordingly, jail confinement served as the major intervention strategy with public inebriates. However, the vigorous emergence of the disease model shifted the focus of attention from vice to sickness. The disease conceptualization effectively exonerated the alcoholic from immorality/criminality charges and legitimized the use of medical interventions. Alcoholics could now be seen as victims of illness rather than as perpetrators of evil. Consequently,

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today's public inebriates routinely find themselves under the supervision of medical personnel rather than local jailers, a highly beneficial change given the life-threatening severity of alcohol withdrawal symptoms. Having altered the perception and handling of alcohol problems, the disease model serves an important sociopolitical function. In many ways, it comes as no surprise that the medical profession has firmly embraced the disease model.

Nevertheless, an alternative model emerging from the scientific research literature has boldly questioned some basic assumptions of the medical/disease model. This scheme embodies an amalgam of conceptual models that together can be summarized under the rubric of behavioral approaches to alcoholism. Before detailing the central features of the behavioral approaches, a thumbnail sketch of the disease model is needed. Construction of a straw man is not our intent. Instead, we wish to furnish a useful theoretical backdrop. The development of the behavioral perspective on alcoholism cannot be fully understood without at least a brief description of the prevailing ideological context.

Jellinek[1] presented an alcoholism typology, specifying the gamma and delta syndromes as disease entities. The four major characteristics of the gamma syndrome, the most predominant type of alcoholism in North America, include (1) acquired tissue tolerance; (2) adaptive cell metabolism; (3) craving and withdrawal symptoms; and (4) loss of control of drinking. The latter feature distinguishes gamma from delta alcoholism. It has also become recognized by some as the central pathognomic sign of alcoholism. The loss-of-control notion states that the "ingestion of one alcoholic drink sets up a chain reaction so that they (alcoholics) are unable to adhere to their intention to have one or two drinks but continue to ingest more . . . contrary to volition" (p. 41). Presumably, the mere presence of alcohol in the bloodstream elicits continuous drinking that culminates in severe intoxication. These are the barest of essentials of the disease conceptualization pioneered by Jellinek.

Scores of writers have further elaborated, speculated, and pontificated on various details of the original model. Slowly, what was originally presented as a "working hypothesis" has become reified as basic truth. However, allies of the model do not always agree on the best presentation and interpretation of it. Despite interpretational nuances, statements of the model reliably express a set of fundamental implications:

1. Alcoholism is a unitary identifiable phenomenon.
2. Alcoholics and prealcoholics differ in important constitutional factors.
3. The alcoholic is a helpless victim of internal physiological mechanisms beyond his/her voluntary control.
4. Abstinence is the only acceptable goal of treatment intervention.

The pervasiveness of and allegiance to this belief system is considerable, as is the sheer power of its political support base. The "alcoholism is a disease" thesis has been adopted in both professional (e.g., A.M.A.) and lay (e.g.,

A.A.) circles. It has become the official stance of the medical profession. However, closer examination reveals a troublesome irony about all this; namely, that the medical profession's approach to treating alcoholism is surprisingly nonmedical. Missing is a focus on systematic physical/physiological interventions based on scientific research. Instead, medically based treatments of alcoholism tend to emphasize moralistic/philosophical arguments and descriptions of the medical complications that follow from continued alcohol abuse. The goal seems to be to instill in the alcoholic a supreme sense of willpower, motivated by fear, sufficient to induce an enduring commitment to abstinence.

Herein lies the "control paradox." That is, "loss of control" is the single most important symptom for diagnosing alcoholism; yet medical interventions attempt to browbeat the alcoholic into a self-controlled abstinence. They are trying to teach self-control to people who, by definition, are incapable of control. This double-bind message betrays a fundamental weakness in the disease model of alcoholism; there is no logical link between the proposed etiology and the treatment interventions. Without an empirically based explanation of etiology, such a tie is difficult to obtain.

There is no reliable empirical evidence to support the physiological pathogenetic process suggested by the model. Researchers have pursued a number of attractive hypotheses concerning allergic reactions, nutritional anomalies, metabolic anomalies, and biochemical lesions. No persuasive empirical trends have emerged from this body of literature. It has now been over 20 years since Jellinek's most influential statement on the disease model, and the search for the missing physiological link continues without success. Nevertheless, despite the absence of adequate conceptual linkage, advocates of the model continue to pair a decidedly medical/physical etiological doctrine with a nonmedical treatment regime. In short, there is no etiological justification for the mainstream treatment strategies promulgated by advocates of the medical/disease model.

In light of these shortcomings, the behavioral perspective presents an important alternative for understanding the etiology of alcoholism. With its emphasis placed squarely on external and internal determinants of behavior, this approach pursues two major questions. (1) How is problem (alcoholic) drinking acquired? (2) How is it maintained? This stance does not deny the importance of physiological processes. Clearly, there are physiological concomitants of excessive drinking that often necessitate medical attention. Individuals do, in fact, become physically dependent on alcohol. However, these properties do not qualify drinking itself as a disease. If they did, then cigarette smoking and coffee drinking would also qualify as diseases: nicotinism and caffeinism. The behavioral perspective views these physiological concomitants as effects, not causes, of "alcoholistic" drinking. For example, an abusive drinking pattern creates physical dependence, not vice versa. Covariation between these physical concomitants and abusive drinking does

not prove that alcoholism is caused by physical factors. Such logic would equally imply that lung cancer "causes" nicotinism (cigarette smoking).

The different approaches subsumed by the behavioral perspective share a common set of assumptions: (1) all patterns of alcohol use and abuse are learned; (2) alcohol consumption is more accurately described as a continuum rather than as a dichotomy (thus, alcoholics and social drinkers differ quantitatively rather than qualitatively); (3) the events that influence the acquisition of drinking behavior can be identified, measured, and altered; (4) alcohol abusers are not seen as helpless victims of biological processes beyond their voluntary control.

Behavioral approaches to understanding alcoholism reflect specific applications of learning principles embodied by more general behavioral models. History reveals that each general behavioral model emerged, flourished, and enjoyed an era of prominence before yielding to the development of a newer model. Wedded to the scientific method, learning theorists relied heavily on empirical research trends to validate the principles of a model, to demonstrate its limitations, and to usher in an improved model. Thus, learning theory has evolved through a number of stages: classical conditioning, drive-reduction theory, operant conditioning, social learning theory, and, more recently, cognitive–behavioral theory.

The overall purpose of this chapter is to trace the evolution of the behavioral perspective on the etiology of alcohol abuse. Each model and its relevant research findings are briefly reviewed. The presentation of the models is organized according to their treatment of nonobservable mediating constructions. Thus, the models have been divided into nonmediational and mediational approaches. A secondary objective is to review relevant animal paradigms of alcoholism. These paradigms are discussed in the context of the most relevant theoretical models. Finally, the cognitive–behavioral model is presented in some detail. The latter approach is more comprehensive because it embraces both behavioral principles and cognitive theory that have been substantiated by empirical validation.

1.1. Animal Models

A few words are needed to describe the role of animal research in our understanding of alcoholism. An abundant amount of alcohol research has relied on infrahuman subjects. The obvious advantage is that it allows implementation of a wide range of procedures that are ethically prohibited with human subjects, for example, surgical interventions, selective breeding, exposure to severe physical and social stressors, postmortem examinations, etc. Consequently, more control can be exercised over organisms' phylogenetic and ontogenetic histories, enabling the researcher to better isolate the variables of interest. Clearly, these considerations are especially accomodating for hypotheses regarding the physiology of alcohol metabolism and abuse.

Thus, it is not surprising that hypotheses derived from the medical/disease conceptualization have guided much of this research. Moreover, many of these endeavors have focused on rather specific features of the alcohol abuse syndrome, having enhanced our knowledge about physiological and biochemical correlates of alcohol use.

However, the ultimate utility of alcoholism research done with animals depends almost entirely on its generalizability to humans. Many human phenomena such as social behaviors (e.g., aggression and parenting) and regulatory behaviors (e.g., food and water) have naturally occurring analogues in the animal kingdom. Animal research in these areas is interesting in its own right as well as in its relevance to the human condition. This is not true with alcohol abuse. Voluntary alcohol consumption and abuse seems peculiar to humans. Therefore, the degree to which a particular line of animal research helps to unravel the mysteries of alcoholism depends greatly on how well the paradigm used models the human condition.

Addressing this concern, various writers[2-4] have specified criteria for an animal model of alcoholism. Summarizing across overlapping sets of criteria, an adequate model must demonstrate (1) voluntary oral ingestion of alcohol, (2) excessive chronic drinking producing substantial blood ethanol elevation, (3) an elective preference for alcohol over other fluid solutions, and (4) signs of physical dependence, i.e., withdrawal symptoms.

Extensive treatment of the animal literature exceeds the scope of this chapter. A comprehensive review of this literature has recently been compiled.[5] Thus, for the current purposes, we limit our discussion of animal research to those endeavors that have concentrated on the operation of learning factors in alcohol consumption, abuse, and related phenomena.

2. Nonmediational Approaches

Hallmarked by its avoidance of mediating unobservable variables, the nonmediational approach to explaining behavior focuses exclusively on the observable characteristics of behavior. The overarching explanatory scheme can be described as an input–output analysis in which environmental events channel into the organism and produce measurable behavioral events. Extreme statements of this paradigm depict an empty organism devoid of independent causation and puppeted by the environment. Thoughts and feelings have no place in this analysis, as they are superfluous for exacting a parsimonious and scientific explanation. A number of researchers and theoreticians have employed these approaches in efforts to understand alcoholism or problem drinking. These endeavors have generated detailed descriptions of the relationship between alcohol consumption and environmental events. They have also been useful in explaining other symptoms of alcohol abuse, particularly tolerance and craving.

2.1. Classical Conditioning Models

Pavlov's classical work at the turn of the century fashioned an explanatory model of behavior that has stood the test of time. Identifying the unlearned reflex as the fundamental unit of behavior, the model capitalizes on the uniformity with which certain events (unconditioned stimuli) elicit these reflexes (unconditioned responses). Briefly, the model postulates that regular contiguous pairing of a neutral (conditioned) stimulus with an unconditioned stimulus enables the conditioned stimulus to elicit an anticipatory (conditioned) response when presented alone. Presumably, the organism has learned an association between the conditioned stimulus (CS) and the unconditioned stimulus (UCS). Moreover, this association is both generalizable and discriminative. It is generalizable in that stimuli similar to the original CS^+ will elicit approximations of the conditioned response (CR) and discriminative in that other stimuli (CS^-) will not. The validity of this general learning model is without question. However, its utility for understanding the etiology of alcoholism is somewhat limited. Clearly, alcohol consumption is not a reflexive unlearned response and thus taxes the explanatory breadth of the model. Not surprisingly, there has been little systematic work toward developing a comprehensive classical conditioning model of alcoholism etiology. But the model has been used to explore tolerance, craving phenomena, and relapse.

2.1.1. A Classical Conditioning Model of Tolerance. Jellinek[1] specified tolerance as a component of the disease syndrome of alcoholism. An individual develops tolerance "as a result of repeated exposure to the drug, so that an increased amount of drug is required to produce the same specified degree of effect, or less effect is produced by the same dose of the drug" (p. 137). Experts distinguish between dispositional and functional tolerance. Dispositional tolerance refers to processes, e.g., metabolic, that reduce the amount of a drug substance that reaches the target tissue. Tolerance not resulting from a diminution in drug level at the receptor site is described as functional. For the present purpose, our use of the term tolerance will be restricted to functional tolerance.

There is no consensus regarding the mechanisms underlying tolerance, but belief in a physiological substrate has been the status quo. According to this position, receptor site tissues habituate to the frequent presence of the drug, thereby becoming less sensitive to its effects. However, recent research by Siegel[6–10] seriously challenges the validity of this explanation and demonstrates the utility of classical conditioning principles in explaining tolerance phenomena.

Siegel's basic paradigm entails presentation of a drug (UCS) in a distinctive situational context (CS). As expected, with repeated exposures to the drug in that context, the animal exhibits tolerance; i.e., the size of the drug effect (UCR) diminishes. If, at this time, the drug is presented in an altogether different situational context (CS^-) which has not been previously paired with

drug administration, then no tolerance is evident; i.e., the size of the drug effect is fully restored. Furthermore, unreinforced presentations of CS, in which the UCS is omitted, can be accomplished by injecting the animal with a placebo substance (e.g., saline) in the presence of the drug cues. This procedure elicits a distinct physiological reaction (CR) that is opposite in direction to the actual drug effect (UCR). It is argued that this CR serves to oppose or compensate for the actual effect of the drug. Thus, observed tolerance can be construed as a simple algebraic subtraction whereby the conditioned compensatory response counteracts actual drug effect. The suggestion here is that the organism learns to use available situational cues to predict the onset of a drug and reacts to that prediction by generating an anticipatory counterresponse that dampens the drug's action. In a series of well-controlled experiments with rats, Siegel[10] has demonstrated the validity of this paradigm in accounting for the tolerance shown by rats to the analgesic and hyperthermic effects of morphine as well as the hypoglycemic effects of insulin.

A recent investigation by Mansfield and Cunningham[11] extends this associative model of drug tolerance to ethanol. In rats, ethanol produces a reduction in body temperature. This hypothermic effect provides a convenient index for assessing tolerance. These investigators alternated injections of ethanol with injections of saline for three groups of rats. The ethanol injections were consistently paired with one set of distinctive environmental cues, and the saline injections were paired with another set of cues. The three groups differed in the amount of handling and motor activity experienced during the injection procedures. A fourth group received injections of saline in both environments. Following this tolerance acquisition phase, all subjects received ethanol injections in order to test for tolerance under both drug cue (CS^+) and saline cue (CS^-) conditions. The data clearly revealed tolerance, i.e., diminished hypothermia in the presence of drug cues. However, these same rats exhibited no tolerance for ethanol when tested under the saline cues. In fact, their hypothermic reactions under the saline cues were indistinguishable from those exhibited by the control animals who had not received ethanol during the acquisition phase.

To test for the presence of a conditioned response, placebo (saline) injections were administered under either the drug cues or the saline cues. Consistent with Siegel's model, animals exposed to drug cues during their placebo injections exhibited a conditioned hyperthermic reaction. That is, body temperature increased in apparent anticipation of the ethanol-induced hypothermia. Moreover, continued presentation of the unreinforced CSs (i.e., placebo injections) led to extinction of the conditioned hyperthermia and loss of tolerance. These findings clearly demonstrate that tolerance to ethanol effects is mediated by a compensatory reaction that is classically conditionable to environmental cues that regularly accompany ethanol administration.

From this animal research, it appears that associative processes are extremely important to the development of ethanol tolerance. Independent work

on an instrumental model of tolerance also supports this contention, as we shall show momentarily. Tolerance has long been recognized as a sound indicator of a physiologically based disease process. The importance of learning principles may be fundamental to understanding the etiology of alcohol abuse.

However, such speculations are premature in the absence of comparable human research. To date, this classical conditioning model of tolerance has not yet been employed with human subjects. Nevertheless, the model has applicability to some of the empirical trends that have emerged from Marlatt's work with the balanced placebo design. The details of this experimental design are more fully described in a later section of this chapter. For our current purposes, it is useful to note that the design involves delivery of a placebo drink under conditions intended to resemble naturalistic drink administration procedures. With certain types of behavior, these placebo drinks are as effective as actual alcoholic drinks in determining subsequent performance.

Marlatt and Rohsenow[12] proffered a classical conditioning interpretation in partial explanation of this robust placebo effect. Presumably, the experienced drinker has undergone repeated episodes where situational drinking cues (CSs) have been paired with the introduction of alcohol (UCS) into the bloodstream. Therefore, a properly administered placebo drink functions as an unreinforced CS that elicits a CR. In light of the research on the classical conditioning model of tolerance, this CR would be stimulatory in nature to compensate for the direct depressive effects of alcohol. This anticipatory CR can be readily interpreted by the drinker as the initial kick or high that routinely follows a first drink. Thus, subsequent behaviors would be influenced by the "knowledge" that one has consumed alcohol.

2.1.2. Craving. Ludwig and Wikler[13] proposed a classical conditioning model of craving, relapse, and loss of control drinking. The model postulates that exteroceptive and interoceptive events that are temporarily contiguous to the occurrence of alcohol withdrawal symptoms (UCR) become conditioned stimuli. Subsequent exposure to these stimuli elicits a "subclinical conditioned withdrawal syndrome." According to this analysis, craving is the "cognitive correlate" of this "miniwithdrawal." Ludwig and Wikler expanded the realm of possible eliciting stimuli to include cues previously associated with heavy drinking episodes as well as those associated with withdrawal experiences. The rationale for this overextension is not made entirely clear and at best represents an overly generous interpretation of the stimulus generalization process.

To summarize the first stanza of their model, presentation of any exteroceptive (e.g., physical setting or drinking companions) or interoceptive (e.g., certain emotional states) stimuli associated with either heavy drinking or withdrawal elicits craving, thereby predisposing the individual to take a drink. Here the model asserts that consumption of the first drink ("appetizer") intensifies the craving ("hunger") experience and precipitates loss of control

drinking ("entree"). Also, the model radically departs from a strict classical conditioning paradigm by evoking cognitive modifiers. The authors suggest that, once elicited, the "subclinical conditioned withdrawal syndrome" is not invariably interpreted as craving by the alcoholic. Supposedly, that interpretation depends on such influences as situational factors, the availability of alcohol, and the alcoholic's ability to correctly label the arousal state. So, the occurrence of craving depends on two processes: the presence of conditioned stimuli that elicit the internal state and the convergence of certain situational factors that determine how the internal state is labeled. If these considerations are active and in synchrony, the alcoholic takes that first drink, craving increases, and he launches into loss-of-control drinking, defined by these authors as a "relative inability to regulate ethanol consumption" (p. 122).

In an empirical test of this model, Ludwig *et al.*[14] permitted 24 detoxified alcoholics to consume a high-dose, low-dose, or placebo drink under one of two sets of labeling conditions. In the label condition, subjects consumed their preferred alcoholic drink in the presence of alcohol-related cues. In the nonlabel condition, subjects consumed a drink containing ethyl alcohol and an artificially sweetened mixer in the absence of alcohol cues. The authors hypothesized that subjects would exhibit more craving and more alcohol acquisition behavior after receiving a low dose than a high dose of alcohol because the high dose would "satisfy" craving, whereas the low dose would function as an "appetizer," thereby enhancing craving. They also predicted that craving and alcohol acquisition behavior would be greater under the label versus nonlabel condition. Craving was measured through subjective reports registered on a "craving meter," a device that allowed the individual to rate his degree of craving on a scale of 0 to 100. To measure alcohol acquisition behavior (presumably an indicator of loss of control), subjects were permitted to work for alcohol on a button-pressing device (although the alcohol could not be consumed until the end of the experimental session).

Methodological and data analysis problems prevent a clear understanding of the results. However, the findings reported did not support the primary hypothesis. Under the label condition, the low-dose group did not differ significantly from high-dose subjects on the craving measure at each of the multiple assessment points. But a main effect for the labeling variable was indicated, with subjects reporting significantly more craving under the label condition. This finding would seem to support the importance of cognitive expectancy factors. That is, perhaps these subjects drank more not because of any actual craving but because they expected to experience strong craving in a situation purposefully saturated with alcohol cues. The findings for the alcohol acquisition measure paralleled those for the craving measure. The investigators' experimental findings failed to substantiate their theoretical propositions. Marlatt[15] has presented a detailed critique of this research and its implications.

With respect to alcoholism, the classical conditioning paradigm is best

known for its treatment applications—aversive conditioning procedures. This treatment approach has been extensively reviewed elsewhere[16–19] and in later chapters of this current volume. Although different learning paradigms (e.g., punishment, escape) dictate different procedural arrangements and explanatory analyses, the seminal work with aversive conditioning was based on classical conditioning principles.[20] Sufficient pairing of alcohol and related stimuli with a noxious stimulus (typically emetics or faradic shock) would, presumably, lead to an aversion for alcohol severe enough to promote abstinence. Empirical support for the efficacy of aversive conditioning has been equivocal; a well-controlled treatment outcome study with emetic conditioning has yet to appear in the literature. Moreover, the limited success that has emerged does not support a classical conditioning interpretation. In fact, W. Miller[18] suggests that cognitive processes may be responsible for the success reported with electrical aversion therapy.

2.2. Operant Conditioning

Like classical conditioning, the operant approach has endured and proliferated. The operant uprising that began in Skinner's laboratory in the 1930s extended learning theory beyond the realm of unlearned reflexes. Instead, the emphasis was placed on freely emitted responses and their consequences. Consequences that altered the future occurrence of those responses were identified as reinforcers. By definition, positive reinforcers increase and negative reinforcers decrease the probability that the preceding behaviors will reoccur in the future. Supposedly, the organism learns that procurement of certain consequences under certain stimulus conditions is contingent on the emission of certain responses. Thus, the response (R)–reinforcer (S*) contingency is further tied to the antecedent stimulus conditions (S) that prevailed when it was established. This S–R–S* association is generalizable and discriminable across both stimulus and response gradients.

The operant model can be used to explain the acquisition, maintenance, and termination of any behavior, including problem drinking. According to the model, the final form of the behavior, in this case excessive drinking, develops or is "shaped" through selective reinforcement of incipient components of the behavior. Once the behavior becomes established, intermittent occurrence of reinforcement enhances the resiliency of the behavior. Termination of behavior requires the removal of positive reinforcers (i.e., extinction) and/or the application of punishment. An enormous corpus of experimental data has accrued validating the predictive validity of these basic operant principles.

2.2.1. Animal Research with Operant Models. The operant approach to alcoholism generated a great deal of animal research. As we shall see, some of it has been geared toward finding a comprehensive animal model of problem drinking. Nonetheless, other work, as in the classical conditioning approach, has sought to unravel the tolerance phenomenon.

2.2.2. An Instrumental Model of Tolerance. Well-controlled experiments by Siegel and others, described earlier, seriously challenged the widespread and traditional belief that tolerance to ethanol and other substances is mediated entirely by changes in physiological sensitivity. An independent line of research employing an instrumental paradigm offers an equally compelling refutation of the long-standing physiological view. (The instrumental paradigm is an operant procedure in which the target response can only occur during single discrete trials delimited by the experimenter.)

Chen[21] operationalized tolerance as the reduction in errors made during performance in a maze under the influence of a drug. Two groups of rats were given three equivalent dosages of ethanol and three trials at the maze task before being tested for tolerance. The groups differed in that ethanol injections were administered either before or after each of the practice trials. Thus, one group performed while intoxicated, and the other group performed while sober. In the subsequent tolerance test, subjects in both groups performed in the maze following ethanol administration. Since both groups underwent equal ethanol exposure, the physiological view would predict that they would be equally tolerant. However, the findings revealed that tolerance was evident only in the group that had previously performed the maze while intoxicated. This result suggested that ethanol tolerance, at least as measured in this experiment, is mediated by a learned adaptation to ethanol's disruptive impact.

LeBlanc *et al.*[22,23] criticized Chens methodology and interpretation. These investigators argued that behavioral practice did not fully account for the observed tolerance but only accelerated the rate at which a fundamentally physiological tolerance developed, hence the term "behaviorally augmented" tolerance. According to this position, rats in Chen's two groups would have reached the same asymptotic levels of tolerance expression if the procedure had been extended over a longer period of time. They tested this prediction using a different experimental procedure. Rats were trained to walk on a treadmill and avoid error-contingent electrical shock. Intoxicating injections of ethanol were administered either before or after practice trials on the treadmill. Daily practice trials were given over an extended period of time with regularly interspersed tolerance tests. As predicted, the group that received intoxicated practice initially demonstrated more tolerance, but eventually the postpractice intoxication group reached an equal tolerance asymptote.

Wenger[24,25] used the same paradigm to further examine the importance of intoxicated practice in the acquisition of tolerance. His initial study demonstrated that the finding of LeBlanc *et al.* was caused by a procedural artifact and suggested that ethanol tolerance could be entirely attributed to the effects of intoxicated practice. Presumably, tolerance is reduceable to a skill acquisition process whereby the organism learns how to behaviorally compensate for ethanol effects. In a series of impressive experiments, Wenger[25] showed that tolerance to pentobarbital and diazepam as well as cross tolerance between these substances and ethanol can be similarly explained. For example,

rats that have received practice while under ethanol intoxication exhibit tolerance when tested under the influence of pentobarbital or diazepam, and vice versa.

To further support a learning interpretation of tolerance, Wenger demonstrated that the development of drug tolerance does not even require prior drug exposure. Using the same paradigm, a mechanical spin treatment that produced ataxia was substituted for drug injections. In two otherwise identical experiments, the animals were then tolerance tested under the influence of either diazepam or ethanol. In both experiments, rats for whom treadmill practice trials were regularly preceded by the spin treatment evidenced tolerance to diazepam and to ethanol. There was no tolerance evidenced for rats who received the spin treatment after their practice sessions. These quite remarkable findings provide a clear indication that the observed tolerance is merely the by-product of the organism learning to adapt its performance to a state of impairment. This experimentation presents an interesting implication for controlled-drinking treatment programs. That is, certain drink-refusal skills may be most effective (in terms of preventing drinking to intoxication) when taught under low-dose intoxication levels.

Summarizing across classical and operant trends, it seems that both physiological and behavioral indices of tolerance can be accounted for by learning principles.

2.2.3. Schedule-Induced Polydipsia. This peculiar anomaly of reinforcement scheduling has been used to construct an animal analogue for alcoholism. Falk and his associates[26,27] described an experimental procedure that reliably generates excessive drinking in rats. The phenomenon was originally observed in 1961. Subsequent refinement has established that delivery of one food pellet every 2 min (fixed-interval 2-min schedule) causes rats to more than triple their fluid consumption. In this arrangement, food delivery is not contingent on drinking behavior. This particular scheduling procedure or some approximation of it is also capable of increasing aggressiveness, pica, chronic hypertension, wheel running, and air licking. Because this "generator schedule" also proved effective in producing chronic excessive ethanol consumption, it became the centerpiece of a general animal model of alcoholism. In an elaborate succession of experiments, Falk and his associates show that rats subjected to the procedure voluntarily ingest (orally) sufficient fluid to produce chronic blood ethanol elevation and physical dependence. They have also shown that the ethanol solution is preferred over water and weak (3%) dextrose solutions. In response to criticism, subsequent experimentation suggests that the phenomenon cannot be explained on the basis of caloric deficit or water–electrolyte imbalance. In sum, the paradigm seems to fit most but not all of the previously outlined criteria for an animal model of alcoholism.

Nevertheless, a number of drawbacks persist and have discouraged wider acceptance of the model. One objection is that the behavioral excess is not specific to ethanol. Ethanol consumption is but one in a class of "adjunctive behaviors" susceptible to schedule-induced overindulgence. Falk and his as-

sociates prefer to construe the specificity problem favorably since it dovetails nicely with the more general notion that all excessive indulgences share common etiologic forces. Polemic posturing aside, the model fails to reconcile the reality that in human alcoholism, the acute and chronic effects of ethanol ingestion are integral and specific to the development of the overindulgence pattern. Cicero[2] raises the objection that schedule-induced ethanol overindulgence is not purely voluntary as claimed, because the excessiveness is mysteriously coerced by procedural forces and, seemingly, not mobilized by inherent properties of ethanol. A third and more fundamental drawback is that there is no understanding of why the procedure works. This explanatory gap limits the model's usefulness in forging a theoretical account of alcoholism's etiology. Furthermore, the model's applicability to humans is questionable, since it rests on the precarious suggestion that human overindulgences result from reinforcement histories characterized by intermittent deliveries of sparse reinforcers. There are considerable difficulties in attempting to convert this proposition into a feasible validational study or a useful treatment intervention.

2.2.4. Concurrent Scheduling Analysis. A recent report by Samson *et al.*[28] describes another operant analysis of ethanol consumption in rats. In their procedure, rats are trained to bar press for water and later, for ethanol. Subsequently, they are confronted with simultaneous presentation of two operant manipulanda: one produces water, the other ethanol. Employment of concurrent fixed ratio schedules revealed a distinct preference for ethanol. That is, they responded more for ethanol than for water. Next, using the same concurrent schedules, ethanol was pitted against increasing concentrations of a dextrose solution. This resulted in increased bar pressing for dextrose and decreased responding for ethanol, reflecting an absolute decline in ethanol's reinforcement value (equals the number of responses for ethanol divided by the number of total responses). The authors interpreted this result as supportive of other findings indicating that in humans ethanol intake decreases "when other reinforcers of greater value to the individual are concurrently available."

In a companion experiment, the ethanol and the highly preferred 5% dextrose solution were administered under concurrent FR8 schedules. Then the response requirement for the dextrose was increased to FR64 and later returned to FR8. The response requirement for ethanol remained unchanged at FR8. The findings demonstrated that the reinforcement value of ethanol increased tremendously when the response cost of the highly desired dextrose solution was increased. This finding suggests that the reinforcement value of ethanol is not only influenced by the values of other reinforcers but also by the "costs" of other reinforcers. That is, an individual may select to overrely on alcohol even though more valuable reinforcers are available (e.g., money or social praise) because he or she may perceive these other reinforcers as too costly in terms of the work expenditure (e.g., social skills) required to obtain them. At the human level, this analysis must entail mediational mech-

anisms, since perceptions of reinforcement values and costs require subjective estimations. Indeed, these findings and the accompanying interpretation are not incompatible with a cognitive social learning analysis of alcoholism.

2.2.5. Human Research with Operant Models. Operant work with alcoholics was pioneered by Mello and Mendelson (review by Mello[29]). They viewed the alcoholic's consummatory response primarily as the reinforcer of alcohol acquisition behavior. In order to quantify subjects' responses, various operant manipulanda were constructed, requiring subjects to "work" for reinforcers. An elaborate series of studies was performed using different reinforcement schedules and comparing the reinforcement value of alcohol with other potential reinforcers, e.g., money and cigarettes. These researchers have demonstrated that although alcohol is a potent reinforcer for alcoholics, the amount consumed varies with the amount of work required to obtain it. In addition, the efforts of Mello and Mendelson generated important findings about patterns of consumption, abstinence, and attendant biological concomitants.

Many of the other human studies have instead viewed alcohol consumption itself as the operant response. These investigations focused on manipulation of antecedent and consequent events in an effort to systematically influence consumption rates.

2.2.6. Manipulation of Antecedent Events. P. Miller[17] suggested that antecedents play a very important role in problem drinking. By virtue of their frequent association with reinforcements derived from excessive drinking episodes, these antecedent or "setting events" acquire secondary reinforcement properties. As a result, subsequent exposure to such cues as drinking buddies, barroom paraphernalia, or even discrete emotional states can potentiate excessive drinking. An experiment reported by P. Miller *et al.*[91] indicated that alcoholics and social drinkers differ in the extent to which situational cues control their drinking behavior. Alcoholics and social drinkers matched on age and education performed an operant lever-pressing task to obtain alcohol under one of two cue conditions. Alcohol-related visual stimuli were either prominently displayed on the response console or absent in the "cue-salience" and "no-cue" conditions, respectively. The results showed that social drinkers emitted a significantly higher number of responses for alcohol under the "cue-salience" than the "no-cue" condition. In contrast, alcoholics failed to discriminate, responding equally under both sets of conditions. Evidently, the social drinkers appropriately regulated their behavior in response to the prevailing stimulus conditions. Their behavior was situation specific. Alcoholics, on the other hand, exhibited a wider range of stimulus generalization. Their responding was less susceptible to the control of situational variables.

2.2.7. Manipulation of Consequences. As the centerpiece of operant paradigms, the response-contingent consequence is viewed as the primary regulator of alcohol consumption. The prototypic investigation in this area systematically applies certain consequences and observes the subsequent impact on alcohol intake. The treatment implications are obvious and direct. In

fact, most of these studies use alcoholic subjects and classify as treatment outcome investigations. A variety of positive (e.g., money, redeemable tokens, social interaction) and negative (e.g., electric shock, social isolation) reinforcers have been employed within a number of different operant paradigms (e.g., punishment, escape, token economies, contingency contracting) to modify drinking behavior. Extensive reviews of these endeavors are available elsewhere[17,18,30] as well as in the current volume.

An emerging consensus is that operant-based manipulations of response consequences demonstrate only limited effectiveness. More specifically, these approaches modify drinking behavior only as long as the contingencies are strictly enforced, as in a rigidly controlled artificial setting. Under the more relaxed contingencies that prevail in the naturalistic environment, these approaches have considerably less efficacy. So, despite their demonstrated success during the active treatment procedures, the effectiveness of operant approaches generally fails to transfer to the posttreatment environment. Hunt and Azrin[31] demonstrate impressively that operant principles are effective in the natural environment if all relevant contingencies are comprehensively assessed, conscientiously imposed, and carefully controlled. However, this degree of environmental regimentation cannot be realistically deployed and maintained. Yet, without it, it appears that operant approaches will not provide the long-lasting treatment effects that were originally hoped for.

In summary, the operant approach has made important contributions to the search for the etiologic origins of alcoholism. Methodologically, it preserved the emphasis on the scientific process as a means of generating, testing, and revising promising hypotheses about alcohol consumption. Conceptually, the operant approach addresses both antecedent and consequent influences on drinking and provides a framework for their integration. In contrast, earlier behavioral approaches, classical conditioning and drive reduction (discussed below), focused almost exclusively on one class of influences. With its dual emphasis, the operant approach produced cogent functional analyses for both the acquisition and maintenance of alcohol abuse. However, treatment procedures based on strict operant analyses have effected variable success and little posttreatment transfer. Thus, it appears that the operant approach and nonmediational approaches, in general, cannot adequately account for the complexities of alcohol abuse.

Efforts to improve on the explanatory power of nonmediational analyses and the therapeutic potency of derivative treatment procedures inevitably evoke mediational constructs. This is apparent in the trend toward "broad-spectrum" behavioral treatments.[30] It is also evident in the continually evolving work of learning theorists, work that is based primarily on animal research. Over the last 15 years, a number of developments have emerged that seriously question the empirical origins and fundamental principles of operant conditioning. In an excellent review of these developments, Bolles[32] disputes the operant contention that organisms learn responses *per se*. He postulates that learning involves the acquisition and synthesis of two types of expectancies:

(1) S–S* expectancy ("primary law of learning"), that certain events predict certain outcomes; and (2) R–S* expectancy ("secondary law of learning"), that certain behaviors predict certain outcomes. As Bolles illustrates, this decidedly mediational stance reconciles a number of experimental anomalies and offers a more compelling system for explaining behavior.

3. Mediational Approaches

"A mediating variable is an inferred factor which relates stimulus input to response output"[33] (p. 19). Unlike nonmediational approaches, theoretical paradigms that utilize mediating variables invest explanatory power in unobservable covert influences. Thus, the veridicality of these paradigms depends on evaluation of indirect indicators of the covert influences proposed. Different paradigms suggest different covert influences and attend to different indirect indicators. The paradigms also differ in regard to the degree of specificity and precision with which the indicators are described and measured. However, regardless of the particulars of the mediational variables used, these approaches are ultimately judged by the same criteria as the nonmediational approaches: predictive accuracy and explanatory force. Evolutionarily, the two mediational approaches presented here represent two distinct eras in psychology. With regard to prominence in the research and conceptual literature, a faithful chronology would show that the drive-reduction approach preceded, whereas the cognitive–behavioral approach followed the operant approach. However, they have been arbitrarily juxtaposed here for the sake of conceptual organization.

3.1. Drive Reduction: The Tension-Reduction Hypothesis

Like other behavioral approaches to understanding alcoholism, the tension-reduction hypothesis was clearly a product of the times. In the early 1940s, Hull presented an elaborate and detailed theory of learning that proved to be remarkably influential. Essentially, Hullian learning theory postulated that the presence of a heightened drive state energizes organismic behavior and that a reduction in the drive state reinforces the behavior. Applying this logic to alcohol-related behavior led to the formulation of the tension-reduction hypothesis (TRH). According to the TRH, alcohol is reinforcing because it reduces tension, and individuals consume alcohol to obtain this reduction in tension.

The TRH, readily accepted within the prevailing Hullian framework, generated a great deal of experimental research with both animals and humans. Seminal animal work by Masserman and Yum[34] and later Conger[35] demonstrated that in cats and rats alcohol facilitated resolution of an approach–avoidance conflict by presumably suppressing the fear-motivated avoidance. However, subsequent studies have failed to provide consistent

support for the TRH. In an extensive and critical review of this work, Cappell and Herman[36] conclude that "negative, equivocal, and contradictory results are quite common if not preponderant" (p. 59). Studies using human subjects (recently reviewed[37,38]) have produced results that are even more confusing and discouraging. In fact, some of these investigations[39–41] reported that alcoholic subjects experienced an increase in subjective tension and anxiety following ingestion of alcohol. Yet the conventional wisdom, as well as some experimental findings,[42] indicate that a sense of relaxation does initially follow alcohol intake.

This discrepancy, though perplexing, is not irreconcilable when the dose–response features embedded in the TRH are considered. The TRH assumes a linear dose–response function in which an increasing alcohol dosage leads to increasing reductions in tension. However, experimental investigations into the specifics of the dose–response relationship suggest a different picture. Doctor *et al.*[43] and others report that alcohol produces a biphasic response pattern. The first phase, initiated by introducing a low dose of alcohol into the bloodstream, is characterized by an excitatory effect that is subjectively experienced by the drinker as a euphoric high. With continued consumption, this phase gives way to a suppressive effect accompanied by subjective dysphoria, tension, and depression. This biphasic pattern has profound implications for understanding drinking behavior, since it describes two distinct effects that differ both in terms of valence and temporal contiguity. The low-dose euphoria effect is experienced as positive and immediately follows the initiation of the drinking episode. Hence, it has a much more potent associative tie to drinking behavior than the delayed negative effects that accompany the high-dose phase. Continued drinking and subsequent arrival at the high-dose experience may be, in part, motivated by a desire to recapture the euphoria effects. In the problem drinker, the association between alcohol and its negative effects can be further weakened by alcohol-induced memory impairment. These considerations suggest that the expected rather than the actual tension-reducing properties of alcohol are most influential in governing human alcohol consumption. That is, perhaps people drink alcohol because they think of it as a relaxing agent when, in fact, any relaxation experienced subsequent to ingestion is a short-lived harbinger of a more enduring state of dysphoria.

The assertion that people drink more when tense represents a corollary of the TRH. In research designed to test this assertion, Higgins and Marlatt[44,45] indicated that expectations do mediate the relationship between tension states and alcohol consumption. In their experiments, an ostensive taste-rating task[46] was used to unobtrusively assess the amount of alcoholic beverages consumed by male subjects. In the first experiment, subjects were threatened with high or low levels of electric shock to create high and low tension conditions. The results failed to substantiate the prediction that high-threat subjects would drink significantly more than low-threat subjects. In the follow-up experiment, tension was induced by threat of social evaluation. High-threat subjects

were told that their behavior in the taste-rating task and a subsequent social interaction with female judges would be critically evaluated by the same group of females. Control subjects were not led to anticipate social evaluation. The results proved the social evaluative threat to be more influential than threat of electric shock used in the previous study. In this study, subjects who anticipated social evaluation drank significantly more alcohol than control subjects.

Combined, the results of these two experiments suggested that increased tension leads to higher alcohol consumption only when the source of tension is meaningfully related to expected alcohol effects. More specifically, individuals have little reason to expect that alcohol intake will moderate an impending adversity as impersonal as electrical shock; however, they are very likely to expect that a few drinks will soften the impact of an impending social evaluation. The prevailing cultural norms, which depict alcohol as a "social lubricant," clearly instill a stronger belief in the latter expectancy than in the former.

Despite its mediational properties, the TRH, as originally formulated, does not adequately explain alcohol consumption. Its fundamental proposition that alcohol directly reduces tension has proven untenable. The research literature provides neither supportive nor reliable empirical trends. The simplistic "alcohol in—tension down" relationship posited by the TRH appears to be a much more complicated picture, clouded by difficult conceptual (e.g., defining tension) and methodological (e.g., measuring tension) problems. The companion notion that people drink alcohol to reduce tension is also without clear support. However, when biphasic dose–response considerations and cognitive expectations are also taken into account, the TRH seems somewhat salvageable. Nevertheless, it seems that without resort to cognitive factors, the TRH, like the nonmediational approaches, cannot provide a sufficient account of problem drinking, its etiology, and its maintenance.

3.2. The Cognitive–Behavioral Approach

In contrast to its predecessors, the cognitive–behavioral approach did not emerge as a uniform conceptual system; nor is it associated with any predominant progenitor. Instead, it represents a confluence of work from diverse empirical and theoretical origins: cognitive psychology, observational and social learning, personality theory, behavior modification, and linguistics. There are three primary characteristics that distinguish the cognitive–behavioral approach. First, it recognizes the subjectivity of perception. People have idiosyncratic ways of construing the environment. Thus, their perception of stimulus events and response contingencies are also idiosyncratic. Therefore, perceived stimuli and contingencies take on as much importance as actual ones. Mischel[47] describes these perceptions as stimulus–outcome and response–outcome expectancies. This distinction directly parallels Bolles'[32] description of S–S* and R–S* expectancies in animal learning

paradigms. In humans, the indication is that individuals believe that the occurrence of certain stimuli and certain responses predict the arrival of particular outcomes. Human language capabilities render these expectancies more amenable to experimental inquiry. Second, the cognitive–behavioral approach sees behavior as the result of interactions between internal (e.g., cognitions and emotions) and external (e.g., situational settings) events. Third, this relationship between behavior and those internal and external events is seen as reciprocal. Behaviors can effect changes in external and internal events; these changes can, in turn, affect behavior. For example, an individual can behave so as to structure the external setting in ways that will increase the probability that certain cognitive expectancies will be strengthened.

Furthermore, the cognitive–behavioral approach embodies a methodological stance that clearly derives from the nonmediational emphasis on measuring observable aspects of behavior. In the case of problem drinking, attention focuses on (1) the frequency and duration of drinking episodes, (2) the amount of alcohol consumed, (3) the situational events that precede and follow drinking episodes, and (4) the nature and severity of problems associated with excessive alcohol use. Through use of self-report techniques, the cognitive–behavioral approach also provides for assessment, though indirect, of unobservable cognitions and emotions, thereby permitting an evaluation of how they contribute to drinking behavior.

According to the cognitive–behavioral position, problem drinking is a multidetermined phenomenon. No single learning process is seen as entirely capable of explaining the etiology of problem drinking. Chief determinants include past learning history, previous experience with alcohol, situational and environmental antecedents, cognitive expectations, and prevailing reinforcement contingencies (both positive and negative). Combined, these determinants yield compelling explanations for both acquisition and maintenance of problem drinking. The etiologic gestalt suggested by this multicausal paradigm has not been empirically validated. However, the reliable empirical trends presented below are persuasively consistent with the various determinants postulated.

3.2.1. Modeling. Research on observational learning indicates that modeling experiences greatly influence behavior. The rich behavioral information provided by vicarious experiences short-circuits the otherwise gradual learning processes depicted by operant and classical paradigms. The implication is that the alcohol-related behaviors of problem drinkers are acquired primarily through observing the alcohol-related behaviors of others, especially significantly others with drinking problems. Indeed, the empirical evidence indicates that adolescent problem drinkers frequently have parents who drink heavily,[48–50] whereas adolescents who are abstainers or light drinkers tend to have nondrinking parents.[50] These trends exceed the explanatory power of genetic arguments and underscore the vitality of social learning processes in accounting for the acquisition of drinking behaviors. Social learning analyses suggest that in families where alcohol use is limited to specific occasions (e.g.,

celebrations and religious rituals), the children learn moderation skills that promote light social drinking. However, family systems where alcohol is used indiscriminately to cope with a variety of emotional and environment stressors predispose the children toward similar patterns of excessive indulgence. These youngsters learn that drinking is what adults do to handle and/or escape life's problems.

This lesson may be further inculcated by media portrayals of alcohol use. Garlington[51] analyzed the alcohol use depicted on television soap operas and reported that most drinking occurred in the home and that straight drinks were more popular than mixed drinks and beer. Lowery[52] also analyzed alcohol use patterns shown in soap operas. The results revealed that in most portrayals, alcohol functioned to facilitate social interaction, manage crises, and escape from chronic stress. Moreover, alcohol use was generally reinforced or had no consequences. Such presentations, like familial models, communicate normative information about alcohol consumption. Certainly, their contribution to the acquisition of drinking behavior can be potent. In sum, influential vicarious sources of alcohol-related information that depict drinking as a means to (1) facilitate social interaction, (2) cope with stress and negative emotional states, (3) enhance sexual attraction, and (4) bolster self-esteem seem to encourage the development of excessive problem drinking.

Modeling experiences are important not only to the acquisition of alcohol use patterns but also to their maintenance. Experimental evidence clearly demonstrates that ongoing drinking behaviors are susceptible to modeling influences. These findings were extensively reviewed by Collins and Marlatt,[53] but the current discussion necessitates a brief review of this literature. In the initial investigation by Caudill and Marlatt,[54] male heavy social drinkers were asked to participate in the previously described "taste-rating" task so as to unobtrusively assess their wine consumption. During the task, each subject was accompanied by a confederate, posing as another subject, who demonstrated either a heavy (700 ml of wine) or a light (100 ml of wine) consumption performance. The congeniality of the subject–confederate pre-task interaction was also manipulated. Another group of subjects participated in a no-model control condition. The results showed that subjects in the heavy-model condition drank significantly more than subjects in the light-model and no-model conditions. The latter two groups did not differ significantly from each other. Alcohol consumption was unaffected by the type of social interaction. In explaining these findings, Caudill and Marlatt suggest that subjects (1) felt competitive or other social pressure to match the model and/or (2) used the model's behavior as a guide for how to perform in an ambiguous situation.

Using a similar design and natural tavern setting, Reid[55] obtained comparable results without the taste-rating task. Each tavern patron used as a subject was exposed to a light- or heavy-consumption model who behaved in a warm or cold fashion throughout the experimental assessment period. The results revealed that subjects exposed to the warm–heavy model consumed significantly more drinks per hour than those in the warm–light model

condition. For subjects exposed to the cold unfriendly model, their consumption rate did not vary with the modeled consumption rates, nor did it differ from that of the no-model control subjects. Similarly, Parks[56] found that a warm sociable model was most effective in influencing alcohol consumption. Other studies have shown that the modeling effect is greatest with male subjects[57,90] and with heavy drinkers.[57] Moreover, the effect is generalizable to black male social drinkers[58] and to alcoholics.[59]

In a more detailed analysis, Hendricks *et al.*[60] required subjects to perform an art-rating and the wine-tasting tasks under one of three sets of procedures: (1) coaction, in which subject and model perform the tasks simultaneously, thereby simulating the procedure used by Caudill and Marlatt[54]; (2) audience facilitation, in which subject and model perform each task concomitantly but in reverse order from each other (one rates wine while the other rates art); (3) imitation, in which the subject performed each task alone after having watched the model perform. In all conditions, the model exhibited either heavy or light consumption during the wine-tasting task. This investigation showed that subjects matched the model's drinking pattern only in the coaction condition. This finding corroborates the evidence from other investigations. Furthermore, it suggests that coaction rather than imitation best describes the modeling effects that have been observed experimentally. Unlike coaction, imitation does not require simultaneous performances. According to Hendricks *et al.*[60] in imitation, the model's behavior is passively observed and later performed in the model's absence.

In light of the evidence concerning the effects of parental drinking habits on offspring drinking, the Hendricks *et al.*[60] finding implies that perhaps imitative and coactive modeling experiences serve different developmental functions. Imitative modeling may be more important to acquisition of an alcohol use pattern, but coactive modeling may be more important to maintenance. During childhood and early adolescence, opportunities for coactive drinking experiences are limited by external restrictions. However, through observation of adult drinkers, e.g., parents, youngsters may acquire, store, and later imitate incipient components (e.g., cognitive expectations) of their evolving alcohol use patterns. In late adolescence and through adulthood, when drinking is not only acceptable but encouraged, coaction becomes the prepotent source of behavioral information about drinking. Coactive drinking experiences tend to be imbued with a host of social pressures and reinforcers that can propel or retard spontaneous consumption rates. Once an alcohol use pattern becomes established, the individual can conceivably seek coactive drinking experiences that will further strengthen or legitimize the established pattern and associated expectancies. Perhaps this explains the anecdotal observation that people tend to drink with companions who share similar use patterns. "Bottle gangs" are an extreme example of the role that coactive modeling plays in the maintenance of alcohol abuse.

The above considerations indicate that observational learning is critical to the acquisition and maintenance of problem drinking. The potency of both

imitative and coactive modeling experiences resides not only in their direct facilitational abilities but also in their encouragement of enduring alcohol-related expectancies.

3.2.2. The Role of Expectancies. Bandura[61] proposed a conceptual system that has proved very useful in clarifying the relationship between cognition and behavior. In some respects, his system combined Bolles'[32] and Mischel's[47] idea of response–outcome expectancies with Rotter's[62] idea of locus of control. Bandura distinguished between outcome and efficacy expectations. As before, outcome expectancies refer to the belief that a given behavior will produce certain desired outcomes. Efficacy expectancies refer to the individual's belief that he or she can execute the behaviors required to generate the desired outcomes. Applying Bandura's system to the problem of alcohol abuse permits a parallel categorization of alcohol-related expectancies: alcohol-outcome expectancies and alcohol-efficacy expectancies. This differentiation serves primarily as a conceptual tool in clarifying the role of alcohol-related cognitions in problem drinking. It also is useful for organizing relevant empirical findings.

3.2.3. Alcohol Outcome Expectancy and the Balanced-Placebo Design. Alcohol-outcome expectancy refers to the belief that alcohol consumption produces certain outcomes, e.g., mood alteration. These sorts of beliefs are primarily transmitted by the culture. Moreover, these beliefs tend to be general rather than specific. That is, the individual believes that the particular alcohol-outcome contingencies in question are applicable to most people and not just to the self. The cognitive–behavioral framework indicates that these expected contingencies can be as influential as actual contingencies. Therefore, the behaviors observed after alcohol consumption have presumably been influenced by both the expected and the actual alcohol-outcome contingencies. However, the advent of the balanced-placebo design has enabled researchers to experimentally separate the expected from the actual effects of alcohol.

Recent reviews have extensively explored the history and implementation[12] and the methodological features[63] of the balanced-placebo design. In brief, this design factorially crosses two alcohol expectancy conditions (expect alcohol versus expect no alcohol) with two alcohol content conditions (get alcohol versus get no alcohol). The resulting four-group design (see Fig. 1) has two true conditions in which subjects get what they expect and two deception conditions in which subjects receive the opposite of what they expect. This fully balanced design has been crucial to explicating the role of alcohol outcome expectancies.

A number of experimental studies[12] examining a variety of behavioral measures have employed the balanced placebo design. In one of the earliest of these studies, Marlatt *et al.*[46] established a laboratory analogue of the disease model "loss-of-control" hypothesis. Alcoholics and social drinkers were randomly assigned on an individual basis to each of the four balanced placebo conditions. Thus, half of all subjects were led to believe that their drinks

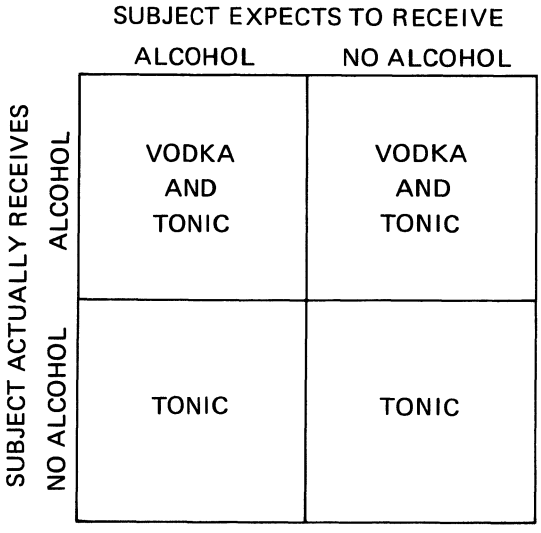

Figure 1. The balanced-placebo design.

would contain vodka and tonic, and half were led to believe that their drinks would contain only tonic. In actuality, half the subjects in each of those groups received a drink containing vodka. As part of the procedure, all subjects consumed a "primer" drink before participating in a taste-rating task. According to the loss-of-control hypothesis, alcoholics who received alcohol would show the greatest beverage consumption during the taste-rating task regardless of what they were told about beverage content. However, the findings revealed a main effect for alcohol expectancy. That is, subjects who were told that their primer drinks contained alcohol drank significantly more than subjects who expected only tonic. Also, alcoholics drank significantly more than social drinkers. The loss-of-control hypothesis was not supported, as the amount consumed was unaffected by actual alcohol content.

Another set of studies has used the balanced-placebo design to evaluate the role of alcohol-outcome expectancies in emotional experiences. Wilson and Abrams[64] investigated social anxiety in male subjects. After alcohol was administered in accordance with the balanced-placebo conditions, subjects were asked to make a favorable impression on a female observer. The authors found that subjects who expected alcohol showed significantly lower heart rates and reported less subjective anxiety than subjects who expected tonic. In an almost identical study using female subjects and a male observer, Abrams and Wilson[65] found that expectancy was again the only significant determinant of anxiety. However, the effect was reversed. Females exhibited higher heart rate and skin conductance when they were led to expect alcoholic drinks. These opposite findings were explained in terms of the fact that female subjects had less drinking experience and therefore may have had different or less clear-cut expectancies about how alcohol effects interpersonal behavior and evaluation anxiety. The literature suggests that more experienced drinkers

have clearer and stronger expectancies about the outcomes that follow alcohol consumption.

Southwick *et al.*[66] conducted a correlational study and found that the heavier social drinkers expect more "stimulation/perceived dominance" and "pleasurable disinhibition" effects from a moderate dose of alcohol than do light social drinkers. At any rate, the two Abrams and Wilson studies clearly illustrate that perceived alcohol ingestion is more effective than actual alcohol ingestion in influencing both self-report and physiological measures of anxiety. Likewise, Lang *et al.*[67] found that expect-alcohol subjects were more aggressive during a shock administration procedure than expect-tonic subjects regardless of actual drink content.

Without adequate control of expectancy effects, early studies revealed an inverse relationship between the amount of alcohol consumed and penile tumescence.[68,69] However, a large and growing number of investigations have employed the balanced-placebo design to further examine this relationship. Wilson and Lawson[70] found that expect-alcohol subjects exhibited greater penile tumescence than expect-tonic subjects in response to films depicting heterosexual and homosexual erotica. Actual alcohol content of the drink was unrelated to penile tumescence. These findings suggest that male subjects generally expect alcohol to enhance sexual responsivity. Presumably, the conviction that they had consumed alcohol activated the alcohol-outcome expectancy, thus facilitating physiological responsivity to sexual stimuli. Subsequent studies show that this expectancy effect, in male subjects, becomes more pronounced as the sexual stimuli become less socially appropriate. Briddell *et al.*[71] reported that the difference in penile tumescence between expect-alcohol and expect-no-alcohol subjects was greatest when the erotic material was deviant (forcible rape and sadistic aggression) rather than nondeviant (heterosexual intercourse).

Lang *et al.*[72] investigated this deviance variable by using subjects who differed in their degree of generalized comfort with sexual material, as measured with Mosher's[73] sex guilt scale. After undergoing the alcohol-expectancy and alcohol-content manipulations, high-, medium-, and low-sex-guilt males were provided with *ad libidum* exposure to a series of slides depicting sexual content. The stimulus slides varied in the explicitness of their content. The time spent viewing and evaluating each slide was unobtrusively monitored. As predicted, subjects high in sex guilt showed the greatest expectancy effect. That is, high-sex-guilt subjects who expected alcohol viewed the slides longer than their counterparts in the expect-tonic conditions. Moreover, the effect increased with the more explicit stimulus slides. Lansky and Wilson[74] were unsuccessful in replicating this effect with slide viewing behavior, but they did find that high-sex-guilt subjects evidenced a significant expectancy effect with penile tumescence. Together, these findings indicate that male subjects generally expect enhanced sexual responsivity as an outcome of alcohol ingestion and that sex guilt represents an important moderator variable. Female sexual arousal is not as susceptible to expectancy effects.[75]

In sum, the findings reviewed above indicate that for a certain class of behavior, perceived alcohol ingestion exerts a stronger influence than actual alcohol ingestion in governing the intensity of the behavioral outcomes. This particular class includes behaviors that have come to be associated with pervasive culture-bound beliefs describing how these behaviors are affected by alcohol consumption. Social anxiousness, aggression, and sexuality certainly belong to this behavioral class. Within this class of behavioral experiences, alcohol-outcome expectancies are generally simple, specific, and potent, especially with male social drinkers. However, with behaviors that are not associated with clear and specific alcohol-outcome expectancies, actual alcohol content is more important than perceived content. This has been illustrated with tests of reaction time,[67] pursuit-rotor performance,[76] and memory recall.[77]

3.2.4. Self-Efficacy, Alcohol-Efficacy Expectancies, and Social Skills. Bandura coined the term self-efficacy, a concept that refers to the belief that a person holds about the adequacy of his/her skill repertoire for impinging situational demands. A person high in self-efficacy believes that he/she can execute the behaviors necessary to successfully generate the desired outcomes in a situation. Conversely, those low in self-efficacy believe that they are unable to execute the actions required to attain the desired reinforcers; for this individual, the behavioral skills needed to generate the desired outcomes are unavailable. Very often, this unavailability reflects one of two sets of circumstances: (1) social skill deficit, in which the individual has never acquired or adequately mastered the social skills needed to obtain the reinforcers available in a given situation; (2) response inhibition, in which the necessary skills are present in the individual's repertoire, but inhibitory cognitions (e.g., fear of failure) or anxiety prevent their expression. The inhibition experienced may be situationally specific or transsituational. When the individual estimates that the prevailing situational demands exceed his/her available skills, then he/she is likely to experience a lowered sense of self-efficacy. As a result, the individual is unlikely to initiate and maintain efforts to produce the appropriate behaviors.

We hypothesize that for the problem drinker, these episodes of low self-efficacy activate a compensatory set of cognitions: alcohol-efficacy expectations, i.e., the individual's belief that alcohol consumption will enable him or her to successfully execute the behaviors required to produce the desired outcomes. This belief may be expressed by such self-statements as: "I don't get going at parties until after I've had a few drinks," "In discos, I'm much better at meeting and dancing with strange women when I've had a couple of beers." Bandura[61] explained that self-efficacy accrues from four sources of information: performance accomplishments, vicarious experiences, verbal persuasion, and emotional arousal. Similarly, alcohol-efficacy expectations arise from the same sources.

3.2.5. Performance Accomplishments. Problem drinkers have probably had experiences in which they behaved in socially reinforcing ways (e.g., "life of the party") while under the influence of alcohol. Recollections of these

episodes may contrast sharply with memories of less rewarding and perhaps even aversive social encounters unaccompanied by the "social lubricant." Moreover, the successful social skills expressed during previous drinking episodes could conceivably have become associatively tied to the presence of a rising blood alcohol level through a "state-dependent learning" process. In a new and challenging social situation, this individual may experience a miraculous reappearance of his social skills after a drink or two.

3.2.6. Vicarious Experience. Witnessing others receive social reinforcement for behaviors emitted after alcohol ingestion strengthens the problem drinker's belief that alcohol will improve his/her social competency.

3.2.7. Verbal Persuasion. Other individuals may actively encourage the problem drinker to use alcohol as a social facilitator, e.g., "don't be so uptight; let me buy you a drink," or "let's have another drink and go talk to those pretty ladies over there." Other problem drinkers may even communicate to the individual that he or she is more interesting or desirable after he has been drinking.

3.2.8. Emotional Arousal. Often, emotional arousal is used as a source of information about personal competency. The individual learns that anxiety impairs social performance. Thus, the presence of anxiety becomes recognized as an indicator of poor performance. Problem drinkers probably maintain the perception that alcohol reduces anxiety and thereby enhances social competency. In the framework of the response-inhibition idea, alcohol has lifted the inhibitory anxiety, releasing the individual's social skills for expression.

These four sources of information converge to convince the problem drinker that the efficacy of his basic social and coping skills is highly reliant on alcohol consumption. Stressful situations that spotlight these skill deficits evoke a lowered sense of self-efficacy and set the state for alcohol-efficacy expectations. To the extent that subsequent consumption of alcohol seems to bestow the necessary skills, it induces momentary feelings of enhanced self-efficacy and self-esteem. Although later, these heightened feelings of self-worth will be disowned and attributed to the alcohol, for the moment they are eagerly embraced.

Unlike the alcohol-outcome expectancies, the alcohol-efficacy analysis lacks direct empirical validation. Such validation requires demonstration that for problem drinkers, in a stressful social situation, alcohol consumption varies negatively with the strength of self-efficacy expectations and positively with the strength of alcohol-efficacy expectations. A cogent demonstration of this would entail a multifactorial design with social drinkers and nonstress control conditions as well as multiple assessments of the strengths of both types of expectancy. To date, no such investigation has been reported.

However, a number of empirical findings have emerged that are germane to the alcohol-efficacy analysis. These findings are consistent with the proposition that alcohol consumption increases in situations in which individuals have limited access to coping responses. Marlatt *et al.*[78] exposed two groups of subjects to an anger-inducing encounter with a confederate who posed as

another subject. One group was then given an opportunity to retaliate against the confederate by administering ostensibly painful electric shocks; members of the other group were not permitted to discharge their anger. The taste-rating task was used to compare the alcohol consumption of these two groups with that of an unangered control group. As expected, subjects who were allowed to exercise a retalitory coping response drank significantly less than angered subjects who were deprived of anger expression. The absence of a coping response for these subjects led to a heightened alcohol intake. The unangered control group consumed an intermediate amount, falling between the other two groups.

The Marlatt *et al.*[78] study used only social drinkers and controlled the availability of coping responses through external procedures. Each subject was assumed to possess an adequate and functional repertoire of coping responses. Investigations using alcoholics as subjects suggest that these individuals inherently lack comparable access to coping skills. Conditions of interpersonal stress accentuate these shortcomings and set the stage for increased drinking. Using a within-subject design, Miller *et al.*[79] exposed a group of alcoholics and a matched group of social drinkers to stressful and nonstressful conditions. Stress was created by presenting the subjects with analogue assertion situations, antagonistic prompts, and negative evaluative feedback. During the nonstress condition, subjects merely discussed recreational interests and activities. Following each exposure, subjects bar-pressed for alcohol. The findings revealed that alcoholics drank significantly more alcohol after the stressful than the nonstressful condition. Social drinkers, on the other hand, tended to decrease alcohol ingestion in the stressful condition. The authors[79] concluded that "the alcoholic has learned to respond to stressful situations by consuming alcohol, whereas the non-alcoholic has learned a variety of more adaptive responses" (p. 71). Other studies by this research group using alcoholic subjects have shown that assertiveness[80] and effectiveness at expressing negative feelings[81] were negatively correlated with alcohol consumption. Similarly, Allman *et al.*[82] found that alcoholics drank most when exposed to interpersonal stress and socialization demands.

Whether or not the increased drinking shown in the above studies was mediated by fluctuations in efficacy expectations or self-esteem is not known, since cognitive experiences were not assessed. However, other investigations have supplied findings that suggest a definite link between alcohol consumption and cognitive–emotional experiences. McClelland *et al.*[83] report on a series of investigations designed to evaluate the role of power needs in alcohol abuse. In an experimental study with social drinkers, they found that increases in alcohol consumption were accompanied by increases in perceptions of control and power as measured by the Thematic Apperception Test. The authors present a well-supported argument for the contention that drinking is primarily motivated by the individual's need for personal power. Alcohol, presumably, fulfills this need by conferring a sense of power and control on the drinker. Deardoff *et al.*[84] employed a scale entitled the Power-

Oriented Semantic Differential and found that problem drinkers describe themselves as having significantly less personal power (dominant–submissive) than nonproblem drinkers. Using Rotter's Locus of Control Scale, O'Leary *et al.*[85] found that among alcoholics, an external locus of control was associated with a high frequency of self-enhancement drinking. Together, these findings indicate that reduced feelings of power, control, or efficacy may play a precipitative role in alcohol abuse.

Other empirical findings pertinent to the alcohol-efficacy analysis come from research examining the conditions under which abstinent alcoholics undergo relapse drinking. In a follow-up assessment of alcoholics who had participated in an aversive conditioning treatment program, Marlatt[86] found that 78% of the subjects had relapsed within 90 days. Details culled from extensive interviews revealed that 50% of all relapse situations fell into one of two categories: (1) situations in which subjects felt angry or frustrated, usually in an interpersonal or social situation, or (2) situations in which the subject was socially pressured to resume drinking. Chaney *et al.*[87] categorized the relapse experiences reported by alcoholics who had undergone skill training or one of two control group procedures 12 months earlier. In this study, 43% of the reported relapses involved a negative emotional state, and 17% involved interpersonal temptation. Frustration/anger situations and intrapersonal temptation each accounted for 15.5% of the relapses; 9% were unclassifiable.

Marlatt and Gordon[88] examined the relapses of a mixed sample of alcoholics, smokers, and heroin addicts who had undergone treatment programs. Seventy-four percent of the 70 alcoholic relapse episodes investigated fell into three categories: coping with negative emotional states (38%), social pressure to drink (18%), and interpersonal conflict (18%). The uniformity of these trends is quite compelling.

To summarize, the alcohol-efficacy expectation analysis has not been tested in its entirety. However, a growing network of empirical findings seems to support the idea that alcohol-efficacy expectancies and subsequent drinking serve a compensatory function for problem drinkers confronted with situations that pose a threat to the individual's self-efficacy and self-esteem.

3.3. Prediction of Excessive Drinking and Relapse

The analyses and findings presented above indicate that excessive drinking and relapse may be best conceptualized as a maladaptive coping response. Extrapolating from these considerations, Marlatt[89] formulated a model for predicting excessive or inappropriate drinking. The proposed model states that the probability of excessive drinking will vary in a particular situation as a function of the following factors. (1) The degree to which the drinker feels controlled by (or feels helpless relative to) the influence of another individual or group (e.g., social pressures to conform, modeling, evaluation, or criticism by others; being frustrated or angered by others) or by external environmental

events beyond the control of the individual (e.g., misfortune, financial loss, feeling bored or depressed). Any situational event that threatens the drinker's perception of control in this sense is defined as a *high-risk situation.* (2) The availability of an adequate coping response as an alternative to drinking in the high-risk situation. If the individual fails to perform an appropriate coping response, a sense of lowered *self-efficacy* develops. (3) The drinker's *expectations* about the effects of alcohol as one means of attempting to cope with the situation. As previously discussed, these expectations can center around the presumed outcome and/or efficacy features of alcohol. *Alcohol-outcome* expectations are more general and simplistic and entail beliefs regarding alcohol's presumed role in directly bringing about reinforcing consequences, e.g., relaxation or sexual arousal. *Alcohol-efficacy* expectations are more specific to the drinker's past experiences and entail beliefs about alcohol's role in enabling behaviors that will be reinforced (e.g., assertiveness). Both types of expectancies are capable of enhancing the drinker's feelings of perceived control and personal power. (4) The availability of alcohol and the constraints on drinking in a given situation.

In a detailed theoretical analysis, Marlatt and Gordon[88] discuss how the same constellation of determinants can precipitate relapse. After undergoing a period of voluntary abstinence, the alcoholic individual who encounters a high-risk situation, feels unable to cope, expects that alcohol ingestion might help, and finds alcohol easily available will very likely have a drink. If he takes that first drink, then the ensuing cognitive and behavioral sequelae maximize the likelihood of continued drinking. Marlatt and Gordon describe the Abstinence Violation Effect (AVE) to explain the progression from the initial violation of abstinence to a "full-blown" relapse. The AVE has two primary components. (1) *Cognitive dissonance:* taking that first drink conflicts with the individual's image of the self as an abstainer and thereby creates dissonance. Continued drinking relieves dissonance, a negative emotional drive state, by reducing subjective anxiety and permitting the alcoholic to bring his self-image more in line with his current drinking behavior ("I guess I'm off the wagon"). (2) *Personal attribution effect:* the individual attributes the violation to internal weaknesses or personal failure (e.g., "no willpower") rather than to situational pressures or lack of coping responses. By way of "self-fulfilling prophecy," this attribution can spur more drinking. The relapse process is further fueled by any perceived positive sensations that follow from that first drink. Such perceptions can restore feelings of power and thereby provide a potent reinforcement experience.

4. Concluding Comments

The behavioral perspective on the etiology of alcohol abuse has not remained static. Instead, it has evolved through a number of distinct stages. The principles presented at each stage have contributed to our understanding

of alcoholism and associated behaviors. Nevertheless, each set of principles eventually gave way to newer ideas that modified and added to the growing body of knowledge. As reliable research trends revealed the conceptual and practical limitations inherent in the various explanatory systems, the evolution continued.

The material reviewed in this chapter asserts that a rigidly nonmediational behavioral perspective cannot adequately explain the complexities of alcoholism. This is not to suggest that these systems are invalid or superfluous. On the contrary, the validity and heuristic value of classical and operant conditioning models are without question. Research and explanatory constructs based on these models will continue to enrich our insights about the psychology and physiology of alcohol effects. However, these approaches have proven inadequate for resolving the myriad of questions regarding the acquisition and maintenance of alcohol abuse. Likewise, the original TRH, despite its intuitive appeal and mediational nature, does not provide a sufficient explanation. By contrast, the cognitive–behavioral approach greatly expands the potency of the behavioral perspective. With its multicausal framework, the cognitive–behavioral approach generates compelling theoretical explanations for both the acquisition and maintenance processes. Furthermore, it furnishes realistic intervention strategies that emphasize the cultivation of coping skills and self-control abilities. This skill training approach is designed to promote greater feelings of confidence, mastery, and self-efficacy for the individual.

As we have seen, the cognitive–behavioral approach embodies a number of explanatory constructs that enjoy empirical support. Nevertheless, the theoretical gestalt and some of its subordinate analyses, presented here, must await research validation. The evolutionary process continues, and the cognitive–behavioral approach will be judged by the same criteria as its predecessors: predictive accuracy and explanatory force.

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6

Behavioral Treatment Methods for Alcoholism

Glenn R. Caddy and Trudy Block

Abstract. This chapter offers a review of the behavioral methodology directed to the treatment of alcoholism. Beginning with an outline of the theoretical bases of behavior therapy and assessment, a review with some historical perspective is undertaken of the chemical, electrical, and covert aversion treatments of alcoholism. Thereafter, the procedures of the social skills-training strategies (including marital skills and assertiveness training) are presented, followed by a discussion of the relaxation and desensitization techniques. The operant methodologies are illustrated by contingency contracting and the community-reinforcement approaches. Within the broad-spectrum procedures, a description of self-control training and an example of a broad-spectrum treatment study are offered. It is noted that although the merits of these various techniques are becoming widely recognized in the alcoholism treatment literature, the behavior therapeutic approaches to alcoholism have yet to receive widespread public acceptance. It is anticipated that future studies of treatment effectiveness will contribute to an increasing appreciation of the advantages of behavioral therapies to the management of alcohol abuse and dependence.

1. Introduction

Historically, virtually all approaches to the treatment of alcoholism have included at least some behavioral prescriptions and proscriptions. Those therapies that are primarily behavioral in their orientation and methodology, however, may be distinguished from other therapeutic orientations in that they involve one or a number of specific techniques that employ psychological (especially learning-based) principles to change behavior. Although even today there is controversy over just which techniques are encompassed by the term "behavior therapy,"[1,2] it is clear that the field is gradually changing its focus from one involving a rather narrow interpretation of behavior staunchly based on presumably "established" principles of learning[3–6] to a more broadly based one reflecting the application of experimental psychological principles to the treatment of the individual case. These more recent formulations permit and, in fact, encourage the study of the role of cognition in mediating the behavior under investigation.[7–10]

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A number of general assumptions are made in the application of behavioral treatment techniques: (1) in comparison to other psychotherapy approaches, behavior therapy focuses on the patient's current behavior with relatively little concern being directed to the presumed underlying causes of the problematic behavior; (2) maladaptive behaviors, as all behaviors, are acquired through learning; (3) the application of psychological (especially learning) principles can be effective in changing maladaptive behaviors; (4) specific individualized treatment goals must be established, and special procedural sequences must be developed to achieve these goals; and (5) although this is not stringently adhered to, the process of ongoing treatment evaluation and follow-up is seen to be an integral part of behavior therapy.

Behavior therapists tend to view alcohol abuse within a social learning model. Within this model, alcohol abuse is viewed as a socially acquired behavior pattern maintained by numerous antecedent cues and consequent reinforcers which may be of a psychological, sociological, or physiological nature. Such factors as reduction in anxiety, increased social recognition and peer approval, enhanced ability to exhibit more varied spontaneous social behavior, or the avoidance of physiological withdrawal symptoms maintain substance abuse. Therapeutic goals within this framework typically include (1) a detailed assessment of the specific antecedent and consequent events related to alcohol abuse, (2) the use of social learning-based treatment procedures to teach social skills that can serve as alternatives to excessive drinking, (3) the rearrangement of consequences for both excessive drinking and sobriety within the community environment, and (4) the short- and long-term evaluation of the outcome of the treatment program using objective measurement procedures.

Over the past 20 years, behavior therapists increasingly have been drawn to the alcoholism field by the challenge of what many have seen as the theoretical as well as technical inadequacies of previous treatment approaches to this most important area of social concern. The work of these behaviorally oriented scientists has been significant, for it has profoundly altered the way many disciplines now perceive the nature, processes, and management of the alcoholic condition. It has led to a questioning of the traditional perspective of alcoholism[11,12] and to a reevaluation of the necessity of and/or the commitment to abstinence as the only acceptable treatment goal for alcoholic patients.[13]

Before beginning a review of the behavior therapy approach to the treatment of alcoholism, it would seem desirable to offer some perspective on the behavioral assessment of alcoholism.

1.1. Behavioral Assessment of Alcoholism

Behavior therapy requires a detailed behavioral assessment of the individual case as a precursor to treatment. In the case of alcoholism, such an assessment permits an understanding of the functional relationships between abusive drinking and various cognitive, emotional, social, environmental, and

physiological events existent within or impinging on the drinker. The nature and extent of the patient's drinking patterns also must be objectively determined. These "base-line" assessments not only provide a basis for the establishment of a treatment plan, they also help to establish criteria for an evaluation of the plan both during and following the intervention.

Miller[14] comments on the issues of behavioral assessment of alcoholism in the following terms:

> In assessing alcoholic drinking behavior, the therapist must use all of the sources of data at his disposal. These sources frequently consist of self-reports, reports based on observations by "significant others," and direct observations by the therapist. In addition, behavior patterns which correlate with abusive drinking are evaluated. These might include number of hospitalizations or arrests for alcohol-related problems. Self-reports of drinking by the alcoholic are the most easily obtainable sources of data. However, due to the possibility of distortion, forgetfulness, or misrepresentation, the reliability of this information must be established.

Of course, the patient's functioning in other (not directly drinking-related) areas of his/her life also must be assessed before and throughout the therapeutic endeavor. Such an assessment is undertaken for several major reasons: (1) alcohol abuse commonly leads to marital, social, emotional, or occupational problems which must be resolved along with the drinking problem, (2) attention to these problems can facilitate the prospect of therapeutic success, and (3) social and emotional problems often precipitate excessive drinking and so commonly maintain its occurrence.

Caddy[15] recently has offered a multivariate behaviorally based model of alcohol abuse assessment. Briefly, this approach recommends that alcohol abuse and dependence be conceptualized and evaluated in terms of dynamic interplay among behavioral, cognitive, incentive, social, and discriminative factors. Irrespective of one's subscription to this particular conceptual and assessment model, it is generally recognized in the behavioral approach to alcoholism that a detailed functional analysis of the social, emotional, cognitive, environmental, and physiological antecedents and consequences of drinking is essential in order to delineate which factors must receive therapeutic attention.

We now begin a review of the behavior therapy approaches to the treatment of alcoholism. In so doing, however, we concentrate on the procedural aspects of the various treatment strategies rather than on evaluating their outcome. (A fine review of the behaviorally oriented treatment outcome literature is available elsewhere in this text; see also refs. 16–18.)

2. The Aversion Therapies

Relatively recent research[19,20] addressing the importance of stimulus relevance and salience to the establishment of taste aversion has pointed to the

biological appropriateness of the use of nausea- and emesis-inducing therapies in reducing the desirability of alcohol consumption; yet, early alcohol aversion programs did not have the advantage of these data. Rather, these programs were founded on the more basic laboratory-based classical conditioning paradigms in which the response of nausea was conditioned to a broader array of alcohol-related stimuli.

Virtually all of the behaviorally oriented aversion studies reported in the alcoholism treatment literature prior to 1970 involved either the introduction of specific conditioning procedures as a component in an otherwise nonbehavioral treatment program or, alternatively, they involved the use of one or occasionally two behaviorally based techniques as part of a narrow-band treatment endeavor.

The basic classical aversive conditioning paradigm involves a simple procedure in which the sight, smell, and taste of alcohol is paired repeatedly with one of a number of unconditioned stimuli (UCSs ranging from electric shock to apneic paralysis). The aim of this procedure is to provoke abstinence by reducing the incentive motivation to drink (mediated via the formulation of conditioned aversive reactions to the various alcohol-related stimuli). In the earlier literature, it was believed that a sensitizing of the patient would occur and that, depending on the UCS employed, the patient would experience nausea or distaste or anxiety and fear on exposure to the alcohol-related stimuli. Subsequent experimentation, both in the aforementioned area of taste aversion and with electrical aversive procedures,[21,22] has led to a reevaluation of this earlier position. This research has shown the most common consequence of aversive conditioning to be a reduction in the motivation to drink rather than the establishment of conditioned nausea reactions. An interesting and provocative critique of a number of theoretical foundations of aversion therapy has been provided by Hallam and Rachman.[23]

2.1. Chemical Aversion

The first scientifically credible alcoholism treatment studies to include a chemical aversion component were conducted by Voegtlin and his associates at the Shadel Hospital in Seattle.[24] These investigators used emetine hydrochloride in a classical conditioning paradigm within a program that included an array of other abstinence-oriented therapies, all of which were provided together with a very strong expectation of positive treatment outcome.[25–29] More recently, Wiens *et al.*[30] and Neubuerger *et al.*[31] also have used chemical aversion treatment procedures and patients similar to those of Voegtlin.

In a typical chemical aversion treatment regimen, especially if apomorphine is used as the nausea-inducing agent, the alcoholic patient is given a stimulant drug such as benzedrine sulfate prior to the conditioning trials. Such a procedure, it is believed, augments the conditioning effect and offsets the possible narcotic effects of the subsequently administered emetic.[32] (In fact, emetine has become favored over apomorphine as the emetic drug of

choice, for the former does not produce the same degree of central nervous system depression as is found with apomorphine.) The conditioning trials typically are conducted in a light- and sound-attenuated room in which the patient is confronted with an array of liquors. At the beginning of the session, the patient is given a warm salt water solution within which emetine has been dissolved. Thereafter, the client is given an injection containing additional emetine (and perhaps containing some stimulant drug as well). A few minutes later he/she is confronted with several ounces of alcohol to smell, taste, swill around in the mouth, and then (for some clinicians) swallow. It is considered crucial to administer the alcohol prior to the onset of emesis even if the temporal lag between the presentation of the alcohol and the effects of the drug is relatively long. If the alcohol is administered following emesis, its consumption may be associated with recovery, which, in turn, may enhance its palatability.[33]

Typically within a few minutes, nausea and vomiting occur. Some authors[29] even have recommended the administration of tartar at this time in order to prolong the nauseous state. After a period of recovery, typically 1 to 2 days, subsequent treatment sessions are permitted to last somewhat longer, with a variety of alcoholic beverages being offered in order to enhance the occurrence of stimulus generalization.

The foregoing paradigm has been employed most commonly in the classical aversion approach to alcoholism therapy. Within this general framework, however, the use of drugs resulting in temporary paralysis also has been reported.[34,35] In this procedure, the alcoholic patient is attached to monitors that record electrodermal responses (EDR), respiration, heart rate, and muscle tension. A saline solution to which succinylcholine chloride dyhydrate (Scoline®) has been added is then intravenously administered.

During the alcohol confrontation sequence, the patient is presented with a series of bottles containing preferred beverages which he/she is required to smell and taste. After several such sequences, the paralytic agent is injected into the drip and an immediate change in the patient's EDR indicates the imminence of paralysis. The administration of alcohol is timed to precisely coincide with the onset of traumatic paralysis. During the brief period of apnea, typically lasting 60 sec, the beverage is held to the patient's lips until regular breathing recurs.

Since abstinence or improvement rates for alcoholic patients subjected to succinylcholine aversion therapy have not exceeded those of placebo groups[36–38] and given the extremely traumatic nature of the procedure, this therapeutic approach currently is not considered viable as a treatment for alcoholism.

A detailed review of studies that have evaluated the outcome of chemical aversive conditioning procedures in alcoholism therapy is beyond the scope of this chapter (but see refs. 39–43). Nevertheless, these and other studies have reflected serious limitations in, and questioned the value of, such procedures in the treatment of alcoholism.

Recognizing the limitations of the traditional chemical aversive condi-

tioning procedures, Lovibond[44] and Lamon *et al.*[45] have reported pilot work involving the development of alcohol aversion induced by motion sickness. The pseudocoriolis effect[46] involves the induction of nausea when an individual tilts his/her head from side to side under certain conditions of visual experience. The effect is achieved by seating the subject within a large cylinder marked with vertical stripes and then rotating the cylinder around the subject while he/she moves the head from left to right and back again. This combination of optokinetic input and vestibular stimulation produces in the subject the illusion that he/she is spinning. This, in turn, results in severe disequilibrium, dizziness, and nausea. Alcohol is administered during this sequence until nausea ensues. Following a recovery period, additional conditioning trials are required. S. H. Lovibond (personal communication, 1980) has succeeded in inducing conditioned nausea with this sequence and has precipitated a response in some of his subjects characteristic of taste aversion learning. At this time, however, there have been no significant outcome studies reported in the alcoholism treatment literature pointing to the success of this rather unusual treatment approach.

2.2. Electrical Aversion

Aversive conditioning procedures employing electrical aversion to suppress drinking behavior were first reported by Kantorovich in 1929.[47] It was not until the early 1960s, however, that the approach began to receive considerable attention, largely because of the simplicity of its application, the degree to which it permitted the control of stimulus onset and offset, and because it was seen to offer less unpleasant consequences for both the patient and therapist than was the case with chemical aversive procedures.

Electrical aversion therapy is based on the principles of classical (Pavlovian) conditioning in which a conditioned stimulus (CS, alcohol) is repeatedly paired with an UCS (electrical stimulation) until a conditioned response (CR, anxiety, according to the two-factor theory[48]) is developed in response to the administration of the CS alone. This anxiety, it is argued, triggers avoidance behavior which is presumably reinforced by anxiety reduction. Generally, escape or avoidance conditioning paradigms have been applied in the electrically based aversive procedures, although nonavoidable response-contingent aversive procedures (punishment) also have been employed.

Blake[49] employed an escape paradigm combining relaxation, motivational arousal, and shock procedures in what he regarded as the successful experimental treatment of 37 alcoholic patients. In this procedure, Blake's patients were attached at the forearm to a shock generator, and various alcohol beverages were presented for them to sip but not swallow. Following each sip, a shock of increasing intensity above the patient's pain threshold was administered. The patient could terminate the shock by expectorating the alcohol. Shock was delivered on a schedule randomized around a 50% ratio. The shock trials were spaced over 4 to 8 days with a total of about 5 hr devoted

to each patient (see also ref. 50). Blake reported that 54% and 52% of his subjects located at 6 and 12 months follow-up, respectively, were sober.

Mills, Sobell, and Schaefer[51] also used an escape paradigm in attempting to train social drinking in a group of voluntary inpatient alcoholics. Based on previous research by these same investigators,[52] Mills and his colleagues attempted to shape "appropriate" drinking behaviors of diluting drinks, sipping, spacing, and stopping when a certain limit had been reached. During the first three drinks of each treatment trial, the intensity of the shock administered to their subjects was a function of the kind of drinks ordered (diluted or straight) and whether they were sipped or gulped. If their subjects exhibited two inappropriate drinking behaviors, a high-intensity shock was administered at once. The emission of one inappropriate behavior resulted in a low-intensity shock. Any drinks ordered beyond the limit of the first three drinks led to the receipt of a 100% intensity shock which began when the patient's hand touched the glass and was terminated when the glass was released. Fourteen experimental sessions, each of which lasted up to a maximum of 2 hr, were conducted. In addition to the abovementioned studies, similar escape paradigm electrical aversive procedures have been reported.[53–57]

MacCulloch, Feldman, Orford, and MacCulloch[58] reported a study involving four alcoholic patients who were treated within an anticipatory avoidance paradigm. In this study, avoidance of the punishing stimulus was contingent on the performance of a specific operant, in this instance, the avoidance of alcohol. This avoidance paradigm differs from that of escape in that in the former, if the subject's reaction to the shock is sufficiently rapid, the aversive stimulation may be avoided entirely. The adoption of the approach by MacCulloch *et al.* was based on the work of Solomon and Wynne[59] who demonstrated active avoidance conditioning to be extremely resistant to extinction (see also ref. 60).

The treatment involved setting up a hierarchy of stimulus situations comprising a range of photographs of beer and spirits, the sight and smell of both corked and open bottles of alcohol, and glasses containing liquors. Taped recordings inviting the subject to enjoy a drink were played, and slides of orange squash were used as relief. In addition, a glass of squash was placed beside the patient to be drunk immediately on CS removal. This battery of stimuli was arranged by the patient into a hierarchy of increasing attractiveness. The patient was then required to work up the hierarchy and was shocked for failure to avoid turning off the stimuli. Therapy was terminated for the four inpatients when they indicated a lack of interest in alcohol and when all stages of the hierarchy had been completed. The number of sessions ranged from 10 to 46. Unfortunately, in all cases, therapy was unsuccessful. A lengthy critique of this approach to alcoholism therapy has been offered by Caddy.[61]

Finally, there are those aversive procedures that involve a punishment paradigm. Whereas in traditional conditioning studies, the UCS is presented after the CS, with punishment (response-contingent aversive stimulation), the aversive stimulus is delivered after the occurence of a response considered

by the therapist to be unacceptable. From the point of view of the subject, punishment permits the retention of some control in the therapeutic situation; if the subject does not respond, he/she receives no aversive stimulation. However, when these procedures are applied in the therapeutic setting, the patient is often instructed to respond in order that the punishment may be administered.

Observation of animals in a punishment paradigm readily demonstrates that if the aversive stimulation is intense, conditioned emotional behavior comes to be displayed in the experimental chamber prior to the onset of the stimulation. It may then be suggested that the major component in the effectiveness of punishment is the classical conditioning of emotional responses to the interoceptive "feedback" stimuli that accompany the performance of the response (and to a lesser extent to the stimuli in the experimental siuation). If this is so, the effective difference between classical aversive conditioning and punishment (when applied to patients in a therapeutic setting) is that in the former, linkages are established between a selected external CS and the UCS. In the latter, the linkages form between the stimuli (both internal and external) produced by performing the response and the UCS.

Hsu[62] reported the use of a punishment paradigm in which 40 alcoholic inpatients were treated with electric shock. On conditioning days, each patient was presented with three alcoholic and three nonalcoholic beverages and was required to drink these in any order he chose. Within ½ to 30 sec after taking the alcoholic drink, a unidirectional electrical pulse ranging from 2 to 5 mA was applied. (Self-administration of these shocks using a portable device was later instituted.) The procedure lasted for 5 days, and during each conditioning period, the patient was permitted to choose five and four (respectively) of the available six drinks in order to permit the establishment of a passive avoidance response. Following the treatment sessions, each patient was released, and 2-day booster sessions at 4 weeks and 5 months were scheduled.

It is not likely that the conditioning of emotional responses to alcohol actually occurred in Hsu's study, for the procedure, which involved between 12 and 15 shocks, did not provide a sufficient number of punishment trials. Furthermore, given that the criterion for success in this study was not alcohol consumption related but involved the number of times patients attended the therapy sessions, treatment success could not be established.

Lovibond and Caddy[63] introduced a variation of electrical aversive conditioning when they subjected 31 alcoholic patients to a discriminated aversive punishment procedure embedded within a broad-spectrum behavioral treatment program. The punishment procedure employed by these investigators was both particularly aversive and unique and, therefore, will be briefly described.

Lovibond and Caddy required their patients to drink preferred alcoholic beverages until such time as they achieved a blood alcohol concentration (BAC) of 65 mg/100 ml. Thereafter, a shock electrode was attached to the neck about 1 inch above the larynx, and a second electrode was positioned on various areas of the neck and face. Patients were told that they may expect

to be shocked whenever they drank with their BAC in excess of the prescribed limit. They were then requested to continue drinking, at which point random high-intensity (up to 8.2 mA) shocks were administered on an 80% delivery schedule. The actual punishment sequences lasted between 10 and 20 min (involving between three and 12 shocks, depending on the capacity of the subjects to withstand the stimulation). The shock trials were distributed over up to 12 sessions, with the early sessions spaced weekly and later sessions spaced fortnightly.

The findings reported by Lovibond and Caddy[63] were most impressive, with 89% of the 28 patients contacted at the end of 6 months showing either moderate improvement or complete success. At the 18th month of follow-up, 66% of the 27 subjects located also were deemed to qualify for inclusion in one of these two success categories.

The aversive procedures presented to this point have involved the imposition of legitimately aversive external stimuli to the alcoholic patient. We shall now discuss procedures in which the introduction of aversive stimuli is covert and under the cognitive control of the patient.

2.3. Covert Sensitization (Covert Aversion)

Covert sensitization[64–66] is based upon the premise that learning principles that influence overt behavior are applicable to covert behavior as well. A plethora of studies based on this premise has provided ample evidence that imagery and other covert processes can be manipulated to modify undesirable or maladaptive behaviors.[6,67–69]

Typically, in covert sensitization, the therapist generates a series of scenes that has been developed in collaboration with the patient. These scenes incorporate the problem behavior, relevant environmental stimuli, and the aversive consequences involved in engaging in the problem behavior. As the maladaptive behaviors to be treated by aversive procedures are generally well established, highly motivating, and intrinsically reinforcing, covert sensitization is frequently conducted in a systematic sequence. In the early stages of this sequence, the patient may be requested to imagine an extremely aversive scene together with a scene in which the problem behavior is only somewhat elicited by the ideational stimuli. As treatment progresses, the problem behavior is gradually more strongly presented.[71] It is crucial that the noxious aspects of the imagined aversive stimuli are consistently paired with the attractive aspects of the problematic behavior.

As applied to alcoholism, the procedure generally involves the pairing of nausea- or emesis-related scenes with images of the act of drinking[33,65,71–73] while the patient is deeply relaxed. The procedure, as initially developed by Cautela,[65] did not extend to the actual imaginary taste and ingestion of alcohol but was related only to preconsummatory responses. Anant,[71] however, emphasized the imaginary tasting of the alcoholic beverages. It is presumed that a sufficient number of presentations of such scenes will establish a conditioned aversion in response to stimuli related to alcohol and its consumption.

A sophisticated version of the covert sensitization procedure has recently been described by Elkins.[20] This procedure incorporates the monitoring of respiration, finger pulse volume (FPV), and the galvanic skin response (GSR) to provide objective evidence for nausea induction. Following screening and the collection of a history of alcohol consumption, Elkins' patients were instructed in deep relaxation using a modification of Jacobson's progressive relaxation technique.[74] Thereafter, base-line scenes were generated consisting of images in which preferred nonalcoholic and alcoholic beverages were consumed in natural settings. No nausea induction was attempted at this time. Subsequent to base-line imagery, covert sensitization was explained, and the patients were given a positive outcome expectation. The aversive procedure was then begun with emphasis being placed on the pairing of nausea suggestions with the desire for alcohol, the sight, smell, and taste of the beverage, the feel of the glass, and the sensations associated with ingestion of the drink. The suggestion of nausea was initially provided after imaginary swallowing and intensified up to the point of, but not including, emesis. Patients were instructed to signal both nausea onset and the intensity of nausea short of emesis. Physiological response monitoring aided in verifying the accuracy of the patients' signals. Signals of intense nausea were followed by suggestions of relief, at which time nausea reduction was paired with rejection of the alcoholic beverage, scenes of a favorite activity, or the consumption of a favored nonalcoholic beverage. Elkins[20] notes that all patients who participated in at least six covert sensitization sessions produced genuine nausea reactions.

Other researchers have used variations of this procedure[72,75–77] and have reported encouraging outcomes. Maletzky[78] treated alcoholics with covert sensitization and valeric acid, a malodorous fluid, which aided nausea development. Less positive results were reported by Wilson and Tracey[79] who found no differences between a group treated with an electrical aversion procedure and a covert sensitization group. However, these researchers used the method developed by Cautela,[65] which did not include the imaginary tasting of the drink. Thus, it may be that the negative results reported by Wilson and Tracey resulted from this less powerful method of conditioning aversion to alcohol ingestion.

An assessment of the efficacy and the attributes of the covert sensitization procedure and the other aversive approaches to the treatment of alcohol abuse and dependence is offered elsewhere in this text and in a variety of other reviews.[18,80,81]

3. Social Skills Training

Social skills training procedures have been used both as the primary treatment strategy in narrow-band approaches to the management of alcoholism and as one of a number of components in broad-spectrum program-

ming.[82–84] Their application to alcoholism arose from studies that indicated that alcoholics are deficient in appropriate interpersonal coping behaviors, from studies showing a positive relationship between degrees of social stress and alcohol ingestion, and from research demonstrating relapse to be a function of a lack of alternative behaviors during interpersonal stress. The two most common social skills training procedures directed toward reducing alcohol abuse are marital skills training and assertiveness training.

3.1. Marital Skills Training

Several theoretical orientations have formed the basis for marital (family) skills training. Some of these focus on alcoholism as the cause of marital difficulties, whereas others stress the view that alcohol abuse is the consequence of family problems. Although these etiologic considerations appear to be of little significance to the behavioral treatment of alcoholism, an understanding of the functional relationships that exist within the alcoholic's marital dyad and his/her family structure are viewed to be crucial.

Miller[85] described the treatment procedure administered to a 49-year-old alcoholic male and his spouse. This package included assertiveness training, increasing problem-solving skills, positive interaction skills, and behavioral contracting. Prior to beginning the therapy, Miller assessed his patients' interactions during interviews and during a 20-min nonstructured video taping session in which they conversed about both problem and nonproblem areas. Further interactions also were evaluated at home by audio taping mealtime conversations. On the basis of these assessments the following general goals were established: (1) to increase the couple's ability to express themselves more directly and to solve mutual problems more efficiently; (2) to increase positive interactional patterns; (3) to decrease conversations regarding negative incidents in the past; and (4) to provide each partner with positive skills needed to increase more desirable behaviors in the other. In addition, the wife specifically requested that her husband (1) abstain from all alcoholic beverages, (2) talk to her more frequently about his feelings, and (3) take her out to a restaurant and/or movie more often. In turn, the husband wished his wife to (1) reduce her nagging, (2) watch television with him on some evenings, and (3) engage in pleasant conversations with him.

The skills necessary to achieve these goals were then taught during conjoint sessions with both a male and a female counselor present. For example, the counselors modeled negotiations for a mutual agreement regarding disulfiram (Antabuse®) intake by the husband and cessation of nagging by the wife. In the presence of the couple, the counselors roleplayed this situation, demonstrated compromise and appropriate use of direct assertive problem solving skills, negotiated a written contract, and, subsequent to successful negotiations, used positive comments to reinforce the other partner. More positive adaptive marital skills were also taught via videotaped feedback and roleplaying together with feedback and social reinforcement from the coun-

selors. Simple instructions, periodic prompts, and behavioral rehearsal fostered these new patterns quite rapidly.

The couple continued treatment on a biweekly basis for 3 months and thereafter monthly for a further 3 months. Based on self-reports corroborated by the wife, the husband remained abstinent from alcohol for 9 posttreatment months. Further, the quality of the relationship between the couple markedly improved.

A similar case study is reported by Eisler *et al.*[86] In this case, an alcoholic husband was taught skills to enable him to cope with marital arguments that triggered his drinking. This type of procedure has been used as a component in a number of comprehensive behavior therapy programs for alcoholism.[87–89]

3.2. Assertiveness

Rimm and Masters[70] posit the following criteria for assertive behavior: (1) assertive behavior is interpersonal behavior involving the honest and relatively straightforward expression of thoughts and feelings; (2) assertive behavior is socially appropriate; and (3) the expression of assertive behavior takes the feelings and welfare of others into account. Assertiveness training includes any procedure aimed at enhancing an individual's ability to conduct him/herself in an appropriately assertive manner including the expression of both negative and positive feelings.

Assertiveness is presumed to benefit an individual by instilling in him/her a greater feeling of well-being and resulting in the achievement of significant social (as well as material) rewards, which ultimately should bring the attainment of more satisfaction from life.

The application of assertiveness training to alcoholism can be illustrated well in a case study reported by Eisler *et al.*[90] The patient was a 34-year-old divorced male with a history of abusive drinking. He had been abstinent for a number of months and had been promoted not long before the therapy began. His inability to deal with the responsibilities of this new position, because of his lack of assertiveness, resulted in his resumption of heavy drinking. In this case, short-term abstinence was reinforced by job satisfaction and promotion. However, long-term abstinence was threatened by his lack of social skills in interpersonal situations accompanying sobriety and occupational success.

Six typical work situations were chosen as the focus during assertiveness training. Prior to training, the patient participated in a videotaped roleplaying sequence to assess his skill in handling each of a series of situations. Training consisted of roleplaying scenes with the patient and the provision of specific instructions regarding possible assertive responses to be used in each of these situations. Sequentially, he was instructed to increase eye contact, decrease compliance, develop more appropriate voice tone and facial expressions, and to increase behavioral requests. While practicing these skills with the therapy staff, he was given feedback on the quality of his performance. Following

this training, the authors report their patient to have achieved success in employing alternative skills rather than responding by drinking.

Using a single-case experimental design, Foy *et al.*[91] have demonstrated the efficacy of assertive training using roleplayed situations to teach alcoholics to refuse drinking. Similarly, Miller[92] described the use of such training when the treatment goal was controlled social drinking rather than abstinence. Group outcome studies employing assertiveness training with alcoholics also have been conducted with promising results.[93–95]

4. Relaxation and Desensitization

4.1. Relaxation

Relaxation training as a treatment technique has been added to the armamentarium of procedures available for the treatment of alcoholism during the past 15 years. The rationale for its introduction was implied by the tension-reduction theory of alcohol abuse which assumes that alcohol consumption leads to a reduction of tension or stress and is, therefore, reinforcing to the drinker.[96]

Although it must be noted that the tension-reduction account of alcoholic drinking has received some damning criticism during the time in which relaxation procedures have been in vogue in the alcoholism literature,[97,98] there is evidence to support the view that many alcoholics drink aperiodically to alleviate anxiety and tension.[99,100] A further reason beyond that of tension reduction for considering the use of relaxation procedures in alcoholism therapy is that the use of the technique can serve as a delay tactic to block the path between the initial thought and the act of drinking.

Rohan[101] reported some success using relaxation procedures in the treatment of alcoholics. Methodological difficulties in this as well as other research studies, however, have made it impossible to separate the effects of relaxation from alternative explanations of treatment outcome. The few studies that have incorporated adequate control procedures have reported results that are modest at best.[102–106] In terms of specific procedures, relaxation training has encompassed a variety of techniques ranging from yoga and transcendental meditation through Jacobson's progressive muscle relaxation[74] to autogenic training.[107] Explanations of these approaches and their application are available in such detail elsewhere that their explicit procedures will not be presented herein (but see refs. 6,74,108–112).

4.2. Systematic Desensitization

In this procedure, relaxation is paired with imaginary scenes depicting stimuli that lead to anxiety or tension. The scenes are developed by the patient (usually 10 to 15 scenes are generated) and are then ordered hierarchically

using a Likert-type scale from least to most anxiety producing. The progression of the hierarchy can be thematic or spatial–temporal. As in covert sensitization, enough relevant stimuli must be included in these scenes in order to make them as realistic as possible.

The procedure is initiated with deep muscle relaxation. Once this is mastered, and the hierarchy has been developed, each scene is presented sequentially to the patient, beginning with the scene producing the least amount of anxiety. During the progression, the patient is required to signal the therapist at the first sign of anxiety, at which time the therapist interrupts the presented scene and returns the patient to a relaxed state. The next scene is introduced only when the patient has successfully visualized the previously presented stimulus situation without experiencing anxiety for a specific interval (usually 5–10 sec). One or two presentations of each scene usually are sufficient to remove the anxiety associated with the scene. Generally, no more than three scenes are presented per session. This avoids fatigue and rushing the patient through the scenes and insures that the patient is not reinforced for not signaling anxiety. The next treatment session is begun by presenting the last successfully completed scene.[70]

Systematic desensitization has been used successfully by a number of investigators to reduce or eliminate alcohol consumption.[113–116] In these studies, the patients commonly were diagnosed as alcoholic but only secondarily or concomitantly with one of a number of social or other anxiety states. The systematic desensitization procedure in each instance was directed primarily to reducing the patient's level of anxiety, which, when successfully accomplished, resulted in a marked decrease in alcoholic drinking.

In vivo desensitization procedures also have produced some encouraging results.[117,118] Hodgson and Rankin,[118] for example, presented a unique *in vivo* desensitization application in which a 43-year-old male alcoholic was administered daily doses of either 40 or 60 ml of vodka. Thereafter, their patient's subjective alcohol craving was monitored, and desensitization was directed at reducing his anxiety regarding these craving sensations. Such a procedure, it was hoped, would reduce the possibility that these sensations might trigger a relapse sequence.

Finally, desensitization and related techniques also have been applied to alleviate fears that are hypothesized to be linked with inability to cope with sobriety. Hall[119] has coined the term "abstinence phobia" to describe the phenomenon.

Outcome data from systematic desensitization procedures have been more encouraging than those from studies using relaxation alone. When used as part of a multimodal approach, desensitization may prove particularly valuable. In this regard, Lazarus[120] recommends that brief cognitive restructuring be introduced prior to the commencement of systematic desensitization. He is convincing in his argument and suggests that such an approach almost definitely contributes to the durability of the desired treatment effects.

5. Operant Methods

Operant treatment procedures, as related to alcohol abuse, are so named because they modify drinking responses by manipulating the consequences of those responses. They involve the contingent presentation and withdrawal of rewards and punishments in order to increase desirable behavior while decreasing undesirable behavior. Though seemingly superficial, operant procedures are quite complicated and sophisticated in that they require base-line measurement of specific behaviors deemed to need modification and the training of observers and contingency managers. They also require careful planning of the contingencies and procedures to be employed, and they require ongoing data collection, assessment, and follow-up.

Crucial to the planning of operantly based therapeutic strategies is the identification of stimuli or reinforcers that maintain the maladaptive behavior as well as the rewards (reinforcers) that may be manipulated to modify the behavior of the individual case. Contingency management is one of the most common applications of operant methodology.

Sulzer's[121] case study represents one of the earliest operant approaches to the management of alcohol abuse. Sulzer's patient was evaluated as being particularly concerned that continued drunkenness would lose him the companionship of two moderate drinking friends. Thus, under the direction of the therapist, it was agreed by all parties involved that the patient and his friends would meet daily for a few drinks. However, if the patient ordered or drank hard liquor, the friends were requested to leave him immediately. It was also stipulated that ongoing social interaction would occur in the homes of all three participants, but an abstinence requirement was applied in this instance.

Following the successful outcome of this case study, a number of other investigations involving contingency contracting have been reported.[122–125] These studies have demonstrated convincingly, at least under laboratory conditions, that the drinking practices of alcoholics can be brought under environmental control. Somewhat paradoxically, Bigelow *et al.*[126] demonstrated that even subsequent access to alcohol may be used as a reinforcing consequence following restricted drinking by alcoholics. These investigators noted that not one of their five inpatients drank above a criterion of 8 oz per day when alcohol on a subsequent day was made contingent on not exceeding this predetermined amount. In contrast, when no contingencies were attached to their drinking, all five patients drank to extreme intoxication.

Perhaps the most interesting of all the operant techniques is the "community reinforcement approach" developed by Hunt and Azrin.[87] This work is particularly important because the study extended the operant findings observed in the laboratory setting to the natural environment. Hunt and Azrin assigned 16 inpatient alcoholic subjects to two matched and essentially equal groups. The subjects in the experimental group received community rein-

forcement counseling along with the standard hospital treatment. Subjects in the control condition received the standard hospital care, which comprised milieu therapy, information about alcohol-related health risks, and counseling regarding interpersonal problems associated with continued drinking. Participation in AA also was available. The community reinforcement counseling, on the other hand, involved direct modification of the subjects' interpersonal and environmental support systems encompassing social, marital, and vocational interactions.

An experienced behavioral clinician aided in the setting of specific goals for each patient and helped each find employment and improvement in familial relationships. This clinician also aided in the structuring of reinforcing social activities for each subject. This effort, which began in the hospital, continued throughout a 6-month aftercare phase during which time frequent home visits were made in order to help the subjects strengthen the reinforcement value of naturally occurring reinforcers. At the 6-month follow-up, it was found that subjects in the experimental group had spent significantly less time drinking, unemployed, and away from home or institutionalized than had subjects in the control condition.

Although data from operant paradigm studies such as reported herein suggest that it is possible to modify the alcoholic's environment or influence the rewards that he/she derives from the environment, it also is clear that it is difficult to influence dramatically the alcoholic's drinking practices in the natural environment. We are still a long way from being able to specify and arrange all the relevant variables and control precisely the behavioral consequences for the drinker and those around him/her in such a way as to provide patients with long-term stability in a sober and reinforcing lifestyle.

An operant method also deserving of some attention is that of time-out. This procedure constitutes a highly generalized withdrawal contingency in which an individual is isolated from as much reinforcement as possible including contact with others and access to tangible items of reinforcing value.

Griffiths, Bigelow, and Liebson[127] conducted a research study in which inpatient alcoholics were given access to a specified amount of alcohol periodically during the day but were subjected to various time-out experiences that were imposed after each drink. Alcohol was available once every 40 min, following which there were restrictions placed on the particular subject's behavior. The conditions compared were: (1) no restrictions (base line), (2) a social time-out during which patients and ward staff could not talk, gesture, or play games with the subject, (3) an activity time-out during which staff and patients could talk to the subject, though he was confined to a particular chair in the day room and could engage in no activities other than smoking, and (4) a combined social and activity time-out.

The results of this study indicated that for the particular problem behavior of drinking, which has its own social ramifications, social time-out occasionally produced a slight reduction in subsequent alcohol consumption, although it

also sometimes actually raised it. On the other hand, activity times-out and the combined social–activity time-outs suppressed alcohol intake in the subjects participating in these procedures to 36 and 24%, respectively, of their base-line alcohol consumption.

6. Broad-Spectrum Behavioral Approaches

Although much of the earlier work in behaviorally based therapeutics was conducted from a narrow-band perspective that focused on a limited segment of behavior, present-day behavioral clinicians generally have moved toward a far more comprehensive view of the dynamics of clinical problems. With this movement has come the development of broad-spectrum approaches. Similarly, in the alcoholism field, the emergence of the multivariate perspectives[15,128] has led to greater recognition of the complexity of addictive behavior and has broadened the focus in alcoholism treatment. Broad-spectrum therapy is based on an integrated systems approach to the unique dynamics of the individual case and encourages multitechnique and multigoal intervention strategies.

Lazarus[82] was one of the earliest behavioral clinicians to propose a broad-spectrum behavioral approach to alcoholism. His treatment combined systematic desensitization with a variety of other behavioral techniques designed to modify both the alcoholic's drinking and the behavioral problems associated with this drinking. The procedure included medical care directed to the patient's alcohol-related physical disabilities, aversive procedures designed to change the patient's motivation to drink, assessment of the specific stimulus antecedents of anxiety (in order to enable construction of anxiety hierarchies for systematic desensitization), assertive training, behavioral rehearsal, and even hypnosis. A therapeutic relationship with the patient's spouse also was recommended to help her perceive and alter her role in the patient's alcoholism. This early commitment to a multicomponent behaviorally based therapeutic strategy foreshadowed a number of subsequent developments in the behavioral treatment of alcoholism.

The literature describing the emerging broad-spectrum behavioral approaches to alcoholism therapy is rapidly growing and is far beyond the capacity of the present chapter to review adequately. Within this body of literature are encompassed techniques involving behavioral self-control training,[70,129,130] BAC discrimination training,[61,63,131] the cognitive strategies which include stress inoculation[132] (R. Novaco, unpublished data, 1975), rational emotive therapy,[7,133–136] and thought stopping,[137] and those broad-spectrum therapies that incorporate a number of the aforestated procedures together with other behaviorally oriented techniques. Competent reviews of the various treatment techniques commonly encompassed within broad-spectrum programming may be found elsewhere.[18,81,138] For the purpose of brevity, we

limit the discussion of the issues within broad-spectrum behavioral programming to behavioral self-control training and to the presentation of an example of one broad-spectrum behavioral alcoholism treatment approach.

6.1. Self-Control Training

Behavioral self-control training has emerged in the behavior therapy literature largely as a consequence of the growing recognition of the role that cognitive processes play in all behavior change. Self-control training aims at providing clients with active coping strategies for dealing with problem situations. Thus, assertiveness training, which we presented earlier, and the cognitive therapies are examples of self-control techniques.

As far as the alcoholism literature is concerned, the most important contribution that behavioral self-control training has made to date is in the area of the restricted drinking research. Lovibond and Caddy[63] were among the first investigators to employ behavioral self-control training as a major component in the treatment of alcoholism. In this study, the component of self-regulation was seen as the fundamental philosophical basis of the entire procedure. It was introduced within a cognitive restructuring framework that demanded that the patient take full responsibility for his/her drinking and related behavior. Thus, in therapy, the patients were provoked to critically analyze their rationalizations and self-justifications with regard to drinking (and other matters) and were encouraged to undertake therapy in the role of students who had much to learn about their own cognitive and skills functioning and their attendant needs for specific change in both areas.

Other investigators also have included behavioral self-control training within their multifaceted inpatient or outpatient programs.[89,129,130,138–141] Typically, these programs also have explored a restricted drinking treatment goal, but with the exception of the work of Caddy and Lovibond,[142] little attempt has been made to tease out the relative contributions made to the overall treatment outcome by the behavioral self-control components.

Miller and his colleagues[143–147] reported the development of a comprehensive behavioral self-control package that included (1) determining the appropriate limits for alcohol consumption via an educational approach combined with specific BAC discrimination training; (2) self-monitoring of alcohol consumption; (3) rate control training, designed to alter the topography of the drinking behavior; (4) self-reinforcement to encourage the ongoing progress; (5) functional analysis of drinking behavior with training in stimulus control techniques; and (6) alternatives training, designed to teach coping skills to be used in situations in which alcohol previously had been used. Miller[145] compared the results of this package with two alternative approaches, an electrical aversive conditioning procedure and a multifaceted program incorporating techniques derived from Lovibond and Caddy[63] and Sobell and Sobell.[89] Miller reported no significant between-group differences during the course of a 12-month follow-up. The author noted, however, that the alter-

native treatment procedure consumed far more therapist time than the more economical behavioral self-control procedure.

In a second study, Miller, Gribskov, and Mortell[148] compared two different approaches to behavioral self-control training: a bibliotherapy (minimal therapist contact) condition and a paraprofessional therapist-administered self-control training program involving ten weekly sessions. Again, no significant differences were found between these two conditions.[106,149–151]

Given the relative lack of research in this area at this time, it is not clear whether the impressive results reported in the behavioral self-control literature are a function of the more mechanical aspects of behavioral self-control training, such as self-monitoring, or whether the potency of these techniques lies in their more basic cognitive components which act on individuals' belief systems regarding alcohol use and the extent to which one sees oneself as capable of overcoming the difficulties that have emerged with drinking.[152] Alternately, the positive effects reported may be a consequence of the interaction between these two general areas.

Irrespective of the primary locus of these positive effects, however, what is particularly striking about the behavioral self-control approach is the extent to which even those alcohol abusers who have been exposed to the traditional perspectives of alcoholism find themselves easily drawn to the learning perspectives that are communicated along with behavioral self-control training and the cognitive strategies generally. If such attraction proves to be more than simply a reflection of enthusiasm for a new approach or the demand characteristics associated with certain unique interviewing situations, it would seem highly likely that behavioral self-control training presented within a learning-based multivariate framework will prove most valuable in treatment generally and especially so in prevention and/or early intervention programs.

We now present a specific example of a multifaceted behavioral treatment program.

6.2. The Patton Study

Mills *et al.*[51] and Sobell and Sobell[89] reported an ambitious and most provocative broad-spectrum alcoholism treatment program which dealt directly with their patients' excessive drinking and emphasized the acquisition of alternative responses to stimulus conditions that previously functioned as having set the stage for heavy drinking. Seventy male gamma alcoholics underwent treatment over 17 sessions on an inpatient ward at Patton State Hospital. The procedure was individualized to reflect each patient's prior drinking history, his treatment goals, and his unique problems. Either abstinence or restricted drinking was available to patients of this program.

The general treatment plan was as follows:

1. Sessions 1 and 2 (videotaping). Subjects were permitted to drink until quite intoxicated. As they drank, they were engaged in discussions

with the staff about their views on alcoholism and themselves generally and on their expectations regarding the treatment in which they were engaged. These sessions were videotaped in their entirety.

2. Session 3 (treatment planning). During this session, the treatment plan proposed for each subject was presented, and an alcohol education sequence was begun.
3. Sessions 4 and 5 (videotape self-confrontation). Each patient's video recordings from sessions 1 and 2 were replayed to them to demonstrate just how inappropriate their drunken comportment really is and to increase their motivation to change.
4. Session 6 (failure experience). Twenty minutes prior to this session, the patients were required to complete a series of impossible tasks. Therapy during this session was focused on how each patient responded to such frustrations and how he dealt or failed to deal with the stresses in everyday life.
5. Sessions 7 to 16 (stimulus control). During portions of each of these sessions, patients assigned to the restricted drinking behavior therapy condition were subjected to aversive (shock) procedures when they exhibited "uncontrolled" drinking (as defined by Schaefer *et al.*[153] and reinforced with alcohol (up to predetermined limit) when they drank as "social drinkers." Behavior therapy subjects whose treatment goal was abstinence were shocked whenever they drank any alcohol. During these sessions also, patients were helped to identify crucial stimulus variables (stressors) associated with their individual decisions to drink, and they were aided via modeling and roleplaying to acquire effective responses which they could use in such stressful future situations.
6. Session 17 (summary and videotape contrast). Edited replays of drunken behavior taken from sessions 1 and 2 were contrasted with videotapes of sober functioning recorded during session 16. Progress during therapy and future applications of what had been learned were discussed, the intention of the investigators to remain in contact with each patient was indicated, and each patient was given a *do* and *don't* list specific to himself and to be kept in his wallet. Thereafter, the patients were discharged from the hospital.

In the most comprehensive and intensive follow-up study yet reported in the alcoholism literature, the drinking, social, personal, and vocational behavior of the Patton subjects was monitored over a period of 3 years. Overall, the findings from these follow-up and aftercare activities reflect positively on both the broad-spectrum behavior therapy approach to the treatment of even these quite depleted alcoholic patients and the possible merit of offering a restricted drinking alternative in the treatment of alcoholism.[89,139,154,155] Of course, the findings of this provocative study also point to the difficulties of treating the seriously advanced alcoholic patient and to the

limitations of our present ability to produce profound and durable changes even with a relatively intense and theoretically well-integrated behavioral treatment program.

Behavior therapeutic approaches to the treatment of alcohol abuse and dependency have come a long way since the early studies of Kantorovich and Voegtlin. These approaches have greatly influenced the alcoholism field but have not yet been accepted widely as the strategy of choice in the management of alcoholism. The biases of the present writers notwithstanding, it seems eminently clear that given the continued rate of growth of behavioral psychology generally and its likely continued growth in addressing addictive behavior, we will see continued rapid advances in the application of behavioral psychology to alcoholism. As this likely scenario unfolds, we can expect the behavioral approach to offer even more clearly than now an evaluation of its merit in the management of the broad range of drinking-related problems. Given such a perspective, scientists, clinicians, and the society in general will be then better able to determine the role that behavioral procedures will have, and the type of behavioral procedures that will be used, in the treatment of alcoholism.

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7

Outcome Studies on Techniques in Alcoholism Treatment

Gloria K. Litman and Anne Topham

Abstract. This chapter attempts to evaluate outcomes of treatment trials subsequent to 1975 which used behavioral techniques as the main treatment modalities. The results of trials using aversion therapy, contingency contracting, broad spectrum behavioral treatments, behavioral self-control and other cognitive therapies are critically evaluated and compared with both conventional treatment and other behavior therapies.

An overview of these trials indicates that where appropriately applied, behavioral treatment programs are at least as effective as more conventional treatment and in some cases their success has been strikingly high. Among the advantages of behavioral techniques is the fact that patients tend to stay in treatment longer and the use of paraprofessional and community resources tend to make these techniques economic in terms of professional time. Given these advantages of behavioral treatments, it is surprising that behavior therapy has not really affected the mainstream of alcoholism treatment to any great extent. The question of why behavior therapy has been confined mainly to isolated pockets of behavioral researchers and clinicians is also addressed.

1. Introduction

In a previous review,[1] we attempted to evaluate the outcome of treatment studies that used behavioral modification techniques and were carried out prior to 1975. The results of this evaluation indicated that behavioral techniques showed great promise in modifying alcohol dependence. However, the promise and enthusiasm for the possibilities of behavioral modification in the treatment of alcoholism were tempered by an equal concern about the standard of methodology used in these studies. As Nathan and Lipscombe[2] point out, the behavioral tradition brings with it a respect for empirical data and a search for validation of the data according to the accepted rules of scientific procedures. Although behaviorists are viewed by some of the more traditional workers in the field of alcoholism as experimenters who would do better to confine their inquiries to the animal laboratories, the scientific tra-

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dition which they have brought to the field of alcoholism requires others also to examine their findings and beliefs in the light of scientific scrutiny.

Our own concern regarding methodology was shared by Emrick,[3] who found that of the 384 studies he reviewed, only 72 randomly assigned patients to two or more treatments groups or matched them on important variables, and by Maisto and Cooper,[4] who looked at the inadequacies of treatment evaluation research. They pointed out that the nonrandom assignment of subjects to treatment conditions makes it impossible to determine whether outcome results from treatment or from pretreatment differences in groups which artificially influence the results. They attributed nonrandom assignment in many of the studies not to practical or ethical considerations on the part of the researchers but simply to poor design. Another basic methodological fault Maisto and Cooper found in many of the studies they reviewed was the failure to collect base-line data on pretreatment information on the subjects, so that posttreatment change cannot be compared with pretreatment behavior. A third source of faulty design lies in the improper or inadequate use of control groups. Finally, the presentation of the data may be contaminated by the high dropout rate of subjects who are not included in the final analyses.

In the previous review too, we examined learning theory and the techniques derived from various learning models and concluded that some of the theories that had evolved from experiments in tightly controlled laboratory conditions were not sufficient to explain such complex behavior as alcohol dependence, treatment outcome, or relapse subsequent to outcome.

This present chapter attempts to evaluate the outcome of those treatment trials subsequent to 1975 that used behavioral techniques. In the context of the criticisms outlined above, we concentrate mainly on those studies that used adequate designs or at least adequate controls, although some of the more recent techniques, which are still in the process of development, are mentioned. We concentrate on those studies that compared behavioral techniques to more traditional treatment programs and evaluate those treatment trials that compared the efficacy of one behavioral technique with another. In the previous review, there was a suggestion that a cognitive behavior model[4a] may be more useful in explaining successful outcome than the earlier classical conditioning stimulus–response model or the operant conditioning response–reinforcement model. Therefore, in this chapter we examine cognitive–behavioral models in the context of empirical findings from treatment trials in an attempt to assess the efficacy of these models.

Finally, we briefly address the question of the ways in which treatment may be affected by the political *Zeitgeist* of the times. During the late 1960s and early 1970s, there was an explosion of basic research and treatment trials based on behavioral models. Despite this, there was a relative dearth of treatment research in the behavioral area in the late 1970s. Some of the possible explanations for this failure of the continuation of such research are considered.

2. Outcome: Its Definition and Measurement

Before we consider the specific treatment trials to be reviewed, let us consider first what is meant by outcome, how it is measured, and how outcome rates affect the interpretation of the data.

Because there are different ways of calculating treatment outcome, the results of treatment studies may be inflated spuriously in several ways. The most obvious is to report the outcome at follow-up and deal only with those subjects who can be located. In fact, there is good evidence[5–7] that subjects who are difficult to locate tend also to be those who are unsuccessful. The differences in the resultant outcome figures are clear. In outcome studies that include only those cases who can be found at follow-up, the success rate is about 32% abstinent and 34% improved, averaging about 66%.[3] However, if all subjects are included, and those who cannot be located are assigned to the "not improved" category, then the success rates fall to between 12% and 45%, with an average of 26%.[7]

The Rand Report,[8] which has been criticized severely on methodological grounds but is nonetheless the most comprehensive survey of treatment outcome, found that at 18-month follow-up, 70% of patients followed up had been abstinent or drinking moderately. The results at 4 years was quite different. At 4-year follow-up, 56% of the subjects treated were rated as problem drinkers. The type of treatment was found to be irrelevant to outcome, but the length of treatment was a good predictor of outcome.

This discussion of outcome and spontaneous remission rates for various treatments is not an academic one in the context of this chapter. In order to evaluate the success or failure of behavioral techniques to modify alcohol dependence and other behaviors, the figures cited above provide a notion of what the base-line rates are for conventional treatment and provide the standard against which the outcome of behavioral treatment may be measured.

3. Aversion Therapy

Aversion therapy consists of procedures in which the individual is given either a noxious substance along with alcohol or an electric shock when alcohol is consumed. One of the models underlying this use of aversion therapy is the Pavlovian or classical conditioning paradigm in which a conditioned stimulus (CS), in this case the alcohol, is paired with an unconditioned stimulus (UCS), the noxious substance or electric shock, until a conditioned response (CR), nausea, vomiting, or pain, follows the administration of the alcohol alone. In this sense, classical conditioning is not contingent on the behavior of the individual undergoing the treatment. The temporal contiguity of the UCS and the CS is thought to be sufficient to produce the changes in the individual, which are presumably automatic and beyond the individual's cognitive control. The two-factor theory then goes on to postulate that during

this process, a conditioned anxiety is developed. If the individual avoids drinking the alcohol, then this conditioned anxiety is reduced. It is this reduction in anxiety that is presumed to be the reinforcer for subsequent avoidance behavior.

Obviously, these principles were recognized in early Roman times when Pliny suggested drowning eels in the wine of excessive drinkers. In modern clinical trials, the two main modalities of aversion therapy have been the use of electrical shock as the aversive condition or the use of chemicals to induce nausea and vomiting. In this section, we first consider the outcome of those trials using electrical aversion as the main treatment and examine the contribution that electrical aversion therapy may make to a broader-spectrum treatment program. We then go on to examine chemical aversion trials and, finally, attempt to compare the results of electrical aversion and chemical aversion.

3.1. Electrical Aversion

If we look at the outcome of earlier studies[9–23] in which electrical shock aversion was used as the main modality of treatment, it seems reasonable to conclude that electrical aversion is only minimally effective in the long term, although it may improve short-term results. When it does work, there is no evidence to indicate that when aversion therapy is effective, the classical conditioning model or the two-factor avoidance model is sufficient to account for the results. Some investigators[11,20,24,25] have reported that patients perceive differences in the qualities of the alcohol during or after aversion, although there has been no change in the physical attributes of the stimulus itself. The role that subjective devaluation of alcohol plays in successful treatment by electrical aversion would not be predicted by conditioning theory.

Lovibond and Caddy[26] report the case history of one patient who was devastated by the shock experience, not because of the shock *per se* but because the therapy brought home to her the fact that she had allowed her drinking problems to become so serious that such drastic treatment procedures were necessary. Because aversion therapy may have some impact on factors such as motivation, expectations, or other cognitive changes, it might be argued that electrical aversion therapy still has a role to play in a broader-based treatment program by serving the initial function of suppressing the deviant behavior, thus acting as a precursor to the more positive aspects of therapy, i.e., the initiation and maintenance of adaptive behaviors that are satisfactory competing alternative responses to excessive drinking. This hypothesis can be tested against three more recent studies.[27–29]

In an earlier study, Lovibond and Caddy[30] introduced the technique of discriminated aversion conditon as part of a broader-spectrum program. This technique was used to attempt to train alcoholic patients to discriminate their own blood alcohol levels by administering an electric shock when the levels exceeded 0.065%. The results of this study were confounded by the high

dropout rate in the control group, so that proper statistical comparisons could not be made. However, in a subsequent study,[31] 60 alcoholics were randomly assigned to one of three treatment groups: an aversion therapy plus self-regulation group, a self-regulation group that did not receive the shock component, and a group that received only discriminated electrical aversion without the self-regulation procedures. Immediately after treatment, the results indicated that the group receiving both aversion and self-regulation training showed the highest rate of improvement, with the group receiving self-regulation training without the shock showing similar but less spectacular results. The group that received discriminated electrical aversion showed the least improvement, with only 20% of this group in the highest success category. However, for the 37 patients who were follow up 12 months after treatment, 76% of the aversion therapy plus self-regulation group were considered either successful or improved, whereas 65% of those who were given self-regulation training but did not receive the shock were improved. These differences are not statistically significant. We can conclude from this trial that the addition of electric shock did not significantly enhance the results of the treatment program.

Vogler and his colleagues[28] also included electric shock procedures in their broad-spectrum treatment program and found their results similar to that of Caddy and Lovibond: the inclusion of electric shock produced marginally, but not significantly, better results than a program that included alcohol education and behavioral counseling only. When they applied these techniques to less seriously damaged problem drinkers, the results were similar. There was no greater improvement in the group receiving electrical aversion than there was in two other groups who did not have this component in their treatment program.

Comparing a behavioral counseling program with a more extensive program that included discriminant electrical aversion,[29] Miller found that there was a marginally superior outcome for the group that did not receive the electrical aversion when they were followed up 3 months after treatment. However, at 12 months, there was no significant difference between the two groups in terms of outcome, indicating that electrical aversion neither significantly enhanced nor diminished the results.

Electrical aversion originally seemed a reasonable procedure on the theoretical grounds of classical conditioning and the two-factor avoidance theory. It was also technically superior to other forms of aversion therapy since the techniques involved enabled greater precision in the control of the subjective pain threshold of the patient, the temporal contiguity between the application of shock and the particular behaviors to be modified, and they permit the use of partial reinforcement schedules which are powerful in shaping behavior in the laboratory. Although the earlier studies indicated that the theoretical basis was not demonstrated, there were cognitive mediational factors that came into play in those patients for whom electrical aversion was successful.

The later studies outlined above indicate strongly that there still remains

little evidence of the efficacy of electrical aversion as a treatment procedure, either as the main treatment or as a component of broader-based treatment programs. Elsewhere,[32] we have advocated the concept of "critical perceptual shift," the sudden, sometimes dramatic change in perception, motivation, and responsibility on the part of alcoholic patients as an important element in treatment success. If changes in expectation, motivation, perception, or other cognitive attitudes are produced in some patients by the use of electrical aversion as Caddy, among others, reports, it might be better to develop strategies for eliciting these changes in ways other than subjecting patients to the kind of pain and distress inherent in electrical aversion procedures. Both on ethical grounds and on the practical grounds that treatment efficacy has not been demonstrated, it is our opinion that electrical aversion should be discarded as a behavioral treatment.

3.2. Chemical Agents as the Noxious Stimulus

As we noted previously, the use of electric shock as the unconditioned stimulus enables greater precision and control of the conditioning procedures. In contrast, chemical agents present particular problems in the aversive control of behavior.

Disulfiram, a drug that leads to nausea, vomiting, tachycardia, marked drop in blood pressure, and other symptoms of massive autonomic arousal if followed by the ingestion of alcohol, is widely used as a treatment agent, with varying degrees of effectiveness.[33] Some mild conditioned aversion to alcohol consequent on disulfiram treatment has been reported. However, the process involved is not a matter of establishing associations between stimulus and response where none existed before but results as a direct pharmacological action of the drug in that disulfiram is said to block the action of a specific enzyme involved in the metabolism of alcohol[34] and to alter levels of certain chemical neurotransmitters in the brain.[35,36] One of the problems in the use of disulfiram therapy is motivational, i.e., insuring that patients who have been prescribed this course of treatment actually take their tablets. An interesting application of behavioral contracting to drug treatment has been reported by Bigelow *et al.*[37] Outpatient alcoholics were required to deposit funds with a clinic. They were repaid in small installments when they attended the clinic to receive disulfiram. These patients showed substantial and positive changes in decreasing their drinking during the course of their contracts.

Emetine and apomorphine as aversive stimuli are much more difficult to control than electrical shock. Because of variable and fluctuating differences in individual response, there is considerable difficulty in controlling the temporal contiguity between the administration of the drug and the onset of nausea. Franks[38] has also pointed out that these drugs may produce a central depressant effect that could interfere with conditioning. Because of the difficulty in controlling time factors, spurious conditioning may result. Bhakata[39] has also highlighted the considerable distress in using these drugs, and Hsu[11]

has cited the risk of such undesirable side effects as cardiac arrest and myocardial failure.

Despite these theoretical and practical considerations mitigating against the use of chemical aversion, reports of trials using these techniques have indicated surprisingly successful outcome in some cases. In 1940[40] and 1950,[41] the successful use of nausea-inducing drugs in the treatment of alcoholism was reported by Lemere and Voegtlin at the Shadel Hospital. Although emetine was used as the unconditioned stimulus, their patients were also given drugs to control serious side effects, oral saline to provide easily vomited stomach contents, and both injected an oral emetine. Just prior to the expected onset of nausea and vomiting, various forms of alcohol were smelled and tasted by the patients. The patients were also given large quantities of nonalcoholic beverages between conditioning sessions to maximize differentiation between the conditioned response to alcohol and their response to other liquids. Booster treatment was given at the patient's request and routinely at the end of 6 months and then after 1 year following treatment. Accurate follow-up data were available on 4096 of 4468 treated patients, a follow-up rate of 92%. Forty-four percent of these patients had remained totally abstinent since their first course of treatment. Of the patients who relapsed, 878 were treated. Of these, 39% were reported as sober since their last conditioning treatment. Sixty percent remained abstinent for 1 year, 51% for at least 2 years, 38% for at least 5 years, and 23% for at least 10 years after their first treatment.

Using similar techniques on a similar patient population, Wiens *et al.*[42] reported a 63% abstinence rate at 1-year follow-up on a group of 261 patients, 92% of whom were included in the follow-up sample. Patients who could not be located for follow-up were considered as failures for this study. More recently, Neuberger *et al.*[43] reported substantially lower rates of abstinence using these treatment procedures on a different population sample. They followed two groups of patients, 275 in one group, 290 in the other, and found that 1 year after treatment, the abstinence rates for the groups were 39% and 50%, respectively.

Because of their concern about the toxic effects of emetine in high doses, Baker and Cannon[44] advocate a more medically conservative procedure combining syrup of ipecac and low doses of emetine. They have demonstrated[45] that there is evidence of alcohol aversion following emetic therapy but not electric shock therapy, consistent with the findings of a large body of animal literature that suggests that taste–illness associations are readily learned, whereas taste–shock associations are not.

A recent treatment trial[46] examined the relative efficacy of chemical versus electrical aversion as components added to a multifaceted inpatient program. Twenty male alcoholic patients were assigned randomly to one of three groups: inpatient treatment only (six patients), inpatient treatment plus emetic therapy (seven patients), and inpatient treatment plus electrical aversion therapy (seven patients). In addition, subjects in both emetic and electrical aversion groups

were offered five booster sessions. Only one subject returned for all five booster sessions in the emetic aversion group, and none in the electrical aversion group. The results were reported in terms of the number of days the patients in each group were abstinent following treatment. At the 6-month follow-up, the patients in the emetic aversion group showed significantly more days abstinence than the electrical shock and control group combined. However, at the 12-month follow-up, there was no significant difference between the emetic group and the control group in number of days abstinent, although the patients in the electrical aversion group showed significantly fewer days abstinent. The authors suggest cautious interpretation of these results because of the small number of patients in each group. The results found in this study are comparable to those reported by Boland *et al.*[47] who found that at the 6-month follow-up, 36% of their emetic patients were abstinent compared with only 12% of controls. For the Cannon *et al.* work,[46] the results were 29% and 17%, respectively. Neither study is consistent with the results reported by the Shadel or Portland group.

When looking at the outcome of emetic aversion treatment, we are confronted with the problem of trying to reconcile two sets of apparently disparate results: this method seems extremely effective for very large patient populations for long periods of time in specific centers reporting mainly uncontrolled trials, whereas in controlled trials using very small patient populations, the addition of chemical aversion seems only marginally to enhance an enriched inpatient treatment program. One of the main factors in the differences between those conflicting results seems to be the type of patient population treated.

It has been stressed repeatedly by the Shadel and Portland workers that patient characteristics are extremely important in successful treatment with emetic aversion conditioning. The treatment at these centers requires a substantial investment on the part of the patient in terms of fees for treatment and in terms of time. Therefore, their patient population, and particularly the most successful ones, tend to be married, highly educated, of high socioeconomic status, and relatively intact. One of the conclusions we can come to is that emetic aversion therapy in these centers is extremely successful for those patients whose prognosis is already particularly good.

In 1950, Lemere and Voegtlin[41] reported discouraging results with 100 patients who were "charity cases" and were not paying fees for treatment. Length of drinking history also seems an important factor, since their successful patients seem to have a longer drinking history irrespective of age, although Lemere[48] reported poor results with patients less than 30 years old.

The contribution of patient motivation to the success rate should also be considered as a factor in successful outcome. Because of the substantial investment of time and money, and because of the extreme unpleasantness of the treatment, patients completing this program would need to be highly motivated indeed to change their drinking behavior. In line with this, abstinence rates were positively related to the number of booster sessions the

patients attended, and periodic reconditioning sessions are important in maintaining the effectiveness of this treatment. As noted above, although the Cannon *et al.*[46] patients were offered booster reconditioning sessions, very few actually attended them.

Although Cannon and Baker[45] have demonstrated that taste–illness associations are more readily produced than taste–shock associations, it is likely that factors other than simple conditioning can account for the results found by the Shadel and Portland group.[40–42] In terms of our examinations of cognitive mediational factors in conditioning procedures, in addition to the motivation of the patients, it is of special interest that the suggestive effects of the conditioning sessions, the therapeutic attitude of the staff, the group support, and group cohesiveness of the patients were noted as important adjuncts to the aversive treatment. Then too, there was a concerted therapeutic commitment to the vocational, social, and recreational rehabilitation of the patients, suggesting the later community reinforcement programs of Hunt and Azrin[49] and Azrin.[50]

The data suggest strongly that with a group of highly motivated, well-educated, fee-paying middle-class patients, emetic aversion therapy along with a therapeutic milieu and community reinforcement are extremely effective in modifying excessive drinking.

3.3. Verbal Aversion Techniques: Covert Sensitization

The method of covert sensitization was introduced by Cautela.[51–53] In this procedure, the noxious stimulus is aversive verbal imagery rather than electrical shock or chemical agents. The patient is first taught relaxation, usually in accordance with Jacobson's[54] procedure, and then asked to visualize very clearly the stimulus (i.e., alcohol) and scenes involving alcohol. As each scene is visualized, the patient is instructed in very graphic and explicit terms to imagine step by step the onset of violent nausea and vomiting, so that scenes involving alcohol and nausea become strongly associated. The patient is usually instructed to practice these sequences between treatment sessions, a form of cognitive rehearsal advocated by Bandura[55] to involve cognitive events in the mediation of adverse physiological reactions.

There have been several case studies involving covert sensitization,[56–58] with reports of abstinence ranging from 6 months to 23 months following treatment by this procedure. However, there have been few controlled trials to attempt to assess the efficacy of this technique.

Ashem and Donner[59] investigated the relative efficacy of covert sensitization, backward conditioning, and no treatment (patients assigned to a waiting list) on a group of 23 male alcoholics whose average drinking history was 18 years, each of whom had been in some kind of treatment for alcoholism previously. The results indicated that the backward conditioning group, consisting of seven patients, were not receiving pseudoconditioning as originally thought but had quickly made an association between alcohol and nausea,

as did the covert sensitization group, which consisted of eight patients. Because of this, the two groups were combined for comparison with the control group of eight patients. At 6-month follow-up, six of the 15 treated patients, but none of the controls, had stopped drinking.

An investigation by Fleiger and Zingle[60] attempted to test the efficacy of covert sensitization in the treatment of alcoholism against a "problem-solving" approach. The sample consisted of 32 patients, 15 of whom were assigned to covert sensitization and the remaining 17 to problem-solving treatment, the standard form of therapy used at that institution. Eight staff counsellors intensively trained in covert sensitization conducted the verbal aversion treatment. This treatment was administered on an individual basis, whereas the problem-solving technique was conducted in small groups. Both groups had 40 1-hr sessions over a 20-day period, and the goal of treatment for both groups was total abstinence.

The results indicated no differences between the two groups on 3-month follow-up in terms of abstinence. However, the authors note that no provision was made for rehearsal for the covert sensitization patients after release from treatment. There was also some indication that female therapists tended to be more effective in covert sensitization than males, a trend that also appeared in the Ashem and Donner[59] results.

The results of 22 subjects who had received covert sensitization in addition to an alcoholism rehabilitation program were reported by Elkins and Murdock[61] who measured autonomic responses and used behavioral observations and subjective reports to determine the onset and the intensity of the nausea experienced by their subjects. These measures seemed to discriminate those subjects who become nauseous in response to images of alcohol alone (conditioned nausea) from those who become nauseous only while imagining feeling ill and vomiting (demand nausea). Elkins and Murdock[61] reported that 22 out of 24 alcoholic patients were able to develop demand nausea, and of these, 15 developed conditioned nausea to images of alcohol alone. The patients in this sample who were able to develop conditioned nausea remained abstinent for a mean of 14.9 months, whereas those who had developed only demand nausea remained abstinent for only 3.7 months. In a further study,[62] about 31% of the conditioned nausea group were found to be abstinent for from 5 to 62 months, whereas none of the other group had remained abstinent. However, when controlled drinking criteria were applied as outcome measures, 11 of the 13 conditioned and five of the six demand nausea patients were not abusing alcohol. Maletzky[63] reported the results of using covert sensitization assisted with valeric acid, a foul-smelling fluid. Military patients were randomly assigned either to halfway house treatment or covert sensitization with valeric acid. At 6 months, the covert sensitization group reported fewer urges to drink, consumed fewer drinks, and had been on fewer reports for drunken behavior than the controls.

In another treatment trial, however, no significant differences in treat-

ment outcome were reported[64] when covert sensitization was compared with insight therapy or systematic desensitization.

There are advantages to using covert sensitization as a form of aversion therapy in that it does not require elaborate equipment, is less traumatic than electrical aversion, and does not involve the possible serious consequences to the health of the patients that chemical aversion techniques do. Since covert sensitization involves imaginal stimuli, no external stimuli need be present. In addition, the locus of control of the treatment is eventually given over to the patient, so that at least theoretically they are taught a technique that may be practiced and used outside the treatment situation and thus may be self-administered.

However, the findings from controlled trials are really not sufficient to assess the efficacy of covert sensitization either alone or in conjunction with other treatments. The assumption by Elkins that a conditioned response was created in some of his patients seems at variance with the findings that the sex of the therapist influenced the outcome of treatment, suggesting that there were other factors operating in the sessions. To our knowledge, there have been no trials involving booster sessions as in electrical and chemical conditioning trials. The powerful visual imagery that is required may be difficult for some patients to create at will, and even greater difficulty may be experienced in achieving the appropriate intensity of the emotional response required. It may be even more difficult to maintain the visual imagery and emotional response over time. Before covert sensitization is dismissed as a technique, more research is required into the type of patient for whom this technique may be appropriate and the contribution of booster sessions to outcome. A most interesting and consistent finding is the achievement of controlled drinking, or at least the nonabuse of alcohol, subsequent to covert sensitization training. Randomly assigning subjects to treatment goals other than abstinence may provide some interesting outcome measures.

4. The Application of Operant Procedures to Alcoholism: Contingency Management and Contingency Contracting

Contingency management procedures refer to the application of the laboratory findings of instrumental conditioning and operant behavior to clinical problems. They are based on the assumption that the consequences of any given behavior govern the probability of emission of that behavior. There are two prerequisites for the application of these techniques. The first is to identify the target behavior to be controlled. The second is to find effective reinforcers that are sufficiently powerful not only to modify the target behavior but also to diminish the value of those reinforcers that are maintaining that behavior. It may also be necessary to identify the reinforcers that are maintaining the undesirable behavior. In essence, this is a scientific reiteration of the simple,

but apparently correct, principle that people will act in such a way as to maximize the rewards and minimize either the punishments or loss of rewards in their lives. This is what makes the continuation of excessive drinking so difficult to understand. Well before they come into treatment, alcoholics have usually been subject to powerful contingencies associated with their drinking behavior. Yet is is surprising how ineffective such serious consequences as marital breakdown, loss of job, social isolation, and even imprisonment can be in the modification of alcohol abuse.

In the laboratory, operant procedures do not usually require the specification of expected changes in behavior before the act. It is assumed that the available reinforcers are so powerful they will effect the required changes. However, as we have noted, outside the laboratory, powerful reinforcers, both positive and negative, are not always sufficient to effect changes in so dominant a behavior as alcohol abuse, and thus other procedures have to be brought in.

We briefly review the laboratory investigations that served as a basis for later treatment trials and then go on to consider some of the more interesting and effective applications of contingency management and contracting to the treatment of alcoholism.

There have been several research programs that have investigated the extent to which environmental contingencies may affect drinking behavior in alcoholics. The early work of Mello and Mendelson[65–67] challenged some of the traditional notions of craving and loss of control in chronic alcoholics. Cohen and her colleagues in Baltimore[68–70] set about to investigate systematically the manipulation of environmental contingencies and their impact on drinking behavior. Based on their conclusion that the kind of attention given to the alcoholic by family, hospital personnel, or even the police following a drinking episode all contributed to the maintenance of drinking behavior, they designed experiments to determine if reversing contingencies for drinking would alter this behavior. Instead of being reinforced with attention for excessive drinking, alcoholics would be rewarded with a variety of environmental and social privileges when they drank moderately, but excessive drinking would result in the removal of these reinforcers. They were able to demonstrate that chronic alcoholic patients will voluntarily moderate their drinking when moderate drinking is reinforced and when excessive drinking results either in punishment or in loss of privileges. Other laboratory studies by Nathan and his colleagues[71–73] and Gottheil and his group[74–77] have also demonstrated convincingly that under laboratory conditions the drinking behavior of alcoholics can be brought under environmental control. These contingency management procedures proved to be remarkably effective in moderating drinking behavior within an institutional setting.

In a previous review,[1] our main concern was the successful extrapolation of these procedures to noninstitutional settings. Contingencies must be applied consistently and repeatedly in order to be effective. Perhaps this is one of the explanations for the failure of powerful contingencies in their life sit-

uations to affect alcoholics' drinking behavior. Spouses, employers, and others rarely adhere to the contingencies as rigorously or consistently as they should for maximum effectiveness. The application of reinforcement procedures to the world outside the institution seems a difficult task, since in real life settings, consistency could seldom be maintained over a sufficiently wide range of activities. However, as Cohen *et al.*[78] pointed out, some of the agencies controlling reinforcers to alcoholics in the outside world—family, employer, and medical, welfare, and rehabilitation services—dispense reinforcements such as money, shelter, medical care, attention, and sympathy contingent only on drinking and its end-stage consequences. Although sobriety may also be reinforced, moderation seldom is. They suggested that the reinforcers cited above may be manipulated as contingencies for moderation as well as abstinence.

Although single case studies have been reported,[79,80] the most interesting application of contingency management procedures, foreshadowed by the suggestions of Cohen, has been the "community reinforcement" program developed by Hunt and Azrin.[49] Sixteen alcoholics were assigned to two groups. The patients in the control group received the standard hospital care, which comprised milieu therapy, alcohol education with emphasis on health risks, participation in Alcoholics Anonymous, and counselling for interpersonal problems associated with continued drinking. In the experimental group, the subjects were given specific behavioral training focused on the improvement of longstanding vocational, interpersonal, and family problems. Role playing, behavioral rehearsal, and cognitive restructuring of attitudes and beliefs about the appropriateness and effectiveness of certain behaviors were among the techniques employed.

In addition, an experienced behavioral clinician contracted with each patient in setting specific goals, helped him to find a job (and trained him in letter writing and interview behavior), aided in restructuring reinforcing social activities, and was involved in improving family relationships. Once the patient could deal more effectively with family, job, and friends and experienced these as reinforcing, these new-found reinforcers were incorporated into a contingency management program. A 6-month aftercare phase was introduced, whereby frequent home visits were made in order to help the alcoholics strengthen the value of these naturally occurring reinforcers; access to hospital procedures, etc. were all made contingent on sobriety. At a 6-month follow-up, those patients who had gone through the community reinforcement program had spent significantly less time in drinking, unemployment, or institutional care than those in the control group who had received the standard hospital care.

Subsequent modifications of this approach were reported by Azrin.[50] The basic format was retained along with the following additions: disulfiram was given to all experimental clients to reduce the likelihood of impulsive drinking which would dilute the effectiveness of the contingency contract. Prior to the dispensing of the drug, behavioral efforts were made to teach clients to view

the use of disulfiram as a positive step; an "early warning" system of clients drinking or other problems was introduced, whereby clients, family, friends, and employers reported regularly to the counselor; a neighborhood "buddy" was elected and trained for peer counseling for continuing social support before and after professional counseling stopped; and to reduce the amount of expensive professional time involved, groups were set up to include two to four clients, their peer–counselors, and spouses.

These efforts to enhance the effectiveness of the community reinforcement program and to extend its therapeutic impact beyond the formal treatment program at reduced cost in professional time yielded strikingly positive outcome data. The ten experimental patients showed significantly more improvement along a variety of dimensions, including drinking behavior, than did the ten patients who had received hospital care.

Contingency contracting has also proved successful with a group of debilitated and chronic alcoholics who were selected from a group of "public drunkeness offenders" while they were still in prison. Miller[81] contracted with ten of these men to provide them with a broad range of goods and services in exchange for their demonstration of attempts to control their drinking when they were released from prison. Another ten men served as a control group and were given the same goods and services regardless of their subsequent drinking behavior. Special arrangements were made with the Salvation Army to house and feed the ten men in the experimental group, efforts to get them jobs were made, medical assistance was provided where required, clothing, cigarettes, and meals were obtainable with the use of canteen coupons at the Veterans Administration Hospital, and the subjects also received counseling sessions geared to advice on practical problems, including money management.

During the 2 months the contingencies were in force, the experimental subjects were tested for blood alcohol level at unpredictable intervals in their natural environment. The goods and services were withheld for 5 days if their blood alcohol level was found to exceed 10 mg/100 ml at any time during the 2 months. As in the Hunt and Azrin studies,[49,50] the results of this manipulation were strikingly positive. The ten control subjects showed no change in their behavior, whereas the subjects who received goods and services on a contingency basis significantly decreased their rate of drinking and mean number of arrests and significantly increased the time spent in employment per week.

It would seem from these results that contingency contracting may be effective with chronic skid-row alcoholics whose drinking behavior had been intractable previously and for whom other forms of treatment had proved ineffective.

Another form of contingency contracting has already been mentioned in connection with disulfiram, wherein Bigelow *et al.*[37] contracted with a group of outpatient alcoholics to report to the clinic to receive their disulfiram and in order to receive back small installments of money they had deposited with

the clinic. Again, substantial positive results were found in drinking behavior over the course of the contract. This same group[82] arranged to contract for abstinence with four hospital employees who were in danger of being dismissed for drinking on the job. These subjects were required to report daily to the Alcoholism Treatment Unit for disulfiram. Failure to report would result in no work and no pay. The results indicated marked improvement in all four employees' job performance and attendance.

Contingency management has been demonstrated to be remarkably effective in moderating alcoholics' behavior within the laboratory setting. Contingency contracting has proved to be a successful treatment with relatively intact alcoholics and with skid-row alcoholics whose condition seemed previously to be intractable. As we have pointed out elsewhere,[83] treatment in the short term may be effective, but it is the relapse subsequent to treatment that is of ultimate concern. Hunt, Azrin, and Miller have all indicated ways in which community resources may be used as powerful reinforcements to enable alcoholics to continue to moderate or terminate their drinking after hospitalization or imprisonment. Nathan and Lipscombe,[2] in their excellent review, point out too that in a very real sense Alcoholics Anonymous aims at the contingent management of alcoholics in that one of the most effective sources of therapeutic effectiveness is the social sanction exercised by members of the group in the form of disapproval.

In the beginning of this section, we pointed out that it is difficult to understand why alcoholics continue to drink when faced with the dire consequences this behavior involves. The effectiveness of contingency contracting sheds some light on this paradox. The strength of rewards and punishments to affect behavior is not reward or punishment *per se* but the individual's perception of their relative importance. Basic to the work on perception is that an organism responds not to some "pure" external stimulus (or stimuli) but to the stimulus as perceived. Mahoney[84] cites several examples in which such cognitive processes as perception, beliefs, and attitude have transformed stimuli, so that the simple assumption cannot even be made that any given set of stimuli is either reinforcing or aversive on the basis of the experimenter's standards. Therefore, although we speak of dire consequences and powerful punishments, these may not be perceived as such by the alcoholic or at least not as sufficiently powerful to dilute the rewards of their drinking behavior. The work on contingency contracting shows that a contingency is effective only when it is specifically articulated, based on mutual agreement between the patient and the clinician, carefully observed, and rigorously carried out.

5. Broad-Spectrum Behavioral Treatments

As we have seen, some of the early behavioral clinical trials concentrated on narrow techniques aimed at modifying only the excessive drinking behavior. In 1965, Lazarus[85] published a conceptual paper in which he suggested

that a "broad-spectrum" approach, which included a variety of behavioral techniques to change interpersonal and intrapersonal behavior as well as drinking, might be far more effective in the treatment of alcoholism. In the 1970s and 1980s, there have been several treatment programs that have used broad-spectrum techniques. For the purposes of this review, we consider only those that have been evaluated sufficiently to enable us to draw conclusions with regard to their efficacy.

In 1972, the Sobells[86] released a preliminary report on their IBTA (individualized behavior therapy for alcoholics) treatment package and have been the center of a maelstrom of controversy ever since. The controversy revolves around the issue of controlled drinking rather than abstinence as a goal of treatment for alcoholics. One of the findings of the Sobells' study was that at 6-month, 1-year, and 2-year follow-up, patients who had been through the IBTA program, regardless of their original treatment goal (i.e., abstinence or controlled drinking), were drinking in controlled fashion significantly more than the patients in the control group.

Forty male "blue collar, gamma" alcoholics were selected for controlled drinking trials from a group of alcoholics who had been hospitalized voluntarily and who had volunteered for the study. The patients were assigned at random to a controlled drinking experimental or a controlled drinking control group.

In addition, 30 patients were selected for abstinence trials and were randomly assigned to two groups, a nondrinker experimental group or a nondrinker control group. Subjects who could identify with Alcoholics Anonymous, had requested abstinence as a treatment goal, and/or were evaluated as lacking sufficient social support to maintain a controlled drinking pattern were always assigned to the abstinence group.

The treatment program consisted of 17 90-min daily sessions. Central emphasis was placed on defining prior setting events for excessive drinking and training the individual in alternative, socially acceptable responses to these situations. During each treatment session, except for five "probe" days, the experimental subjects were also given aversion training contingent on appropriate drinking behaviors, i.e., diluting, sipping, spacing, and stopping after three drinks. In addition, there were three noncontingent sessions consisting of videotape self confrontations, one when drunk, the other sober, and two "stimulus control" sessions in which shock contingencies were not in effect. An education session was included early in the program to inform the patient of the treatment program, to advise him of the contingencies under which shock would be administered, and to give him an explanation of the treatment rationale of drinking as learned behavior, occuring under certain stimulus conditions and not others, and controlled by its consequences. In addition, social skills and assertiveness training were given, particularly geared towards training the subjects to resist social pressures to continue drinking. One session consisting of an "artificial failure" experience was also included.

Control subjects received conventional hospital treatment which included group therapy, AA meetings, chemotherapy, psychotherapy, etc.

Contact was maintained with subjects and their cohorts every 3 to 4 weeks after treatment. Subjects were classified as abstinent, controlled, or drunk based on defined criteria in amounts drunk on 6-month, 12-month, 18-month, and 1-year follow-up.

At the 1-year follow-up,[87] experimental subjects in both the controlled drinking and abstinence groups were functioning significantly better than the control group in terms of drinking, social stability, vocation, physical health, etc. Two-year follow-up[88] indicated that of the 19 experimental controlled drinking subjects located, approximately 92% were abstinent and/or controlled drinkers. The 2-year data on the abstinence groups[88] indicated that of the 13 experimental subjects located, approximately 63% were abstinent, and a further 8% were controlled drinkers. An independent 3-year follow-up[89] with 49 of the original subjects provided evidence that subjects in both the abstinence and controlled drinking IBTA experimental groups continued to function better than control subjects who had received conventional hospital treatment.

Readers who are interested in controversial issues surrounding this study are referred to criticisms by Emrick,[90] Hamburg,[91] Nathan and Briddell,[92] and Pendery *et al.*[92a] As the results stand now, the IBTA program has proved far more successful than did conventional hospital treatment. Although the results of this study are impressive, and the procedures warrant further attention, so many procedures were involved that it is difficult to sort out the relative contribution of each, but "educated" guesses can be made from the results reported in the Sobells' book[93] and from other reports from this group. It is the contention of the investigators that the stimulus control sessions not only constituted the majority of the treatment sessions but were also the primary factors responsible for satisfactory results. In other words, the patients were trained individually to a whole new repertoire of social behaviors antithetical to continued alcohol abuse. With the option of controlled drinking rather than abstinence as a treatment goal and the emphasis on alcoholism as a learned behavior, the patients were given new perceptions and beliefs about their problem. The stimulus control sessions afforded a means of translating changes in perception and beliefs into changes in their behavior. Also, the follow-up program would almost certainly have been instrumental in maintaining treatment gains. Although official follow-up was every 6 months, apparently patients were contacted every 2 or 3 weeks and thus had low cost continuing care after treatment.

The "integrated behavioral change techniques" described by Vogler and his colleagues[28,94,95] incorporate some of the elements used in the Sobells' treatment package, although the sequencing of the elements differed. The success of this package was tested by comparing chronic hospitalized alcoholics treated by integrated behavioral change techniques with a control group

who were given alternative training, behavioral counseling, and alcohol education. Although the group given the entire package was treated for approximately twice the length of time of the comparison group, the results at 1-year follow-up indicated that the two groups differed significantly in the amount of ethanol consumed, but there were no significant differences between them on other outcome criteria. However, it should be noted that 65% of the patients in both groups met the criteria for success.

These techniques were then applied to four groups of problem drinkers who had never been diagnosed as alcoholic or been hospitalized for alcohol-related problems but were consuming sufficient alcohol to produce legal, vocational, and/or marital problems.[95] One group (23 patients) received the entire integrated behavioral change techniques program; a second group (19 patients) was given blood alcohol level discrimination training, behavioral counseling, alternatives training, and alcohol education; a further 21 received only alcohol education; and 17 received alcohol education, behavioral counseling, and alternatives training. The goal for all 80 subjects was moderation rather than abstinence. At 1-year follow-up, 50 of the 80 subjects completing treatment and follow-up were considered moderate drinkers, and three had remained abstinent. Overall, the subjects had decreased their ethanol consumption by between 50 and 65%. However, there was no significant difference among the groups on any outcome variables. The specialized behavioral techniques given to the patients in the first and second groups did not significantly increase the effectiveness of the treatment. Although the overall results obtained by Vogler and his colleagues are impressive, the failure to find significant differences in treatment impact raises grave doubts about the cost effectiveness of such an expensive and labor-intensive broad-spectrum program.

A program designed to moderate the drinking of middle-class, well-motivated, socially, psychologically, and vocationally intact problem drinkers was carried out by Pomerleau and his colleagues.[96] Of the 32 subjects selected for treatment, 18 were randomly assigned to behavioral treatment, and 14 to traditional treatment. Both treatments were conducted weekly in small groups for 3 months. Five additional sessions were scheduled at increasing intervals for 9 months after treatment. Patients in the behavioral treatment group were required to pay for their treatment in advance and to pay a $300 "commitment fee" which could be earned back contingent on their rigorously adhering to treatment instructions, regular attendance, commitment to follow-up procedures, etc. The patients in the traditional treatment group were not required to do so. Perhaps as a result, of the 18 patients assigned to the behavioral treatment group, only two failed to complete treatment, whereas only eight of the 14 patients in the traditional treatment group remained in treatment for the full course. Another factor in keeping patients in the behavioral treatment program may have been that they were given a choice of goals, i.e., abstinence or controlled drinking, whereas the patients in the traditional treatment group were given the goal of abstinence only.

Miller and Hester,[97] in their exhaustive review, have summarized in table form (pp. 82–85) the results of 19 studies that used broad-spectrum treatment approaches. Of these, at least 15 met the criteria for adequate research design that were discussed at the beginning of this chapter: the use of proper comparison groups, random assignment of patients to treatment, specified criteria for outcome, and the use of corroborative data sources as well as patients' reports on outcome.

In terms of successful outcome criteria for reduction of ethanol consumption and other measures, the Sobells' work[86–88,93] has shown the most consistent findings of superior outcome over a conventional treatment program with follow-up at 3 years. Caddy *et al.*[98] and Alden[99] have found a multimodal treatment program to be more effective than behavioral self-control training, which is discussed below. Caddy and Lovibond[31] reported that their complete package was more successful than two alternative narrower versions of the same program. On the other hand, as in the studies discussed above, the major advantage of broad-spectrum behavioral treatment approaches seems to be to reduce the dropout rate of patients in treatment. However, although the results of these programs have been strikingly successful, the cost effectiveness of these broad-spectrum programs over less intensive intervention has not been demonstrated. Much more streamlined programs could be designed, incorporating the most useful elements of these programs and discarding techniques such as aversion therapy and others that contribute little if anything to the overall success.

6. Behavioral and Self-Control Training and Other Cognitive Therapies

The underlying assumption of cognitive behavior therapy is that both behavior and emotions are governed by perceptions, beliefs, attitudes, and other thought processes. Thus, cognition is presumed to be the crucial process in affecting emotion and behavior, rather than *vice versa*. Beck's seminal work on depression and other affective disorders,[100] Ellis' rational–emotional therapy,[101] and Mahoney's application of cognition to behavior modification[102] have all provided conceptual bases for the development of techniques designed to lead to cognitive behavioral change. In this section, we review the recent application of these techniques to the modification of excessive drinking.

Although other programs have incorporated some of these techniques,[27,86] the most comprehensive program focused on cognitive behavioral change has been that developed by Miller and his colleagues.[91,103–107] Their program includes goal setting, specific information about the effects of varying levels of alcohol consumption on the body and on behavior, external-cue blood alcohol concentration training,[106a,107a] self-monitoring by the client of alcohol consumption, training to teach diluting drinks, sipping, spacing, and stopping,[86]

training in stimulus control procedure, and skill training designed to teach coping skills other than use of alcohol. This program has been evaluated in a series of trials. No differences in effectiveness were found between the results of this program and complex broad-spectrum programs incorporating techniques from Lovibond and Caddy[26,30] or the Sobells' program[87] of electrical aversion therapy.[104] Miller notes that the broad-spectrum programs involved at least three times as much therapist contact as did the behavioral self-control training program.

Comparing two different approaches to behavioral self-control training, Miller and his colleagues[108] found no significant differences in 3-month outcome between a bibliotherapy condition that required minimal therapist contact and a ten-weekly-session behavioral self-control program administered by a paraprofessional. In the most recent reports from this group,[109] a comparison between bibliotherapy with a behavioral self-control orientation and two more extensive broad-spectrum behavior therapy programs showed no statistically significant outcome differences between groups. The outcome results of Miller's program have been very impressive, since about 70% of the patients assigned to the behavioral self-control training program made significant improvement in the studies reported. As mentioned previously, Alden[99] also found a success rate of 70% in the problem drinkers she treated using a program similar to Miller's. However, she reported finding significant differences between her broad-spectrum multifaceted program and her behavioral self-control training package.

Brandsma et al.[110] compared the outcome of 104 alcoholics who were randomly assigned to a rational behavior therapy group, an insight therapy group, an Alcoholics Anonymous group, or a no-treatment control. The results on 12-month follow-up indicated that patients in all three groups had improved more than subjects in the control group. Those receiving rational behavior therapy, who had received treatment from either professionals or paraprofessionals, were at least as successful as those receiving insight therapy from a highly experienced professional.

McCourt and Glantz[111] report case studies of four male alcoholics in which attempts were made to change maladaptive thought processes similar to those found by Beck[100] in depressives. The therapy benefited all four patients in terms of drinking behavior at 1-year follow-up. However, it did not benefit all subsequent patients who participated in the therapy as part of an inpatient program consisting of groups of eight patients meeting twice weekly for 10 weeks. The authors are unable to specify which patients would be helped by this form of treatment.

Litman and her colleagues[112] found that cognitive control as a coping behavior was the strongest discriminant between relapsers and survivors in the group she studied, and Sanchez-Craig[113–115] has described methods for teaching coping skills that included cognitive restructuring and covert rehearsal of coping behaviors. Finally, Marlatt[116] has proposed a method of alcohol relapse prevention based on a cognitive behavioral therapy model.

The results of the behavioral self-control program of Miller and his colleagues have been impressive. However, to date, there is not yet evidence that the efficacy of this program results from cognitive restructuring, although as Caddy and Block (Chapter 6, this volume) note, it is striking that alcohol abusers who have been long exposed to traditional perspectives of alcoholism relate more easily to the learning perspectives that are communicated with behavioral self-control techniques and cognitive strategies in general. The success of the application of cognitive strategies to the treatment of depression and other affective disorders suggests that they may be useful in the treatment of alcoholism. However, since there have been so few controlled trials within the field of alcoholism, the promising results must be interpreted with caution.

7. Behavioral Techniques in the Treatment of Alcoholism: An Overview

In comparison with our previous review,[1] we now have a respectable body of research literature on the use of behavioral strategies for the treatment of alcoholism based on clinical trials whose methodology meets at least some acceptable standard of design, control group selection, defined outcome measures, and follow-up procedures to make evaluation possible. Many of the studies reviewed in this chapter have indicated that when appropriately applied, behavioral programs are at least as effective as more conventional forms of treatment.[18,31,40–42,49,50] A number of the successful outcome rates have been strikingly high.[28,31,40–42] Some of the gains of behavioral over more traditional treatment include the fact that patients tend to stay in treatment longer under behavioral regimes, and the high cost of professional time may be reduced by the use of paraprofessional and community resources.[49,50,81] In two of the studies,[95,96] behavioral techniques have been shown to be effective with individuals whose drinking is problematic, although they were not clinically labeled as alcoholics; this implies that these techniques may be used in secondary prevention.

However, the success of some behavioral techniques in the treatment of alcoholism does not invite complacency, since there are many questions yet to be answered. Much of the work has been done by small, enthusiastic, innovative teams, and the "experimenter effect" on their patients has yet to be evaluated. There is still no satisfactory data base from which to assess which of the ingredients of the more successful broad-spectrum treatment programs are more efficacious than others and how they should be programmed sequentially to attain optimum efficiency. Although there have been many references to "individualized" behavioral techniques, the systematic matching of patient to treatment has yet to be explored (see ref. 117).

With the current emphasis on cognitive mediators, cognitive therapies, and skill training, there seems to be an urgent need to assess the role of cognitive deficit in the institution and maintenance of cognitive behavioral

changes in alcoholic patients. Acker[118] has emphasized in his review that there are certain specific areas in which problem drinkers and alcoholics may be deficient in cognitive functioning. Aside from the gross memory and concentration deficits seen in individuals who have been drinking large amounts of alcohol over long periods of time, in others there may be more subtle deficits in memory and concentration as well as deficits in cognitive flexibility and perseveration.

To our knowledge, there has yet been no systematic exploration of how to teach coping skills effectively to an individual whose thinking has been either grossly or subtly impaired in certain areas, although Sanchez-Craig[114,119] has noted that most of her "halfway house" subjects failed to recall the coping skill strategy 1 month after treatment, although they were able to retain the information until the end of the program. She suggests that the simplicity and generalities of her coping skills program, originally designed to be its main advantages, in the end turned out to be disadvantages in that they were not sufficiently relevant. She suggests that in designing instruction techniques for clients whose cognitive abilities are limited, more attention should be paid to the use of modeling techniques and specific behavioral guidance. In studies of coping skill training,[120,121] it was found that the benefits of this training fall off after 3 months. Litman[83] has suggested that a hierarchy of coping behaviors from simple avoidance to more complex cognitive control be taught over a period of time to assure relevance and to promote retention. However, this remains to be investigated systematically.

Although, as we have seen, research into the application of behavioral techniques to the treatment of alcoholism has continued during the last 5 years, it has been confined mainly to isolated pockets of behavioral researchers and clinicians without really affecting the mainstream treatment of alcoholism treatment to any great extent. Compared to the earlier enthusiasm, excitement, and promise of the late 1960s and early 1970s, there has been a relative dearth of innovation, breadth of ideas, and depth of exploration. Why did the initial momentum not result in the wider application of behavioral treatment to the field of alcoholism, particularly since some of the outcomes reported are far superior to that of conventional treatment?

Most of the traditional regimens that are widely available are based on the assumption that alcoholism is a disease and are carried out by workers who adhere to this concept. These workers see behavioral researchers simply as experimenters whose emphasis on learning models is seen as treating the symptom and not the underlying pathology and whose emphasis on proper data collection is misunderstood in itself and seen as threatening to the old order. Both the contempt generated by learning models and the fear generated by assessment are conveyed not only to clients in the treatment centers but affect funding of behavioral research at the political level as well.

Although not all behavioral researchers espouse "controlled" drinking, there has been a fundamental linking of behavioral approaches and "controlled" or "nonproblem" drinking since the early 1960s. The concept of con-

trolled drinking is, of course, opposed to the concept of alcoholism as a disease. It would be difficult indeed to use hyperbole to describe the intensity of the emotional reaction of traditional workers to the idea that alcoholics could learn to control their drinking rather than remain abstinent. From the initial reception of the Davies' paper[122] to the recent maelstrom of controversy surrounding the latest Rand Report,[123] the hue and cry surrounding this issue still rage. However, it should also be noted that although speculations abound, there has been no definitive work by behavioral researchers as to the type of patients who may be more suitable for controlled drinking than abstinence goals. This lack of adequate delineation has resulted in behavioral researchers outlining patient characteristics for controlled drinking that can be seen more as placatory concessions to the establishment than as scientifically based observations. Thus, they themselves dilute the potential value of their contribution.

However, the positive contributions of behavioral researchers are continuing. The increasing sophistication and precision of methodology and treatment procedures will continue to address some of the questions we raised earlier in this section regarding optimum combinations of techniques sequenced for maximum effectiveness, the matching of patient to treatment and to treatment goal, and the continuing exploration of cognitive behavioral techniques and their application to the treatment of alcoholism.

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8

Contributions to Behavioral Treatment from Studies on Programmed Access to Alcohol

Glenn R. Caddy and Edward Gottheil

Abstract. Major developments in research involving the alcohol–alcoholic interaction are presented against a backdrop of the traditional perspective of alcoholism. Studies explaining the effects of alcohol, factors influencing drinking patterns, the significance of alcohol for the alcoholic, expectations regarding alcohol use and its affective and social consequences, the parameters of alcohol use, the question of control of alcoholism, and a number of treatment related issues are reviewed. It is concluded that much of this research is provocative and may recommend a reassessment of current thinking about the nature of alcohol abuse and dependence. Such a reassessment, however, is seen to be difficult to make, for the task likely will require a paradigmatic shift.

1. Introduction

The contribution of basic research to the study of alcohol dependence prior to 1960 has been characterized by Mendelson[1] in the following terms: "Our heritage from years of scientific neglect is profound ignorance concerning even the basic behavioral and biosocial concomitants of alcoholism" (p. 1681). Concern about the lack of fundamental scientific knowledge was also apparent in the dry humor underlying the proposition offered by Keller[2] following his review of the literature on the defining characteristics of the alcoholic. According to Keller, "The investigation of any trait in alcoholics will show that they have either more or less of it" (p. 1147).

It was not that alcoholism failed to receive any research attention prior to 1960 but that few of the studies that had been undertaken before the pioneering work of Mendelson attempted to explore the basic dynamics of the relationship between alcohol and the alcoholic or to investigate the antecedents, correlates, or effects of observed drinking by alcoholics under sys-

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tematic, controlled conditions. Typically, the studies that were conducted involved either hospital studies of alcoholics in the absence of alcohol or laboratory studies of alcohol with the alcoholic absent. The lack of studies involving direct scrutiny of alcoholic drinking and the processes associated with it was probably a reflection of the *Zeitgeist*, the prevalent models of alcoholism, and the scarcity of professionals and scientists interested in the area of alcoholism.

2. Emergence of the Disease Concept

In the early part of this century, a group of physicians began to lobby for the proposition that alcoholism was a disease and should be treated accordingly. This proposition, however, was met with considerable criticism and did not gain public recognition.[3] By 1960, though, with the support of a paradigmatic change that had been building since the founding of Alcoholics Anonymous in the 1930s, a similar movement influenced greatly by the thinking of Jellinek[4] came to the fore in both professional and lay circles.[5,6] Interestingly, the sociopolitical changes that led to the development and acceptance of the disease concept of alcoholism occurred within a scientific context that offered little additional data on which to base acceptance of the disease model than had existed 50 years previously.

Various perspectives of the disease process were described in the alcoholism literature.[4,7–11] In all of these, however, there was a general acceptance of the notion that alcoholism existed as an identifiable entity that could be characterized as follows: alcoholics are different from nonalcoholics; this "difference" either leads to or includes psychological/sociological and/or biochemical/physiological changes; these changes become part of a progressive and irreversible disease process; the disease is characterized by an "inability to abstain" and/or a "loss of control" over alcohol. It was hypothesized that the supposed difference between alcoholics and others was based on a psychological predisposition,[12–14] an allergic alcohol reaction,[7,15,16] or some nutritional deficit which may or may not be genetically influenced.[17–20] Whatever the ultimate loci of the presumed differences that distinguish alcoholics from other drinkers, it was assumed that loss of control was a major discriminant of alcoholics (especially gamma type).[4,21]

Since alcoholism, in this view, was a chronic and progressive disease, it seemed reasonable to assert that until a "cure" could be found, the only logical approach to management was to promote life-long abstinence in an attempt to arrest the disease. Thus, the controlled administration of alcohol to alcoholics for the purpose of scientific study, although possibly useful for the advancement of knowledge, was seen to offer the alcoholic only negative consequences and so was considered to be unacceptable.

It was within this social and political context that the early research involving programmed access to alcohol appeared so provocative.

3. The Effects of Alcohol: Early Research

Prior to 1960, clinical observation of the acute effects of alcohol ingestion provided valuable information on the effects of chronic alcohol abuse. Studies of the withdrawal syndrome,[22,23] of tolerance,[24] and of cross tolerance[25] had offered important insights into the nature of alcohol dependence. Additionally, psychologically oriented retrospective studies of the effects on the self-concept of acute intoxication[26] had been conducted. In none of this work, however, had alcoholics been administered alcohol.

Although Diethelm and Barr[27] had examined the therapeutic value of interviewing the alcoholic patient during intoxication, and Doctor and Bernal[28] had explored the psychophysiological effects of sustained alcohol consumption in alcoholics, the first extensive program of research involving the administration of alcohol to alcoholics was that of Mendelson and subsequently Mendelson and Mello and their colleagues at the National Center for the Prevention and Control of Alcoholism.

Mendelson's early work[29] was provoked by a desire to explore the controversial nature of the alcohol withdrawal syndrome. During the 1950s, it was held widely that this syndrome reflected intercurrent illness and nutritional deficiency in the alcoholic rather than resulting from the cessation of drinking. Despite challenges to this interpretation by the clinical observations of Victor and Adams[22] and the studies by Isbell *et al.*,[23] it was not until Mendelson's[29] work in which he administered precribed daily amounts of alcohol in divided doses every 4 hr to inpatient alcoholic subjects that withdrawal signs and symptoms were shown to appear in healthy and well-nourished alcoholics solely as a function of the cessation of drinking. With this demonstration, a major part of the controversy surrounding the nature of the alcohol withdrawal syndrome was resolved.

Further study conducted within this early program of research[30] also generated important findings regarding the very nature of alcoholism, the significance of which is examined later in this chapter. These investigators undertook the systematic observation of ten alcoholic subjects who were administered up to 40 oz of 86-proof whiskey in programmed doses over a period of 24 days. The results of this study indicated, contrary to existing statements and theoretical constructions in the contemporary literature, that anxiety was increased rather than decreased under conditions of increased alcohol dosage. Further, this study also indicated, contrary to expectations derived from the literature, that "craving" did not appear with the first drink but only "after large quantities of whiskey" had been consumed over an extended period of time.

Given the wealth of new and important findings from this first research program, Mendelson and his colleagues set about to examine systematically the nature of alcoholic drinking. The programmed method of alcohol administration employed in the first set of studies, although it permitted precise control over the subject's alcohol intake, did not lend itself to the investigation

of the subjects' drinking patterns. Thus, in most of their subsequent research, Mendelson and his colleagues employed a variety of operant procedures[31] in which alcohol was used to reinforce successful performance on assigned motor tasks. By working at these tasks, the subjects could exert a degree of control over the amount and rate of their drinking, and their patterns of working and drinking could be studied.

Mello and Mendelson[32] developed an operant sequence in which two inpatient alcoholics were required to key press under a variety of schedules of reinforcement in order to obtain either money (15 cents) or its equivalent in bourbon (10 ml). Throughout the study, both subjects maintained relatively stable blood alcohol concentrations (BACs), and yet they complained constantly about the monotony of their task. Provoked by these complaints, the investigators became concerned that such an unpleasant task might disrupt spontaneous drinking. Thus, in a subsequent yet similarly oriented study, Mendelson and Mello[33] employed a simulated driving task in which their subjects could earn points for competent "driving" performance. If a subject performed on this device with maximum efficiency, it was possible to earn points resulting in consumption of up to 5 oz of 84-proof spirits per hour.

As in the 1965 study, the two subjects of the present study maintained relatively stable BACs (within the range 150–250 mg/100 ml and 200–300 mg/100 ml, respectively). Also, as previously, neither subject appeared markedly intoxicated nor showed severe ataxia, dysarthria, or stupor. An observation that Mendelson and Mello initially believed to be related to the maintenance of BAC stability also emerged from this work. The subjects of both studies showed a temporal shift in their pattern of working and drinking throughout the studies. During the first 4 days of both drinking sequences, the subjects worked primarily during the day. As the drinking periods progressed, however, they tended to work more frequently and in shorter sessions and to work increasingly more at night.

Summarizing the observations from both the Mello and Mendelson[32] and the Mendelson and Mello[33] studies, these authors concluded[34] (1) that all their subjects had shown a striking behavioral tolerance for alcohol and yet also had indicated severe withdrawal signs on cessation of drinking; (2) that even though vast quantities of alcohol were available to every subject, no subject attempted to drink himself into oblivion, and, even after the achievement of quite high BACs, no subject reported an uncontrollable urge to continue drinking; and (3) that social interaction factors played an important role in determining both the consumption parameters and the periodicity of the drinking behavior of their subjects. (In the second study, for example, one subject decided to stop drinking because his cohort had ceased drinking 2 days previously.)

The idiosyncratic patterns of alcohol use exhibited by each of the subjects in these two studies and the sheer number of factors that were believed to contribute to the observed fluctuations led Mendelson and Mello away from studying further the spontaneous drinking patterns of their alcoholic subjects.

Rather, they began to direct their focus to the identification and assessment of the forces that influence drinking within a drinking episode.

In the same year as Mendelson and Mello reported their second study (1966), a London psychiatrist, Julius Merry,[35] published an account of a double-blind study using a within-subjects design to test what he termed the "loss of control myth." In this experiment, nine gamma alcoholic inpatients were administered an orange-flavored vitamin mixture to which small quantities of vodka were added and removed on a 2-day no-alcohol/alcohol schedule. The beverage was administered early each morning over a total of 17 days. Then, in the late morning, the subjects were required to rate on a 5-point scale the strength of any craving experience they could detect.

Merry found that his subjects' craving scores were identical on the no-alcohol and standard dose alcohol days. Even more significant, however, from the point of view of examining the loss of control concept, on the last day (the day on which all subjects were given a slightly larger alcohol dose), the total craving score actually dropped quite markedly. Thus, in this first direct test of the loss of control concept, Merry concluded that if loss of control occurs in alcoholics, it is unlikely to be precipitated by only one drink. Further, he suggested that psychological and environmental factors are likely to play a part "more important than alcohol" in the initiation of alcoholic drinking. Again, in the same year and also subsequently, the most serious scrutiny and criticism of the concepts of craving and loss of control were presented in the writings of Pattison and his colleagues.[36–38]

4. Factors Influencing Drinking Patterns and Valence of Alcohol

Mello *et al.*[39] reported a series of studies emanating from the same laboratory in which they explored the relationship between drinking and motivational parameters measured in terms of the work required to obtain alcohol. The procedure required inpatient alcoholic subjects to attend continuously to a vigilance task, correct responding on which led to the accumulation of reinforcement points. Incorrect responding or a failure to respond, on the other hand, led to a loss of all the points accumulated. Additionally, a risk-taking option incorporated within the procedure permitted subjects to settle for the number of points they had acquired or, alternatively, they could gamble for double or nothing. The design involved two groups, each of six alcoholic subjects, who were required to make 16 and 32 consecutive correct (fixed ratio, FR) responses, respectively, in order to obtain a single reinforcement of 10 ml of alcohol or its then monetary equivalent, 15 cents.

Results from this study were most enlightening. First, it was found that the mean BAC of the subjects in the FR 16 group was twice as high as the same figure calculated across subjects in the FR 32 condition. Such data provided strong support for the notion that the volume of alcohol consumed by an alcoholic can be manipulated in rough proportion to the work required to

obtain an alcohol reinforcement. Clearly, these observations do not reflect loss of control and/or craving in these alcoholic subjects.

Most subjects earned between 1 and 2½ oz before drinking and thereby terminating a reinforcement sequence. Also of interest was the observation that subjects neither hoarded alcohol nor consumed a single dose as soon as it became available. Further, unlike subjects in the previous studies, and despite a relatively short drinking period (7 days), only 11 of the 14 subjects worked for and consumed alcohol throughout the entire drinking period.

Turning now to the relationship between gambling and the level of intoxication in the above studies, Mello *et al.* found no relationship between their subjects' BACs and the initiation of gambling. In fact, their subjects regarded the gambling contingencies within the experiment as involving an unnecessary level of risk, especially given that each subject could acquire all the alcohol he could drink without gambling. Finally, only one subject showed any sustained preference for money over alcohol as a reinforcer, a finding that is most reasonable considering that the lifestyle of virtually all of the subjects in the study was one that did not provide any expectation of sustained acquisition of money, even for the subsequent purchase of alcohol.

Using research paradigms similar to the preceding studies, Mello and Mendelson also have increased our understanding of the relationship between laboratory-based and longer-term drinking and working behavior.[40] Further, they have explored the consistency of drinking patterns over time,[41] they have examined the interactions of fluctuations in BAC and the emergence of withdrawal phenomena,[42] they have studied the relationship between drinking history and pattern interactions and the appearance of alcohol withdrawal,[43] they have studied the relationship between caloric intake and alcohol metabolism as well as, more generally, the biochemistry of alcohol dependence,[44,45] they have extended our knowledge of the effects of alcohol consumption on the sleeping patterns of alcoholics,[29,47–50] and they have explored memory function and dysfunction under conditions of intoxication.[51,52] Although we further address some selected aspects of the research of these investigators later in this chapter, the research areas mentioned above are not examined in detail herein.

Cohen and her colleagues at the Alcoholism Research Unit of Baltimore City Hospital also investigated the valence of alcohol for alcoholics, but they did so within the context of a token economy program. In a series of studies that related the cost of alcohol, expressed in terms of the work output required to purchase alcohol, to the amount of alcohol consumed, Liebson *et al.*[53] first required two inpatient alcoholic subjects to undertake the monotonous chore of stuffing envelopes in accordance with a series of reinforcement schedules, the payoff being alcohol. With a fixed-ratio schedule requiring 50 envelopes to be stuffed for a payment of one ounce of alcohol, the subjects earned a daily average of 20 to 32 oz of alcohol, respectively, over periods of 14 and 9 days. When the cost of the alcohol was varied among fixed-ratio programs with schedules of 100, 50, 10, and free access, the amount of alcohol consumed was found to be inversely proportional to the work demands of the task.

Overall, the evidence from studies that have compared the relative value of alcohol with other potential reinforcers for alcoholics shows consistently that alcohol is preferred in a variety of forced-choice situations. This has been the case even in a situation in which a money alternative could be used to buy alcohol.[39]

An important corollary of the issue of just how much an alcoholic will expend in order to obtain alcohol is the matter of the relative worth of abstinence and/or limited drinking for such individuals. Cohen *et al.*[54] reviewed the three studies conducted by their team that addressed this issue. In all three studies, these investigators explored the effects on abstinence of either a delay in reinforcement or a priming dose of alcohol and the magnitude of monetary reinforcement required to reinstate abstinence when it was disrupted by these manipulations. Also, in all experiments, inpatient alcoholic subjects were given the option to purchase up to 720 ml of 95-proof alcohol at 25 cents/30 ml every third day. Additionally, abstinence was reinforced by cash, which in turn permitted the payment of fines levied for ward infractions as well as the purchase of luxuries and outings. Data on each subject were collected throughout his stay in the program.

In the first experiment,[55] subjects were offered 25% of their in-ward savings if they abstained from alcohol for 1 day. On subsequent days, the amounts of money they were offered depended on whether they drank or abstained on the previous day. If they drank, payment for abstinence was increased. If they abstained, it was decreased. The results of this manipulation indicated that although the range of the various payments was considerable (7–20 dollars/day), every subject had his price for abstinence.

In the second study,[55] two of the subjects used in the earlier experiment were offered the same payment for which they had abstained previously. In this case, however, payment for abstinence was delayed from 3 to 21 days. Within this framework, again, payment for abstinence was increased if the subjects drank. However, if they abstained, the delay period for reinforcement was increased. As may be anticipated, the results of this second study also indicated that each subject had his price. One subject required a 14-day delay of reinforcement in order that his abstinence be disrupted. The other subject required only 3 days without payment before drinking began. Overall, the results from both subjects suggested that the longer payment was delayed, the more likely the alcoholic was to drink. All the same, increases in the magnitude of the reinforcement consistently reinstated abstinence for both subjects.

In the third study,[55] the two subjects who participated in the second study were required to drink priming doses of alcohol (up to 300 ml of 95-proof ethanol) every third day with cessation of drinking being contingent on the dose that had resulted in a cash payment. If the subjects drank beyond the priming dose, payment for stopping after that dose was increased the next day. If they abstained, however, the priming dose was increased. Results of this third experiment indicated that the subjects' abstinence was disrupted by priming doses of 180 and 300 ml, respectively, but that, again, increases

in the magnitude of the reinforcement reinstated the abstinence condition. The authors concluded[54]:

> That a chronic alcoholic can stop drinking after he has started is contrary to what the alcoholic often reports and suggests that current contingencies, as well as personal history and recent drinking, may determine consumption (p. 752).

From such a perspective, and given the background of the previous research, Cohen *et al.* conducted a further series of experiments to determine if moderate drinking could be elicited through contingency management for periods longer than 1 day. In the first study of this series,[56] five inpatient alcoholic subjects had access to 24 oz of 95-proof ethanol each weekday for 5 successive weeks. During the first, third, and fifth weeks, moderate drinking (less than 5 oz of ethanol) was differentially reinforced via access to an enriched environment consisting of social, recreational, and work privileges as well as a more palatable diet. Drinking in excess of 5 oz of alcohol per day, on the other hand, led to revocation of the privileges of the enriched environment for periods of 24 to 48 hr. During this restriction, the subjects were required to remain in their rooms; they could not work, socialize with staff, or enter the day room, and their food was pureed. The second and fourth weeks of the experiment were noncontingent weeks, with no differential consequences for moderate or excessive drinking. During these periods, the subjects remained in the impoverished environment. Results from this study showed the emergence of significant differences in the mean volumes of alcohol consumed by each of the five subjects during the contingent and noncontingent weeks, with a marked reduction in consumption being observed during the contingent weeks.

In the second experiment in this series,[56] the first, third, and fifth weeks of the experiment were identical to those described immediately above. Also, four of the subjects from the previous study were employed in this experiment. However, the procedure of the second and fourth weeks was different. In the second study, the subjects were maintained in the enriched environment no matter how much they drank during the second and fourth weeks. Again, the results presented in terms of the mean volumes of alcohol consumed by each subject showed significant differences, with each subject drinking moderately during the contingent weeks and excessively during the noncontingent weeks (see also ref. 57).

In the final experiment in this series,[58] three inpatient alcoholic subjects were placed in the enriched–impoverished contingency condition for from 14 to 17 days with alcohol available throughout. As previously, all three subjects, none of whom had participated in any of the other studies, maintained moderate drinking as long as the contingency was in effect.

The results from this series of experiments and others from the Baltimore group[59] as well as the previously noted work by the Mendelson and Mello team indicate that although drinking is a particularly high-valence activity for

alcoholics, this behavior can be controlled or otherwise contingently manipulated within a ward environment. The data also indicate, however, that alcoholics commonly will drink to excess if there are either no immediate negative consequences imposed on such drinking or no positive consequences provided to refrain from such drinking.

5. Affective Consequences of Alcohol Consumption

Prior to the experimental work involving chronic alcohol intoxication, it was widely held as self-evident that alcoholics consumed alcohol in order to neutralize anxiety or to escape from stress.[60] Horton,[61] for example, asserted that an "unequivocally significant property of alcohol was that it reduced anxiety." Although statements such as this no doubt conform with the observations of many nonalcoholic individuals, the evidence in support of such assertions with respect to all or even many alcoholics is lacking.

Diethelm and Barr[27] had noted that the effects of alcohol on an alcoholic subject were largely a function of the context in which the alcohol was taken, but it was Mendelson, LaDou, and Solomon[30] who first challenged the notion that alcohol reduces anxiety and depression in the alcoholic. These investigators reported that although some degree of anticipatory anxiety was observed in their subjects prior to the administration of alcohol, and although little impact on mood was noted following alcohol intake early in the study, all ten of their subjects became progressively more tense and anxious as the study progressed. Although one subject showed severe depression early in the study, by day 19 of the 24-day period, anxiety levels had increased markedly for all subjects, "with evidence of depression, hyperaggressiveness, and other psychopathology." During the withdrawal phase, many subjects showed mixed relief and apprehension, and all verbalized considerable ambivalence about their drinking (see also ref. 47).

In similar vein, Tamerin and Mendelson[48] described progressive depression, guilt, psychic pain, increased sexuality, and aggressive behavior with continued drinking, and Tamerin *et al.*[49] reported that their subjects saw in themselves more aggression, sexuality, and dysphoria while drinking than they had anticipated seeing. Titler and colleagues[62] also observed that ten of their 11 subjects spontaneously reported hysterical anxiety, depression, and hostility and a lowered energy level by the second or third day of drinking. None of these effects had been noted during the first several hours of drinking (see also ref. 63).

In a research program designed specifically to focus on mood changes occurring during programmed versus free-choice drinking, Davis[50] subjected nine and eight alcoholic inpatients, respectively, to programmed and free-choice drinking sequences. In the first study of this program, four and five subjects, respectively, participated in programmed drinking sequences. The second study involved two sequences of free-choice drinking, each with four

subjects. Under the programmed drinking conditions, drinking lasted 5 days, during which time approximately 16 oz of 50% ethyl alcohol was administered in divided doses every 4 hr throughout the day. This period was followed by a withdrawal period lasting 5 days which, in turn, was followed by a further 5 days of drinking. During the second drinking sequence, each subject was administered, at 4-hr intervals, a total of approximately 26 oz of alcohol each day. Following this second drinking sequence, a further 5-day withdrawal period was provided.

Assessment of the mood of each subject in the programmed drinking sequence was done twice daily prior to and following the administration of the 11 a.m. alcohol dosage. This assessment required the sorting of a set of 68 cards drawn from the Mood Adjective Checklist.[64] In addition, a diary was kept of ward events that may have affected the subjects' mood ratings. In the free-choice drinking format, a base-line period of 8 days was followed by a 5-day drinking period and a 5-day withdrawal phase. At 11 a.m. each day during the drinking period, each subject was provided with three chips that could be inserted into an alcohol dispenser to obtain a total of 3 oz of 50% alcohol. At 1 p.m., a further 21 chips were given to be used for free-choice drinking until 3 a.m. the next morning, at which time all drinking ceased until 11 a.m. that day. As in the programmed sequence, the card-sorting mood assessment was required of all subjects in the free-choice drinking sequence.

Results from subjects undergoing both programmed and free-choice drinking showed a decrease in "carefree" scores and an increase in fatigue from the first to the second drinking period. Likewise, increases in depression, unfriendliness, anxiety, guilt, and fatigue within each drinking period were noted over time. This study also provided data that indicated both a transitory positive affective change with anticipation of the first drink and a decrease in mood in response to drinking. Davis concluded that when alcoholic subjects experience an initial euphoric affect or anticipate such an affect from alcohol, under nonrestrictive environmental conditions they are likely to attempt to maintain it, or, failing to do so, they continue drinking in an attempt to regain it.

6. Social Facilitation and Alcohol Consumption

Although the social facilitating effects of alcohol have been known widely for centuries, the effects of alcohol on the interpersonal behavior of alcoholics have been studied under controlled conditions only very recently. Diethelm and Barr[27] observed two alcoholic inpatients who were permitted to drink for 14 consecutive days as part of a study of the effects of drinking on the psychotherapeutic relationship. In both cases, these investigators noted their subjects to be relatively uncommunicative when sober. When drinking, however, the subjects talked spontaneously and extensively and were demanding

and assertive with each other and with the clinicians. Mendelson[29] also observed an increase in the sociability of his chronic alcoholic subjects under conditions of alcohol consumption of up to 30 oz daily. The level of social interaction of his subjects dropped markedly, however, when the alcohol dosage was increased to 40 oz daily.[28,67–69]

Mendelson *et al.*[70] studied the role of social forces on individual drinking patterns. In this study, an attempt was made to stimulate a number of the social forces existent in a skid-row bottle gang. Using an operant paradigm, the study was designed so that all alcohol earned by each of three alcoholic subjects was dispensed into a common reservoir from which each subject could draw. Observations made throughout the 30 days of the study indicated that each subject adopted different roles in relation to his two peers and that each deposited and withdrew different quantities of alcohol from the common supply. The investigators concluded that at least some of the significance of alcohol for their alcoholic "catalytic" effect as far as the facilitation of complex patterns of social interaction was concerned. It appeared, in fact, that within the context of group drinking, each subject had taken on one or more roles that may not have been characteristic in other life situations.

Subsequent research from the same laboratories by Steinglass and his colleagues has examined further the roles taken on by drinking alcoholics, this time in the unique and complex area of family interaction. Steinglass *et al.*[71] and Weiner *et al.*[72] examined the family as a relatively stable drinking group from which they hoped to gain a greater understanding of the interactional issues in alcoholism. In both of these studies, the procedure involved a related subject pair undergoing a period of inpatient experimentation which comprised a 5-day predrinking period followed by a 14-day drinking period and, finally, a 5- to 7-day withdrawal phase. During the drinking period, each subject could make daily purchases of up to 1 quart of 100-proof beverage alcohol from a continuously operating dispenser, each purchase being supported by tokens which also were supplied daily.

The most striking finding to emerge from both of these studies was that in each dyad observed, alcohol and drinking had come to be used as a basis for the definition of roles. To quote Steinglass and Weiner[73]:

> Whereas, superficially these pairs seemed to be exhibiting a wide diversity of individual interactional behavior, on closer examination it became apparent that in each case this behavior rigidly adhered to a set of rules which predetermined the form the behavior would take (p. 702).

Also of considerable interest, especially from the perspective of general systems theory, was the finding that within 2 weeks of the completion of the study, each of the above-noted dyads had separated. In response to this fact, Steinglass *et al.* have speculated that it was the intense scrutiny to which each dyad was subjected that brought about the dissolution. In each dyad, it seems, the relationship between the members was so brittle as not to be capable of dealing with the pressures provoked either by the scrutiny or the insight that followed it.

Subsequently, Steinglass *et al.*[74] have extended the perspectives that emerged from the previous work by developing an experimental treatment program in which ten couples with one or two alcoholic members were placed in an intensive 6-week multiple-couples group therapy program. Of special significance to the present discussion was the fact that following an initial 2-week outpatient phase, a 10-day inpatient phase was instituted during which three couples were simultaneously admitted to the laboratory. In what amounted to a "simulated apartment setting," these couples were required to reproduce as accurately as possible their usual interactional behavior, including their drinking practices. Again, the rationale was to permit the therapist to gain a first-hand understanding of the role that alcohol consumption was playing in the couples' lives. The treatment program utilized a variety of techniques to examine patterns of interaction exhibited by each couple during both periods of sobriety and periods of intoxication. These techniques included videotape recording and replay, role-playing techniques, the use of feedback from observers, analysis of speech and communication patterns, emphasis on nonverbal behavior, and postural analysis. Other techniques used commonly by family therapists in more traditional settings also were employed.

Although Steinglass and his colleagues have advised caution regarding the outcome from this experimental study, the evidence to date is that all couples responded quite positively to the treatment approach. All ten couples completed the study, and all reported a profound emotional impact derived particularly from the inpatient experience (see also ref. 75). Also of major importance in our developing understanding of the role of social forces on the drinking practices of alcoholics had been the early work of Nathan and his colleagues within the Alcoholic Research Unit of Boston City Hospital.[76,67] In a series of three studies involving 20 inpatient alcoholics, 3-day periods of free socialization were alternated with similar duration isolation periods during base-line, drinking, and then withdrawal phases of the study. The drinking periods in the three studies lasted 6, 12, and 18 days, respectively. The operant task, required of all subjects in this series of experiments, involved interrupting a photocell beam. Three thousand such responses yielded either 20 ml of bourbon of 15 min out of social isolation. Additionally, points could be accumulated throughout the base-line period for expenditure during the drinking phase.

Throughout the series, the subjects' drinking patterns did not appear to be unduly affected by the isolation conditions. Although five subjects drank slightly less during isolation, the remainder drank approximately the same as during socialization periods. Although considerable variability in BAC was reported across days, again this did not correspond in any systematic fashion with the conditions of isolation or socialization. In fact, the only significant effect of the isolation condition during the entire series was that it produced an elevation in anxiety and depression scores as determined from the self-rated Mood Adjective Check List. Interestingly, it appeared that despite a general pattern of increasing affective distress, subjects in these studies did

not attempt to alter their mood states by increasing or decreasing their alcohol consumption.

Examining the above outcome, Nathan *et al.*[78] hypothesized that perhaps the previous subjects had not modified their drinking in relation to the socialization manipulation because although alcohol was readily available, other important discriminative stimuli for both socialization and alcohol consumption, namely, the existence of a bar and the presence of a bartender, were not. Moreover, they suggested that the lack of an isolation effect in the previous research may have reflected the fact that the subjects had prior knowledge of the length of each isolation period. Thus, Nathan *et al.*[78] ran an additional four subjects over an 18-day drinking period during which the 3-day alternations of socialization and isolation were not explained. Further, in this additional study, a bar was incorporated into the laboratory, and a bartender was employed.

Data from this new procedure showed considerably more socialization (at the bar) than had occurred in the previous studies. Additionally, subjects now drank somewhat less than had subjects in the previous studies. Nathan and his colleagues interpreted these observations as being related to their subjects' uncertainties about the prospects of abrupt withdrawal. Finally, and this observation is in concert with much of the evidence reviewed previously in this chapter, these investigators indicated that the choice between alcohol consumption and socialization appeared primarily to be a function of the number of points (income equivalent) that their subjects had accrued prior to the time at which expenditure began. When a large number of points had been acquired, the subjects expended their savings for both alcohol and socialization. When the amount in savings was minimal, or when it was necessary to work for each drink, drinking occurred to the exclusion of socializing.

An additional study by Bigelow[79] examining and quantifying the phenomenon of increased social interaction by alcoholics during drinking is also worthy of note at this time. In this study, alcohol was made available essentially randomly to five alcoholics in a residential research setting over a 20-day period, and their rates of social interaction on drinking and nondrinking days were compared. The behavior of all subjects was recorded at random intervals throughout the day. When a timer sounded, the staff would record whether a subject was awake or asleep, and if he was awake, whether he was interacting socially or not. Bigelow found that his subjects interacted socially 11% of the time on nondrinking days and 39% of the time on drinking days (see also ref. 80).

7. Alcoholics versus Nonalcoholics

7.1. Social Facilitation

As is obvious from much of the research examined in this chapter, the free-operant paradigm in which a subject acts as his own control has been

used extensively in much of the laboratory-based research involving the use of alcohol by alcoholics. Such a method, however, does not allow meaningful comparisons to be drawn between the behavior of the sober or drunk alcoholic and a matched sober or drunk nonalcoholic. Thus, in a second phase of the work at Boston, Nathan and O'Brien[81] used a matched-subjects design focusing on the complex interactions that exist among drinking patterns, drinking effects, and social behavior in both alcoholic and nonalcoholic subjects. These investigators conducted a series of two experiments involving four skid-row alcoholic men and four nonalcoholic subjects matched with the alcoholics in terms of lifestyle and socioeconomic status. In each study, an 18-day drinking period followed a 9-day predrinking and preceded a 6-day postdrinking phase.

As previously, the present studies also were divided into alternating three-day socialization and interpersonal isolation periods. Additionally, in both studies, the task involved work at an operant panel to which the subjects had unlimited access during the predrinking period. Points earned at the task permitted the purchase of 86-proof bourbon and/or relief from isolation. Data were collected in relation to four functional categories: operant rate, drinking behavior, affective behavior, and social interaction.

The results indicated that although both the alcoholic and nonalcoholic subjects reached similarly high BACs early in the drinking sequence (a finding that would not be expected with most nonalcoholics), the alcoholic subjects remained at these levels for longer and returned to them more often than did the nonalcoholics. Thus, the alcoholic subjects drank almost twice as much as their controls. The alcoholic subjects also established a pattern of 3- to 5-day "spree" drinking followed by a longer "maintenance" drinking period. The nonalcoholic subjects, meanwhile, exhibited maintenance drinking but showed no spree drinking. Additionally, unlike the nonalcoholics, at least these alcoholic subjects proved to be social isolates before, during, and after drinking. They became significantly more depressed and less active and showed more psychopathology when drinking than did the nonalcoholics. Finally, Nathan and O'Brien noted that their alcoholic subjects preferred straight rather than mixed drinks, that typically they gulped rather than sipped their drinks, and that they chose not to concern themselves with the "niceties" of social drinking such as the use of ice and bar napkins.

Overall, individual differences notwithstanding, the evidence from studies involving the administration of alcohol to alcoholic subjects shows the general social facilitative effects of such administration. As with mood, however, there seems an upper limit to this facilitative effect, and under conditions of considerable intoxication, the sociability of alcoholic subjects appears markedly reduced.

7.2. Drinking Parameters

Like Nathan and O'Brien, Schaefer *et al.*[82] also had noted significant differences in the base-line drinking behaviors of their alcoholic subjects in

comparison with a group of social drinkers. To further explore this observation, Sobell *et al.*[83] compared the drink preferences, sip magnitudes, amount of time taken to consume drinks, and time between sips in a sample of 23 social drinkers and 26 hospitalized alcoholics. The procedure permitted the consumption of up to 16 oz of 86-proof liquor in the time allotted. Again, the results showed the alcoholic subjects ordering more drinks (means of 15.3 and 6.7 drinks, respectively), taking larger sips (typically two to three times the volume of their nonalcoholic peers), and drinking faster. However, the alcoholic subjects took longer between sips than did the social drinkers.

A quantitative analysis of the drinking patterns of alcoholic subjects also has been reported by Mello and Mendelson.[84] In this instance, the focus was on prolonged drinking, with an effort being made to determine if it was possible to categorize reliably and predict from this categorizing the drinking patterns of alcoholics. In previous research examining the consequences of programmed versus spontaneous drinking, Mello and Mendelson[42] had found support for the view that the behavioral and biological effects of alcohol on alcoholics were determined more by the patterns of consumption than by the duration of drinking. (In fact, they observed that the spontaneous drinking paradigm consistently produced the more severe, varied, and prolonged withdrawal signs and symptoms.) In the present research, Mello and Mendelson studied 15 inpatient alcoholics in groups of four to five throughout a sequence that involved a 1-week base-line phase followed by a 9- to 12-day spontaneous drinking phase and then a recovery period. During the drinking phase, each subject was permitted to purchase, with supplied tokens, up to 32 oz of spirits.

Overall, the results of this study indicated considerable variability in daily alcohol consumption both within and across subjects. Interestingly, however, the average interpurchase interval within the study was 135 min, which corresponds quite closely to the time that it takes the average man to metabolize 2 oz of liquor,[85] which, in turn, was the average size of each liquor purchase. These observations, together with other alcohol consumption data from other ward studies by these same investigators, have led Mello and Mendelson[84] to conclude that the limits in the variability found in the drinking patterns of alcoholics are determined primarily by the physiological–pharmacological actions of alcohol and that within these limits, the variability in alcohol consumption noted in alcoholics is largely a function of the interactions of the social and affective forces imposing on and existing within these individuals at the time of their drinking.

Additional findings from this study[84] also are particularly important to our understanding of the drinking patterns of alcoholics. Mello and Mendelson noted that their subjects consistently showed a decreased ability over time to deal with the effects of alcohol. For example, they noted, paralleling findings we have discussed previously, that their alcoholic subjects became increasingly dysphoric and progressively more ambivalent about their drinking. Further, they reported that equivalent amounts of alcohol produced greater degrees of intoxication over time. Related to this, they observed a decrease in food consumption over time. Finally, Mello and Mendelson found that

their alcoholic subjects tended to consume progressively less rather than more alcohol over a prolonged period.

This last observation by Mello and Mendelson, together with much of the laboratory-based evidence that we have examined to this point and additional evidence that we review later in this paper, do not seem capable of convenient reconciliation with the concepts of craving and loss-of-control. It is to this issue that we now turn.

8. The Loss-of-Control Hypothesis and the Prospect of a Paradigmatic Shift

Limitations in the generalizability of laboratory-based approaches to the study of alcoholism notwithstanding, by the early 1970s, the accumulated evidence from experimental investigations of the consequences of administering alcohol to alcoholics (both in the laboratory and in experimental treatment programs, as we see later) revealed drinking patterns and effects that were inconsistent with traditional views of alcoholism. Yet, for reasons perhaps best articulated by Kuhn,[86] relatively few specialists within the alcoholism field were provoked to reevaluate the traditional perspective or suggest a view more consistent with the emerging evidence.

Jellinek[4] had hypothesized that with loss of control, the alcoholic is affected by alcohol so that

> the ingestion of one alcoholic drink sets up a chain reaction so that [they] are unable to adhere to their intention to "have only one or two drinks" but continue to ingest more and more—often with quite some difficulty and disgust—contrary to their volition (p. 41).

Although the widespread acceptance of Jellinek's view no doubt helped some alcoholic individuals refrain from experimenting with alcohol, empirical evidence to support the hypothesis was lacking. Mello and Mendelson, of course, pointed out[84] that despite the accumulated data that "are inconsistent with the notion of craving . . . this construct [continues to] form the basis for the usual therapeutic goal of total abstinence for the alcoholic patient" (p. 680). Similarly, following his review of the development of the loss-of-control construct, Keller[87] concluded that it was fascinating that it should be thought necessary to devise and execute experiments to disprove a misinterpreted claim with no foundation at all in observed reality.

A number of additional studies appearing at about the time of Keller's review and confronting directly the loss-of-control hypothesis also are worthy of attention. Sobell *et al.*,[88] for example, summarized the results from a program involving experimental drinking by alcoholics. Of 214 voluntary gamma alcoholic subjects housed on an inpatient open ward, only 3.3% left treatment seeking additional alcohol. Many subjects participated in as many as 15 drinking sessions and on some occasions drank as much as 16 oz of 86-proof alcohol.

In a questionnaire section of the study, 30 subjects all answered that they believed they would ingest one drink and then stop as long as they resided in the hospital, and 90% thought they would be able to stop even if they consumed 16 oz of liquor while hospitalized. On the other hand, 43% of the same subjects thought they could consume one drink and stop if they were not hospitalized. This figure diminished to 23% when 16 oz was the quantity in question. The authors urged a restatement of the loss of control concept on the basis that treatment of the alcoholic

> could be facilitated by making him aware that he became drunk by his own choice, as opposed to describing him as the victim of a strong set of physiological circumstances whereby his only prognosis after the first drink was to become drunk (p. 123).

(See also refs. 89–93.)

Two significant attempts have been made to revise the loss-of-control hypothesis to incorporate the conflicting experimental findings. Glatt[94] suggested that rather than considering loss of control to be an all-or-none phenomenon based on the administration of any alcohol, each alcoholic might have his/her own critical blood threshold level. Loss of control would emerge only if drinking exceeded that critical level. Such a revision, however, does not explain the wealth of evidence that failed to show loss of control drinking by alcoholics who had consumed quite large volumes of alcohol.

The other revision was offered independently by Keller,[87] Sobell *et al.*,[88] and Paredes *et al.*[95] Allowing for slight differences in phraseology, all of these authors suggest adding a qualifying motivational statement to the original hypothesis. Thus, an individual who is an alcoholic is likely to drink to excess after consuming an initial drink on some occasions depending on the circumstances. In all of these formulations, loss of control is interpreted as an acquired process rather than a physiologically determined one (also see the review, ref. 96).

It was research involving the administration of alcohol to alcoholic subjects that led to a questioning of the loss-of-control hypothesis. This enabled and led to further research that seemed to indicate that for at least some alcoholics, abstinence need not represent the only possible treatment alternative (see, for example, refs. 93, 97–101) and that abstinence does not necessarily indicate improvement in other areas of life health.[36,38,102,103] On the basis of these observations, some investigators have suggested that there is a need to change the paradigm of alcoholism to what they term a multivariate perspective of alcohol abuse.

A detailed presentation of the multivariate approach is beyond the scope of the present chapter. Briefly though, Pattison *et al.*,[104] on the basis of a rather thorough review of clinical and laboratory research evidence in the alcoholism field, have concluded the following: (1) Alcohol dependence includes a variety of syndromes defined by drinking patterns and the adverse physical, psychological, and/or social consequences of such drinking; these syndromes are

best considered as a serious health problem. (2) Alcohol dependence syndromes can be considered as lying on a continuum from less to more pathological. (3) A variety of factors may contribute to differential susceptibility to alcohol problems, but these factors, although predisposing, do not *per se* produce alcohol dependence. (4) Any person who uses alcohol can develop a syndrome of alcohol dependence. (5) The development of alcohol problems follows variable patterns over time and does not necessarily proceed inexorably to severe or fatal stages; the problems may progress or be reversed through either naturalistic or treatment processes. (6) Alcohol problems are typically interrelated with other life problems, especially when alcohol dependence is long established. Although these investigators have avoided the hazards of model building, they do stress that the dynamic complexity of alcoholism cannot be assessed unless the disorder is conceptualized multidimensionally.

From a similar perspective, Caddy[105] has extended the multivariate approach by proposing a therapeutically oriented analysis of alcohol dependence. This idiographic strategy permits an evaluation of the current dynamics of the patient in terms of behavioral, cognitive, social, incentive, and discriminative domains. Although the work by Pattison *et al.* and Caddy has been influenced heavily by learning-based theory, it has been data from the alcohol administration studies that has provided the impetus for the emergence of the multivariate approach (see also refs. 106–109). At the present time, however, there appears little evidence that a paradigmatic shift in theory or treatment away from the more conventional views of alcoholism has occurred. No doubt, further investigation of the issues confronting both the traditional and the multivariate perspectives will provide additional insight regarding the nature of the complex we label alcoholism.[110–112]

9. Research on Blood Alcohol Concentration Discrimination Training

The evidence from the parametric studies of the drinking behavior of alcoholic and nonalcoholic subjects that we reviewed earlier suggests that alcoholic drinking becomes patterned and that once the decision to drink has been made, the actual behavior involves relatively little thought. Although we know that within certain limits increasing intoxication is paralleled by recognizable and consistent changes in subjective states,[113] it is conceivable that as drinkers acquire an increase in tolerance, their ability to discriminate slight changes in intoxication is reduced, and with continued drinking, significant changes in BAC are required in order that increments in intoxication be perceived. Further, it may be that the parameters of alcoholic drinking necessarily produce BAC increments of such magnitude that the subtle and changing internal cues noticed by the moderate drinker during a drinking occasion are lost to the alcoholic. Thus, given a pattern of ingesting "straight"

drinks of high alcohol content in a short period and the inevitable delay between ingestion and the introduction of alcohol into the circulatory system, a state exists wherein the alcoholic, even without further drinking, will continue to experience an increasing BAC until the rate of absorption into the blood supply is matched by the rate of the elimination processes. Under such conditions, having consumed alcohol, the alcoholic truly may be said to be incapable of preventing a continued increase in his/her BAC. Attempting to reach a level of intoxication previously associated with pleasant and positive affect, the individual may overshoot the mark without realizing it.

It was reasoning such as this that provoked the early work aimed at exploring the possibility of training drinkers, and particularly alcoholics, to monitor accurately their BACs by focusing on their patterns of consumption and the concomitant subjective effects of such drinking.

The earliest work examining training in BAC discrimination was conducted with nonalcoholic subjects in the late 1960s.[114] This research, which involved the comparison of pre- and posttraining scores for subjects who underwent a training sequence of drinking followed by BAC estimation followed by rapid BAC determination and feedback, indicated that nonalcoholics could learn to estimate accurately their BACs within as little as one training session (see also refs. 115–117). That finding, in turn, led Lovibond and Caddy[118] to develop a multicomponent alcoholism treatment program that included BAC discrimination training. We discuss this and other treatment-oriented research subsequently.

Although Caddy's early BAC discrimination work with nonalcoholic subjects had been conducted within a framework of controlled experimentation, the BAC discrimination training program developed by Lovibond and Caddy for alcoholic subjects had not. In an effort to investigate the determinants and efficacy of BAC discrimination in alcoholics, Silverstein *et al.*[119] undertook a controlled study with four male gamma alcoholics whom they observed over a 36-day period with the Rutgers Alcohol Behavior Research Laboratory. This study was divided into two phases. The goal of the first phase (which lasted 10 days) was to examine some of the factors involved in training alcoholics to estimate their BACs accurately. Drinking was programmed in five 2-day cycles, so that the subjects' BACs rose on the first day to around 150 mg/100 ml then fell overnight and over the next day to zero. During the first (base-line) two-day cycle, the subjects estimated their BACs approximately ten times daily without receiving any feedback regarding their estimation accuracy. During the following three 2-day cycles, subjects were continuously alerted to the emotional and physical correlates of changing levels of blood alcohol while receiving feedback (1) after each BAC estimate; (2) after 50% of their estimates; and (3) after 50% of their estimates, with positive reinforcement delivered contingent on accurate BAC estimation. During the final 2-day cycle of this phase of the study, which represented a return to the base-line condition, subjects were again required to make their BAC estimates in the absence of training, feedback, or contingent reinforcement.

During the 26-day second phase of the study, three of the four subjects who had participated in the study's first phase were trained to drink to, then maintain, a prescribed BAC of 80 mg/100 ml. Three converging behavioral shaping procedures were utilized for this purpose. (1) Responsibility for control over drinking was gradually shifted from the experimenter to the subject. (2) The range of positively reinforced BACs was successively narrowed closer and closer to the BAC target (80 mg/100 ml). (3) All reinforcement and feedback were gradually faded out over the nearly 4 weeks of this phase of the study.

Data from the first phase of this study showed that the most powerful factor influencing BAC estimation accuracy was, singly, the presence of absence of accurate BAC feedback. Whether this feedback was continuous or intermittent or accompanied or unaccompanied by reinforcement for accuracy was unimportant. During the second, control-training phase of the study, subjects were able effectively to regulate their drinking within the prescribed range, but only as long as BAC feedback was provided. According to Silverstein *et al.*, BAC feedback "apparently served as an information anchor in that its presence to even a minimal degree kept estimates accurate" (p. 13). These investigators also noted that

> our subjects said they learned to discriminate internal cue states associated with different [BACs] . . . nonetheless . . . the precise degree to which subjects relied on these internal cues is questionable (p. 13).

Procedural and subject differences between the Silverstein *et al.* study and that of Caddy[114] or Bois and Vogel-Sprott[115] make any comparison of results rather difficult. It may be that BAC estimation difficulties experienced in the absence of BAC feedback by the subjects of Silverstein *et al.* were related to a reduced interest in the task, especially as the subjects' BACs approached 150 mg/100 ml. Alternatively, adaptation effects, which, over a prolonged drinking sequence, may blur the "base-line anchor" or effects resulting from an inability on the part of high-tolerance alcoholic subjects to perceive small changes in BAC may account for these observed limitations.

Although Lovibond and Caddy[118] reported considerable success in training their alcoholic subjects to discriminate accurately various BACs, their study employed both restricted BAC and time ranges. Additionally, their study had the advantage of the incorporation of a comprehensive alcohol drinking education program, which was designed to supplement the estimation of BACs based on the perception of primarily "internal" alcohol-related cues.

Paredes *et al.*[120] also reported on BAC estimation training, in this case with one alcoholic. Extremely high BAC estimation and self-titration accuracy scores were obtained when they provided their subject with BAC feedback on all sessions.

Lansky *et al.*[121] set out to explore further the extent to which alcoholic subjects could learn to estimate accurately their BACs and to evaluate the role played by internal and external cues in the mastery of this task. Lansky

and his colleagues housed two groups of four chronic alcoholics within their laboratory for a period of 3 days, during which time their subjects were administered alcohol (7 oz of vodka with tomato juice taken over 3 hr) and taught to attend either to internal or external BAC cues. During a single training session, each subject received BAC feedback immediately following each of his BAC estimates. As in previous work, the subjects received no BAC feedback during the pre- or posttraining sessions.

The results from this study indicated that prior to the training session, subjects in both groups were equally inaccurate in their BAC estimations. During the training phase, as expected, BAC estimation accuracy increased markedly for subjects in both groups. During the posttraining session, however, only the externally trained alcoholic subjects maintained the ability accurately to estimate their BAC and to monitor its changes. The authors concluded that unlike the nonalcoholic subjects studied in the same laboratory by Huber *et al.*,[116] their alcoholic subjects had difficulty learning to discriminate their BACs on the basis of internal feelings and sensations, even though they could do so easily by referring to external cues.

Of course, neither this study nor any of those cited previously have insured the separation of the various internal and external drinking-related cues, and so the significance of the hypothesized difference between alcoholics and other drinkers regarding their sensitivity to internally registered BAC cues, although intriguing, remains to be demonstrated.

10. Treatment in the Presence of Alcohol

The research we have examined to this point has generally explored the effects of interactions between alcohol and the alcoholic. With few exceptions, therapy has not been a focus of this research. We now change the focus of our review and turn to an examination of treatment-oriented research in which the alcoholic has received therapy in the presence of alcohol.

With the exception of the administration of minor amounts of alcohol in the early and subsequent aversive conditioning therapies (e.g., refs. 122–124) and in treatment in which alcohol was administered after disulfiram (Antabuse®) to provoke nausea, the alcoholic patient has traditionally been treated in the absence of alcohol. In fact, under hospital conditions in which the overt signs of the disorder were not permitted to occur, Canter[125] suggested that inpatient therapy for the alcoholic patient involved primarily shelter from temptation and responsibility.

There are some reasons to believe, however, that the administration of alcohol to alcoholics in therapy may yield important therapeutic, as well as research, benefits for some alcoholics. For example, earlier in this chapter, we addressed research that called into question the ability of the alcoholic to perceive him/herself accurately under conditions of intoxication. We noted the tendency of alcoholic subjects to hold positive expectations about the

effects of drinking which routinely go unfulfilled and we also noted the alcoholic's tendency to restructure unpleasant inebriated experience in terms of wistful expectancy (see also ref. 66). Such phenomena would seem especially likely to be responsive to therapeutic procedures involving audiovisual feedback of the immediate consequences of programmed intoxication.

10.1. Drinking and Self-Image Confrontation

The earliest audiovisual confrontation studies reported in the alcoholism literature were those of Carrere[126–128] who took motion pictures of his patients while they underwent delerium tremens. Carrere aimed to shock his patients into halting their use of alcohol.

It was not until the work of Paredes and Cornelison,[129] however, that audiovisual recording was used in concert with the administration of alcohol in an effort to explore the therapeutic potential of the "self-image experience." In their first study, seven alcoholic subjects were administered small doses of alcohol (120 ml at 80-proof) and underwent between six and 12 audiovisual confrontation sessions. Additionally, three subjects who became intoxicated soon after their discharge from the hospital were persuaded to return and were again videotaped, this time under conditions of marked inebriation.

Results of the study indicated that even those subjects who were recorded under conditions of only limited alcohol use brought forth considerable quantities of what Paredes and Cornelison regarded as "emotionally significant" material that might not have been elicited under alcohol-free circumstances. Further, for those subjects who were filmed while intoxicated, the effects appeared far more profound, with each of these subjects reporting the objectively perceived effects of intoxication to be far greater and far more distressing than they had been perceived subjectively. Although these authors were enthusiastic about the potential of their new therapeutic element, controlled studies remain to be done.

Incidental to the present topic but nevertheless interesting in that it contributes to our understanding of the influence of the loss-of-control concept, Paredes and Cornelison[129] reported that initially they were quite concerned about the possible "loss-of-control" consequences of administering alcohol to alcoholics. Having completed the study, however, they noted that: "Our apprehensions were quickly dispelled. None of the patients demanded more alcohol immediately or during the following week of hospitalization" (p. 88).

In a subsequent and yet quite similar clinical trail of the impact of videotaped self-confrontation,[130] this time with 66 alcoholic or problem drinker subjects who were administered only 3 or 4 oz of 80-proof vodka, the subjects responded in a similar fashion to that noted for the subjects in the previous study. Again, the subjects failed to respond to the administration of alcohol by exhibiting a craving for alcohol, and, again, the lack of controlled experimentation and adequate follow-up procedures notwithstanding, Paredes *et al.* asserted their conviction that their subjects became more receptive to the

clinical interview process following their observation of their "evasive attitude" while intoxicated.

Significantly, whereas Paredes and Cornelison[129] had first administered alcohol to their alcoholic subjects with considerable trepidation, by 1970, they were asserting[131] that

> the incorporation of alcohol in certain phases of the treatment program of alcoholics . . . will help them evaluate the role of alcohol in their thinking and feeling, in a setting in which the social consequences of being intoxicated are being controlled (p. 710).

Subsequent work using videotaped self-confrontation procedures by Schaefer *et al.*[132] at Patton State Hospital has also explored the effects of the technique with drinking alcoholics. These investigators assigned 52 hospitalized male alcoholics to either a long (30-min), short (5-min), or no videoplayback condition and then administered quantities of alcohol adequate to induce intoxication. The results indicated that their subjects considered the video replay of drunken comportment to be truly aversive, yet there was no evidence that the subjects' actual drinking practices were affected by the procedure. In other work at the same hospital, however, Baker *et al.*[133] observed that videotaped confrontation of drunkenness can have a positive impact on the subsequent drinking practices of alcoholic subjects if the procedure is used as part of a multicomponent therapeutic program to induce motivation for change.[134]

Interestingly, a decade after the early research of Schaefer *et al.*, and with no substantial subsequent research on the effects of self-image confrontation in the treatment of alcoholism, M. B. Sobell (personal communication, 1980) now is developing a new program of research to explore further the possible merits of the approach.

10.2. The Fixed-Interval Drinking Decisions Program

It was observed earlier that a major component of many treatments for alcoholism involves a sheltering of the patient from temptation and responsibility. Under such conditions, often the alcoholic is comfortable, may resolve never to drink again, and is likely to show little interest in receiving additional treatment for a condition that is no longer causing him/her any immediate discomfort. It was because of these very circumstances that Canter,[125] among others (see also ref. 135), suggested that drinking be permitted and even employed in the treatment situation, so that those factors that result in a decision to drink at certain times and not at others might be observed and studied.

It was in response to just such reasoning, together with concern over the appropriateness of programmed and free-choice drinking paradigms for the study of alcoholic drinking behavior, that led Gottheil and his colleagues[136,137] to develop a program involving fixed-interval drinking decisions (FIDD). Men-

delson and Mello[33] had noted that although the programmed administration of alcohol permitted precise control over a subject's alcohol intake, such a procedure bore little resemblance to the way in which an alcoholic drinks outside of the laboratory. Similarly, although the free-choice drinking paradigm was particularly suitable for the study of spontaneous drinking patterns, it was somewhat limited when drinking decisions became the focus of investigation. In another respect as well, free-choice drinking may not have been entirely free, for in all of the experimentally induced intoxication studies noted previously, the alcoholics were recruited to serve as experimental subjects. Thus, presumably, they were "supposed" to drink. Finally, demand drinking, even over a considerable period of time, may result in very few spontaneous drinking decisions. Once drinking has commenced, for example, the patient with alcohol always available might make one decision to drink; then, becoming drunk, he/she might remain drunk and oblivious throughout the program. Thus, subsequent decisions might be made when the individual's critical faculties are considerably impaired or when further drinking is prompted by the discomfort of partial withdrawal.

In the FIDD program, the decision to drink or not to drink became the focal point of the endeavor and a major component of their concurrent multidisciplinary therapeutic effort. Gottheil *et al.*[137] have suggested that alcoholism provides a special opportunity for the study of a psychiatric disturbance. If the subjects are allowed to decide whether to drink or not, then, instead of the honeymoon effect of enforced abstinence, their patterns of drinking may be observed, and factors and cues that affect their decisions may perhaps be identified. Personality and behavioral correlates of drinking and their temporal relationships may be assessed, and studies undertaken of positive and negative reinforcers. The most promising aspect of a research model in which the patient at fixed intervals may decide to drink or not, however, would seem to be that both patient and therapist may observe the effects of any experimental or therapeutic maneuver on day-to-day drinking behavior.

The FIDD program, which is still operating at the Coatesville Veterans Administration Hospital (Pennsylvania), is conducted entirely on a closed ward. During a 1-week predrinking phase, the patients are introduced to the ward routine in groups of about ten; they are required to undergo a number of psychological investigations, and the treatment program is begun. The next 4 weeks constitute the drinking phase, during which the patients may elect to abstain or to drink 1 or 2 oz of 80-proof ethyl alcohol, neat or with water, each hour on the hour from 9:00 a.m. to 9:00 p.m. Monday to Friday. This schedule provides 13 decision points daily and a maximum intake of 26 ounces of alcohol. The BAC of each patient is measured four times daily and additionally if it appears that any drinker is becoming particularly intoxicated (an upper BAC limit is set at 250 mg/100 ml).

Throughout the drinking phase, the staff adopts a neutral attitude regarding alcohol, neither encouraging nor discouraging its use. They do, how-

ever, encourage the patient to think about his problem and about his drinking decisions. During the sixth and final week of the cycle, the initial psychological assessment sequence is repeated, and discharge, transfer, or other posttreatment plans are developed. The therapeutic program surrounding the drinking decisions component includes individual and group therapy, physical therapy, administrative ward meetings, and educational and religious seminars. Alcoholics Anonymous meetings, music therapy, marital casework, and recreational and occupational therapy also are included.

Gottheil *et al.*[136] reported the results of the first three cycles of the FIDD program which comprised 29 patients. Overall, these investigators observed a braoder range of drinking behavior than had been noted in previous studies of experimentally induced intoxication, and they also noted a greater degree of control by their subjects after drinking than had been anticipated. In the first analysis involving 25 cases, 36% drank moderately throughout the drinking phase, and 16% took almost every drink every day. Some patients drank mainly in the evenings, some took a drink before meals, and some took only two or three drinks to test their control.

Interestingly, in each of the three groups, there developed the attitude that group members should stop drinking. Social pressure to abstain was exerted on those patients who continued to drink, and social support was provided to those who were attempting to stop. In contrast, several patients developed the rationalization that they were drinking only to help the research. Despite repeated attempts to correct this misinterpretation, some patients persisted in this belief. Further, the investigators reported that their patients did not like drinking under ward conditions; they complained about taking straight ethanol, and they objected strongly to being limited to one drink in an hour. They asserted that if they could drink only in that manner outside the hospital, they would choose not to drink at all.

Follow-up information at 6 posttreatment months was obtained on 92% of the subjects in these first three groups. The results indicated that only two cases had remained completely abstinent since discharge (This result is not too surprising when one considers that abstinence had not been presented as a treatment goal of the program.) However, 52% of the patients polled at follow-up reported that they were drinking less than twice a week, and 70% indicated that they had not been intoxicated more than once in the preceding month. Patients who did not drink during the program tended to do better than patients in the other two groups, but this trend did not prove statistically significant.

A series of reports have been published by the Coatesville team as they have studied the behavioral responses of additional subjects within the FIDD model. Gottheil[138] reported the results of subsequent analyses comparing the nondrinker, "stoppers," and continuously drinking patients. There had been no previous data to help predict the effects of resisting available alcohol while on a closed ward in the presence of other drinking patients, and so it was

anticipated that this situation might be difficult and anxiety provoking. Gottheil reported, however, that the discomfort scores of the nondrinking patients decreased significantly during the study, that the extent of their sleep disturbances remained unchanged, and that their self-esteem scores increased significantly. Subjects who stopped drinking during the program reported no strong craving for alcohol on discontinuing, and no change was noted in their sleep disturbance indices. Instead, they reported a decrease in discomfort scores and a marked increase in self-esteem scores. In contrast to the results of Mendelson *et al.*,[30] Vanderpool,[139] and Mello and Mendelson,[140] Gottheil reported no significant changes in discomfort, sleep disturbance scores, or self-esteem scores in the continuously drinking group (see also ref. 141). This was interpreted as possibly being caused by the treatment aspects of the program.

Of significance also was the finding that the three drinking subgroups did not differ with respect to age, education, marital status, occupational level, or past drinking history of the subjects within them. Additionally, subjects' base-line self-esteem, discomfort, and sleeping difficulties scores also did not discriminate those who chose to abstain, to drink and then cease, or to drink continuously. Further, Gellens *et al.*[142] examined their patients' scores on the Eysenck Personality Inventory, the Drinking Behavior Inventory, the MacAndrew Alcoholism Scale, and several other measures. These scores also were found to be nonpredictive of the drinking practices of individual patients during or following the FIDD program.

As increasing numbers of patients have completed the FIDD program, additional outcome data have pointed to a relationship between drinking during the program and subsequent functioning. Skoloda *et al.*,[143] for example, reported a follow-up based on 91 of 98 patients who had completed 6 post-treatment months. Fourteen patients (15%) abstained completely throughout the follow-up period, and 18 (20%) reported that they had not used alcohol in the preceding 30 days. Further, 26 (29%) stated that they were drinking twice weekly or less. Analyzing the comparisons between program nondrinkers and drinkers, these investigators noted that program nondrinkers were more likely than drinkers to be abstinent at follow-up and that they generally tended to drink less often following treatment than either moderate or heavy drinkers. Nevertheless, many patients who drank during the program also appeared to benefit from treatment. Interestingly, although drinking during the program predicted subsequent drinking, with one exception, family relationships, program drinking failed to predict psychosocial adjustment at the 6-month follow-up.

Alterman *et al.*[144] have presented the most recent follow-up data available from the FIDD program. By the time of this report, 249 subjects had passed through the program and follow-up data at 6, 12, and 24 months were available on 87, 81, and 67%, respectively, of this total. First, these authors noted that 20.9% of all those who drank on the program consumed no more than 10% of the alcohol available to them throughout the drinking period. Thus, a small

percentage of the subjects were demonstrating moderate drinking under the conditions of the program.

The 12-month treatment outcome was evaluated in terms of frequency of drinking and frequency of intoxication during the preceding 30 days. The data were analyzed in terms of program abstainers (n = 120), drinkers who never consumed more than 8 oz of alcohol daily (low-maximum, n = 19), and drinkers who exceeded an 8-oz daily program maximum (high-maximum, n = 110). The results indicated that the patients in the low-maximum group did as well, or nearly as well, as did program abstainers and that both of these groups appeared to be functioning considerably better than patients in the high-maximum group. Thirty-four percent of the abstainers and 32% of the low-maximum drinkers reported being abstinent during the 30-day window, whereas only 20% of the patients in the high-maximum group reported abstinence during the 30 days preceding the follow-up. Perhaps more significantly, 50% of the abstainers, 53% of the low-maximum drinkers, and only 28% of the high-maximum group reported not having been intoxicated over the preceding month.

Results at the second posttreatment year were quite similar to those at the first-year follow-up, although there had been some decline in the relative proportions of patients drinking at what might be considered "acceptable" levels. Alterman *et al.*[144] do make the point, however, that the 2-year results are somewhat different from those obtained at 6 months. At 6 posttreatment months, the evidence indicated that abstainers fared somewhat better than moderate program drinkers. Although the two-year findings continued to suggest a relationship between program drinking and treatment outcome, the previous distinction between abstainers and moderate drinkers no longer appeared tenable.

Although the follow-up results of the FIDD program indicate in general that program abstainers or moderate drinkers fared better than did other drinkers, methodological limitations notwithstanding, it is unclear whether the better outcome of the abstainers and limited drinkers resulted from the nature of their drinking behavior *per se* or from the other, possibly ego-enhancing, effects of voluntarily refusing alcohol over a period of up to 20 days.

Thornton *et al.*[145] undertook a comparison of outcome measures on 30 "involuntary" abstaining patients (who underwent treatment within the FIDD program but were required to abstain for medical reasons) and 93 drinking decisions patients at 6 posttreatment months. Of this latter group, 46 patients were voluntary abstainers, and 47 chose to drink in varying degrees. When the results of the total drinking decisions group were compared with those from the involuntary abstainers, no differences were noted on any of the six alcohol-related indices used. When the drinkers and abstainers within the drinking decisions group were compared with the involuntary abstainers, however, the latter subjects reported a more favorable outcome than did the drinkers but a less favorable outcome than did the voluntary abstainers. Compared with the voluntary abstainers, the involuntary abstainers reported a

significantly lower abstinence rate during the last month of the follow-up period, they tended to drink earlier in the follow-up period, and fewer remained abstinent throughout the entire follow-up period.

With some caution provoked by the lack of an adequate experimental design, Thornton and his colleagues concluded that their findings provide additional support to the notion that undergoing the process of deciding to drink or to abstain within therapy contributes significantly to improvements noted following the completion of therapy. It is to the research addressing the issue of drinking versus abstinence as a goal in the treatment of alcohol dependence that we now turn.

10.3. Restricted Drinking as a Treatment Procedure

Until this point, treatment-oriented research has been presented only *en passant* in this chapter. Further, in those instances when treatment has been mentioned, its goal typically has been lifelong abstinence from all alcohol. Yet we also have indicated that the research that we have examined has provoked a questioning of some of the tenets of the traditional perspective of alcoholism. Moreover, in at least some circles, this questioning has brought a further questioning of the necessity and/or the appropriateness of the fundamental commitment to abstinence as the only acceptable treatment goal for all alcoholic patients. There are, for example, data suggesting that abstinence does not insure adjustment or well-being in all alcoholic patients.[36,37,102,103,146–150] There also are data indicating that alcoholics who commit themselves to abstinence and violate that goal do not necessarily fail to benefit from treatment.[152,153] Finally, there are data showing that at least some diagnosed alcoholics are capable of engaging in restricted drinking following treatment aimed toward abstinence.[37,93,97,98,101,154–158]

The work of Lovibond and Caddy[118] was the first treatment-oriented research specifically aimed at training alcohol-abusing patients to restrain their drinking rather than to abstain entirely. Their procedure, known as discriminated aversive conditioning, employed an electric shock-based punishment paradigm which was administered contingent on their subjects' BACs exceeding 65 mg/100 ml. This procedure was administered as part of a broad-spectrum behavioral treatment program. These investigators randomly assigned 31 alcoholic subjects to the experimental procedure and 13 subjects to an additional noncontingent shock condition. At the 12-month follow-up, these authors reported 21 of the 28 experimental subjects who completed the study to be "completely successful," with an additional three cases "improved." In the control condition, a particularly high drop-out rate (8 of 13) neither permitted nor required the application of statistical procedures to evaluate the differential treatment effects.

In a subsequent study, Caddy and Lovibond[158] conducted a components analysis of their previously developed program. In this second study, 60

alcoholic subjects completed treatment following random assignment to one of three treatment groups: an aversion plus self-regulation group, which was identical to the original procedure but excluded the use of shock, and an aversion group, which was similar to the original procedure but omitted the self-regulatory procedure. The results immediately after treatment showed subjects in the aversion plus self-regulation group to be progressing best, with 85% making some gains and 65% in the highest success category. The self-regulation group demonstrated similar though less striking improvement, and the aversion condition showed the least improvement, with only 20% of subjects in the highest success category and 55% showing some improvement.

Six months after treatment, the aversion plus self-regulation group again showed intermediate improvement, with subjects in the aversion only group again considerably less improved. Only 37 patients had completed 12 post-treatment months at the time of the publication, yet the trends apparent in the earlier data were maintained at this time also. The removal of the shock aversion component from the entire package resulted in a modest decline in overall improvement (60% versus 80% improvement at the sixth month and 65% versus 76% at the 12th posttreatment month). There also was a trend noted in these data for the subjects in the self-regulation group to perform better than subjects in the aversion group based on a criterion of relapse, although this trend did not prove statistically significant.

In a somewhat similar study, Vogler *et al.*[133] found that the results from their broad-spectrum program, which included electrical aversion, were somewhat superior (though in most cases not significantly so) to the results they reported in a group that received alcohol education and behavioral counseling only. A subsequent, and again similar, study by this group,[159] but this time employing a sample of nonalcoholic problem drinkers, concluded that subjects in the group receiving electrical aversion showed no greater improvement than did subjects in two other groups who received similar broad-spectrum behavioral programming but without the aversion therapy.

Miller and his colleagues also have explored the goal of restricted drinking with alcohol-abusing clients.[160–164] These investigators have reported the development of a comprehensive behavioral self-control package that included (1) determining the appropriate limits for alcohol consumption via an educational approach combined with specific BAC discrimination training, (2) self-monitoring of alcohol consumption, (3) rate control training, designed to alter the topography of the drinking behavior, (4) self-reinforcement to encourage ongoing progress, (5) functional analyses of drinking behaviors with training in stimulus control techniques, and (6) alternatives training, designed to teach coping skills to be used in situations in which alcohol previously had been used. Miller[163] compared the results of this package with two alternative approaches, an electrical aversive conditioning procedure, and a multifaceted program incorporating techniques derived from Lovibond and Caddy[118] and Sobell and Sobell.[151] Miller reported no significant between-group differences

during the course of a 12-month follow-up. The author noted, however, that the alternative treatment procedures consumed far more therapist time than the more economical behavioral self-control procedure.

In a second study, Miller *et al.*[165] compared two different approaches to behavioral self-control training: a bibliotherapy (minimal therapist contact) condition and a paraprofessional therapist-administered self-control training program involving ten weekly sessions. Again, no significant differences were found between these two conditions, although here the follow-up period was only 3 months (see also refs. 166, 167). Finally, in the most recent report from this group,[168] a comparison between bibliotherapy with a behavioral self-control orientation and two more extensive broad-spectrum behavior therapies was described. These authors report in a preliminary analysis, done at 6 months following treatment, that no statistically significant differences between their groups emerged. And, as was true of Miller's other studies, about 70% of the subjects assigned to the behavioral self-control training program made significant improvement.

At this point, the restricted drinking research studies, exploring the impact and outcome of administering alcohol to alcoholic subjects, tend to merge with studies employing behaviorally oriented treatment strategies in which alcohol use is authorized in attempts to develop controlled drinking behavior. Although these behavioral approaches, exemplified in the work of the Sobells and their co-workers,[151,169–172] Alden,[173] Pomerleau *et al.*,[174] Sanchez-Craig,[175–177] Marlatt,[178] Caddy,[179] and others, do not fall within the purview of this chapter, they are mentioned here because they are viewed by many as outgrowths or applications of the research findings previously described.

Suffice it to say that during the 1970s, the behavioral therapies seem to have changed from narrow-band single-component approaches to more broad-spectrum endeavors. Simplistic and generally inadequately conceptualized behaviorally influenced treatment programs have for the most part given way to more genuinely integrated behavioral programs based on a multivariate perspective and emphasizing patients' unique clinical histories and present dynamics. Some of these programs have reported promising outcome results. We need more well-designed studies of these approaches to determine the relative effectiveness of the different programs: which components of the programs are helpful and which are not, which components of which programs are effective for which types of patients, and, most importantly, which particular patients are suited for which programs?

11. Discussion

Of necessity, this review has been selective rather than exhaustive. For example, we did not focus on the interesting studies of McClelland and his co-workers[180] on the relationship between power motivation and drinking, of Rubin *et al.*[181,182] on pupillometry as a possible predictor of drinking behavior,

of Jones *et al.*[183] on the differential responses of males and females to alcohol, and many others. Nor did we report on the many physiological and biochemical findings that have emerged from studies of the acute and chronic administration of alcohol to alcoholics and nonalcoholics. Instead, we emphasized psychological, social, behavioral, and clinical studies that generally have been more controversial in terms of their implications for commonly held notions and theories about the effects of alcohol and the nature of alcoholism.

Many of the studies that have been described would have been unthinkable as little as 15 years ago. Clearly, however, a great deal has been learned from observations of alcoholics in the presence of alcohol under a variety of controlled conditions. New and often surprising findings have emerged, previously untested ideas have been challenged, and more and more questions are being translated into testable hypotheses. We firmly believe that, even though earnestly held traditional beliefs may be disputed, such research should not only be permitted but encouraged and supported.

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9A

Current Status of the Field: Contrasting Perspectives

The Behavioral Therapist's View

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Abstract. Over the past decade, behavioral research and treatment approaches to alcohol problems have become an integral part of the alcohol field. Particularly valuable contributions have included the provision of alternative treatment methods and goals for nonaddicted problem drinkers, the development of relapse management and prevention techniques, the refinement of assessment and treatment outcome evaluation methodologies, and an overall enhancement of our understanding of the nature of alcohol problems. Viewing the progression of research studies over the last 10 years, it is evident that the behavioral perspective on alcohol problems has matured beyond ideological boundaries. The greatest strength of behavioral approaches relates to a reliance on the scientific method. Most likely, behavioral approaches will continue to contribute meaningfully to the field of alcohol studies.

1. Perspective

In the early 1970s, the alcohol field experienced a strong influx of behavioral research and treatment approaches. With reports of early successes, these approaches held strong promise for the treatment of alcohol abusers. A decade later, we must ask whether behavioral approaches have, in fact, lived up to these early expectations. From the following review, it should be evident to the reader that in slightly over one decade, behavioral treatment approaches have had a marked impact on the alcohol field. Behavioral approaches and methods have become accepted as reasonable clinical procedures, and behavioral research has substantially contributed to knowledge about the nature and treatment of alcohol problems.

By reviewing the status accorded behavioral treatment approaches in the series of special reports on alcohol and health prepared by the National Institute on Alcohol Abuse and Alcoholism,[1–4] one can quickly gain an appreciation of their impact over the years. Readers familiar with the process by which the NIAAA reports were prepared and reviewed for policy and political

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implications will likely agree that statements in these reports are cautious and conservative appraisals of the state of the art in treatment. In the first report, in 1971, behavioral treatments (other than aversion therapy, which predated the field of behavior therapy by many years[5]) were described as having ". . . only begun to be developed experimentally . . ." (p. 75). The next report, in 1974, indicated that such methods were " . . . still experimental . . ." (p. 152), and by 1978 it was clear that behavioral treatments had gained clinical as well as research acceptance: "The trend in behavioral approaches is away from development of theory in favor of comprehensive, eclectic treatment plans . . ." (p. 75). Finally, by 1981, behavioral treatments were fully acknowledged as a major treatment orientation: "There appears to be an acceptance of behavioral therapy techniques, if not theory (p. 152)." Also, the 1981 report described behavioral assessment and treatment as one of only three areas of "Refinements in Treatment Methods," the others being pharmacotherapy and family therapy. It should also be noted that behavioral studies are well represented among the newer family therapy approaches.[6–8]

No doubt, recognition of behavioral treatments by the alcohol field was greatly facilitated by the conduct of sound research. At a time when the value of any treatment for alcohol problems is unclear,[9–11] behavioral methods have gained acceptance because they have been used in some of the few well-designed studies found to demonstrate positive treatment effects. For example, Emrick[12] reviewed the relative effectiveness of alcohol abuse treatments for the Department of Health, Education and Welfare's Alcohol Program and Policy Review Project. He evaluated all (there were 90 total studies) "randomized controlled trials reported in the English treatment evaluation literature from 1952 to 1978" (p. 71) and arrived at only two conclusions[12]:

> (1) the effectiveness of nonbehavioral treatments is not increased by giving more than very brief care when such therapy is applied to heterogeneous groups of alcohol abusers, and (2) some behavioral approaches have been found to be relatively effective in reducing problem drinking. (p. 82)

In earlier reviews, Emrick[13,14] had concluded that although any treatment seemed to have limited beneficial value for persons with alcohol problems, it made little difference what treatment orientation or procedures were used.

Given the above, it would be fair to say that over the past decade behavioral treatment methods have gained acceptance as reasonable clinical procedures that have empirical support in the treatment research literature. The contributions of behavioral investigators have gone far beyond treatment techniques, however, and over the long run, these ancillary contributions are likely to influence the way we think about alcohol problems and to enhance our understanding of alcohol problems.

Behavioral research has been coincident with and has augmented a radical change in the way alcohol problems are conceptualized.[15,16] Laboratory demonstrations that even chronic alcoholics could control their drinking under

appropriate circumstances (reviewed by Caddy and Gottheil[17]), coupled with repeated published reports that many individuals with alcohol problems could successfully acquire a pattern of nonproblem drinking (reviewed by Sobell[18]), have led to an increased emphasis on the role of learning factors in the etiology of alcohol problems. It is interesting that this research emphasis was actually foreseen by Jellinek, who noted[19] that ". . . a learning theory of drinking in the well-defined terms of psychological discipline is essential to all species of alcoholism, including addiction" (p. 77). He also noted that the learning and disease approaches were complementary rather than antagonistic to one another. Thus, the recent research on the role of learning factors in drinking problems is not as great a departure from tradition as it first appears.

The ways in which behavioral research has contributed to the alcohol field are surprisingly diverse and go well beyond the simple development of treatment techniques. One of the major contributions has been the development of treatments for problem drinkers—individuals who have distinct alcohol problems but who do not drink so as to become physically addicted to alcohol and whose overall impairment usually is less severe than that of more chronic, addicted alcoholics. Although epidemiologic studies have repeatedly shown that there are a vast number of such individuals (e.g., Cahalan and Room[20]), problem drinkers tend to be underrepresented in formal treatment programs and Alcoholics Anonymous. Men in their early 20s, for example, have the highest incidence of alcohol-related problems,[20] yet constitute a disproportionately small proportion of admissions to treatment programs[9] as well as of those attending Alcoholics Anonymous meetings.[21] Behavioral clinical research has clearly been at the forefront of efforts to develop treatments specifically designed for this population.[22–24] Presumably, these efforts relate to research on the use of nonabstinent goals, since the available research, although correlative in nature, indicates that nonproblem drinking outcomes are more often achieved by persons who have less serious drinking problems at the start of treatment.[18]

Why nonbehavioral research has failed to make substantial contributions to the treatment of problem drinkers is unclear, as there is no *a priori* reason why nonbehavioral approaches would not be effective. Clearly, the need for such services exists, and the provision of services is most consistent with an emphasis on secondary prevention (which has gained some favor in the field). One possible reason for the relative lack of nonbehavioral programs for this population is the widespread belief that alcohol problems are progressive in nature and that interventions need to be focused on "raising the bottom" rather than on providing a different type of treatment for persons with less serious problems. The available data, however, suggest that for the majority of persons with alcohol problems, the disorder is not progressive, but simply that problems will be recurrent with varying levels of severity.[10,15] Whatever the reasons, behavioral approaches have offered some of the few clear alternative treatments tailored specifically to problem drinkers, and the early evidence regarding the effectiveness of these treatments is very encouraging.[22]

Another major contribution of behavioral approaches to the treatment of alcohol problems is premised on three conceptual thrusts in behavioral research. These are (1) Bandura's[25] separation of treatment effectiveness into immediate treatment effects, their generalization, and their maintenance, (2) the development of cognitive behavior therapies and research on the role of cognitions in explaining alcohol use and abuse,[26] and (3) Marlatt's combining of these factors into a conceptualization of the relapse process.[27,28] These lines of thought led to research on the precipitants of relapse and to the development of methods of relapse prevention and management. The treatment outcome literature convincingly documents that relapse, or continued problems, tends to be the modal outcome for most treatments even when clients show some improvement over time.[10,13,14]

Curiously, once again, there seem to be few nonbehavioral approaches that specifically address the problem of relapse, a problem that seems central to the treatment of alcohol problems. Generically, of course, all treatments are aimed at relapse prevention, but few treatments have been specifically developed based on conceptualizations of the mechanisms involved in the relapse process and with the explicit purpose of providing clients with ways of dealing with relapse situations. Perhaps behavioral researchers have taken the lead in relapse prevention research because they are willing to accept the data indicating that multiple relapses occur in most cases, and that improvement can be conceptualized most realistically as a gradual reduction in alcohol problems rather than an immediate absence of further problems following entry into treatment.

An emphasis on the importance of cognitive factors is central to many of the newly developing behavioral treatments (e.g., relapse prevention). There are probably two reasons for this. First, the influence of cognitive factors on behavior has become an area of intensive behavioral research over the past decade.[29,30] Second, the alcohol field was truly ripe for such research, since strong and diverse beliefs exist about whether persons who have drinking problems can control their drinking. Cognitions regarding control, for instance, were found by Orford[31] to be related to nonproblem drinking treatment outcomes. Finally, this research emphasis has been considerably strengthened by Bandura's[32] restatement of social learning theory to include self-efficacy as a mediator of behavior change. Perceived self-efficacy is a person's personal expectation that he or she can effectively execute a particular behavior pattern. Efficacy expectations, likewise, are hypothesized to be a function of several other variables including cognitive appraisals of the difficulty of enacting a given behavior pattern in a specific situation and whether the individual has successfully coped with similar situations in the past. Thus, beliefs regarding personal control over behavior and one's ability to cope with risk situations have become pivotal conceptual foci in the development of relapse prevention and other behavioral treatments. Needless to say, results from such research, whether or not supportive of self-efficacy therapy, are likely to enhance our understanding of the etiology and character of alcohol problems.

There are many other ways in which behavioral research has contributed to the alcohol field. The development of evaluation methodology has clearly had a marked impact on the way treatment outcomes are evaluated.[33] Measurement of drinking has become much more precise, and the validity of alcohol abusers' self-reports has been extensively evaluated. In general, it has been determined that, in contradiction to popular beliefs, alcohol abusers' self-reports of drinking and of drinking-related events are quite valid when gathered in an appropriate clinical setting, unless the individual is intoxicated when providing the reports. Here we have another example of behavioral researchers having pioneered research that should have been conducted long ago. It is paradoxical, if not ludicrous, that for so long the prevailing belief was that alcohol abusers' self-reports could not be trusted, although in nearly all treatment programs, self-reports constituted the major or sole source of data on which clinical assessments and treatment outcome conclusions were based. We believe that these sorts of contributions by behavioral researchers to the field of alcohol studies are best considered as simply reflecting the application of sound scientific method to fundamental research questions.

The technology that has evolved from these studies, used in combination with other technologies and orientations, now allows broader lines of investigation, such as the relationship of daily drinking behavior to various liver function tests. In this sense, it also can be said that the interests of behavioral investigators have broadened considerably over the past several years so that behavioral methods have become part of a more eclectic approach to the study of alcohol problems—one predicated more on empirical clinical science than the narrow application of learning theory to explain all the nuances of alcohol problems. Behavioral research has increasingly provided useful tools for biomedical research, epidemiology, and other approaches to alcohol studies.

A final contribution of behavioral approaches has been to increase our understanding of normal drinking.[34–36] This research may well have a future impact in the area of prevention,[37] an area that has recently become a national priority.[3,4] Studies intended to identify sources of influence on drinking are likely to have value for the developing of prevention strategies.

To be sure, behavioral treatment of and research on alcohol problems have their deficiencies. The most notable problem is a relative lack of well-controlled clinical treatment trials that include long-term follow-up. This problem is not confined to behavioral research or to the study of alcohol problems, however. It is a broad deficiency in clinical research that probably relates to the academic structure within which most clinical research is conducted.[26] A great many pressures militate against conducting this kind of research: research that is necessary to answer clinical questions but is time and effort consuming as well as relatively high risk (in terms of personal career advancement). Without fundamental changes in the support system so that stable resources and increased incentives are provided for the conduct of meaningful clinical research, it is unlikely that any field of clinical research will realize its full potential.

2. Summary

Behavioral research has had a marked impact on the field of alcohol studies and on the treatment of alcohol problems. In just over one decade, behavioral approaches have gained acceptance as a legitimate approach to the treatment of alcohol problems, and behavioral research has broadened our understanding of alcohol problems. In this sense, behavioral approaches have matured rapidly and are now considered part of the mainstream in alcohol treatment research and practice; behavioral researchers, likewise, have matured in that they recognize that a comprehensive (i.e., biopsychosocial) view of alcohol problems is a necessary precursor for sound behavioral research.

The greatest strength of behavioral research has been its overriding emphasis on good science. This is not to imply that science methodology had been lacking in the alcohol field, but in the area of clinical research, it was surely in short supply. To the extent that behavioral research contributes meaningfully to our understanding of the nature and treatment of alcohol problems, everyone benefits—most of all, people with drinking problems. Such an impact is the ultimate goal of all approaches and must transcend disciplinary and ideological boundaries. As these trends continue, we hope that such boundaries will increasingly come to be viewed as a thing of the past. Based on the foregoing, we have considerable enthusiasm for what the future holds.

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9B

Current Status of the Field: Contrasting Perspectives

The Future of Behavioral Interventions

S. H. Lovibond

Abstract. It is suggested that in the future alcohol abuse is likely to be treated as part of a program of general health management designed to change a number of health-related behaviors simultaneously. The overall program will emphasize detailed contingency and stimulus management procedures in the early stages, with control passing to generalized reinforcers and new cue stimuli as training proceeds. The client/patient will be an active participant from the start and will be given ultimate responsibility for managing his/her own behavior. There is likely to be a resurgence of interest in aversive procedures as a means of assisting behavioral self-management.

1. Introduction

Developments within the field of behavioral control of excessive drinking over the past decade have paralleled developments in other areas of behavioral intervention. It is not unlikely that similar trends will be observed over the next decade. Hence, it is of interest to question whether it is possible to discern developmental trends within the wider field of behavioral change that may suggest future shifts in emphasis in the field of alcoholism.

In the early years of behavior therapy, the emphasis was very clearly on the application of single techniques or procedures. In almost all cases, the problems tackled were relatively circumscribed, and, for the most part, the medical model was implicitly accepted. Thus, the patient or client was allotted a passive role. In the last decade, the range and complexity of problems tackled has increased to the point where virtually all behavioral problems have become the focus of behavioral intervention programs. At the same time, multimodal interventions have become the norm, and the most favored procedures are no longer direct conditioning techniques. Usually the medical model

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is rejected, either explicitly or implicitly, and the patient or client is an active participant in the total program.

As a natural further development, the focus of attention is shifting from exclusive concern with problems of psychological health to problems of physical health that directly reflect the individual's lifestyle, i.e., eating habits, levels of physical activity, use of tobacco and other drugs, and self-imposed stress.

At the same time, increasing interest is being directed towards the possibility of instituting not merely preventive measures but positive health management in individuals who are clinically healthy. Thus, instead of tackling, say, obesity or cigarette smoking as a single problem, each problem is targeted as part of an overall health management package.[1]

I believe the time is ripe for a similar shift in our orientation to problem drinking. Instead of directing our multifaceted programs towards control of drinking behavior, we would, in consultation with the client/patient, design a behavioral program of general health management to include training in appropriate dietary habits, abstinence from smoking, adequate exercise, and control or elimination of alcohol use.

There is, of course, abundant evidence that the behavioral excesses of alcoholics are seldom limited to alcohol abuse. Indeed, there is related evidence that indulgence in appetitive behavioral excess is a quite general factor of behavior. Hammond's longitudinal study[2] of 350 males over a period of more than 20 years from age 10 is highly relevant. A wealth of data from multiple sources provides a striking picture of some aspects of behavioral development. In particular, a clear-cut factor of indulgence in a range of appetitive behaviors emerges. Heavy drinking, heavy smoking, and experience with other drugs all show high loadings on the self-indulgence factor. The young men with high self-indulgence factor scores are not yet alcoholics, but it is a reasonable hypothesis that future alcoholics in the total sample will come from their ranks.

Support for such a proposition comes from a number of other sources. For example, in Holloway's detailed study of 50 alcoholics,[3] it was found that not a single one was a nonsmoker, and there was evidence of excessive use of drugs other than alcohol in 29 of the 50 cases. Studies of drinking drivers (e.g., Raymond[4]) reveal a similar pattern.

Several conclusions can be drawn:

1. In only a small proportion of alcoholics is abuse of alcohol likely to be the only life-style component that poses a serious health risk.
2. The early-stage alcohol abuser is likely to be as reluctant to give up other behavioral excesses as he is to give up alcohol. Consequently, only the threat of legal sanctions (arising, for example, from a drinking–driving offence) or a clear and imminent threat of pronounced deterioration in physical health is likely to get the early al-

cohol abuser into a program designed to encourage positive life-style changes.

3. Behavioral strategies directed towards change of appetitive motivation are likely to be required.

In view of the difficulties posed by control of alcohol abuse alone, it may at first sight appear paradoxical to attempt to control a wide range of health-related behaviors, including excessive drinking, simultaneously. There are, however, several lines of evidence that suggest the adoption of such a strategy.

First, and most importantly, the behavioral excesses are usually closely interrelated psychologically in the sense that they have interlocking sets of control stimuli. For example, the behaviors of alcohol consumption, eating junk foods, and smoking not only tend to occur in the same situations and thus share common cue stimuli, but often provide cue stimuli for each other. Changing only one of the behaviors may thus leave many of the cue stimuli for that behavior intact.

Second, if the individual is convinced of the need for behavioral change, acquisition of control over a wide spectrum of health-related behaviors simultaneously is likely to result in changes in perceived self-efficacy that will act to further reinforce the new behaviors.

Third, the reinforcing effect of an increased sense of well being is more likely to result from change in a number of unhealthy behaviors.

As health care becomes increasingly a matter of modifying health-disrupting behaviors and establishing health-promoting life styles, it is evident that behavioral management programs must assume increasing importance. The potential role of the behaviorally sophisticated psychologist is highlighted by the very limited success of nonbehavioral public health programs directed towards life-style change.

2. Specific Techniques

2.1. Social Skills Training

As Caddy[5] has noted, social skills training and, in particular, assertiveness training have received increasing attention in the behavioral literature over the past 10 years. Many workers in the field of alcoholism have included some form of social skills training in their treatment programs as an adjunct to other procedures. Intagliata,[6] however, found that social skills training conferred only marginal benefits, and there was a marked tendency for subjects to forget, quite rapidly, significant portions of the training material.

Few workers believe that social skills training alone will ever provide an effective treatment for alcoholism, but as an adjunctive procedure for particular subjects, social skills training makes a lot of sense.

2.2. Cognitive Therapy

The 1970s have also seen a burgeoning of interest in so-called cognitive behavioral therapies in widely diverse fields, including alcoholism. In general, the cognitive therapies emphasize covert events that are assumed to underlie behavior and deemphasize overt behavior. The potential contribution of cognitive strategies has yet to be determined, but it would be naive to believe that procedures such as training the individual to change self-statements will ever, in themselves, provide a sufficient means of controlling excessive drinking. The inclusion of cognitive components in the treatment package is, of course, a different matter. Certainly, the specific procedures used need to be nested in an overall cognitive framework that has meaning for the client/patient.

2.3. Self-Control Methods

Behavioral self-control training has received increasing emphasis in the behavioral change field over a number of years. Self-control training may be regarded as a natural development in the shift away from the medical model of therapy which casts the client/patient in the role of a passive recipient of therapies devised and implemented by experts in one-to-one clinical settings. Acceptance of the principle that individuals ultimately must assume responsibility for their own behavior implies that clients/patients must become active participants in the therapeutic process from the start and must learn to apply behavioral control principles to their behavior not only during clinical sessions but constantly in daily activities.

To date, there have been few studies of either the contribution of the self-control component to the effectiveness of overall treatment packages, or of the critical mechanisms of self-control. There is an urgent need for detailed experimental investigation of self-control training, but there is little doubt that future behavioral change programs will continue to emphasize self-control in one form or another.

2.4. Contingency Management and Stimulus Control

Procedures that involve manipulation of the consequences of the behavior to be changed and control of the stimuli antecedent to the behavior have long formed the basis of most comprehensive behavioral interventions. A trend that is likely to continue into the future is the shifting of responsibility for contingency management and stimulus control onto the client/patient as part of a self-control package. In order to assume responsibility for his/her behavior, the subject must acquire a basic knowledge of behavioral principles, must participate in the process of behavioral analysis and selection of reinforcing consequences and stimulus control strategies, and must carry out the pro-

cedures required by the program, including detailed monitoring and recording.

If such a set of procedures is to be made acceptable to a sophisticated adult, several conditions must be met:

1. As far as practicable, primary emphasis must be placed on the intrinsic reinforcement resulting from subgoal achievement.
2. The magnitude of the behavioral unit to be reinforced must be steadily increased from a small base.
3. The client/patient must be made aware of the possibility of ultimately moving beyond detailed contingency management and stimulus control.

In order to reach the point at which it is no longer necessary to specify, monitor, and reinforce particular behaviors or to employ particular stimulus control strategies, two conditions must be present. First, reinforcers such as a sense of mastery, a sense of well being, and social approval must support the new behaviors. Second, and possibly even more importantly, new forms of stimulus control must have been acquired. Whereas previously many stimuli served as cues for behavioral excesses, as training proceeds, more and more stimuli should become cue stimuli for reduced alcohol intake and other moderate appetitive behaviors. Thus, to the extent that the training program has been successful, stimulus control now will favor behavioral moderation rather than excess.

The generalized reinforcers are, of course, not closely and specifically associated with the new behaviors to be maintained. Consequently, they are less powerful than specific immediate reinforcers. Nevertheless, when acting in conjunction with newly developed control stimuli for healthy behavior, the generalized reinforcers should maintain the new healthy patterns of behavior and free the subject from the necessity to continue detailed self-management procedures. If, however, reversion to old unhealthy behaviors occurs, specific self-management procedures can always be reinstituted until control over behavior is reasserted.

2.5. Aversive Procedures

Over recent years there has been a sharp decline in interest in the use of aversive procedures, not only in the field of alcoholism, but quite generally. The reasons are not far to seek. First, evidence that aversive procedures make a worthwhile contribution to behavioral interventions has not always been found when the issue has been addressed directly (see review by Caddy[5]).

Second, the use of aversive procedures has been identified historically with the medical model of treatment in which an authority prescribes and administers a specific procedure to a passive recipient.

Third, and probably most important of all, aversive techniques have had

a very bad press over the past 10 years. Interestingly, during the same period, laboratory work with both animal and human subjects has thrown a good deal of light on the mode of action of aversive procedures in the control of appetitive behaviors and has heightened our understanding of the circumstances that favor effective aversive conditioning.

Recent work permits the following tentative conclusions:

1. The term aversion therapy is inappropriate. The use of aversive procedures seldom results in conditioned aversions in human subjects. Rather, when they are effective, aversive procedures typically reduce the motivation to perform the behavior to be controlled.[7–9]
2. Conventional thinking seriously underestimates the extent of stimulus control in addictive behavior. Aversive procedures probably act principally by reducing the incentive value of the stimuli that control the addictive behavior. Consequently, emphasis should be placed on pairing of the aversive event with the stimulus/fantasy/behavioral sequences leading to the behavior to be controlled.
3. Because powerful inhibitory controls cannot ordinarily be developed, it is essential that aversive procedures form part of a comprehensive behavioral management plan. Used in such a manner, aversive procedures may often play a useful role in the control of addictive behaviors, including alcohol abuse.
4. The issue of biologically relevant aversive procedures[10] in therapeutic interventions is still unresolved inasmuch as electrical aversive stimuli appear to produce stimulus devaluation[7,9,11] or motivational change in much the same way as emetic drugs. If, however, as suggested earlier, the aversive stimuli are to be paired with stimulus/fantasy/behavioral sequences leading to excessive drinking, only the technique of covert sensitization would seem to offer the flexibility required. Elkins[12] has demonstrated that, with sufficient perseverance, covert sensitization may produce conditioned nausea in the more susceptible subjects. For general use, however, it has been suggested that it may be wise to employ covert sensitization boosted by prior induction of mild motion sickness.[8]

Given the decline in the social movements that provided the backdrop for the attacks on the use of aversive procedures for therapeutic purposes, the lack of new, more obviously potent therapeutic procedures, and the new laboratory data on aversive conditioning, the time may well be ripe for aversive conditioning to be revisited.

Any future use of aversive procedures, however, is certain to form part of a comprehensive behavioral control program in which the individual client/patient is an active participant at all stages. The purpose of aversive procedures in such a program will not be to provide a built-in control over alcohol abuse that relieves the individual of all responsibility for his/her behavior.

Rather, the procedures will be seen as one means open to the individual to assist his/her efforts at self-control.

3. Conclusions

The problem of alcohol abuse should be attacked as part of a general behavioral health management program.

Self-management or self-control should be the principal focus of behavioral intervention, and the client/patient should participate actively in the definition of the problem, the setting of goals, the selection of intervention strategies, and the implementation and evaluation of the program.

Intervention programs should be based on comprehensive and detailed contingency and stimulus management procedures which are phased out when the point is reached at which generalized reinforcers and new cue stimuli are able to maintain the new behaviors.

Reduction of appetitive drive by aversive procedures is likely to be the most generally useful adjunctive procedure in the control of alcohol addiction.

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9C

Current Status of the Field: Contrasting Perspectives

A Medical Clinician's Perspective

Robert A. Moore

Abstract. Because alcoholism is viewed and treated as a bad habit, almost all treatment approaches are covertly behavioral. Behavior therapy as a specific and overt approach to alcoholism is widely written and talked about. Nevertheless, it is not the major approach utilized in the majority of treatment programs. This is a result of a trend toward deprofessionalization in the field manifested by a reluctance to accept ideas not compatible with accepted dogma, a reluctance of professionals dynamically oriented to accept behavioral approaches, and a lack of belief in the efficacy of behavior therapy. It is vital to keep the field open to new ideas and facts; if we do, behavior therapy has a bright future in the treatment of alcoholism.

1. Perspective

In the broadest sense, it is difficult to conceive of an alcoholism treatment program, or even a technique, that does not employ a behavioral approach. Alcoholism is, after all, a form of behavior we wish to eliminate or modify. More accurately stated, the behavior is the drinking of ethyl alcohol.

After all the effort we have spent over the past several decades in arguing that alcoholism is a disease, we remain unable to identify it except by its behavior. Hope continues that biomedical research will ultimately find one or many physiological factors that is or are critical in the development of this disease, probably genetically transmitted. If we are fortunate, we will also be able to develop a "magic bullet" treatment that has laser beam accuracy. When that happy day arrives, we can use a treatment approach that is not entirely behavioral. We do not have that choice at the present time.

When I suggest that all current treatment is behavioral, I do not refer to specific identifiable behavioral therapy techniques. Rather, I refer to the traditional treatment forms that have changed precious little in recent years.[1,2] An alcoholic comes to treatment because his behavior is bothering others and

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possibly himself. If he or she does not get help, there will be unpleasant consequences. A treatment contract of varying formality and sophistication includes a tacit contingency: compliance will lead to a reduction of unpleasant consequences; failure to comply will lead to a continuation or increase in unpleasant consequences. Compliance will also lead to immediate reward—the favor and support of the therapeutic person or staff, of the other patients, and of the family.

If psychotherapy is the, or part of the, treatment, the psychotherapist clearly supports behavior that reassures the psychotherapist of his own competence. The same motivation holds for all involved in the treatment such as group therapists, nurses, activity staff, alcoholism counselors and so forth, and all will subtly or overtly convey their pleasure or disappointment to the alcoholic patient. Alcoholics Anonymous has at its cornerstone of expectations that the alcoholic will become and remain abstinent. Rewards are given in the form of friendly acceptance and "birthdays" for success; for failure, the person is shunned until abstinence is regained. Or, put another way, although AA colleagues seek to assist in that outcome, the full fruits of the fellowship are withheld. When we prescribe disulfiram, the alcoholic is made aware of the dire consequences of acetaldehyde toxicity.

Success or failure in treatment or in spontaneous recovery is met by such contingency variables as continued marriage or divorce, a job or joblessness, freedom or jail, friends or loneliness, self-esteem or shame, life or death. What other disease presents such opposite poles? What it comes down to is that, although we call alcoholism a disease, we really view it as a bad habit and treat it that way. The confusion and contradiction in the field is illustrated by saying this is a disease that does not need doctors manifested by uncontrolled drinking that the victim should stop. Is the uncontrolled drinking a result of alcoholism or is alcoholism the result of uncontrolled drinking?

Having placed behavioral approaches in this context, let us now discuss the role of specific behavioral treatment approaches. They are certainly widely talked and written about, requiring long reviews to encompass the literature. A comparison of the amount of coverage of the subject in the four editions of the *Special Report to the U.S. Congress on Alcohol and Health*[3–6] is only slightly less scientific than much of the treatment literature. The first report had 570 words on behavior therapy, the second 506, the third only 189, whereas the fourth had more than recovered with a figure of 863. Any book or review written about the treatment of alcoholism would be incomplete without a discussion of behavior therapy.[7–10] Pattison states, "Perhaps the most important treatment development of this decade has been behavioral methods."[2] Why, then, is formal behavior therapy not more widely utilized?

To start with, we do not know accurately how widely it is used. It is my impression that as a or the major treatment modality, it is not a part of the majority of programs despite its coverage in the literature.[1] The literature emanates primarily from academic centers or programs located in academic centers. Here are found the professionals who think, research, and write.

Much of this literature is unknown to the treatment staffs of the majority of treatment programs or, if known, is often rejected. There are a number of reasons for this.

In my judgment, the most important reason is the deprofessionalization of the alcoholism treatment field. Although there were some notable exceptions, this field had been ignored by the professional disciplines until after World War II. Alcoholics Anonymous began filling this vacuum in the late 1930s and has continued to do so at an accelerating rate since. In the 1950s and decades to follow, professional treatment personnel were drawn to the field as a new challenge to be met, encouraged by a better reimbursement system and by state and federal funding. An uneasy alliance was established between these two movements, each learning from the other, but each very protective of its territory and treatment modalities. Unfortunately, there is now developing a rejection of professionals in the field. It is not just a matter of demedicalization but a broader move to deprofessionalization. How far this will go is not yet clear, but the present concern with cost cutting suggests that there is more to come. The natural outcome of this is greater standardization of programs around the traditional mixture of AA, alcoholism counseling, group therapy, and alcoholism education. Effective behavior therapy, whatever its form, requires professionals who are trained in these approaches; such people will not be found in the majority of programs.

Another effect of deprofessionalization is the discouragement of new ideas, especially those that are counter to established dogma. New ideas that fit this dogma are welcome, such as research in genetics and the fetal alcohol syndrome. But what about "controlled" drinking? Unfortunately for behavior therapy, it has been linked in the minds of many in the field with attempts to use "controlled" drinking as a treatment objective. The negative reaction to the 1976 Rand report is still fresh in our minds. Whatever the merit of "controlled" drinking as an objective, the more important point is the refusal by so many to even consider this "new" idea. In all candor, I must confess that this reluctance extended to many old-line professionals in the field also. Does this suggest that they have become deprofessionalized? In one sense, yes. Although devoted and skilled in their professional work, all too many have allowed another facet of the professional to atrophy, namely, the eagerness to entertain new ideas and to withhold judgment until the facts are known. Attending conferences and workshops on the treatment of alcoholism is a tedious experience for the most part. Old ideas, some very worthwhile, are rehashed repeatedly, sometimes with new names affixed as though they were new ideas. An outside observer dropping in once a decade would chide us for our lack of movement.

Less important now but still significant are the turf problems between the professional disciplines, in this case, psychology and psychiatry. Strange that I should put it that way, because the issue would be more accurately defined as between the dynamicists and behaviorists. Obviously, there are psychologists and psychiatrists to be found on either side. Still, the overriding

orientation of psychiatrists is dynamic, and behavior therapy is more likely to be in the hands of psychologists. There remains a conflict between these viewpoints, and to the extent psychiatrists influence alcoholism treatment programs, there may be a bias negative to behavior therapy. To caricature the conflict, the behaviorist sees the brain as a black box to be ignored, concentrating on what goes in and what comes out; the dynamicist cares only about the inner workings of the box, not seen as black, and worries not or little about what goes in and what comes out. This is manifested in a viewpoint that should long ago have been extinguished: if the underlying neurotic problems are resolved, the symptom alcoholism will wither away. But perhaps this statement is made in haste. This may be an idea that has a future when we learn more about what actually is in the black box. Fortunately, recently trained psychiatrists are more familiar with and sympathetic to behavioral approaches.

Finally, behavior therapy has not yet been perceived as more effective than traditional treatment. I say "perceived" because that has more impact than whatever the facts may be. Anyone who thinks about it realizes what a monumental task it is to truly determine what treatment is best for a given individual. Alcoholism is a multifaceted disorder manifested by people each as unique as people without the disorder. How do we line up a specific and uncontaminated intervention with a definable personal characteristic and then keep the model pure for a long enough time to make the outcome relevant to the intervention?

Some years ago an interesting conference was held at the National Academy of Sciences in Washington sponsored by the National Institute on Alcohol Abuse and Alcoholism. Concerned by the dearth of new research ideas, a group of Nobel laureates and other noted scholars were assembled to hear about where we were in the hope that their congregated wisdom would open new vistas for investigation. A series of speakers presented the state of the art in the various fields—brain, liver, ethnic groups, etc. My subject was treatment. My question was how do we learn which treatment is best suited to a particular individual. The answer, paraphrased, was "If you don't know which treatment is best for a given person, why not give each person all the treatments?" Why not, except that it is expensive, one treatment may wash out the effect of another, and we will never learn what works best for whom. Behavior therapy will not be advanced as a technique if it is part of a therapeutic smorgasbord.

2. Prospects

Where do we advance from here? It is vital that professionals in the field do that which they find most repugnant: get involved in the politics of treatment. The motivation must not be merely to protect jobs and "turf," although these have certain admirable aspects, but to keep the field from stagnation

and to encourage its receptivity to new ideas. Then the new ideas must be generated, tested, and accepted or rejected. I remain an optimist. As we gradually learn more about the answers to the question with which the Nobel laureates struggled, the field will, must, open up to that knowledge. The two tracts that offer the most hope are behavior therapy and the development of specific biomedical interventions.

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9D

Current Status of the Field:
An Anthropological Perspective on the Behavior Modification Treatment of Alcoholism

David Levinson

Abstract. This chapter discusses behavior modification treatment for alcoholism in terms of the potential influence of cultural factors on the treatment process. It is suggested that cultural factors may be an important determinant of treatment involvement and treatment success. Four categories of cultural factors are identified and discussed: (1) folk beliefs about the cause and treatment of illness; (2) beliefs about control of one's behavior: (3) socialization techniques; and (4) acculturation. It is also suggested that behavior modification treatments, compared to other general treatments, do less harm to an individual's cultural identity while, at the same time, they provide an opportunity for cultural factors to be used in the treatment selection process.

1. Introduction

An anthropological perspective rests on the belief that cultural factors influence the alcoholism treatment process. For the purposes of this discussion, cultural factors are defined as learned, shared, patterned beliefs, values, and behaviors that are characteristic of a particular group of people. That group of people may be an entire society like the United States, a specific ethnic group such as Americans of Irish ancestry, a distinct cultural group such as the Navaho, or specific treatment populations such as women, women who are homemakers, military personnel, adolescents, or the handicapped. The key point about these groups and others like them is that the individual members of each group may share certain beliefs, patterns, or a common group identity which may influence the appropriateness and effectiveness of alcoholism treatment.

From this cultural or cultural factor perspective, we can draw three general conclusions about the behavior modification treatment of alcoholism as it is currently utilized in the United States. First, compared to other treatment

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approaches such as Alcoholics Anonymous and some rehabilitation programs, behavior modification treatments probably do less damage to the cultural identities of the individuals receiving treatment. Alcoholics Anonymous, in particular, implicitly requires members to give up their cultural identity and adopt instead the identity of AA member or recovered alcoholic. Behavior modification treatments, since they often have specific treatment goals aimed at specific behaviors, are most likely to leave a person's cultural identity intact following treatment.

Second, because behavior modification treatments focus on drinking behaviors or behaviors associated with drinking, other behaviors, which are often culturally patterned such as relationships among family members, are left relatively undisturbed. However, in this regard, it should be noted that the techniques or goals of treatment must mesh with group norms. Heath,[1] for example, points out that assertiveness training would not be an appropriate treatment choice for Pueblo excessive drinkers, as the Pueblo social order is based on interpersonal cooperation and emphasizes group rather than individual needs.

Third, cultural factors seem especially relevant to the selection for treatment process in behavioral treatments. Cultural factors seem especially relevant for three reasons: because behavioral treatments taken as a group and broad-spectrum approaches in particular offer a wider range of treatments and utilize a wider range of treatment techniques than do other standard treatments for alcoholism, because behavioral programs offer a wide range of treatment goals including behavioral change, abstinence, and controlled drinking, and because behavioral programs are offered in a variety of settings such as inpatient facilities, outpatient clinics, and the community. In short, because behavioral programs are especially broad in approach and goals, they are especially suitable for utilizing cultural factors in assigning individuals to appropriate treatments and in selecting appropriate treatment goals.

The three general conclusions about behavioral treatment listed above are based on the assumption that cultural factors matter in the treatment of alcoholism. I say this is an assumption because there is actually little empirical evidence that demonstrates a link between cultural factors or cultural identity and treatment involvement or outcome. Of course, there is also no evidence that suggests that cultural factors do not matter. The simple truth is that there has been very little research conducted to date that examines the role of cultural factors in alcoholism treatment. This is especially regrettable since the alcoholic treatment population in the United States and other complex societies is a highly diverse group composed of people from a variety of cultural and other special populations.

To a large extent, our lack of knowledge about the role of cultural factors in alcoholism treatment is the result of the often mutually exclusive interests of anthropologists, on the one hand, and treatment providers, on the other. As Waddell[2] notes:

> Treatment-oriented persons see the people who are more likely to be in "suspended ethnicity," where culture is a secondary issue and treating the immediate psychophysical problems is more critical. Field anthropologists, on the other hand, see ethnicity or cultural factors as a primary consideration in any treatment mode. The clinician sees persons as related to symptoms, the anthropologist sees persons as culture bearers.

Evidence concerning the degree to which cultural factors have been ignored in alcoholism treatment research comes from two recent reviews, one of prognostic factors and the other of behavior modification treatment.[3,4] The tables compiled by Gibbs and Flanagan show that only ten of the 45 prognostic indicator studies they surveyed considered cultural factors. And, then, the only factors considered were race (black versus white), nationality, and religion (generally Protestant versus Catholic). The only firm conclusion that emerged was that white people tend to have a better treatment outcome than do black people.

The situation in regard to behavior modification outcome research is about the same. Of 129 studies published between 1940 and 1980, only two considered the influence of cultural factors on treatment outcome. McCance and McCance[5] in a trial of electrical aversion with 194 men in Scotland, found that one predictor of treatment success (as measured by duration of posttreatment abstinence) was affiliation with a subculture in which heavy drinking is not common. And Twemlow and Bowen[6] reported that in a sample of 62 men, those with a strong religious orientation tended to produce the highest self-actualization scores at the completion of a 6-week rehabilitation program emphasizing EEG biofeedback training. It should be pointed out, though, that at least a dozen other studies considered the influence of individual prognostic factors such as marital status, type of alcoholism, or social stability which may be indirectly influenced by cultural factors. Obviously, we have much to learn about the role of cultural factors both in alcoholism treatment in general and in behavioral treatment in particular.

2. Approaches

There are three approaches we can follow in attempting to increase our understanding of the role of cultural factors in alcoholism treatment. First, we can follow the design suggested by Westermeyer[7] and conduct controlled clinical trials, holding the ethnic identities of the subjects constant while varying the treatments. In that way, we can determine if ethnic or special population identity influences treatment outcome. Second, we can treat ethnic identity as an intervening variable between treatment type and treatment outcome and test its influence statistically. Although less sensitive than the first approach, this design would also give some indication of the effect of ethnic or special population identity. Third, we can ignore ethnic or special

population identity and focus instead on specific cultural factors that can be tested in terms of their links to specific behaviors, treatment techniques, or treatment goals. Aside from its relevance to behavioral treatment, I think this last approach will prove to be the most productive, largely because approaches one and two require us to assign accurate ethnic or special population labels to individuals.

Categorizing people who live in a large, complex, culturally heterogeneous society by ethnic identity is no easy task. There are a number of conceptual and methodological problems. For one, anthropologists have not yet identified the cultural criteria that can be used to distinguish among different populations in urban societies. Part of the problem is that the criteria traditionally used to distinguish among small-scale, non-Western societies, such as language, geographical proximity, subsistence practices, and level of cultural complexity, do not apply in complex societies. A second problem with the study of ethnic or special population groups is that the structure and nature of the group and the group identity of individual group members may change over time or from one setting to another. For example, Stiver's analysis[8] of meanings associated with Irish drinking patterns show that at one time heavy drinking was an indicator of Irish identity; it later became symbolic of American identity and then became an amalgam of the two identities.

As regards the influence of social setting on ethnic identity, Westermeyer's[9] description of Chippewa drinking behavior shows how easily ethnic patterns of behavior can change. The Chippewa display both "white" and "Indian" drinking styles. The "white" drinking, characterized by restraint, is used in white drinking establishments; the "Indian" drinking pattern, characterized by mood swings, blackouts, fighting, hilarity, etc., is used in Indian drinking contexts. And in mixed contexts, Westermeyer reports that both styles are used, depending on whom the drinker is interacting with.

Largely because ethnicity or special group identity is so difficult to pin down, I think our initial emphasis ought to be on (1) identifying cultural factors relevant to alcoholism treatment and (2) testing the actual effect of those factors. Once the relevant factors are identified and their effects documented, we can begin looking for patterns of cultural factors that may then enable us to divide alcoholism treatment populations into meaningful groups.

3. Cultural Factors

There are any number of cultural factors that are potentially relevant to the alcoholism treatment process. Heath[1] has listed three categories of factors: (1) meanings of alcohol use; (2) rules governing alcohol use; and (3) behaviors permitted or prohibited with alcohol use. Information about specific factors falling within these general categories can come from a variety of sources including descriptive reports on the drinking behavior of various ethnic groups,

observations of drinking behavior in natural settings, and identification of group differences in expectancies associated with alcohol use. As regards behavioral treatment, I suspect that much relevant information about these kinds of cultural factors is already gathered in the course of treatments such as behavioral counseling, broad-spectrum programs, and self-monitoring, which focus on the antecedents and consequences of drinking as well as on drinking behavior itself.

Four other categories of cultural factors also seem relevant both to treatment in general and to behavior modification treatment in particular: (1) folk beliefs about the cause and treatment of illness; (2) beliefs about control of one's behavior; (3) socialization techniques and practices; and (4) degree of acculturation.

Alcoholism is widely believed to be a disorder that might be alleviated through some sort of treatment intervention. Thus, two cultural factors that might influence what treatments people choose are beliefs about the cause of illness and how illness is best treated. Although we in the United States generally attribute illness to natural causation (infection, trauma, etc.), it is important to recognize that folk theories of illness followed in many societies encompass a much wider range of causal factors ranging from fate to witchcraft to spirit possession.[10] Similarly, from a cross-cultural perspective, we find a wide variety of beliefs and rules governing the treatment process, including rules about who may provide the treatment, where and when treatment may be provided, what techniques may be used, and patient responsibility in the treatment process. Rodin's analysis of beliefs about the cause and treatment of alcoholism in a middle-class Chicago suburb[11] indicates that theories of cause may be related to treatment choice, although the relationship is clearly influenced by individual factors such as age, sex, and socioeconomic status. Rodin reports that those who cite physical causes more often recommend Alcoholics Anonymous, those who cite lack of individual willpower more often recommend personal involvement such as talking to a friend, and those who cite psychological factors more often recommend involvement with mental health professionals.

The second category of relevant cultural factors concerns culturally patterned beliefs about individual responsibility for one's behavior. This factor is especially relevant to behavioral treatment, as behavior treatments can be roughly divided into two groups: (1) those that seek to control behavior through external means and (2) those that seek to control behavior through internal means. Aversion therapies, contingency management, and hypnosis are examples of externally oriented treatments. Internally oriented treatments include behavioral counseling, relaxation training, self-control training, and some drinking control training programs. From a cultural perspective, we would expect people from a group that stresses individual responsibility and self-control to choose and, perhaps, to benefit more from an internally oriented program, whereas people from a cultural group in which external constraints are the major control mechanism will prefer an externally oriented

treatment. I do not think it is an accident that in Eastern European countries with a strong central government, the prevailing treatments are aversion and hypnosis, whereas a wider choice is offered in western societies. The notion of control orientation has not gone unnoticed by behavioral researchers, a number of whom have attempted to link locus of control to treatment outcome, although their findings are not yet conclusive.[12–14]

In discussing alcoholism treatment, it is important to keep in mind that alcoholism is often seen as both an individual and a societal problem. In fact, discussions of the deleterious effects of alcoholism and alcohol use often focus on economic loss, crime, etc. From this perspective, alcoholism treatment can then be viewed as functioning as both a treatment for an individual problem and as a mechanism of social control. The question, then, in cultural terms, is to what extent does the treatment approach mesh with the prevailing socialization practices in a particular group. Does the treatment emphasize punishment or reward? Does it create or alleviate anxiety? Does it take place in an informal or formal setting? Does it provide a conversion experience? The degree to which the treatment reflects these and other socialization practices will determine its appeal and effectiveness.

The fourth and final cultural category concerns the extent to which a particular group is integrated or acculturated into mainstream society. The importance of this category of factors is suggested by the Honigmanns' research[15] in the Arctic and Ferguson's research[16] among the Navaho. The Honigmanns found that men with a "stake" in modern society as measured by steady employment or a modern home, unlike other men in the community, tended to avoid arrest for drinking-related offenses, even though they consumed as much alcohol as the other men. Ferguson tested the same idea with a sample of Navaho men involved in a disulfiram maintenance program. The findings were less clear than the Honigmanns', as the best treatment results were obtained in men whose stake was either in old Navaho ways or in a mixture of new and old. Men with a stake in modern society had the poorest outcome, perhaps because there was little support within the culture for modern ways.

As with all of these cultural factors, the major task at hand is to develop operational measures that can be applied in a clinical setting. Operational measures of degree of acculturation might include, for example, use of native language, marriage to a traditional spouse, place and type of employment, place of education, membership in community voluntary organizations, and the cultural identities of persons in a social network.

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III

Social Mediators of Alcohol Problems

Movement toward Prevention Strategies

Alfonso Paredes, Section Editor

Overview

Alfonso Paredes

There are certain research areas that may provide an empirical basis for methods of preventing alcohol problems. The information derived from such studies offers what may at first appear to be tantalizing opportunities to translate knowledge into social policy. At the outset, however, we must be aware that premature insistence in translating research findings into social policy may lead to frustration and to a loss of credibility for investigative efforts. A significant body of research is necessary before the findings may be considered sufficiently valid to support such a worthwhile purpose.

The topics to be addressed in the following pages include a review of the methods of estimating the prevalence of alcoholism in the population, the relationships between availability of alcoholic beverages and alcohol-related problems, price and income elasticity of the demand for alcoholic beverages, and a review of the current status of our knowledge regarding the relationships among youth, alcohol use, and traffic accidents.

Perhaps one of the most intriguing topics is that of alcohol availability. Availability may be understood as the relative ease of access to alcoholic beverages as determined by legal, economic, and physical factors.[1] It is reasonable to assume that easy access to alcoholic beverages will affect consumption and, in turn, will impact on several social problems. Alcohol control laws are presumed to regulate several dimensions of availability. Potentially, these laws could be made more oriented toward public health issues.[2]

Interactions have been observed between alcohol consumption and availability and several health and social indicators.[3–5] In a study with data derived from counties in one state, Paredes and Gregory[6] investigated the relationships between several measures of health and social problems and alcohol availability. The measures included county rates per 1000 population regarding number of recipients of public assistance, number of children in correctional schools, reports of neglected children, divorces, births out of wedlock, unemployment, deaths from liver cirrhosis, accidents, suicide, alcohol- and non-alcohol-related admissions to public psychiatric institutions, people in prison, juvenile delinquency, alcohol-related arrests, number of retail liquor

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stores, and the number of individual members of ethnic minorities such as American Indians and blacks in the population. The statistical analysis indicated that the number of arrests related to the misuse of alcohol correlated positively with the number of persons receiving public assistance ($r = 0.61$), unemployment ($r = 0.44$), and the number of Indians in the population ($r = 0.55$). The relative number of retail liquor stores by county was significantly correlated with the numbers of deaths from accidents ($r = 0.67$) and with the proportion of the population in prison ($r = 0.40$). However, alcohol- and non-alcohol-related admissions to public mental institutions showed no significant relationships with several other county-based measures.

The number of persons receiving public assistance in the counties investigated was highly correlated with unemployment ($r = 0.65$), the number of Indians in the population ($r = 0.64$), alcohol-associated arrests ($r = 0.61$), and births out of wedlock ($r = 0.53$). However, only a moderate relationship was found among deaths from cirrhosis of the liver, the number of persons receiving public assistance ($r = 0.23$), and the proportion of Indians in the population ($r = 0.21$). Analysis further showed that alcohol-related offenses were moderately correlated with the number of retail liquor stores ($r = 0.35$) and alcohol admissions to public alcohol treatment units ($r = 0.28$).

The investigators, therefore, documented interrelationships among several social indices of problematic behavior and drinking measures, including measures of alcohol availability. The statistical analyses were carried out further, a stepwise multiple-regression analysis was performed to select those variables that best predicted alcohol-related arrests, admission to hospitals for alcoholism treatment, and the number of retail liquor stores per unit of population for individual counties. The best predictors of arrests for alcohol offenses in a given area were found to be a high proportion of recipients of public assistance, the number of youths in correctional schools, the proportion of Indians in relation to the general population, and the number of retail liquor stores in the area. Admissions to public treatment facilities for alcohol-related problems were best predicted by measures such as low rates of employment and high death rates from suicides and liver cirrhosis. We were able to predict the relative number of liquor stores per 1,000 population from items such as high rates of death from accidents, the percentage of the county's population in prison, and the proportion of alcohol-related admissions to public treatment facilities. Although our findings were obtained within the context of a needs assessment analysis and will require more rigorous testing, the information highlighted how important it is to examine the relationships between different parameters of availability and alcohol problems.

In every state of the Union, there are rather elaborate regulatory systems for the control of manufacture, distribution, and sales of alcoholic beverages. It could be assumed that the time has come to examine the feasibility of incorporating epidemiological and social ecological considerations into regulatory decisions. Eventually, this would give a firmer basis and public health perspective to the measures of control of alcoholic beverages. Lately, theories

have been debated on the relationships between the level of consumption of alcoholic beverages by the population and the prevalence of alcohol-related health problems including alcoholism.[7] Economic factors may either facilitate or hinder access to alcoholic beverages. It has been argued that by manipulation of the price of beverages in relation to disposable income, consumption could be influenced. However, consumer behavior is affected by other variables besides price of beverages. For instance, the price of substitute goods, income, consumer tastes, and advertising must be considered. It is possible that fluctuations in the price of substitute goods may have a greater impact on the demand for alcoholic beverages than the price of the beverage alone. It is therefore desirable to examine more comprehensively the complexities of consumer behavior. Otherwise, our suggestions on how to affect the consumption of alcohol may result in simplistic and poorly validated recommendations.

The possibility also exists that the reinforcing properties of alcohol are of a different nature in those who are dependent on alcohol than in the average person who does not have problems associated with drinking. Within this possibility, alcohol-dependent individuals may be substantially less sensitive to price or availability constraints. If this is the case, attempts to reduce overall consumption by limiting availability through taxes or price policies could well be less effective.[8] There are other things to consider. Price elasticity of demand in the instance of beer is very low. It is unlikely that manipulation of price through increased taxation of this beverage, which, incidentally, is popular among the members of certain ethnic and lower socioeconomic groups, may lead to undesirable results: consumers may spend less on articles such as food and clothing to compensate for the increased cost of beer without changing the level of consumption.

Attention has been called to the fact that causality runs from sales to advertising and not vice versa. Furthermore, advertising may lead to perference shifts without an increase in total consumption. This should be carefully considered by those who readily assume that it might be possible to control the use of alcoholic beverages through advertising controls.

A most urgent issue needing to be addressed is the frequency of traffic accidents involving youths. Traffic accidents are responsible for more fatalities among the youth of this nation than any other cause of death affecting this group. Once more, the complexity of this problem needs to be appreciated. It has been found that young drivers beyond high school age who become involved in alcohol-related automobile collisions are more likely, on the average, to have a history of prior traffic violations and convictions, previous traffic accidents, marital problems, and a history of unemployment. Social problems and alcohol difficulties have many behavioral antecedents. Jessor and Jessor[8] suggested that problem drinking is located in a structure of problem behaviors within a larger system of behavior. According to this line of thinking, problem drinking is functionally linked with other problem behaviors, covaries with them, and relates inversely to conventional behavior. If

this theoretical framework is supported by future research, it will have important implications. Such findings would raise serious questions about alcohol-specific prevention efforts. If, as the Jessors have demonstrated among the youth, alcohol use and abuse are intricately interwoven with other behaviors such as drug use, sexual intercourse, and general deviance (aggression, stealing, lying), a prevention approach can not ignore these linkages and expect success.[8]

The methodological problems inherent in the present approaches to the estimation of the prevalence of alcoholism constitute a critical issue in epidemiology. Statistics of prevalence are often quoted without concern for the precision of our definitions or the reliability of the methods of estimation. It is important to critically examine the methods available as a necessary step to take before more reliable techniques are developed. The problem of alcoholism is very serious and does not need to be overstated through the use of poorly validated statistical figures.

We have raised just a few of the issues that will be treated in considerably more depth in the chapters that follow.

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10

Estimating Alcoholic Prevalence

Charles J. Furst

Abstract. Alternative methods for estimating the prevalence of alcoholism and alcohol problems are reviewed and evaluated. No standard method has emerged to fill the void left by the Jellinek formula, now generally recognized as invalid. Currently viable methods include techniques based on alcohol mortality data, alcohol consumption data, data from general population surveys, and data on clients in treatment. Different methods contain implicit and differing definitions of alcoholic populations, and the choice between alternative techniques may depend on the purposes of prevalence estimation. The disaggregate nature of societal problems with alcohol suggests that a single number may not capture the totality. However, for the practical applications of estimating need for treatment services and for monitoring the level of alcohol problems and alcoholism across local areas and across time, projections based on per capita consumption offer the most useful and valid figures.

1. Introduction

Alcohol researchers, treatment providers, and program administrators have a common interest in the prevalence of alcoholism and problem drinking across different geographic areas, across different population subgroups, and over time. However, the field of alcoholism epidemiology is in general disarray, with no consensus on the best method or even on the definition of the diseaselike entity whose prevalence is to be measured. To some extent, this confusion arises from the lack of any well-developed measuring instrument, but the problem also reflects ideological and political divisions within the professions dealing with alcoholism and alcohol abuse.

Before the early 1950s, the best attempts to make estimates of the number of alcoholics in a given area were based on *case-finding* methods (also called *agency surveys*). Case finding refers to the practice of surveying persons or organizations that might come into contact with alcoholics (hospitals, physicians, clergymen, alcoholism clinics, police, employers, etc.) and having each list the alcoholics known to them in the community. After some attempt to correct for multiple listings of names, an estimate would be generated of the size of the known alcoholic population.[1]

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Case finding never was very satisfactory for epidemiologic purposes because it did not measure the presumably large number of "hidden alcoholics"—those not detected by official agencies, physicians, clergymen, etc. During the 1940s, a formula was developed for this purpose by the pioneer researcher E. M. Jellinek.[2] The Jellinek Formula quickly became the preferred method for estimating the prevalence of alcoholism and was adopted officially by the World Health Organization in 1951.[3] Jellinek's formula, based on the number of yearly deaths reported for cirrhosis of the liver, was justified on the basis of Jellinek's historical studies of cirrhosis trends and their relationships to times of alcohol scarcity.

The Jellinek method was the accepted technique for measuring alcoholism prevalence during the 1950s and 1960s, but, although it is still widely used, in recent years it has fallen into general disfavor. There were several reasons for its decline:

1. During the late 1950s, methodological critiques appeared questioning the methods used in estimating the constants in Jellinek's formula and, more importantly, questioning the assumption that they were indeed constant.
2. The research community came to recognize that there is a broad spectrum of alcohol abuse beyond the chronic addicted alcoholic of the disease model formulated by Jellinek and estimated by his formula. Critics point out that alcohol problems and alcohol dependence can occur in different ways, from a variety of factors—psychological, social, and biological.[4]
3. Recent legislative mandates have required alcoholism funds to be apportioned to local areas (states and counties) on the basis of local need.[5] Thus, official estimates need to be made for small areas. Since the Jellinek formula is statistically unreliable for small populations, a demand was created for methods based on other sources of data.

The past decade has witnessed the appearance of several contenders attempting to fill the void left by the Jellinek formula. Some of these contenders try to patch up the flaws by changing Jellinek's particular method while still maintaining the basic approach of inferring a population of alive but "hidden" alcoholics from the number of certified dead alcoholics. Other contenders base prevalence projections on alcohol sales data and its per capita average: *apparent consumption*. Still another major approach is the use of data from general population surveys of alcohol consumption and alcohol problems.

Each of these methods has strengths and weaknesses, but none has thus far emerged as a standard technique in the sense that the Jellinek method provided a standard. The current state of the art of alcoholic prevalence estimation is reviewed in the following pages.

2. Mortality Methods

2.1. The Jellinek Formula

Jellinek's formula takes the number of liver cirrhosis deaths reported each year (D) and multiplies this by the proportion of cirrhosis deaths resulting from alcoholism (P). This number ($P \times D$) should be equal to N, the number of living but hidden alcoholics, times the probability (K) that each of these persons dies of cirrhosis in a given year. That is,

$$N \times K = D \times P$$

Solving for the prevalence of live alcoholics,

$$N = D \times P/K$$

The number D is obtained directly from mortality records. The parameters P and K are estimated from studies of clinical populations. Jellinek later modified this formula to accommodate the emerging conception of alcoholism as defined more broadly by chemical dependency rather than by medical attention (which was implicit in the ways in which P and K were estimated). What was required was a larger number which included all alcoholics, not just those likely to need medical attention. A scaling factor (R) was introduced—the ratio of all alcoholics to those with medical complications. The final formula was thus

$$N = (D \times P/K) \times R$$

The controversy over the Jellinek formula centered around the methods used for estimating the parameters P, K, and R and the constancy of these estimates. Several critical reviews have shown that the data bases used for the estimates were inappropriate[4,6–8] or that the parameters changed over time or from place to place.[9,10]

A second source of error was that cirrhosis deaths are almost certainly underreported, possibly by as much as 50%.[11] Only a small percentage of all deceased are autopsied, and the stigma of alcoholism often deters physicians from listing cirrhosis as a clinical diagnosis. This reporting bias leads to a relative underrepresentation in prevalence estimates of alcohol abusers with higher incomes, since these would be more likely attended by a family physician. High variability from region to region in diagnostic and reporting procedures for causes of death is a second factor that makes regional comparisons problematical.[12,13] Jellinek himself finally recognized the seriousness of these critiques and suggested that his formula be abandoned in favor of population surveys.[10]

Curiously, one of the standard criticisms of the Jellinek approach now appears to have less force. This is the objection based on the supposed inertia or time lag inherent in mortality data, given that the clinical course of cirrhosis is 15 or 20 years. It has recently been shown, however, that cirrhosis rates in a population do respond rapidly—within about a year—to increases in alcohol consumption.[14] The reason, presumably, is that heavy drinking predisposes certain persons in the population to be at risk from dying of cirrhosis and that any increase in heavy drinking will rapidly manifest itself in mortality increases because of a priming effect. The time-lag problem would still make mortality methods insensitive to rapid incidence (increased prevalence) of alcoholism resulting from rapid population expansion as, for example, with heavy in-migration of young and middle-aged groups.

2.2. The CSM Formula

A recent attempt to overcome some of the objections to the Jellinek formula has resulted in a more up-to-date approach oriented toward computer data systems. This is the formula developed under contract to the National Institute on Alcohol Abuse and Alcoholism by a group called Creative Socio-Medics (CSM).[15] The CSM formula is based on a statistical model of the alcoholism treatment process. By tracking alcoholics in treatment through subsequent readmissions to the treatment system and ultimately to a death record, the CSM model provides a way of estimating the main constant of its formula for local areas. This constant is $E(t)$, the expected "alcoholic lifetime" from first admission to treatment until death. As in the Jellinek formula, the constant allows the estimation of the number of living but hidden alcoholics from the yearly number of reported deaths from alcohol-related causes. Several elaborations of the CSM model allow for local variations in mortality reporting practices and for changes in incidence.

Although it starts from different premises, the CSM model shares with the Jellinek formula many of the same properties, shortcomings, and advantages. The method is potentially inexpensive, being based on agency statistics. It uses the number of deaths for alcohol-related diseases in a general population, together with parameters derived from studies of clinical populations, to infer the number of living alcoholics. Both methods assume that clients seen in clinical settings represent the larger number of alcoholics to be estimated. (There is a certain pleasing simplicity to this assumption, but it is inherently conservative; it tends to ignore categories of alcohol abusers who might benefit from a clinical service if they could be induced into treatment.)

The use of mortality data to infer the prevalence of "hidden" alcoholics is appealing, but as a practical matter, the usefulness of cirrhosis mortality data is limited by the instability of these statistics from year to year. The cirrhosis death rate in the United States is in the range of 10 to 60 per 100,000 population, which means that the yearly sample of cirrhosis deaths in a county of 100,000 population is small and unreliable[16] (see Fig. 1). Although the use

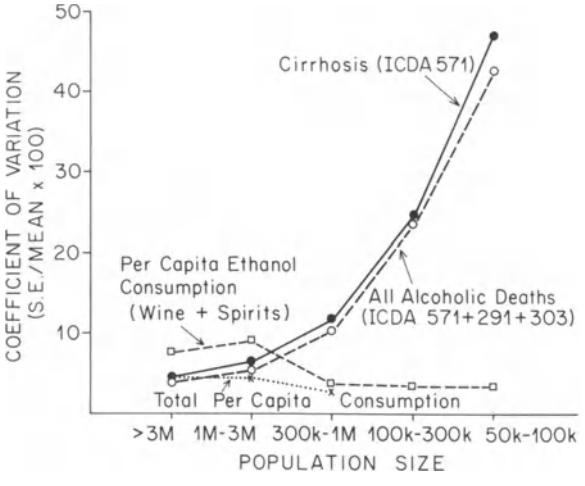

Figure 1. Yearly statistical sampling fluctuation of alcohol prevalence data. Sampling fluctuations (coefficient of variation) of alcohol mortality increase dramatically for areas with population under one million. Consumption data provide a more stable basis for estimating prevalence. (From Furst and Beckman.[16])

of 3-year averages alleviates this problem somewhat, alcoholic mortality data are still too unstable to provide meaningful prevalence estimates for many local areas.

3. The Consumption Method

A second approach to the estimation of the prevalence of alcoholism is based on the amount of alcohol consumed in a population, calculated from sales figures for alcoholic beverages. The method was developed in France by Ledermann[17] to estimate the number of persons whose health is at risk from excessive consumption. Ledermann proposed that the distribution of alcohol consumption among individuals in a population could be described by a lognormal curve; i.e., that the logarithm of the amount of alcohol consumed yearly by individual drinkers is described by a normal probability distribution. Ledermann also proposed, tentatively, that certain other constraints made this distribution, which is generally a two-parameter curve, describable by a single number—the mean level of consumption. Thus, given a criterion for excessive consumption, the Ledermann model provides a method for estimating the percentage of "excessive drinkers" from readily available data on total sales, converted to a per capita statistic.

Advocates of the method have convincingly showed that a heavy-consumption criterion is useful for defining populations at risk or in need of treatment. There is a strong link between heavy consumption and many societal problems with alcohol, including alcohol mortality.[11,18–20] In this sense, the consumption approach is theoretically more neutral than the Jellinek approach with respect to the definition of the population in need of treatment.

The Ledermann method is illustrated in Fig. 2. Given three different

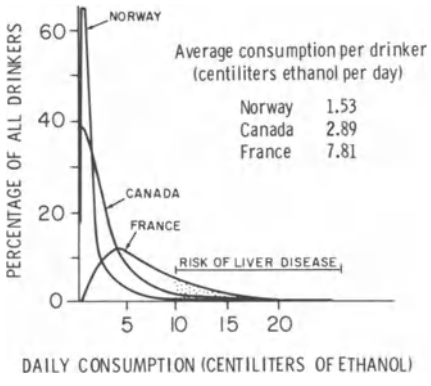

Figure 2. Theoretical Ledermann distribution curves of alcohol use in Norway, Canada, and France in 1968, calculated from average daily consumption. (Adapted from Kalant and Kalant.[21])

populations with different mean levels of consumption (daily intake of absolute alcohol per drinker),* three different lognormal curves are generated. The extent of the alcoholism problem for each population is estimated by the number of drinkers falling in the upper tails of each distribution, that is, by the number of persons who consume in excess of some criterion, say 10 cl daily.

Actually, since it is difficult to specify a universal criterion of consumption that separates an alcoholic or a problem drinker from others, an arbitrary cutoff must be adopted. The criterion most frequently employed is a rate equivalent to 15 cl of absolute alcohol daily.[17] This figure is obtained from studies of self-reported consumption behaviors of clinical alcoholics and describes the lower end of the consumption range for clinical groups. Different cutoff values can be used for estimating different heavy-drinking groups. Applying the criterion of 10 cl daily to Fig. 2, different percentages of excessive drinkers are derived from the upper tail of each distribution.

The statistical criticisms that have been leveled at the Ledermann model are (1) that a lognormal distribution does not accurately describe individual consumption and (2) that although consumption may be distributed lognormally, the special case of a one-parameter lognormal proposed by Ledermann does not accurately describe alcohol consumption.

The lognormal distribution was adapted from economics, where it has been found to describe distributions of consumption of a wide variety of products.[22] The validity of the lognormal curve for describing alcohol consumption was justified by Ledermann on the basis of several sets of data which have since been widely criticized as being inappropriate for various

*Absolute alcohol consumption is obtained by assuming standard percentages for each class of beverage, beer (4%), wine (15%), and distilled spirits (45%), and then converting beverage volume sales figures for each on this basis. Per capita consumption is actually per drinker, so the proportions of drinkers and abstainers in a population must be obtained separately, usually by a drinking survey.

reasons.[23,26] Some of Ledermann's samples were too small to adequately test hypotheses about distributions. Another sample, a population of hospitalized men, could not be presumed to represent a nonhospitalized population, since heavy alcohol use is a predisposing factor for many of the medical conditions listed.

Ledermann[17] and others[27] have observed that the lognormal curve fits the distribution of blood alcohol concentrations (BACs) of randomly selected drivers in roadside surveys, but it has been objected that no linear correspondence between BACs and average daily consumption has been demonstrated and, therefore, cannot be assumed.[24] deLint and Schmidt[28] found that the distribution of individual alcohol purchases follows a Ledermann curve, but Skog[25] has objected that this measure may not equate to individual consumption. Recent statistical tests of a two-parameter lognormal versus other two-parameter curves, using consumption reported in general population surveys, favors, on balance, the lognormal.[29]

A second and more serious objection has been to the assumption that only one parameter, the mean consumption, is sufficient to specify the whole distribution. In the general case, the lognormal is a two-parameter curve: for every value of mean consumption, there is a family of lognormal curves of differing dispersion or variance. Ledermann argued that the dispersion was limited by the fact that the upper end of the curve must be limited at a specific point, related to lethal alcohol dosage, and that the percentage of people consuming more than this amount is constant, independent of the mean consumption. This latter is a curious assumption, but it establishes a functional relationship between the mean and the variance of the distribution which allows the calculation of one-parameter lognormal curves with which one can predict levels of high consumption on the basis of mean consumption alone.

Critics point out that, in general, two parameters are necessary to specify the consumption curve, especially in populations with heterogeneous subgroups.[24–26,30,31] If two parameters are necessary to specify the distribution of alcohol consumption, then the usefulness of the model is destroyed, since mean consumption figures are readily available from sales data, but the variance among individual drinkers is hidden. Advocates of the Ledermann method counter that empirically observed dispersions appear to be close to constant, at least for populations consuming under 10 liters/year per capita.[18] However, even the slight differences in dispersion known to exist would lead to an overprediction of heavy consumers, especially in low-consuming populations, if one uses Ledermann's one-parameter assumption.[25,26] Schmidt and Popham,[27] proponents of the method, conceded that the Ledermann one-parameter distribution will not precisely describe consumption in the general case. However, they argue that for all practical purposes it is a good approximation.

As Fig. 1 shows, the Ledermann method is advantageous for estimating prevalence in small areas, where it has greater sampling stability than mor-

tality rates. Another advantage is its greater sensitivity to changes in the incidence of alcohol problems. A serious drawback is that the method does not allow the breakdown of prevalence rates by sex, age, or other demographic variables of interest, because individual consumption is not measured directly but rather is inferred from alcohol sales data.

The Ledermann technique offers a workable and potentially inexpensive method for estimating the prevalence of alcoholics and persons at risk. However, it cannot presently be implemented in most small geographical areas, because data on local alcohol beverage sales are not generally available. The simplicity and inexpensiveness of the Ledermann technique would make these local alcohol sales data desirable, if laws were passed which authorized the collection of these data.

4. Population Survey Methods

Recent years have witnessed a conceptual shift within the field of alcoholism epidemiology that parallels a similar shift in alcohol research. Jellinek's pioneering efforts were based on his conclusions about alcoholism as a coherent disease entity. Since the Jellinek formula's parameters were estimated from groups of clinical alcoholics, the population whose size is estimated by the formula is for the most part people like clinical alcoholics, which is to say middle-aged chronic alcohol abusers. However, surveys of the drinking practices of the general population based on random-sampling techniques have revealed a broader spectrum of alcohol abuse in different segments of the population.[32–35] These surveys suggest not only a diversity of alcohol-related problems but also a great deal of turnover for individuals in type of alcohol problem and in problem drinking status itself.[36]

Keller[37] has pointed out that the "alcoholics" estimated by survey techniques and those counted by the Jellinek formula appear to be different groups. Within the larger group of survey "problem drinkers" lie those whose heavy drinking has progressed to a point where they lose control over drinking and thus are identified as classically addicted alcoholics. The larger group of problem drinkers represents the population at risk—those whose drinking patterns are like those who eventually go on to become alcoholics *per se*. The figures given by Keller[37] for United States prevalence in 1972 were 5.5 million "disease" alcoholics—a modified Jellinek estimate—and 9.5 million problem drinkers, presumably including the 5.5 million alcoholics. The 9.5 million figure was derived from an application of national survey data in which 9% of all drinkers fell into the most extreme problem drinker category.

Cahalan[38] argued that Keller's conceptualization of the relationship between chronic disease-type alcoholics and problem drinkers may be wrong. The age group found in surveys to have the highest number of persons with alcohol problems in 21–24, with almost twice the rate of problem drinking found in any other age group. Yet clients in alcohol treatment programs—

the "disease" alcoholics of the Jellinek model—tend to be middle-aged. To further complicate matters, Room[36] found that the patterning of problems in the two groups differs. Survey problem drinkers tend to have one or two alcohol-related problems, whereas chronic alcoholics seen in clinics tend to accumulate numbers of problems over a long period of time. What emerges is a picture of two somewhat distinct groups of alcohol abusers: one estimated by the Jellinek formula, the other by household surveys of drinking. The difficulty for the purposes of epidemiology is that the extent of the overlap between these groups is unknown.

In surveys, one source of error in detecting chronic alcoholics lies in the methods of sampling commonly used. Household and telephone surveys systematically exclude transients and people in institutions, two groups that are presumed to have high rates of alcoholism. Another source of error is the well known underreporting of self-reported alcohol consumption, which may result from either deliberate distortion or forgetting. In surveys done in the United States, Finland, and Canada, rates of self-reported consumption, when projected to the whole population, account for only about 40 to 50% of total alcohol sales.[39] Schmidt[40] found that underreporting may be especially severe among those whose heavy buying patterns suggest alcoholism; very heavy purchasers grossly underreported their consumption to the extent that they were classified on the basis of survey responses as light drinkers or abstainers. The limited data available on the validity of self-reported alcohol problems also suggest that here also there is an underreporting bias comparable to the underreporting of consumption.[41]

Recent attempts to define problem drinking on surveys have relied on various combinations of reported consumption and reported problems related to drinking. Scales of alcohol consumption are based on combining items that ask the respondent to specify usual frequency of drinking over a given time period and items that ask the usual amount consumed. Aggregate consumption scores of quantity times frequency (designated as "Q–F" scales) are calculated for each beverage class separately (beer, wine, and spirits), with conversion factors applied. The aggregate Q–F scale is in terms of daily (or yearly) consumption of pure ethanol. First used by Straus and Bacon,[42] the Q–F technique has more recently been modified to account for patterns of binge drinking by taking into account maximum amounts consumed on any drinking occasion.[33]

Other items of drinking-related problems relate to psychological dependence and loss of control over drinking, preoccupation with alcohol, symptomatic drinking (e.g., sneaking drinks), motives for drinking (e.g., escape versus recreation), and social consequences of drinking (problems with family, friends, employer, police). Summary scores of drinking problems are constructed and a criterion adopted for defining a respondent with a severe problem. Problem drinking criteria for epidemiologic purposes can be specified in terms of high intake separately or high problem score separately or

as some combination. For example, Cahalan *et al.*[33] used heavy drinking together with "escape" motives for drinking to obtain the problem drinking designation "heavy–escape" drinking.

Alcohol problems are known to vary in severity and in kind among different population subgroups. The major advantage of surveys are that they can provide separate prevalence estimates for demographic subgroups of the population. Such breakdowns of population rates are often useful for planning and allocation purposes. For example, Knupfer[43] reported that the drinking problems most prevalent among those with high socioeconomic status tend to be excessive intake and dependence, whereas low-status respondents tended to report problems with the social consequences of their drinking. Differences found among different populations subgroups, including ethnic minorities, may be useful in designing prevention and treatment services tailored to the needs of these groups.

A major drawback to the survey technique, compared to indirect techniques, is its high cost. The cost factor is especially salient if prevalence estimates are desired for small areas within a larger jurisdiction (e.g., counties within a state), where a separate random sample for each small area would be prohibitively expensive. Recent legislative mandates require allocation of funds on the basis of a local need, and this legislative pressure has led to the development of a method that uses existing survey data to estimate prevalence in small, local areas.

The method is known as synthetic estimation, a technique using large-area survey data together with local census data to synthesize an estimate of local prevalence. Rates of problem drinking are obtained from the large-area survey separately for each of a number of sociodemographically defined subgroups of the population. The survey sample is cross classified into a table of different subgroups on the basis of such variables as sex, race or ethnicity, age, employment status, income, etc., and for each cell in the table, a separate problem drinking rate is calculated. In a second table, cross classified into the same subgroups, census data provide information about the composition of the local population with respect to each cell. The table of problem drinking rates is then multiplied by the population table, yielding a third table which gives the expected number of problem drinkers in each category. Numbers of problem drinkers by category are useful for program-planning efforts, and total prevalence can be obtained by summing the numbers in this third table (see Table 1).

An early application of the synthetic technique is the Marden[44] formula, which used national survey data and the demographic table approach described above. Other synthetic estimation techniques are described in a recent monograph.[45] Recently, it has been shown that the use of a state-level drinking survey is preferable if county-level estimates are required.[46] National survey data may not accurately reflect local drinking practices or local demographic composition.

There are several cautions that must be exercised in using synthetic es-

Table I. Schematic Outline of the Synthetic Estimation Method Simplified to Two Variables: Sex and Age

	Rates of problem drinking		Population composition		Synthetic prevalence estimate	
Age	Male	Female	Male	Female	Male	Female
15–20	a_1	a_2	b_1	b_2	a_1b_1	a_2b_2
20–30	a_3	a_4	b_3	b_4	a_3b_3	a_4b_4
30–50	a_5	a_6	b_5	b_6	a_5b_5	a_6b_6
>50	a_7	a_8	b_7	b_8	a_7b_7	a_8b_8

timation. The first is that blacks and other minority groups are substantially underenumerated in the census. It is known that the 1960 census missed about 9.5% of the nonwhite population.[47] This undercounting is especially marked in certain age groups. For example, 17% of the nonwhite males aged 20–39 were missed by the 1960 census, yet this is precisely the group for which problem drinking rates are highest.

Secondly, the goodness of a synthetic estimate depends on the amount of the total variance in problem drinking scores accounted for by the sociodemographic variables used in synthesizing the estimate.[45] If the variables of age, sex, race, etc. do not account for much variation in rates of problem drinking, then there is no improvement over simply applying an overall population rate (e.g., 9%) to the total population of the local area. The amount of variance explained by multiple regression of sociodemographic variables on problem drinking in surveys is typically low—around 10% to 15%.[34,45] A related problem, and one frequently encountered in synthetic estimation, is that the local areas must themselves be diverse with respect to demographic composition. If area A and area B, through vastly different in population size, nevertheless each contain about the same proportions of blacks, Hispanics, and whites, in about the same age distributions, etc., then synthetic estimation of total prevalence has no advantage over applying an overall population rate.[45]

Studies of the empirical validity of the synthetic estimation approach reveal that although these prevalence figures are related to indices of drinking-driver problems, they are unrelated or even negatively related to other social indicators of alcohol problems.[46,48,49] In fact, the lack of strong interrelationships among different alcohol problem indicators—such as admissions to treatment, apparent consumption, cirrhosis deaths, and drunken driving—suggests that alcohol problems are disaggregate in nature.[13,50] Rather than a coherent disease entity, there is found to be a more loosely defined conglomerate of societal problems with alcohol. Survey-based techniques seem more appropriate for estimating a heavy-drinking matrix out of which alcoholics are presumed to emerge rather than estimating alcoholics *per se*.

5. The Capture/Recapture Method

One final prevalence-estimation technique deserves mention because, although largely undeveloped, it holds promise for filling the void left by the Jellinek formula. This is the *capture/recapture method* (also called the ecological method). It is adapted from such studies of wildlife populations as, for example, the problem of estimating the number of fish in a lake from successive samples which are tagged and then released.

Suppose it is desired to estimate the total number of fish (N) in a lake. One first captures a group of n fish, tags them with some identifiable mark, and then releases them into the lake. At some later point in time, a sample of fish of size S is taken from the lake, and it is observed that of these, x have the tag. Under the assumption that the S fish are a random sample of all the fish in the lake, a simple equation can be set up relating these four numbers:

$$n/N = x/S$$

Solving for N ($= nS/x$) yields an estimate of the total population.

This is an attractive method for the estimation of alcoholic prevalence because, like the fish in the lake, the total population of alcoholics is presumed hidden, and what is observed is a smaller sample of the total, alcoholics in treatment. An alcoholic admission to a treatment facility would be equivalent to a tagging operation.

Like the Jellinek approach, the capture/recapture procedure attempts to estimate the number of persons in the general population who are like persons seen in existing treatment facilities, and this is arguably the most useful definition of the population to be estimated for the purposes of allocation and program planning. The method has recently been applied to the problem of estimating a population of heroin addicts.[51] The capture/recapture method is attractive in light of the advent of computerized client-information systems, which make available the necessary kinds of data. Separate prevalence estimates could also be made for demographic subgroups of the population.

Unfortunately, the ecological method cannot be directly applied to alcoholic prevalence estimation, because a crucial assumption—that of randomness and independence of the two successive samples—is obviously violated. Since relapse rates for alcoholics are high, the repeaters seen at treatment facilities cannot be presumed to be captured independently. Also, the chances are much greater for someone in treatment to be readmitted at a future time for whatever reasons led to entering treatment in the first place than for an alcoholic who had never before been treated. (It must be pointed out, however, that this second problem, violations of randomness in sampling, probably exists even in wildlife applications, where whatever factors led a fish into the net the first time—factors such as speed or curiousity—would also more probably lead it to be trapped again.)

The first objection, that of nonindependence of successive samples, could

be overcome if the ecological model were elaborated by the incorporation of a relapse process. That is, rather than assuming strict independence of successive samples of clients in treatment, these samples would be regarded as coming from two separate alcoholic pools: one in which the probability of being "captured" is determined by a random sampling parameter and another in which a relapse process was operating. The second pool would include only clients who had been treated previously; the first pool would include new and old clients alike. The relapse process could be modeled in terms of "failure-time" distributions, a statistical description that appears to describe alcoholic relapse.[52]

The development of a more sophisticated capture/recapture model, incorporating deviations from randomness in successive "recaptures" of alcoholics into treatment, may provide the type of figures most useful for practical applications of alcoholic prevalence estimates.

6. Summary and Conclusions

Competing methods for estimating the prevalence of alcoholism rely on differing definitions of the target population. Mortality methods and the capture/recapture technique attempt to estimate the group of persons who have characteristics like clients seen in treatment facilities, largely middle-aged chronic alcoholics. Survey-based methods do not capture this group very well but rather encompass a larger population of problem drinkers and a broader definition of treatment need.[53,54] The consumption method seems to target both.

The disaggregate nature of societal problems with alcohol has been shown in a number of studies.[23,46,50,55] The search for a single number that describes the total prevalence of alcoholism and alcohol problems is motivated more by the political objectives of funding and allocation than by research requirements. Nevertheless, current methods of prevalence estimation for alcoholism (mortality, consumption, and survey) are each useful for roughly indicating the *relative magnitude* of alcohol-related problems across different regions over time. Extreme caution should be exercised in interpreting these estimates as indicating absolute numbers of alcoholics. Survey methods are subject to the biases and distortions of self-reports and are known to underenumerate persons seen in alcoholism clinics. Mortality estimates suffer from time lags and parameter inconstancies and are inapplicable to small geographical areas. The capture/recapture method, like mortality techniques, provides a focus only on clinical populations and is in need of further development. Consumption estimates, although questionable on theoretical grounds, offer the simplest and most valid of the current alternatives for comparisons across geographic areas and over time.

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The Role of Alcohol Availability in Alcohol Consumption and Alcohol Problems

Jerome Rabow and Ronald K. Watts

Abstract. The major theoretical approaches that have provided competing explanations for the distribution and nature of alcohol problems are reviewed and assessed. The sociocultural model and studies are briefly discussed with major emphasis given to the particular studies that have focused on different conceptions and dimensions of availability. Attempts to integrate the sociocultural model and the distribution of consumption model are also reviewed. A recent integrated model finds specific relationships between physical availability or the different types of outlets, with social area characteristics of different populations, and some four different alcohol problems, including cirrhosis, public drunk arrests, and misdemeanor and felony drunk driving arrests. Specific social area characteristics representing social class, minority status, and other structural features such as unemployment and women's labor force participation become implicated in alcohol problems independently of the effects of differing levels of availability. Additional related research on licensing and enforcement practices, beliefs, and attitudes of ABC personnel are also reviewed in relation to the general issue of availability. Other recent research on social and psychological dimensions of availability are also reviewed. Cross-sectional time series modeling is suggested as a technique for determining causal processes.

1. Introduction

Currently, two major theoretical approaches are competing for dominance in the field of alcohol studies. They have been referred to as the sociocultural model[1] and the distribution of consumption model,[2,3] and both have been recently examined.[4] The former has typically concentrated on explaining by reference to norms the observed differences in patterns and rates of alcoholism among various social and cultural groups and differences in normative patterns among those groups.[5–8] Other research using the sociocultural model has usually followed the path of exploring ethnic and religious subgroup norms as primordial sources of learned drinking behavior.[9–13] More recently,

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the distribution of consumption model has provided an alternative approach to the older sociocultural model. It focuses on the statistical distribution of consumption and emphasizes the relationship between the mean level of per capita consumption and the prevalence of cirrhosis of the liver mortality in a population.[2,3,14,15]

These models have generated seemingly contradictory recommendations for the prevention of alcohol problems. The sociocultural model has given rise to the "integration of social norms approach" to alcoholism, which characterizes drinking as a mundane aspect of everyday life, well defined by prescriptive norms of moderation and proscriptive norms against excessive consumption.[16,17] By introducing children at an early age into responsible drinking, they will, it is argued, come to accept drinking as they mature as an ordinary part of everyday life, and eventually, the mystique surrounding drinking will be lost. This implies a policy of encouraging alcohol consumption by a wider number of people, thus increasing the overall level of intake.

The distribution of consumption model, on the other hand, suggests that by lowering the overall level of consumption in a population, the rate of cirrhosis of the liver mortality will, of necessity, decline. Policies aimed at reducing the overall level of consumption, however, appear to contradict the objectives of the sociocultural approach.[18–20] Although Whitehead[20] has called for an integrated approach to prevention, there is some evidence that the two approaches may not be contradictory at all but two different ways of interpreting the same phenomena. In giving a "sociological interpretation" of the Ledermann[21] curve, on which is based the distribution of consumption model, Beauchamp[22] has suggested that the curve may be a graphic representation of conforming behavior to norms regarding drinking. Beauchamp states:

> Of course, there is no single set of norms regarding alcohol use; there is wide variation between the sexes, ages, regions, ethnic groups, and strong urban–rural differences. Despite this variation, however, the overall structure of drinking norms is still supportive of the social order—the dominant norms restrict alcohol use; heavy or alcoholic drinking is not the norm for any group of consequence.[22]

2. The Availability of Alcohol

The issue of alcohol availability derives directly from the distribution of consumption approach. Whereas Prohibition was based primarily on religious and moral sentiments regarding drink, the renewed interest in effective alcohol control policies that would limit consumption is based on a growing awareness of the mounting evidence that aggregate consumption poses an increasing threat to public health. This awareness has grown most rapidly in Canada because of the work of de Lint and Schmidt[2,3,14,15,19] and in Finland, where an international team of researchers has produced an important study outlining alcohol control policy.[23] In several recent publications, Beauchamp[22,24–26]

has carefully analyzed the semantic and logical implications of the idea that differences in individual traits are the deciding factors as to who does or does not become an alcoholic. He concludes that the issue is misdefined; most individuals are never actually exposed to the risk of becoming an alcoholic, and it is therefore inappropriate to attempt to explain a societal problem such as alcoholism through reference to "individual power, capacity or ability."[24] Beauchamp concludes that "this misdefinition of the problem obscures the fact that alcohol problems are a function of existing inadequate public and private controls over availability and use of alcohol."[24] It is becoming increasingly clear that the distribution of consumption model provides the basis for alternatives to the older sociocultural model of prevention.

3. Definitions of Availability

Researchers have tended to concentrate on specific aspects of availability related to their respective disciplines. Reviewing the theoretical literature on availability, Smart[27] discerned four different kinds: economic availability, subjective or individually perceived availability, social availability, and physical availability. The first and last of these have been extensively explored. Economic availability generally refers to the effect of governmental manipulation of price on consumption and alcohol-related problems. Here one considers the aggregate demand for various types of alcoholic beverages in relation to both price increases, through taxation, and disposable income. Seeley[28] was one of the first to argue for a program of prevention based on the control and manipulation of price. Others have pursued this avenue of analysis,[29–31] but there remains considerable controversy about the efficacy of price manipulation as a control mechanism. Reviewing the literature on price, Ornstein[32] concluded that, all things being equal, beer is price inelastic, distilled spirits price elastic, and wine indeterminate.

More recently, some researchers working in the distribution of consumption tradition have turned their attention to the issue of physical availability *per se* as an influence on both consumption and alcohol-related problems. Smart[27,33] undertook a study examining overall availability, per capita consumption, and alcoholism rates in 50 states and the District of Columbia. For the purposes of the study, he employed an eight-item index of physical availability, which included

> minimal legal age for purchase, limitations on availability for off-premise sales, limitations on availability for on-premise sales, density of outlets for on-premise purchase, Sunday retail sales on premise, weekday closing hours for on-premise sales, Sunday retail off-premise sales, and weekday closing hours for off-premise sales.

Smart found that income and urbanization were more closely related to consumption and alcoholism rates than was availability. Parker *et al.*,[34] using

essentially the same data but substituting a more narrowly defined measure of availability (on-premise and off-premise outlet rates), found strong effects of outlet availability on both per capita consumption and alcoholism rates in states not having restrictions on the number of outlets per unit of population. Harford *et al.*,[35] limiting their measure of physical availability to rates of on-premise outlets, found that, when income and consumption were controlled, outlet rates and urbanism were significantly related to rates of alcoholism in 38 states and the District of Columbia.

Although these ecological studies are important to an understanding of the relationships between the physical availability of alcohol and its consumption, they have generated results that are at times in conflict, and, in at least one respect, they are deficient: they fail to account for the influence of different tourist rates on apparent consumption. Tourism or travel to the various states and the District of Columbia has been shown to be differentially distributed and accounts for more variation in consumption than does availability, urbanism, or income.[36]

An additional problem with analyzing state aggregate data is that there is moderate heterogeneity of outlet rates, consumption patterns, and sociodemographic features within states that is not revealed at such a highly aggregated level. Interestingly, although both Parker *et al.*,[34] and Harford *et al.*,[35] were critical of Smart's[27,33] index of availability, they proceeded to use the same unit of analysis as had Smart. They thereby continued to mix conceptions of physical availability by not controlling for other factors such as hours of sale and Sunday sales.

An effort was recently made to control for confounding effects of other components in Smart's index by studying cross-sectional data for counties and cities in California. Rabow and Watts[37] studied two types of off-premise and five types of on-premise outlet rates in relation to social area characteristics, public drunkenness arrest rates, misdemeanor and felony drunk driving arrest rates, and cirrhosis of the liver mortality for 51 of 58 counties. Watts and Rabow[38] subsequently extended the level of analysis to approximately 200 cities in California. Since California is a state with no (or little) local control over alcohol licensing, the local variations of Smart's index were eliminated.

Employing a social area approach first utilized by Donnelly,[39] Rabow and Watts[37] found that in 51 counties in California, specific types of alcoholic beverage outlets were correlated both with specific social area characteristics and particular types of alcohol problems. They tested several hypotheses derived from previous research on social status, urbanization, minority status, household composition, and family structure. The hypotheses were tested in relation both to physical availability of alcoholic beverages and to alcohol-related problems. They also tested the hypothesis that physical availability is related to indicators of alcohol problems not of sociodemographic differences.

The results of the county analysis provided support for most of the hypotheses tested. Indicators of social status, including median income, median education, percent in low occupational status, percent in high occupational

status, and percent of families in poverty, were generally related inversely to alcohol-related problems, and, in particular, percent in poverty was found to be consistently related to public drunkenness arrests and to both misdemeanor and felony drunk driving arrests after controls for urbanization and median income were made. However, social status was found to be virtually unrelated to cirrhosis mortality rates.

Although Rabow and Watts[37] found little support for the hypothesis that urbanization is positively related to alcohol problems, they did find moderate support for the hypothesis that minority status is positively associated with such problems. Public drunk arrests were significantly correlated with percent of household population black, percent of household population Hispanic American, and percent of population other nonwhite and unrelated to percent of population foreign-born or native of mixed parentage. Felony drunk driving arrests were unrelated to any of the minority status variables. And, after controls for urbanization and income were made, only percent of population Hispanic American was significantly related to misdemeanor drunk driving arrests. After controls for urbanization and income were made, cirrhosis mortality was found to be significantly related to three of the four race/ethnicity indicators, with all zero-order correlations increasing after removal of the effects of urbanization and income. Only percent of population Hispanic American remained unrelated to cirrhosis death rates.

The hypothesis relating household composition and family structure to alcohol problems found little support in the county data, particularly with respect to arrests. It was expected that to the degree individuals are integrated into relatively more traditional family-oriented settings, alcohol-related problems would be reduced. For the most part, the household composition and family structure variables, including, for example, percent of households made up of husband–wife families, percent of divorced or separated males and females, sex ratio, female-headed households, youth and aged dependency ratios, and the like, and indicators of familism such as percent of single-person households and percent of large households (six or more persons) showed few consistent relationships with the arrest rates, with some exceptions.

Cirrhosis mortality rates provided considerable support for the hypothesis of an inverse relationship with traditional family structure and familism. Only two of the nine correlations for cirrhosis mortality and the household composition and family structure variables were insignificant (sex ratio and aged dependency ratio), and most of the significant coefficients were significant at a $P < 0.01$ level. The familism indicators provided strong evidence that cirrhosis mortality is related to traditional/nontraditional families. Percent of large households (six or more persons) was inversely related in a significant way to cirrhosis after controls at the $P < 0.05$ level and was positively related to single-person households at the $P < 0.001$ level after controls, confirming recent national findings for 389 cities.[40]

Relatively few studies have concentrated in any comprehensive way on

specific types of alcohol beverage outlets in relation to social area characteristics. Previous studies of the correlates of alcohol control policies by state have generally been limited to comparisons between "monopoly" and "license" states.[41,42] One Moreland Commission study gave attention to the number of liquor sales outlets per unit of population.[42] Other studies have concentrated on consumption and selected socioeconomic and demographic characteristics and their relationship to cirrhosis mortality at the state[43] and county[39,44] levels. Though Donnelly[39] pursued the social area approach using a number of sociodemographic indicators, he did not consider varying outlet rates across county jurisdictions. The work by Smart,[33] Parker *et al.*,[34] and Harford *et al.*,[35] as noted above, is a useful starting point for the consideration of physical availability, but they considered only a few sociodemographic variables in their analyses.

Rabow and Watts[37] considered a wide range of sociodemographic variables in relation to seven types of on- and off-premise outlets and found that specific outlet types were related to specific socioeconomic and demographic characteristics. They hypothesized that there are differential relationships between the various outlet types and social area characteristics. On-premise outlets located in *bona fide* eating places were expected to be positively related to social class and urbanization and inversely related to minority status, to household composition, family structure and familism, and unemployment. Such outlets as off-premise, general (all types of beverages sold), on-premise beer bars (beer only), and on-premise, general (all types of beverages sold), on the other hand, were expected to be inversely related to social class and positively related to minority status, nontraditional household composition, low familism, and other variables indicating social disorganization.

The results of the analysis for California counties confirmed most of the hypotheses about the relationships between specific outlet types and sociodemographic characteristics. Thus, restaurants with on-premise licenses were positively related to social rank and either inversely or insignificantly related to race/ethnicity. And, as predicted by the hypothesis, restaurant outlets were inversely related to structural features such as unemployment but positively and strongly related to women's labor force participation at the $p < 0.001$ level after controls by urbanization and income were made. Furthermore, the household composition and family structure variables, as well as the familism variables, showed support for the idea that social areas with traditional family structures have relatively fewer on-premise restaurant outlets.

Although on-premise general outlets were unrelated to social class after controlling for urbanization and income, and off-premise, general outlets were found to be positively related in a modest way to social class, beer bars were inversely related to social class variables. After controls for urbanization and income, beer bars remained substantially correlated with percent of families in poverty at the $p < 0.001$. Less support was found for the hypothesized positive relationships between on-premise, general outlets, on-premise beer bars, and off-premise, general outlets and the race/ethnicity variables. Only

after controls were made were significant relationships revealed between off-premise, general and on-premise, general and both percent of population black and percent of population other nonwhite. Beer bars were significantly related to percent of population Hispanic American after controls. This lends support to recent research that suggests that beer is the beverage of choice among Hispanic Americans.[45] Solid support was found for the hypothesized inverse relationship between traditional family structure and off-premise, general outlets, but both beer bars and on-premise, general outlets provided little support for the hypothesis.

Familism was related to off-premise, general and on-premise, general outlets in the hypothesized way. However, beer bars were related to familism in an inverse manner. Of all outlet types considered, beer bars showed the strongest correlation with percent living in crowded dwelling units (1.01 or more persons per room).

Rabow and Watts[37] further investigated the relationship between the seven outlet types (on and off sale combined with different types of beverages) and alcohol problems. Briefly, they found that of the seven outlet types, only beer bars were significantly related to both public drunk arrests and misdemeanor drunk driving arrests after controls for urbanization and income were made. Felony drunk driving appeared to be related to several outlet types, whereas cirrhosis mortality was related specifically to off-premise, general and on-premise, general outlets.

A similar analysis was carried out on data at the city level in California by Watts and Rabow,[38] and corroboration was obtained for most of the county results, with some exceptions. Since reliable data were unavailable for consumption at the substate level, both county and city analyses were hampered by reliance on taxable sales for packaged liquor stores, an inadequate measure of consumption.[37,38]

Most of the above studies of physical availability have used cross-sectional data from available sources. Cross-sectional studies are quite useful in revealing the extent to which variables covary across the research unit of analysis. Other techniques are becoming increasingly available to researchers interested in the study of time-ordered phenomena.[46] Douglass *et al.*[47] employed time-series-correlational analyses to study relationships between

> specific kinds of increments in retail alcohol availability . . . and increments in acute alcohol-related mortality; specifically accidental deaths in the working place, domestic accident mortality, and traffic accident mortality.

They also employed data on the distribution volumes of package and draught beer, distilled spirits, and wine. Among their findings were that licensing activities have less of an impact on beer distribution than on wine or distilled spirits, although the authors were unable to examine possible confounding effects on the relationship between licensing and distribution such as population changes and economic fluctuations.

Furthermore, Douglass *et al.*[47] were able to establish fairly well the predictability of alcoholic beverage distribution by employing time series modeling. They found that "draught beer distribution is remarkably consistent with stable and dominant seasonal cycles," whereas wine and distilled spirits showed less seasonal dominance, although "linear trend (growth) was stable and predictable." Establishment of regular and predictable time series patterns in the data is important, they concluded,

> because such stability in the data on beverage distribution accommodates analytic requirements to evaluate the impact of specific changes in licensing rules and regulations on the distribution of alcoholic beverages.

Another aspect of their work concerned the relationship between alcohol beverage distribution and the incidence of health and social problems over time. They found through cross-correlational analysis that both draught and package beer distributions were related to accidental mortality measures such as vehicular and work-place mortality, and draught beer sales weakly related to homicides. They concluded that their results strongly suggest causality between the distribution of beer and total accident mortality, traffic accident mortality, and accidental deaths in the workplace.

Although Douglass *et al.*[47] showed that licensing activities are directly related to alcoholic beverage distribution volumes, particularly for distilled spirits and wine, and that beer distribution is implicated in several types of accidental mortality, they were forced to admit the presence of a paradox in their results: "the availability of beer is the most strongly related to accident mortality but it is the category of alcoholic beverages least effectively regulated by the state." Furthermore, Douglass *et al.* were well aware that their study of Michigan data represented only one jurisdiction's statistical experience. Nevertheless, they concluded, as had Parker *et al.*,[34] that "alcohol availability should become a public health issue."

In his review of the theoretical and empirical literature on availability, Smart[27] has shown that the majority of empirical studies on alcohol availability and consumption have concentrated on changes in alcohol control policies and the impact of those changes on both consumption and alcohol-related problems. Unfortunately, as he has shown, it has been difficult to measure the "detectable effects" from single or a few changes in specific alcohol control policies. Smart reviewed the numerous changes that have occurred in Ontario in the past quarter century and concluded that it is nearly impossible to isolate the effects of discrete alterations in policy, especially when many came simultaneously. In his review of "small changes in availability,"[48–54] Smart[27] concluded that relatively small changes in availability "typically have small or insignificant effects on drinking and drinking problems."

Large changes in availability have occurred primarily as a result of prohibition and wartime interruptions and occasional changes in policy that have brought about the introduction of alcoholic beverages to areas formerly dry." Smart[27] concluded that although prohibition was certainly successful in re-

ducing consumption and consequent problems such as cirrhosis, drunkenness, and mortality rates, it generated other problems by its implementation and is not a viable policy alternative in the present world. Smart reviewed selected studies on wartime restrictions, primarily in World War I Britain, and concluded that several factors including restrictions on production and importation, increased working hours, reduced physical movement, patriotic fervor, and reduced policing and medical manpower combined to produce not only a drop in consumption but also a reduction in public drunkenness and cirrhosis of the liver.

In Finland, a number of studies have been made on the introduction of alcoholic beverages into "dry" rural areas. Kuusi[55] studied the effect of the introduction of alcoholic beverages into a number of rural towns, comparing them with other towns where alcohol remained unavailable. He found that excessive drinking and drunkenness were unaffected by greater availability but that there were increases in the frequency of drinking in the town studied. Interestingly, beer and wine became somewhat more popular in Finland, a nation that traditionally consumed distilled spirit beverages. Smart concluded that Kuusi's study demonstrated that the policy of increased availability in rural areas and small towns was of "uncertain social value."

One other social experiment was attempted in Finland in the early 1970s.[56] Public opinion called for the liberalization of control laws to allow for the introduction of liquor stores in rural areas and for an increase in the number of restaurants with licenses to serve all types of beverages. In response, the government decided to increase the number of liquor stores and to place many of the new licenses for the first time in rural areas. The number of comprehensively licensed restaurants was almost doubled, and over 17,000 new medium-beer (3.7%) shops and 3000 medium-beer bars were opened. The purpose of the increase in beer availability was to make available low-alcohol-content beverages that would be an alternative to high-alcohol-content distilled spirits. It was hoped, ultimately, to reduce "rapid" intoxication from spirits. The results of these changes showed that rather than choosing beer as an alternative to distilled spirits, the Finns tended to choose beer in addition to traditional spirits. Beer consumption accounted for a substantial portion of the 48% increase in per capita consumption in the first year. However, there was virtually no reduction in spirits consumption. Furthermore, the number of heavy drinkers actually increased. Smart[27] concluded that the rapid increase in availability undoubtedly was the major factor in the increase in consumption but that preexisting public opinion certainly had been demanding greater liberalization of the law regarding availability. This last factor is another confounding issue in research on the availability–consumption nexus.

It has been very difficult to sort out the cause–effect relationships between availability and consumption. Smart recognized this in concluding in his review of the availability literature that world trends in consumption suggest that consumer demand may be a major factor in increasing availability and that increasing availability may well increase consumption. He called for fur-

ther cross-sectional and longitudinal or time-series studies that would consider such related factors as increased urbanization and urbanism, increased disposable income, and the social psychological and sociability aspects of drinking behavior.

Smart[27] has presented two other conceptions or dimensions of availability for which, as yet, there have been very few empirical studies. Subjective availability, according to Smart, "could refer to subjective or individual differences in how accessible people feel alcohol is to them." Citing some evidence to show that people often do or do not act on the basis of perceived or subjective estimates of costs and risks rather than actual costs and risks,[27] Smart argued for a conceptualization of subjective availability in terms of the perceived "energy and resources" that an individual might spend in order to obtain and consume alcoholic beverages. He conjectured that small changes in availability have probably had little effect on personal consumption because they have had virtually no impact on subjective availability. It would seem that subjective availability may well depend on relatively larger changes in the physical availability of alcoholic beverages. Certainly in the United States, beverage alcohol is pervasive and readily available in most urban areas. In some states, supermarkets are generally well stocked with a wide range of alcoholic beverages, and there is very little stigma attached to the purchase of alcoholic beverages as part of routine grocery shopping.

Social or family availability was also proposed as an area of study by Smart.[27] Social availability refers to norms of reciprocity or "sociability" in small reference group settings. To what extent is there an "obligation to serve" in small group settings? What are the precise mechanisms by which availability of beverages in the home or social settings gets translated into a felt need to offer beverages to others, say, when family or friends are visiting? Smart[27] suggested that these and other issues need serious attention.

Clearly, what Smart[27] has proposed is a much wider investigation of the various "dimensions" of availability including not only the relative presence or absence of alcoholic beverages (physical availability) but the social norms of collectivities and the social psychological "calculations" of individuals and groups regarding the acquisition and consumption of those beverages. This appears, in a sense, to return the issue of availability, consumption, and alcohol problems at least partly to a consideration of how social norms regarding drinking behavior are generated and maintained. This suggests a reconsideration of the sociocultural model in conjunction with the availability–distribution of consumption model as a unified approach to the study of alcohol problems and their prevention.

Brenner,[58] commenting on Smart's[27] review of the availability literature, emphasized the need to consider both the formal legal restrictions on availability and "those components of the social control process that are informal and constitute the usual norms or conventions by which any society lives." Legal restrictions, in Brenner's view, are only one part of the total normative system that operates to control both the availability and consumption of al-

coholic beverages. He further argued that the diversity of social norms that bear on the demand for alcoholic beverage consumption is not only situationally determined but is determined also by various aspects of the social structure, "including the social status and roles occupied by persons of differing age, sex, occupation, income, education," and, we might add, religious preference and beliefs as well as racial and ethnic membership. He argued that the encouragement or restriction of alcohol consumption is as much, or more, a function of "society's norms, values and beliefs. . . . Availability, in a word, is almost entirely what a society considers to be proper at any given time."

Brenner[58] concluded his remarks by calling for a unified approach to the study of alcohol issues—one based on more sophisticated research and statistical methodologies which incorporate a host of variables thought to be relevant to alcohol availability, consumption, and problems. The alcohol literature is replete, according to Brenner, with studies of a limited number of factors implicated in the alcohol process. It does not contain many studies that consider the "interacting effects of policy considerations with sociodemographic, sociocultural, or psychological variables."

4. Prospects for Theoretical and Methodological Integration

Given Brenner's[58] call for a unified or single multivariate model to study alcohol issues, what are the prospects for theoretical and methodological integration of the sociocultural and distribution of consumption approaches? Some researchers have proposed that the two approaches may be integrated in some specific ways. Whitehead[20] has suggested that there are three different approaches that might be taken to provide an integrated approach to the prevention of alcoholism:

> increasing the integration of drinking practices without significantly changing per capita consumption, reducing per capita consumption without significantly changing the integration of drinking practices, and simultaneously increasing the integration of drinking practices and reducing per capita consumption.

Some specific suggestions that Whitehead[20] makes for each of these three preventive approaches are: encouraging legislative changes that would allow parents to serve alcoholic beverages to their children in their own homes and purchase alcoholic beverages for them with meals in restaurants. This would lead, according to Whitehead, to an increase in the integration of drinking practices by removing the "forbiddenness" of alcoholic beverages, and, from an early age, children would learn to use alcoholic beverages in a healthy family setting, and the "former legal controls against minors would continue to operate."

Whitehead also offers ways by which per capita rate of consumption

might be reduced without affecting the level of integration of drinking practices. He suggests both lowering the alcohol content of beverages and making alcoholic beverages available in smaller containers. Both measures would be aimed at reducing the consumption of absolute alcohol and assume that purchase patterns would remain essentially the same.

A combination of policies aimed at increasing the level of integration of drinking practices and reducing per capita consumption would be directed at advertising policies and contents. He argues that advertisers should direct their attention to new types of advertising of alcoholic beverages that "convey messages that would foster norms of moderation and encourage the use of such drinks in those settings where it is likely to be most healthful."

All in all, Whitehead suggests a number of policies that, in combination, ought to be able to maintain, or even increase, the level of integration of drinking practices while decreasing the mean level of consumption in a population and, by extension, the incidence of alcoholism. Beauchamp,[22] as we have already discussed, has concentrated not so much on the preventive aspects of the two models but on ways in which the sociocultural perspective and the distribution of consumption perspective can be reconciled as two alternative ways of looking at the same phenomena. He has shown that the Ledermann curve, the basis for the distribution of consumption argument, is merely a graphic representation of the "norms and sanctions restricting or regulating the use of alcohol." If the Ledermann curve is accepted as an accurate and valid description of the distribution of consumption, and Beauchamp has argued that the evidence for the general shape of the distribution is substantial, then any shift in consumption is indicative of a change or relaxation of the prevailing norms of alcohol use.

Beauchamp has provided a reasonable explanation for the apparent "disparities" in the two approaches. He has shown that the two perspectives are really not competitive but are two alternative and complementary ways of conceptualizing the factors influencing alcohol consumption and alcohol problems.

Several researchers have undertaken to test a number of hypotheses derived from both the sociocultural and distribution of consumption models in a multivariate framework, as suggested by Brenner.[58] Recently, Frankel and Whitehead[59] separately tested hypotheses derived from the sociocultural and distribution of consumption traditions and then elaborated on an earlier synthesis of the sociocultural and distribution of consumption approaches made by Whitehead and Harvey[60] by employing path analysis. They found relatively more support for the distribution of consumption arguments, and their analysis represents an important advance in efforts to study the two approaches in an integrated manner.

The chief drawback to Frankel and Whitehead's[59] work is that it is based on data obtained by Bacon *et al.*[61] for 139 preliterate non-European societies, most of which are small scale. Consequently, their work offers little assistance for those interested in processes occurring in advanced industrialized societies

where populations are heterogeneous in composition and factors such as high degrees of urbanism and income[58] are at work.

Another attempt to integrate the sociocultural and distribution of consumption models was made by Rabow and Watts[37] and Watts and Rabow.[38] Employing cross-sectional data from a variety of sources on physical availability of outlets and sociodemographic characteristics, as we noted above, they used a social area ecological framework to study physical availability and differential distributions of social structural groupings such as age, sex, social class or rank, racial–ethnic composition, household composition and family structure, familism and other structural variables, and their net effects on selected alcohol problems. At both county and city levels of analysis, they found important specific relationships between overall physical availability and alcohol problems and between social area characteristics and alcohol problems. Also, different types of physical availability (outlets) were found to affect different types of alcohol problems just as did selected relevant sociodemographic characteristics. They employed correlation, partial correlation, and regression techniques to analyze their data. In almost every case, specific types of outlet availability were implicated in specific types of alcohol problems when selected sociodemographic characteristics were controlled. A major assumption in their analysis was that differentially distributed social area characteristics are "proxies" for varying subgroup norms and normative patterns, the direct measures of which were unavailable.

Similarly, they found that specific social area characteristics representing social class, minority status, traditional versus nontraditional family structures, and other structural features such as unemployment, women's labor force participation, and physical density were variously implicated in selected alcohol problems as were the effects of differing levels of outlet availability.

They concluded, as had Donnelly[39] before them, that social area analysis offers a promising tool for developing integrated and comprehensive models for testing a number of hypotheses in the sociocultural and availability–consumption traditions.

Other promising areas of research are currently under way at the Alcohol Research Center at UCLA. Rabow, Schwartz, Stevens, and Watts[62] have been investigating neglected "dimensions" of availability through a pilot survey of 580 Southern California residents. Questions concerning price considerations, physical availability or "propinquity," sociability or obligation to serve, purchasing patterns, and sociodemographic data have revealed that sociability appears to have the greatest net effect on individuals' frequency and quantity of consumption. These results were replicated in a Los Angeles neighborhood with physical availability held constant at a high level.[65] Greater alcohol consumption was associated with economical expenditure of resources. For example, heavier drinkers were the most price conscious, bought closest to home, and combined alcohol purchases with the regular grocery shopping. Since sociability was a consistently strong variable in both samples, efforts are currently under way to explore its meaning. Correlations suggest that, in

the very least, it represents the keeping of alcoholic beverages on hand as a household staple. Other research on social availability is being pursued by Rabow and Neuman.[66]

Another neglected area that has recently received attention at the UCLA Alcohol Research Center is the licensing and enforcement process of the State of California Alcoholic Beverage Control (ABC). Rabow, Johnson-Alatorre, and Watts[63] employed a mail survey sent to all 167 ABC investigators in the 25 State of California ABC offices designed to assess investigators' decisions to recommend issuance or denial of alcoholic beverage licenses. The return rate was 81%. The instrument gathered background data on investigators and attitudinal vignettes developed by the authors. The three vignettes involved a license application that violated a proximity to residence rule, a license application that involved an undue concentration rule, and a license application that involved an applicant's moral character. Vignettes were varied as to the number of community protests. Findings indicated that investigators, although sensitive to community protests, will tend to recommend a license despite the existence of a rule violation. Investigators also tended to perceive the administrative law judge as more likely than themselves to recommend issuance. Investigators would personally prefer to be more strict than they publicly recommend. The authors conclude that the effectiveness of formal rules designed to facilitate the community's interest is minimal; the rules do not present barriers to obtaining an alcohol license, even though investigators are personally concerned about community protests.

In a related study, Rabow, Johnson-Alatorre, and Watts[64] focused on the attitudes of investigators and attempts to link attitudes with licensing decisions. They used a responsibility scale, a power assessment scale, and a host of organizational attitudes to develop meaningful factors. Stepwise discriminant analysis was performed to assess the contribution of the seven factor scores and other personal background factors. Though not conclusive, the results of this analysis showed that only personal satisfaction did not contribute to any of the discriminant functions. The ABC rules were shown to invoke different sets of attitudes, which are linked less to the licensing evaluation than to personal influences.

5. Conclusions and Suggestions for Further Research

This review of the literature on alcohol availability and consumption and the problems that result from the abuse of alcohol has attempted to show that the phenomena relating to alcohol and drinking behavior are complex and multifaceted. The issue of availability has received increasing attention in recent years by many researchers interested in promoting the improvement of the public's health. The tendency has been to promote the distribution of consumption model over the older sociocultural model as the way to study policies aimed at preventing the growth of and, we would hope, reducing alcohol problems.

Although the focus of this review has been on various issues of availability and consumption and problems, we have tried to show, as have other researchers, that a single-minded attachment to one approach or the other is not productive. A realistic model of complex alcohol phenomena will necessarily employ a host of variables in a multivariate framework. Some attempts have been made in this direction. Furthermore, cross-sectional studies, although important in determining statistical relationships at one point in time, need to be supplemented with sophisticated new time-series modeling techniques. There is even the possibility of employing "cross-sectional time series" models which follow a number of variables for any number of jurisdictions over time. The rapid advancement in statistical programming has made this entirely feasible.

The mathematical and statistical techniques are available. However, the data currently are not, in any comprehensive way. Every effort should be made to develop a national epidemiologic system of alcohol-related information that could be integrated with a comprehensive set of sociodemographic information such as that employed by Donnelly.[39]

Science proceeds cumulatively. More and better research is needed if we are going to be able to isolate from among the multitude of normative and regulatory variables those factors that show the greatest promise for conscious manipulation to improve the overall level of living and health of the public in advanced industrial societies.

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12

Price and Income Elasticities of Demand for Alcoholic Beverages

Stanley I. Ornstein and David Levy

Abstract. Estimating the demand for alcoholic beverages represents a difficult statistical problem. A number of studies have attempted to estimate the demand for beer, wine, distilled spirits, or total alcohol consumption. The results vary widely according to country of study, data used, and model and statistical technique. For the United States, most studies find the demand for beer to be relatively price inelastic, at around -0.3. The demand for distilled spirits appears to be unitary price elasticity or somewhat greater, around -1.5. The evidence on wine is too sketchy to draw any conclusions. There is no strong evidence of substitutability among beer, wine, and distilled spirits based on econometric models, nor evidence that advertising plays a strong role in the aggregate demand for beer, wine, or distilled spirits. The main policy implication is that price increases to control consumption will have a stronger impact on the consumption of distilled spirits than on beer.

1. Introduction

Alcoholic beverages have been some of our most heavily taxed consumer goods for centuries. Taxes have frequently represented more than 50% of the final consumer price. In previous centuries, before the advent of income and sales taxes, when governments were heavily dependent on foreign trade duties for the bulk of their revenues, taxation of alcoholic beverages through import duties was a primary source of government revenue. In some cases, these duties were also designed to protect domestic alcohol beverage manufacturers from foreign competition. In this century, in the United States, legislative discussion of taxation has centered on three goals: to increase government revenues, to control illegal production and consumption, and to control the consumption of alcoholic beverages.

In recent times, public discussions have focused on a fourth goal. It is argued that for goods that create large negative externalities or social costs, such as those created by alcohol abuse, taxation to reduce social costs is justified. And, moreover, the monies so collected should be used to cover the costs of heavy drinking, including treatment and rehabilitation programs,

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accident costs, medical costs, and employment losses, and to provide support for research into the causes and cures of alcoholism. At issue is the impact of tax increases on consumption in total and in various income classes and incidence of drinking groups. For example, if the social costs of drinking are caused almost exclusively by heavy drinkers, approximately 10 to 15% of all drinkers, it is necessary to know the sensitivity of this group to increases in prices by tax rate increases. (There is also the question of whether the remaining 85 to 90% of drinkers should be taxed for the social costs created by others.) This leads to a requirement for having precise estimates of consumption sensitivity to price changes, which is measured by the price elasticity of demand.

The price elasticity of demand has been a central theoretical and empirical issue of those concerned with the control of alcohol consumption for many years. Price elasticity has been of pivotal importance in competing theories on the control of heavy drinking. For example, the widely tested and debated single distribution model of consumption states that there is a constant relationship between per capita or mean consumption and the incidence of heavy drinking (for a recent debate on this model see Parker and Harman[1] and Schmidt and Popham[2]). Assuming this to be true, knowledge of the mean of the distribution of consumption in any society is sufficient to know the proportion of a population that are heavy drinkers. Moreover, changing the mean has a predictable and known effect on the proportion of heavy drinkers. Thus, such straightforward policies as changing the mean of consumption by raising price through tax rate increases have wide appeal to adherents of this model. If the demand for beer, wine, and distilled spirits is price elastic (inelastic), proportionally price increases in each beverage will reduce consumption more (less) than proportionally to the price increases. This will also have predictable effects on government revenue and consumer expenditures. At issue, however, is whether demand is price elastic or inelastic and whether it is the same for all subgroups of drinkers—occasional, light, moderate, and heavy.

The purpose of this study is to examine elasticities for beer, wine, and distilled spirits. Initially, we had intended to estimate elasticities for California consumers. We decided against the original estimation since (1) no good price series on each beverage exists for California, and we did not have the resources to construct original series; (2) no quantity–price data by incidence of drinking groups was available, and it was felt that price elasticity by drinking-group incidence was the most relevant data for public policy; and (3) a large number of studies have estimated price elasticity for each alcoholic beverage and comprise an abundance of information on estimation techniques and estimated elasticities that, apparently, has been largely overlooked by many alcohol researchers interested in controlling consumption by the price mechanism. As a sidenote, in the debate between Parker and Harman[1] and Schmidt and Popham,[2] only three studies on price elasticity were cited. Ironically, the

same study, that of Lau,[3] was used to support both sides, and it has been shown to have a major flaw in some of its results (see Lidman[4]).

As a consequence, we chose to examine this body of studies for methodology and results. The questions guiding our review were: (1) Is there a similarity of results across countries? (2) Is there a similarity of results across studies for each beverage; for example, is wine consistently found to have price-elastic or -inelastic demand? (3) What was the estimation technique used, and was it reliable in the sense of producing unbiased, relatively error-free estimates of elasticities? The goals of the study are to (1) examine existing methodologies so as to provide the reader with a guide to the design of an original estimation of elasticities that would avoid the pitfalls of previous studies and (2) compile all the estimated elasticities in a series of tables for purposes of comparison and draw conclusions on the true nature of elasticities for each alcoholic beverage.

The format of this study is as follows. The first section presents the fundamental aspects of elasticities, how they are defined, how they are measured, problems in estimation, and what elasticity estimates are expected in alcoholic beverages. It is intended as an introduction to many of the topics. The second section presents a review of a number of empirical estimates of elasticities for beer, wine, and distilled spirits. It is of a somewhat technical nature by necessity. An understanding of econometric methods and problems is assumed. The last section summarizes the results in a series of tables and discusses their public policy implications.

2. Elasticities and Their Measurement

2.1. Elasticities Defined

Elasticity measures the responsiveness or sensitivity of quantity demanded to the determinants of demand—price, income, advertising, consumer preferences, and so on. The sensitivity of the quantity demanded to changes in the price of a given good, all other variables held constant, is known as the price elasticity of demand or own-price elasticity. Analogous elasticities exist for income, advertising, and other determinants of demand. The effect of changes in the price of substitute or complement goods on the quantity demanded of a given good, holding all other variables constant, is known as a cross-price elasticity of demand. Price elasticity, e_p, can be measured by

$$
\begin{aligned}
e_p &= \text{percentage change in quantity demanded/percentage change in price} \\
&= [(Q_2 - Q_1)/Q_1]/[(P_2 - P_1)/P_1] \\
&= [(Q_2 - Q_1)/(P_2 - P_1)] \cdot P_1/Q_1
\end{aligned}
$$

where Q_1 and Q_2 are quantity demanded in periods 1 and 2, and P_1 and P_2 are price in periods 1 and 2.[5]

Elasticity is measured in percentage terms; that is, for a given percentage change in price, there will be a given percentage change in the quantity demanded. Elasticities are thus comparable between beverages or geographic markets for a given period. For example, the price elasticity of spirits in California could in principle be compared to the price elasticity of spirits in New York or the price elasticity of wine in California. These points can be illustrated by the use of an example. Assume that the price of beer in California averaged $1.70 per gallon in 1977 and fell to $1.50 per gallon in 1978 because of the Supreme Court's invalidation of the fair trade laws. As a result, the quantity demanded rose from 500 million gallons in 1977 to 520 million gallons in 1978. Assuming that there were no changes in disposable income, the price of substitute beverages, the weather, and all the other determinants of the demand for beer in California, the price elasticity could be estimated as:

$$e_p = [(520 - 500)/500]/[(\$1.50 - \$1.70)/\$1.70] = -0.3$$

This says that for a 10% increase in price, the quantity demanded will fall by 3%, a less than proportionate amount. Note that the price elasticity is negative. Since all demand curves are downward sloping (except a perfectly vertical or horizontal demand curve), quantity decreases when price increases and *vice versa*, making all price elasticities negative. For simplicity in the remaining discussion in this section, we drop the negative sign and speak of price elasticity in terms of absolute value.

Goods are classified as price elastic, price inelastic, or price unitary elastic. For a price-elastic good, elasticity is greater than 1, and a given percentage change in price causes a more than proportionate change in quantity. For a price-inelastic good, elasticity is less than 1, as in the beer example above, and a given percentage change in price will cause a less than proportionate change in the quantity demanded. For a unitary elastic good, elasticity equals 1, and a given percentage change in price will cause an identical percentage change in quantity demanded.

There is also a direct relationship between price elasticity and total consumer expenditures on a good. If a good is price elastic, a rise in price causes a fall in total expenditures, and a fall in price causes a rise in total expenditures. If a good is price inelastic, a rise in price causes a rise in total expenditures, and a fall in price causes a fall in total expenditures. If a good is unitary elastic, a rise or fall in price has no effect on total expenditures. These relationships are important for public policy purposes in order to assess the impact of tax changes on the total alcohol expenditures of various income classes.

Proponents of the single distribution model claim that demand for alcoholic beverages is price elastic. Hence, a rise in price will lead to a more than proportionate fall in the quantity demanded and a fall in total expen-

ditures on alcoholic beverages. However, if demand is price inelastic, a rise in price will lead to a less than proportionate fall in quantity demanded and a rise in total expenditures, seriously hampering the control of consumption by price changes.

2.2. The Estimation of Demand

Elasticity can also be described graphically. A downward sloping demand curve is depicted in Fig. 1. This linear demand curve can be expressed algebraically as:

$$Q^d = a + bP$$

where Q^d is the quantity demanded, a is the intercept with the vertical axis, b is the slope or steepness of the straight line, and P is the price. The intercept, a, denotes quantity when price is zero. The slope, b, denotes a change in quantity for a given change in price. If price changes from P_1 to P_2, then quantity changes from Q_1 to Q_2, and the slope $b = (Q_2 - Q_1/(P_2 - P_1)$. This means that a unit increase in price, say 1 dollar, will result in an increment in quantity in the amount of b. Following the first equation of price elasticity above, it is clear that once the slope is known, price elasticity may be found by multiplying the slope by the ratio of initial price to initial quantity, that is, $e_p = [(Q_2 - Q_1)/(P_2 - P_1)] \cdot P_1/Q_1$.

In actual practice, demand curves are estimated by multiple regression analysis. In the case of the linear demand curve above, the form of the regression equation would be: $Q^d = a + bP + u$, where Q^d, a, b, and P are as defined above, and u is an error term representing measurement error and the statistically unexplained portion of Q^d. This equation says that quantity demanded is explained by price. But this equation is clearly incomplete, for demand is a function of many other variables. The economic theory of demand states that demand is a function of own-price, the price of substitute goods, income, consumer tastes, and other relevant exogenous variables such as

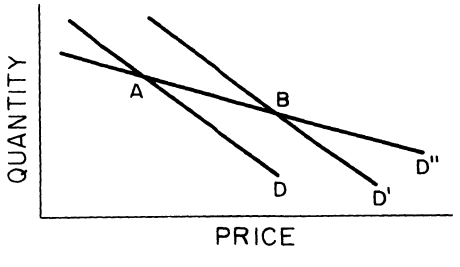

Figure 1. Measuring the price elasticity of demand.

advertising. A linear regression model incorporating these additional variables would appear as:

$$Q^d = a + b_1P + b_2P_s + b_3I + b_4T + b_5A + u$$

where the new variables are the price of substitute goods, P_s, income, I, consumer tastes, T, and advertising, A. Each of the slope coefficients, b_1 through b_5, can be used to calculate an elasticity with respect to its corresponding variable.

This equation allows us to satisfy the "holding all other variables constant" condition when measuring a particular elasticity. In other words, by including these other variables in the demand equation, the effect of price on quantity demanded can be isolated from the effects of these other variables on demand. These other variables can lead to shifts in the demand curve. By ignoring their effects, price elasticity will be biased in regression estimation. For instance, if income increased during a specific time period, demand would shift in Fig. 2 from D to D'. If we observed price and quantity demanded corresponding to point A in the beginning of the time period and price and quantity corresponding to point B at the end of the period, we would estimate the demand curve as D" if the effect of income were ignored. As can be seen, the incorrectly estimated slope of the demand curve is less than the actual demand curve slope.

The elasticities can be estimated directly by transforming the variables into their respective logarithmic values. The b coefficients then measure their respective elasticities directly. Estimating the equation in log form assumes that the demand equation is nonlinear and that elasticity is constant over the whole demand curve. The exact form of the demand equation to be estimated should be derived from economic theory and can be tested for goodness of fit to the data. This is rarely done in practice, but the log form of demand estimation is commonly used because of its convenience.

The variables for demand estimation are generally measured over time, for example, monthly or annually. This is known as time-series analysis. An alternate data set is one measured at a point in time over different geographic areas, such as states. This is known as cross-section analysis. In some cases, the two samples are combined to obtain pooled time-series cross-section samples.

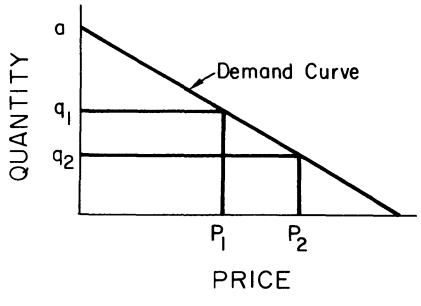

Figure 2. Shift in demand and regression biases.

The interpretation of own-price elasticity, as showing the percent responsiveness of quantity to a given percent change in own-price, holding all other variables constant, is analogous for the other variables in the log form of the demand equation. For example, in log form, b_3 is an estimation of income elasticity, which is defined as a percentage change in quantity demanded for a given percentage change in income. Income elasticity is generally positive; however, in some cases it may be negative. For instance, as disposable income rises, consumers may eat less potatoes and more steaks or consume less cheap wine and consume more fine wines. In these examples, potatoes and cheap wine would have negative income elasticities. A good is said to be income elastic if the elasticity is greater than 1. If a good is income elastic (inelastic), as income increases, the total proportion of income spent on a good rises (falls).

The effect of substitute good prices on consumption is measured by b_2 and is the cross-price elasticity. The effect of the price of other goods on the quantity demanded may be positive or negative. Two goods are called substitutes if the effect of a rise in the price of one good is to increase the quantity demanded of the other good. In this case, cross-price elasticity is positive. For example, if the price of wine is raised while the price of spirits stays constant, spirits consumption will rise, and wine consumption will fall, if the goods are substitutes. Two goods are called complements if the effect of a rise in the price of one good on the quantity demanded of the other good is negative. For example, if the price of butter rose while the price of bread and all other demand variables remained constant, bread consumption would fall. In this case cross elasticity is negative, and the goods are complements.

In summary, the choice of variables to be included in the regression equation determines which effects are isolated. In principle, all relevant variables should be included in the equation. Otherwise, the equation is misspecified, and the results are subject to error. In practice, this is rarely done, since certain variables are unmeasurable or too costly to obtain. The choice of variables included in the equation will be seen to significantly affect the various elasticity estimates.

In estimates of the demand for alcoholic beverages, the included explanatory variables are generally own-price, price of substitute goods, personal disposable income, and a time trend variable to account for changes in consumer tastes and all other miscellaneous changes correlated with time. In a few studies, an advertising variable is included. Other studies include a one-period lagged quantity variable, that is, quantity at the end of the previous year, to account for short-run and long-run adjustments in demand to changes in the explanatory variables. This is done because long-run price elasticity is greater than short-run price elasticity because more information on price changes and substitute products is available in the long run. A few studies also attempt to control for the influence of sociodemographic factors on demand, such as age, educational level, ethnic identity, and religious affiliations of consumers. Finally, specialized variables are included in some studies to account for such effects on demand as labor strikes, inventory adjustments, and illegal pro-

duction and sales. These variables are tested in various mathematical or function forms, including linear, logarithmic, and dynamic models. As will be seen below, the results vary widely depending on the variables included and the sample and period tested.

2.3. Some Problems in the Estimation of Demand

The actual estimation of demand curves is full of difficulties, making the estimated elasticities, at best, only approximations of true elasticities. The most serious problem is that a true demand curve, that is, a curve that shows how much will be demanded at various prices, is never observed. What is observed is how much is purchased at a given price for a given period of time. The necessary experiment of changing price to identify a given demand curve is rarely, if ever, performed by sellers. Estimates of demand are, therefore, generally a rough average of the interaction of demand and supply curves over time. This is obviously far from ideal but is the closest approximation available.

A second set of major problems deals with the econometric problems of statistical estimation of demand curves. These statistical problems create errors in the regression results, which, if not corrected, can so seriously bias the results as to make them useless. These are the classic econometric problems of measurement error, multicollinearity, autocorrelation, heteroscedasticity, and simultaneous equation bias. An explanation of these problems is beyond the scope of this study, but suffice it to say, they can singly or in combination destroy the reliability of estimated elasticities.[6]

Measurement error is a particularly serious problem in the estimation of demand for alcoholic beverages. The quantity and price variables in particular are subject to large potential error. As stated above, of most interest would be estimates of elasticities by incidence of drinking group. This would entail having data for each major explanatory variable, income, quantity, price, and so forth, by incidence of drinking group. Needless to say, such data do not exist as yet in the United States. Most studies are confined to using broad product categories, such as all alcoholic beverages or beer, wine, and distilled spirits, for the entire population. Some studies restrict the population of drinkers to adults only, persons above legal drinking age, or to the population 15 years of age and older to capture drinking by minors. However, a large segment of the population, possibly 30 or 40% never drinks alcoholic beverages, making per capita consumption figures highly inaccurate. In addition, state per capita consumption figures may be strongly affected by interstate traffic in alcoholic beverages and, during certain periods, illegal production and sale of such beverages.

In theory, errors in the dependent variable are taken up by the error term and lead to larger unexplained variation in demand. In practice, it is important to know the true nature of the dependent variable, since price elasticity relative to the dependent variable is at issue. Thus, whether the dependent variable

includes both drinkers and nondrinkers, is restricted to legal drinkers, or is limited to heavy drinkers is crucial in interpreting the meaning of price elasticity estimates.

Price series are also notoriously poor. The official United States price indices as constructed by the Bureau of Labor Statistics are generally sampled over only a few brands, which may be unrepresentative of total sales. For example, in distilled spirits, the whole class of nonwhiskies: vodka, gin, rum, tequila, and so on, are excluded from official data. Similarly, in wine, only certain types of red wine were sampled for years, and white wines were excluded. In some studies authors attempt to construct their own price series from prices and sales of a few leading brands. Again, this may be an unrepresentative sample. In short, existing data on price, quantity, and other explanatory variables are potentially subject to large measurement error, which may bias the results.

A final factor to be considered in interpreting price elasticity estimates is at what point on the demand curve the estimate is being made. In the case of straight-line demand curves, price elasticity varies throughout its range, from elastic in its upper regions to inelastic in its lower portion. This does not apply to logarithmic demand curves which have the unique property of constant elasticity throughout their range. Since elasticity is measured at a point on a demand curve, it matters where that point lies when working with straight line demand curves. In regression analysis, price elasticity is usually estimated at the mean values of price and quantity. For time-series data, this can be approximated by the midpoint of the period if the annual movements are steadily upward or downward. However, for public policy purposes, this means that elasticities should be evaluated at today's price and quantity, since average price and quantity over many previous years may be quite different from today's levels. This also means that for heavily taxed goods, such as alcoholic beverages, taxes may push these goods into the elastic regions of their industry demand curves.

3. Hypotheses to be Tested

Prior to a review of existing estimates of elasticities, a discussion of expected results is in order. Of crucial importance to the elasticities expected is the definition of the economic good being examined. Some studies estimate elasticities for alcoholic beverages in total, some for beer, wine, and distilled spirits separately, and some for more narrowly defined beverages, such as red wine under 14% alcohol. Price elasticities, in particular, will differ according to the level of product aggregation being examined. This is a result of the fact that price elasticity is an increasing function of the number of close substitutes available; the more close substitutes, the higher is price elasticity.

The first result expected is that price elasticity will be negative and statistically significant, confirming the notion that quantity demanded is in-

versely related to price. With regard to whether demand curves are price elastic or inelastic, this is largely a function of the product level being tested. The demand for alcoholic beverages in total is expected to be highly price inelastic because there are no close substitutes for alcoholic beverages. It is much more difficult to predict, *a priori*, the price elasticity of demand for beer, wine, and distilled spirits.

On the one hand, heavy taxation of each beverage, in particular wine and spirits, has been characteristic of governments for centuries. Since governments are generally interested in maximizing revenue, it is unlikely that they would choose price elastic goods, since price increases on such goods would reduce total tax revenues. This suggests that price elasticity is inelastic for beer, wine, and distilled spirits. Since distilled spirits are the most heavily taxed in this country, then wine, and then beer, this may indicate that distilled spirits are most inelastic and beer is least inelastic. Alternatively, there may be greater political support for taxing higher proof beverages. On the other hand, the price elasticities for beer, wine, and distilled spirits should be less inelastic than for total alcoholic beverages, and possibly elastic since they are, in principle, substitutes for one another. The extent of substitutability will determine the extent of elasticity. In short, it is not clear *a priori* whether individually beer, wine, and distilled spirits are inelastic or elastic. Finally, to carry this substitution argument one step further, the demand for a given brand of beer should be price elastic since there are many close substitutes across beers. In similar fashion, price elasticity across brands in wine and in distilled spirits should be price elastic.

Income elasticity, according to economic theory, is positive for normal goods. These are goods in which more is purchased as income rises, albeit, beyond some point at a decreasing rate. Inferior goods are those in which income increases lead to a decrease in consumption, for example, gruel. Income elasticity is expected to be positive and significant for all three beverages. Some may argue that beer is an inferior good, with consumers shifting to wine and spirits as income rises. We hypothesize that beer consumers will shift primarily to higher priced beers as income rises, just as consumers of wine and spirits will shift to more expensive brands. Thus, we expect positive income coefficients for each beverage. Needless to say, this is conjecture, subject to empirical tests. No predictions on whether income is elastic or inelastic can be made.

Cross-price elasticities, if they are statistically significant, are expected to be positive, reflecting substitutability. The extent of substitution should be, in part, determined by alcohol content. Thus, wine and distilled spirits should be more cross-price elastic than beer and distilled spirits. None of the cross elasticities are expected to be negative, reflecting complementarity among beer, wine, and distilled spirits. One rarely observes joint consumption of these beverages.

At the level of aggregation of existing studies, advertising is expected to be insignificantly related to quantity demanded. Advertising is hypothesized

to have little, if any, effect on interindustry shifts in demand. Advertising's prime influence is thought to be within industries, that is, between beers, between wines, or between spirits, and not as a determinant of shifts in demand between beer, wine, and spirits.

4. Demand Estimation Studies

A search of the literature uncovered 18 available studies published in English. Of this group, eight are studies of demand in foreign countries. There are undoubtedly many other foreign studies that are not available in English, especially from the Scandinavian countries which have long pioneered in alcohol research.[7] The tables summarizing the elasticities include the results of two of these studies plus two others, resulting in a total of 22 studies. All the major United States studies over the last 20 years are reviewed. The studies are presented in chronological order starting with studies from foreign countries.

4.1. Foreign Studies

One of the earliest studies of demand estimation was by Stone[8] for a number of commodities in the United Kingdom. Included in the study were estimates of demand equations for beer and distilled spirits based on annual data for the period 1920 to 1938. Beer demand was estimated by regressing consumption in barrels on own-price of beer, a price index of all other goods, real aggregate income before taxes (that is, deflated by a cost-of-living index), the strength of beer, and a time trend variable. The strength of beer was measured by the original gravity of beer before fermentation. Stone found that income had a negligible influence on beer consumption and that almost all the variation in consumption was explained by own-price and the price of other goods. Strangely, the price of other goods was found to have a larger influence on consumption than the price of beer. The price and income elasticities found for beer were -0.73 and $+0.14$, respectively, both apparently inelastic. However, no tests for statistical significance were given. The strength of beer was also positively related to consumption with an elasticity around 1. The time trend variable suggested little variation from factors not introduced explicitly.

The demand for distilled spirits was estimated by regressing proof gallons of consumption on the ratio of spirit price to the price index of other goods, aggregate real income, and a time variable. The price of spirits was deflated by the general price index, since spirit prices were controlled by the government and did not change from 1920 to 1938. Using proof gallons as the dependent variable implicitly introduced strength of the beverage. The results were a price elasticity of -0.72 and income elasticity of 0.54. Again, no tests of significance were given.

Stone's estimates are likely to be unreliable and possibly biased. First, Stone admits the existence of strong serial correlation among the residuals, which tends to increase the sampling variance of the estimates. Second, Stone did not hold population constant by using per capita figures, thus misspecifying the regression equations. The equations are further misspecified by not accounting explicitly for the price of close substitute beverages. Third, there is measurement error in the income variable, since it is not measured as disposable income; that is, personal income after taxes, thus biasing income elasticity.

In a subsequent study, Stone[9] replicates his earlier regressions using first difference equations to correct for serial correlation. In addition, Stone gives confidence intervals around each elasticity at the 0.05 level, which can be used to test for statistical significance. The results are remarkably similar to the 1945 results. The own-price elasticity for beer was -0.69, significantly greater than zero and less than one. Income was not included in the regression because of multicollinearity. The all other price variable and the strength of beer were also significantly greater than zero. For spirits, price and income elasticity were -0.57 and 0.60, respectively, both significantly greater than zero and less than one. Once again, no attempt was made to control for population or price of close substitutes. In addition to these results, estimates for imported wine were made using own-price, price of all other goods, and income as explanatory variables. Own-price elasticity was -1.17, and income elasticity was 0.98, both significantly greater than zero but not significantly different from one. The results suggest that both beer and spirits were price and income inelastic over this period, but imported wine was of unitary price and income elasticity. Stone offers no economic explanation for these results.

Prest[10] estimated price and income elasticities for United Kingdom expenditures on beer and spirits over the period 1870 to 1938. Prest regressed quantity demanded per capita on real national income per capita, price relative to a cost of living index, and a time trend variable to account for changes in tastes and all other miscellaneous changes. By deflating the variables by population and cost of living, Prest avoids the obvious biases inherent in Stone's estimates.

In the beer equation, quantity is measured in barrels per capita, and price is adjusted for bottles and draught and for consumption in cheap and expensive bars. Prest estimates demand in linear, log, and log first-difference form (to reduce autocorrelation). The first-difference results are preferred, and Prest concludes that price elasticity was -0.66 ± 0.08 and income elasticity was $+0.23 \pm 0.12$ over this period. No statistical tests of significance were given; however, the confidence limits are probably at the 95% level and indicate both price and income to be highly inelastic and significant. Prest also found that relative price had a stronger influence on demand than income. In short, over this period in the United Kingdom, beer appears to have been both price and income inelastic, and changes in price had the most influence on changes in the quantity demanded.

For spirits, quantity is measured in gallons per capita, and price is adjusted for the proportion of sales in bottles and glasses. The demand equation is estimated in log, log first-difference, and linear form. Prest concludes there is no way to determine which estimates are the most reliable, but he leans toward the log estimates as being more consistent with consumer behavior. The price and income elasticities in log form were -0.57 and $+0.70$, respectively, but these changed in linear form to -0.03 and 0.95, respectively. Again, no significance tests were given. Prest's results are similar to Stone's, although Prest extended the period of analysis by 50 years. Both studies found beer and distilled spirits to be highly price and income inelastic, with price explaining most of the variation in consumption.

Malmquist[11] estimated price and income elasticities for spirits in Sweden, using annual data on liquor sales from 1923 to 1939. The study was unique in that over this period, Sweden had a system of limiting sales by the use of licenses to purchase alcoholic beverages and the use of rationing books. The ration given each buyer was a function of consumer age, income, marital status, previous consumption abuse, and other special factors. For much of the period, spirit purchasers were limited to 4 liters per month, but there was no limit on wine. The number of "limited purchase" ration books as a percent of all ration books for men increased continuously over the period from 69% in 1923 to 79% in 1940. There was also a major change in age requirement in 1933, when legal age was dropped from 25 to 21 years of age, but Malmquist did not control for this in the regressions.

In the initial regressions, Malmquist regressed quantity of spirits sales by liquor stores on price of spirits, consumer income, the average ration per ration book holder, and a cost of living index, all in log form. Price was measured as average price over all sales, and income was based on tax records. Rather than deflating price and income by the general price index, Malmquist included the general price index as a separate explanatory variable. Similarly, rather than deflating quantity and income by population or the number of ration books, the deflator was included as a separate variable. In subsequent regressions, Malmquist used real prices and incomes, but the results were quite similar to those using nominal values.

Using real price and income, price and income elasticities for spirits were -0.369 and $+0.300$, respectively. Estimates using alternative price series, for example, the price of a leading brand and average price adjusted for shifts in the types of spirits purchased, yielded similar results. No tests of statistical significance were provided. In a separate equation regressing wine sales on the price of spirits and income, cross-price elasticities for spirits and wine ranged from -0.64 to -1.01, depending on the measures of the variables used. These results suggest that wine and spirits are complementary goods.

There are a number of problems with these estimates. First, Malmquist acknowledges a problem of autocorrelation. Second, the price of substitutes is not in the spirits equation, and the price of wine is not in the wine equation. Third, and most important, the system of rationing in use in Sweden strongly

conditioned consumers' reactions to price and income changes. Hence, these are estimates of elasticities with a rationing constraint, and they cannot be compared to those under a distribution system free of government rationing.

Walsh and Walsh[12] estimated price and income elasticities for beer and distilled spirits in Ireland using time series data from 1953 to 1968. Their model regressed consumption in barrels and gallons per capita on own-price, the price of beer relative to the price of spirits, disposable personal income per capita, and a time trend variable. All terms were appropriately deflated. The authors estimated their model in its fully specified form and also with the time trend and price of substitute goods deleted. They found that income explained more than 90% of the variation in both beer and spirits consumption. There was little difference in the results between linear and log-linear models in terms of the elasticities observed. In six alternative regressions for each beverage, the price variable was never statistically significant. It was negative in each case for spirits, but in only three cases for beer. These results are rather implausible. The estimates of income elasticity for beer were all statistically significant and ranged from 0.50 to 0.79. For spirits, the income estimates were all significantly greater than zero and ranged from 1.48 to 2.06. Thus, changes in income had a decidedly larger influence on consumption of spirits than of beer. Their best income estimates for beer and spirits were 0.79 and 2.04, respectively, both significantly greater than zero, but neither was significantly different from 1.

The elasticities of price are much less reliable than those of income. First, they were all statistically insignificant, and half the beer estimates had the wrong sign. Second, there was extremely high multicollinearity between income and the price of both beer and spirits, which can seriously affect the reliability of all three coefficients and their standard errors. Given the significance of income, this effect seems to have fallen on price. The author's best estimate for beer price elasticity was -0.167, and for spirits it was -0.57, both highly inelastic. However, by including the ratio of beer price to spirits price in each regression, they unwittingly confounded the own-price elasticities, since the price of beer appears twice in the beer equation, and the price of spirits appears twice in the spirits equation.[13] Using theIr full model in logs, the corrected own-price elasticities are 0.158 for beer and -0.446 for spirits. The beer result is inconsistent with economic theory and obviously in error, and the spirits result is of questionable validity.

Lau[13] estimated price and income elasticities for beer, wine, and distilled spirits in Canada using annual data for 1949 to 1969. Lau's initial model was identical to that of Walsh and Walsh, except that adult population over 15 years of age was used as a per capita deflator. Estimates were made in linear and log-linear form with no *a priori* preference stated. Each form, of course, assumes a different demand curve, a straight line in one case and a curved constant elasticity demand curve in the other case. In general, Lau found no significant difference between these functional forms. The best estimates chosen for each beverage were those shown in Table I.

Table I. Price and Income Elasticities in Canada[13]

	Beer	Wine	Spirits
Price elasticity	−0.0312	−1.6530	−1.4491
Income elasticity	0.2048	1.4273	0.6795

These were all elasticities estimated at the means using linear equations. The wine and spirits price and income elasticities were each significantly different from zero and 1. When wine was estimated in logs price elasticity fell to −1.293 and income elasticity to 0.7359. Equivalent changes in spirits when estimated in log form were −1.173 and 0.5860, respectively. No significance tests for the beer elasticities were given because of the method of estimation used. That is, the beer equation was estimated after correcting for potential autocorrelation problems and after transforming the explanatory variables by the method of principal components to reduce the extent of multicollinearity.

This indirect method of estimation yields dubious results when precise estimates of elasticities are desired. Unfortunately, Lau made the same error as Walsh and Walsh by including the own-price variable twice in the spirits and beer equations. (Lau's best estimates for wine did not include beer and spirit price indices, although other wine regressions did.) Correcting the log-linear form of Lau's preferred spirits equation yields a price elasticity of −0.162, in contrast to the highly elastic estimate above. The beer price variable in the spirits equation is negative and significant at −1.011, implying that spirits and beer are complements. Correcting those beer equations where adjustments are possible shows beer still to be highly price inelastic and beer consumption to be insignficantly related to the price of spirits.

In an earlier study of Canadian alcoholic beverage demand, Johnson and Oksanen[14] estimated elasticities for beer, wine, and distilled spirits across time, over the period 1955 to 1971, and across Canadian provinces. Their study is unique in using pooled time-series cross-section data and in using sociodemographic variables to explain differences in per capita consumption. Their demand equation consists of consumption per capita 15 years of age and over regressed on the price of each of the alcoholic beverages, income per capita, consumption of the previous year to account for habit persistence, a time trend variable to account for changing tastes, dummy variables for each province, dummy variables for industry strikes, the percentage of the 15 and over population that is in the 25–54 age group, and seven ethnic, six religious, and three educational variables. All value terms were deflated by the Canadian CPI. The authors maintain that the simultaneous equation problem does not exist in Canada because prices are set by the government and are thus predetermined; that is, demand does not affect prices. However, the theoretical justification for this rationale is questionable, since a monopolist

Table II. Price and Income Elasticities in Canada[14]

	Price elasticities at the mean			Income elasticities at the mean
	Beer	Spirits	Wine	
Beer consumption	−0.224	−0.112	−0.033	0.035
Spirits consumption	−0.127	−0.910	0.210	0.227
Wine consumption	0.231	0.138	−0.502	−0.008

(the government) should react in principle to changes in demand just as readily as a perfectly competitive firm, unless one postulates that the government does not attempt to maximize net profits. They tested for autocorrelation and found only the wine equation to require correction.

By including lagged consumption as an independent variable, Johnson and Oksanen transformed their elasticities into 1-year or short-run elasticities. The short-run elasticities reported were as shown in Table II.

The own-price elasticities, the diagonal row in Table II, were all significantly different from zero and 1 at the 0.05 level, indicating that each beverage had short-run price-inelastic demand. The cross-price elasticities yield ambiguous results. The only significant coefficients were beer in the wine equation and wine in the spirits equations. These results indicate that beer is a substitute for wine and wine is a substitute for spirits. However, wine is not significant in the beer equation and has the wrong sign, and spirits are not significant in the wine equation. Finally, the signs on beer and spirits cross elasticities indicate that they are complements, consumed together, such as bourbon and a beer chaser. The only short-run income elasticity that was significantly different from zero was for spirits.

The long-run own-price elasticities for beer, spirits, and wine were −0.379, −1.599, and −1.301, and the corresponding income elasticities were 0.60, 0.399, and 0.022. Statistical significance was not reported. The long-run own-price elasticities suggest that only beer is price inelastic, but Johnson and Oksanen do not report whether the estimates are significantly different from 1.

Of the remaining variables, the province dummies were all highly significant, indicating differences in consumption relative to Ontario, the omitted province; ethnic group, represented by European heritage, was highly significant and positive in the beer and wine equations; religious affiliation, represented by various denominations, was positive and significant in the wine and spirits equations; level of education was negative and significant in the spirits equation; the age–demographic variable was positive and significant for spirits only; and the strike variables were consistently significant. Using the short-run own-price elasticities, Johnson and Oksanen conclude that a tax increase to decrease consumption would reduce spirits the most and beer the least, whereas a tax increase to raise revenue should be levied

against all three beverages, with the largest increase for beer and the smallest for spirits.

In a subsequent paper, Johnson and Oksanen[15] modified their first paper by (1) using the principal components method to reduce multicollinearity among the sociodemographic variables, (2) expanding their strike dummy variables to include direct effects within the province the strike occurs, strikes in adjoining provinces, and strikes in substitute alcohol beverages, (3) estimating three different versions of their model using ordinary least squares, least squares with dummy variables, and generalized least squares, and (4) using first-differencing as a first approximation correction to autocorrelation. This study is probably the best econometric work on price and income elasticities for alcoholic beverages.

The results confirm the own-price elasticities and income elasticities of the first study, but the cross elasticities differ. Beer remains a substitute for wine, but spirits are now a complement with beer, and wine is no longer a substitute for spirits. The ethnic effect is generally significant in all equations; the religion effect is never significant; and schooling is significant for beer only. The strike dummies are all highly significant. Finally, there is no evident superiority in any of the three estimation techniques, with ordinary least squares just as effective as the other two (Table III).

The own-price elasticities are all significantly different from zero and 1, but, as in their earlier study, only the spirits income elasticity is significantly different from zero. In contrast to the earlier results, spirits is now price elastic and more income inelastic.

The corresponding long-run own-price elasticities for beer, spirits, and wine were -0.33, -1.77, and -1.78, respectively, consistent with the earlier paper with the exception of wine which is now more elastic. The long-run income elasticity for spirits was 0.17, far lower than their earlier estimate.

It is instructive to compare Lau's revised results for Canada[13] with those of Johnson and Oksanen.[15] The revised results of Lau show both beer and spirits to be highly price inelastic and wine to be elastic, whereas Johnson and Oksanen find beer and wine to be price inelastic and spirits to be price elastic in the short-run. Lau's figures show that a doubling of the price of beer and spirits would lead to roughly a 3% fall in beer consumption and a 16% fall in spirits consumption. These changes are so low as to be highly suspect. This comparison highlights the sensitivity of regression results to

Table III. Ordinary Least Squares Short-Run Elasticities at the Means[15]

	Own-price	Income
Beer	−0.27	0.002
Spirits	−1.14	0.11
Wine	−0.67	0.04

the specification of the model tested and shows how cautious one should be in interpreting studies of elasticities for public policy purposes.

4.2. United States Studies

Niskanen conducted one of the earliest studies of alcohol beverage demand in the United States.[16,17] He estimated single-equation models of demand and multi-equation or simultaneous equation models of supply and demand. The simultaneous equation model includes seven interrelated equations, three supply equations, and a separate equation for the supply of illegal beverages. The simultaneously determined or endogenous variables are the three price variables (which are included in each demand equation), the three quantity variables (deflated by the number of adults), and the capacity of illegal stills seized per adult. The exogenous variables are the separate producer inventory variables for spirits and wine, separate tax variables for each of the three beverages, a time trend, disposable personal income per capita, and demand deposits and currency per capita. The demand equations showed consumption as a function of own-price, substitute product price, illegal production, disposable income, and currency and demand deposits. The supply equations showed quantities as a function of own-price, own-taxes, producers' beginning of the year inventories, and a time trend variable. Niskanen's contribution was in specifying a series of demand and supply equations and estimating them jointly to correct for simultaneous equation bias. No subsequent study, however, seems to have adopted this model.

The equations were estimated in log, semilog, first-difference, and linear forms, and tests for autocorrelation were conducted. The linear form was chosen, and the presence of autocorrelation was rejected. Over the periods 1934 to 1941 and 1947 to 1960, 22 annual observations were used.

The ordinary least-squares and the simultaneous equation model (three-stage least-squares) results are given in Tables IV and V.

The simultaneous equation estimates of nonprice elasticities are uniformly higher than the ordinary least-squares estimates. Niskanen preferred

Table IV. Direct Least-Squares Estimates of the Demand Elasticities

Quantity	Price spirits	Price beer	Price wine	Income	Currency and demand deposits
Spirits	−0.925[a]	0.646[a]	−0.408	0.621[a]	0.339
	(0.375)	(0.300)	(0.259)	(0.247)	(0.240)
Beer	0.416	−0.333[a,b]	−0.440[a]	−0.270[a,b]	0.174
	(0.222)	(0.182)	(0.161)	(0.128)	(0.133)
Wine	−0.543	0.240	−0.353	0.798[a]	0.310
	(0.484)	(0.396)	(0.351)	(0.283)	(0.290)

[a] Significant at the 0.05 level.
[b] Significantly different from 1.

Table V. Estimates of the Third-Stage Demand Elasticities Assuming a Constant Real Distributors' Margin

Quantity	Price spirits	Price beer	Price wine	Income	Currency and demand deposits
Spirits (legal)	−2.027 (0.445)	0.294 (0.270)	0.078 (0.353)	0.608[a,b] (0.115)	0.398[a] (0.140)
Spirits (total)	−1.773 (0.389)	0.249 (0.229)	−0.068 (0.309)	0.532[a] (0.101)	0.349[a] (0.122)
Beer	0.552 (0.349)	−0.502[a,b] (0.207)	−0.563 (0.279)	−0.333[a,b] (0.115)	0.123 (0.132)
Wine	0.952 (1.160)	1.258[a] (0.575)	−1.585 (0.785)	1.451[a] (0.305)	−0.182 (0.380)

[a] Significant at the 0.05 percent level.
[b] Significantly different from 1.

the simultaneous equation estimates since, in theory, they yield unbiased estimates. However, this only applies for large samples, and Niskanen's sample is very small, with only 22 observations, suggesting that the choice between the two procedures is arbitrary at best. Using the full model, Niskanen reports estimates of the own-price elasticities as −2.027 for spirits (legal), −0.502 for beer, and −1.585 for wine. All are significantly different from zero, and the beer and spirits estimates are significantly different from 1. Niskanen placed the least confidence in the wine estimates because of large variability in results across various wine equations.

Niskanen's analysis assumes that the three beverages comprise one market. However, the results indicate otherwise. The reduced form equations (regressing each endogenous variable on all the exogenous variables) show the consumption of each beverage to be generally unrelated to the tax rates of the other beverages. The cross elasticities presented above provide further evidence of how each beverage is affected primarily by its own market conditions. Beer is significant and positive in the wine equation, indicating that beer and wine are substitutes, but wine is not significant in the beer equation and is of the opposite sign. Because the cross-price elasticities are generally insignificant, Niskanen was only willing to conclude that "beer and spirits appear to be weak substitutes."

The income elasticities in both sets of results are all significantly different from zero, and the spirits and beer elasticities are significantly different from 1. However, the beer income elasticity is of the wrong sign for a normal good, that is, a good in which more is purchased as income rises. Another strange result is that the spirits category is income inelastic, whereas wine is not significantly different from unitary elasticity.

Niskanen estimated an alternative model dropping out the cross-product terms since they yield inconsistent results. This model also omitted the tax rates and producers' inventories. The retail own-price elasticities were about −2.0, −0.6, and −0.7 for spirits, beer, and wine, respectively. They are

roughly consistent with those above with the exception of wine. For largely unexplained reasons, Niskanen preferred these estimates. The income elasticities, however, were quite different from those above, being 0.23, −0.27, and 0.68 for spirits, beer, and wine, respectively, with only wine being significant.

Niskanen's major conclusions were: (1) consumption of each beverage is significantly related to its own-price and little affected by changes in the price of substitute alcoholic beverages; (2) the own-price elasticities of spirits, beer, and wine are about −2.0, −0.6, and −0.7, respectively; and (3) consumption of each beverage is significantly related to the level of personal disposable income, with income elasticities for spirits, beer, and wine of 0.4, −0.3, and 1.0, respectively.

Comanor and Wilson[18] used a two-equation model to estimate the United States demand for malt beverages, wine and brandy, and distilled spirits, using IRS data for the period 1948 to 1964. In constrast to previous studies, Comanor and Wilson included advertising as an explanatory variable. In fact, their chief concern was in estimating the impact of advertising on demand, and the simultaneity problem they identify is between advertising and quantity demanded. Thus, their model includes a demand equation and an advertising equation for each product. The demand equation regresses industry sales per capita, deflated by the wholesale price index, on industry advertising as a percent of total manufacturing advertising, own-price deflated by the Gross National Product (GNP) implicit price deflator, total per capita expenditures deflated by the GNP implicit price deflator, and lagged industry sales per capita. The advertising equation regresses total industry advertising on total sales, lagged total sales, own-price deflated by an advertising index, the latter variable lagged, and lagged advertising expenditures. This equation is not deflated by population or a price index, since Comanor and Wilson maintain that short-run decisions are made in current dollars, and advertising responds to current dollar changes in demand, not constant dollars. Both justifications are unconvincing, and, in addition, lack of deflation causes dimensionality problems in their two-stage least-squares estimation. Another weakness in their model was failure to account for changes in the price of close substitutes, a clear misspecification of the demand equation.

Comparing the results of two-state least-squares estimation with ordinary least squares, Comanor and Wilson found the biases in price and income elasticities to be very small and chose the single-equation results as their best estimates of elasticities. The estimated short-run (SR) and long-run (LR) elasticities are shown in Table VI.

For short-run estimates, the price elasticities were statistically significant for beer and wine but not for spirits; income elasticities were all insignificant; and advertising elasticities were significant in spirits and wine but not in beer. The lack of significance for income elasticities is inconsistent with most other studies of demand estimation. The wine price elasticity was significantly less than 1, but the beer and spirits elasticities were not significantly different

Table VI. Short- and Long-Range Elasticities[18]

	Price		Income		Advertising	
	SR	LR	SR	LR	SR	LR
Malt beverages	−0.562	−1.392	−0.184	−0.456	0.004	0.010
Wine and brandy	−0.680	−0.842	0.407	0.503	0.972	1.202
Distilled spirits	−0.253	−0.295	0.179	0.208	0.641	0.745

from 1. Thus, it is not clear from these results whether beer and spirits were elastic or inelastic. Two final observations are: (1) beer has a negative income elasticity, suggesting that it is an inferior good; and (2) advertising is shown to be related to sales of wine and spirits but not of beer, a totally inexplicable result. The results, to say the least, are weak and ambiguous. One suspects that there are serious problems in the data used and methods of estimation.

In a reexamination of the Comanor and Wilson model, Grabowski[19] criticizes their use of IRS data for advertising and sales figures and their use of Bureau of Labor Statistics (BLS) price data coupled with national income account data on income. The use of IRS data suffers from aggregation errors, since corporation consolidated financial statements are assigned to a particular industry. Given changes in corporate diversification over time, firms will be reassigned to other industry categories. Grabowski argues that this will produce a spurious positive correlation between sales and advertising over time. Grabowski also maintains that mixing IRS data with BLS price data and national income account measures of disposable income biases the correlation of price and income with quantity toward zero. Finally, he criticizes Comanor and Wilson for misspecifying their two-equation model by deleting variables for estimation purposes.

Grabowski attempts to rectify these problems by using national income account measures for sales, price, and income and advertising data from media trade sources. A consequence of using this new data is that Grabowski is forced to use more highly aggregated industry classes, resulting in his estimating the demand for alcoholic beverages in total rather than by beer, wine, and spirits. Therefore, we would predict inelastic price estimates, since there are no close substitutes for alcoholic beverages.

Grabowski estimated a demand equation in both single and simultaneous equation models for the period 1956 to 1972. The demand equation regressed consumption expenditures per capita on own-price, disposable income per capita, lagged consumption, and advertising expenditures measured either in per capita terms or relative to total national advertising expenditures. The advertising equation in the simultaneous equation model assumed that current industry advertising is a function of industry sales, lagged advertising expenditures, and the industry discount rate, measured as the AAA bond rate from Moody's.

For alcoholic beverages, the single-equation estimation yielded a price

elasticity of −0.08, insignificantly different from zero but significantly less than 1. Income elasticity was highly inelastic and significant at 0.26. Advertising elasticity was insignificant and varied between −0.01 and −0.02, indicating that advertising was unrelated to alcoholic beverage consumption. In the simultaneous equation results, the estimates vary according to whether advertising is measured in per capita or relative terms. In per capita terms, price elasticity was insignificant at −0.39, income was again the dominant explanatory variable and was highly inelastic and significant with an elasticity of 0.34, and advertising elasticity was insignificant at −0.008. Measuring advertising in relative terms, price elasticity was −0.52 and significant, income elasticity was 0.38 and significant, and advertising was insignificant at 0.082.

Grabowski's major finding is that, in contrast to the results of Comanor and Wilson, advertising is insignificant in the demand equation, but demand is significant in the advertising equation, suggesting that causality runs from sales to advertising and not from advertising to sales. This, of course, applies only to interindustry shifts in demand. With regard to the demand for alcoholic beverages, as expected, both price and income appear to be highly inelastic, although price is disturbingly insignificant in most regressions.

Other studies of alcoholic beverage demand in the United States focused on one beverage. We turn now to a review of these studies starting with beer and then proceeding to distilled spirits and finally to wine.

4.2.1. Beer. Horowitz and Horowitz[20] estimated the demand for beer across states in 13 separate regressions for the period 1949 to 1961. Because of a lack of price data by individual states, they used state taxes on beer as a proxy for beer price; that is, they assumed that differences in state taxes reflected differences in beer prices across states. Their rationale was that to the extent that taxes are passed on to consumers, inter-state tax differences reflect price differences. They also argue that taxes are not wholly exogenous, being partly determined by a state's attitudes toward beer consumption. As a result, they specify a demand equation and a tax equation and use indirect least squares to estimate a two-equation model. Their demand model regresses consumption in physical quantity per capita on state taxes, state income per capita, and lagged consumption to allow for partial adjustment of consumption for habit. They found tax (price) coefficients to be of the correct sign in nine out of the 13 annual regressions, but none were significantly different from zero, indicating that tax rates are not related to consumption. However, seven of the nine coefficients were significantly less than −1.0, and the authors conclude that the demand for beer is probably price inelastic. The income elasticities were significant for 8 years, from 1949 to 1956, but inexplicably insignificant in the 5 years thereafter, from 1957 to 1961.

The findings of Horowitz and Horowitz are likely biased downward because of measurement error in the use of state tax rates as proxies for beer prices. In addition, they fail to control for changes in price of close substitute products, misspecifying their equation. All in all, their estimates lack credibility.

Hogarty and Elzinga[21] estimated price and income elasticities for beer using data for 48 states and the District of Columbia over the period 1956 to 1959. The price data were derived from a Justice Department suit challenging the 1958 merger of Pabst and Blatz [*United States v. Pabst Brewing Co., 233 F. Supp. 475 (1964)*]. Adding Federal and state excise taxes (four states with significant local taxes were dropped) to FOB mill prices of Pabst Blue Ribbon and Blatz Pilsner, a weighted average price of beer was calculated for each state based on the amount of packaged and draught beer consumed in each state. The basic regression equation used apparent consumption in cases per adult, the price series deflated by the CPI, and state per capita real income. The authors argue that a simultaneous equation problem does not exist because there was large excess capacity during 1956 to 1959 and thus supply was perfectly elastic.

The authors estimate their equation in log-linear form using pooled data for all 4 years. The price and income elasticities were -1.128 and 0.926, respectively, significantly different from zero but not significantly different from 1. Adding proxies for the prices of spirits and wine did not change the elasticities, and the cross elasticities showed beer and spirits to be complements, which the authors rejected as implausible. Adding an ethnic identity variable, the percentage of a state's population that was foreign born, lowered the price elasticity to -0.89, still not significantly different from unity, and the income elasticity to 0.4, which is significantly different from unity. Thus, Hogarty and Elzinga conclude that the price elasticity of demand for beer was unitary during the late 1950s and highly income inelastic. Whether these elasticities hold today in light of changes in the demand for beer is questionable. Consumption per capita was declining throughout the 1950s, whereas it has risen steadily since 1963.[22]

Norman[22] used IRS time-series data for 1946 to 1970 to estimate the demand for beer faced by manufacturers, that is, the demand of beer wholesalers. Norman hypothesized that demand for beer, measured in total barrels, is a function of real price per barrel, real disposable income, the price of substitutes, total real advertising expenditures, and the total population aged 20 to 44. Price is measured as total revenues divided by total barrels sold and thus is a composite price over all brands, all breweries in the IRS income statements, and over draught and packaged beer. The price of substitutes was measured as the ratio of the CPI for all alcoholic beverages to the CPI for packaged beer. Since packaged beer is part of the composite price, this latter variable confounds the price elasticity estimates with no possibility of correction. This error leads to an upward bias in Norman's price elasticity estimates.

Norman estimated his demand equation in stages, adding and then deleting variables. At no point was the full model estimated. Results using just own-price and income yielded a significant price elasticity of -1.07, but it was not significantly different from 1, and a significant income elasticity of 0.30, significantly less than 1. Including the price of substitutes and adver-

tising variables lowered own-price elasticity to −0.88 and raised income elasticity to 0.35 but did not change their statistical significance. The substitute product price was positive and insignificant. Deleting the substitute price variable changed advertising to positive and significant. This experiment suggests substantial multicollinearity. However, this last regression was run over more years, and this may account for the large change in the advertising variable. In the final regression, Norman included the population variable with own-price and income, but the population variable proved insignificant. One problem in all but the last regression was that population changes were not controlled for by using per capita consumption and income, thereby biasing the results. A final problem was possible simultaneous equation bias resulting from interactions between sales and price. In short, there are sufficient shortcomings in the study to be skeptical of the results.

Fortunately, Norman included the raw data he used in his study, allowing us to examine some of these issues. We reexamined his model by: (1) attempting to replicate his original results, (2) estimating the full model Norman hypothesized but did not report on, (3) extending the time period of analysis to 1975, and (4) testing numerous other variations on his model including using per capita consumption and income figures, including the CPI price indices of spirits and wine as alternative substitute price variables, adding a time trend variable, using a partial adjustment model, and testing for a shift in functional form.

In the process, we discovered a fundamental error in the data and found drastically different results by testing Norman's full model. Norman presented the data as if they were calendar year data when, in fact, they were fiscal year data. The error stems from a lack of congruence between quantity in barrels of beer and sales in total dollars. These series are out of phase by 1 year, the sales data always being one fiscal year ahead of the quantity data. When the data are corrected and the regressions rerun, price elasticity is considerably lower, in the range of −0.35 and −0.45, and significantly greater than zero and less than 1. In the full model, income elasticity is 0.13 and insignificant, far different from Norman's estimate. The results using the full model with uncorrected data are similar to these latter figures, with price elasticity of −0.44 and income elasticity of 0.06. Extending the period of analysis to 1975 and using corrected data yielded a smaller but still significant price elasticity of −0.35 and income elasticity of 0.06, again insignificant. The population variable was significant, advertising was insignificant. The test of a shift in functional form between the periods 1953 to 1962 and 1963 to 1975 resulted in the finding of a significant shift between the two periods when the model was tested in per capita terms. This shift, most likely caused by changes in the age distribution of the population, went unexplained by Norman's regressions.

Norman's results are thus misleading. Based on his corrected data and full model, price elasticity is close to −0.4, and income elasticity to 0.10. The results are more in line with those of Johnson and Oksanen[15] than those of Hogarty and Elzinga.[21]

4.2.2. Distilled Spirits. The consumption of distilled spirits presents some unique problems in measurement. Because of the relatively high price of spirits and the high rate of spirits taxation, there are strong incentives to reduce costs by illegal activity. The two main illegal activities are interstate traffic by consumers to affect differential price savings and illegal production, both of which distort state per capita consumption figures. As a result, several innovative attempts to estimate demand for distilled spirits have been made.

Simon[23] notes a number of potential problems with distilled spirits time-series studies, the most serious being (1) that tastes change, leading to demand shifts over time, which result in simultaneous equation bias in single equation models, and (2) that there is little fluctuation in current prices over time, leading to weak regression estimates of price elasticity. Cross-section studies present another set of problems, the most serious, according to Simon, being large differences in consumer preferences across states owing to social, cultural, religious, and so on, differences, which are hard to control for in regression analysis. In an attempt to avoid these problems Simon developed a novel, nonregression, approach to estimating price elasticity. In Simon's words,

> The essence of the method is to examine the "before" and "after" sales of a given state, sandwiched around a price change and standardized with the sales figures of states that did not have a price change. The standardizing removes year-to-year fluctuations and the trend. We then pool the results of as many quasi-experimental 'trial' events as are available.

More specifically, the price elasticity of each state is calculated in three steps. First, compute the percentage change in state per capita consumption between 12-month periods before and after a state tax change, using tax as a proxy for price changes. Second, deduct a similar calculation for a group of comparison states in which no price change occurred, in order to eliminate the effects of outside influences on consumption. Third, divide the result by the price change expressed as a percent of the state's retail price of a medium-priced fifth of spirits, using Seagram's 7 Crown as an index of spirit price in each state. Simon attempts to reduce error by selecting tax (price) increases of 2% or more, excluding states with large illegal consumption, and excluding state price changes that occurred around changes in Federal excise taxes.

Based on 23 independent observations over the period 1950 to 1961, Simon obtained price elasticities ranging from -4.35 in Alaska to 0.95 in Rhode Island. Some states appeared twice with inconsistent results. For instance, Ohio either had a price elasticity of 0.82 or -1.32 and Maine's was either -0.12 or -0.84. Needless to say, this does not bolster confidence in Simon's technique. California's price elasticity based on one observation for July 1, 1955, was -0.96. The median estimate over all observations, which Simon regards as his best estimate for the United States, was -0.79.

Simon readily admits that his estimates are subject to error as a result of poor population estimates, different price effects on off-sale and on-sale consumption, interstate traffic purchases, plus other measurement problems. In

addition, the method does not take explicit account of changes in income, price of substitutes, and other exogenous changes differing across states. Unless these other variables are changing in the same direction and to the same extent in the comparison states, the price elasticities will be biased. Other problems are the use of one observation in most cases to estimate a state's elasticity and the use of a single brand price to represent all brands. No subsequent studies have adopted Simon's method, an indication of its reliability.

Wales[24] attempted to correct price elasticities of distilled spirits for interstate consumption traffic. This concern with interstate traffic may seem unrealistic to those in California, but in the East, with much smaller states, to the extent price differentials are large relative to transportation costs, illegal interstate traffic is to be expected. One state, New Hampshire, is known for placing its state-owned stores on its borders to attract sales from adjacent states, especially from residents of Massachusetts. Wales' contribution is in developing a method for determining the extent of purchases in a state by out-of-state residents. He estimates the cost of transportation relative to price differentials between adjoining states and the population within a geographic area that will be affected by potential cost savings. These effects of in-state and out-of-state consumption were separated indirectly in his regressions by replacing a state's price and income variables with functions of these variables and price and income of adjacent states. This necessitated a complicated estimation procedure.

The sample consisted of 42 observations for 1960. Consumption was measured in number of cases sold, income in disposable income per adult, and price was a weighted average index based on the price and sales of nine leading brands. Other estimates for population, distance, and area were required. Two regressions were run, one taking interstate traffic into account and the other ignoring it. Ignoring interstate traffic yielded significant price elasticities for individual states generally greater than 1. Accounting for interstate traffic yielded an insignificant price elasticity over all states of approximately zero. Average income elasticity was approximately 1.7 under both regressions. Wales interprets these studies as showing that a model that ignores interstate consumption generates spurious results because high per capita consumption in low-price states includes purchases from other states, and low per capita consumption in high-price states reflects residents going out of state to make purchases. Thus, price elasticity estimates without an adjustment for interstate traffic are generally overstated. However, Wales' traffic-corrected estimates may be biased downward. As Smith[25] points out, the Wales method of decomposing the price and income variables to control for in-state and out-of-state consumption produces an identification problem (the independent variables are all correlated with the disturbance term) and yields biased estimates. In addition, Wales neglects interstate differences in sociodemographic variables, the price of substitutes, and illegal production, thereby misspecifying his demand equation.

Smith[25] examined the problem of illegal markets in demand estimation for distilled spirits. Smith identified three sources of illegality: (1) Federal excise tax evasion by underreporting of sales by legal firms and illegal production by unlicensed firms, (2) illegal importation from neighboring states, and (3) evasion of state taxes by underreporting of sales by distilled spirits wholesalers. Smith chose to examine the last source of illegality. His main purpose was to test for evidence of substitution between legal and illegal markets. After developing an elaborate theory to examine the effects of a tax on legal and illegal markets, Smith concludes, "increases in the tax rate reduce legal quantity demanded more than equal-sized increases in the net legal price." The reason for this phenomenon, he argues, is substitution of legal by illegal consumption as taxes rise in order to evade higher taxes.

To test this hypothesis, Smith estimates a demand equation which includes, as separate explanatory variables, the tax rate per gallon and the legal price net of taxes per gallon. The tax rates were taken from state legislation for license states, and an implicit tax was calculated for monopoly states based on state store profits. The legal price series introduced some measurement error problems since it consisted of manufacturers' suggested retail prices and not actual prices and only covered blended distilled spirits, thereby excluding 80% of spirits sales in the year analyzed. The other explanatory variables were per capita state income, the fraction of the population between the ages of 25 and 45, state enforcement budget per 1000 adults, and the lowest adjacent state market price. The latter variable was included to control for interstate consumption effects. The quantity demanded variable was measured as legal consumption in gallons per 1000 adults. All variables in dollar terms were deflated by a specially constructed state cost of living index. To control for price being simultaneously affected by quantity, a supply equation was estimated along with the demand equation. It was found that single-equation and simultaneous-equation estimates could be considered to be identical according to statistical tests, so Smith presents the single-equation estimates, since they offer greater precision. No attempt was made to control for substitute products.

The model was tested using cross-section observations for 45 states in 1970. Smith's results appear to confirm his hypothesis of a differential effect between tax price increases and nontax price increases. The tax rate coefficient is significantly different and higher than the net legal price variable. Both variables were significantly greater than zero. The estimates of price and income elasticities were -1.59 and 1.75, respectively, both significantly different from zero but not significantly different from 1. The border price coefficient was positive but insignificant. However, the tax rate variable may be picking up part of the effect of interstate consumption, since most of the price differential between neighboring states reflects differences in tax rates.[25] Moreover, Hause, in a comment on Smith's study, runs an additional regression which shows no statistical difference between legal price and tax rate but a significant and negative relationship between the difference in own-state tax

rate and adjacent-state tax rate and consumption. This suggests that illegal interstate traffic is the main evasion, and not intrastate underreporting of sales. Of the remaining variables, all are positive, but only the enforcement budget variable is statistically significant.

The Smith study is one of the most complete cross-section analyses, since it attempts to correct for certain illegal consumption effects and checks for simultaneous equation bias. Of greatest interest for our purposes is the possible finding of both price- and income-elastic effects on spirits consumption, a finding in opposition to many other studies of distilled spirits.

In his study, which professes to estimate price and income elasticities in distilled spirits for California, Lidman[4] regresses California spirits consumption per capita on the Bureau of Labor Statistics national price index for distilled spirits, the equivalent national index for packaged beer, state per capita disposable income, a time trend variable, and two state spirit tax variables, one a continuous variable of the deflated tax rate increases in 1955 and 1967. The years covered were 1953 to 1975. Each term in dollar units was deflated by the CPI. The tax variables were included because the national spirits price index does not reflect changes in California taxes.

The regressions were run in both linear and log-linear form with similar results. In the model with tax dummy variables, income, time, and the 1955 tax variable, all were significant and of the correct sign. The price elasticity of spirits was -0.07, insignificantly different from zero. The price of beer was positive and insignificant, implying substitutability with spirits. Beer was also shown to have a larger effect on spirits consumption than the price of spirits, a rather perverse result. The income elasticity was not significantly different from unity, at $+0.9$. In the second model, in which tax is a continuous variable, the spirit price variable was positive and insignificantly different from zero at 0.02, and the tax variable was negative, significantly greater than zero and significantly less than one at -0.113. This indicates that past tax increases have had slight effect on consumption. The beer, income, and time variables yielded similar results to the first model, with income elasticity significantly different from zero at 1.017. Lidman's finding of a positive and insignificant own-price elasticity suggests some serious problems in the data and/or method of estimation.

4.2.3. Wine. Separate studies of United States wine elasticities are rare. In a recent study, Labys[26] estimated elasticities for the United States wine industry and compared them to studies of wine elasticities in other countries. Labys estimated a simple demand model in which consumption per capita is a function of the price of wine and national income per capita. He also postulated consumption to be a function of substitute products, such as beer, but claimed that insufficient data prevented inclusion of a substitute product price index. Labys estimated two regressions for the United States, one with wine prices measured by the BLS wholesale price index for red wine and another using an imported wine price index. Presumably, the appropriate

Table VII. Wine Elasticity and Consumption Levels, 1965–1971[26]

County		Price elasticity	Income elasticity	Per capita consumption (liters)
France		−0.062	−0.148[a]	120.7
Italy		−1.003[a]	0.276[a]	110.7
Portugal		−0.678	0.054	87.0
Spain		−0.366[a]	0.143[a]	60.4
F.R. Germany		−0.379[a]	0.508[a]	13.7
United States	BLS price	−0.440	2.345[a]	3.7
	Import price	−1.654[a]	3.343[a]	

[a] Statistically significant at the 0.05 level.

consumption quantities were used in each regression, but this was never stated.

Labys' estimates of elasticities for 1954 to 1971 are shown in Table VII. The price elasticities are generally inelastic with the exception of Italy and United States imported wines. Those significantly less than 1 are for France, Portugal, Spain, Germany, and the United States domestic price regression. Labys reports the results of an earlier study by Farrell and Blaich[27] which estimates United States wine price elasticity as −0.6. The income elasticities are all inelastic with the exception of the United States which is highly income elastic. The high per capita consumption countries have low income elasticities, with France having a perverse negative relationship between income and consumption.

It is difficult to assess the validity of these estimates, but one suspects that they are of poor quality. First, the inconsistent results for the United States suggests a misspecification problem. Second, with the exception of one equation, no tests for serial correlation are given. Third, simultaneous equation problems are ignored. Fourth, some of the results are implausible. For example, it is hard to believe that consumption is unrelated to price or that demand is extremely price inelastic in France, whereas consumption is quite sensitive to price, and demand is unitary price elastic in Italy.

5. Evaluation of the Results

The foregoing is, to say the least, a bewildering set of results. Any reader expecting to find a single, true estimate of price and income elasticity by type of beverage should justifiably be feeling despair at this point. Elasticities are seen to differ widely by country, time period, estimation techniques, and numerous other factors. In this section, we attempt to bring some order to the results by establishing a reasonable range for price, income, cross-price, and advertising elasticities. In certain cases this cannot be done, but in others

Table VIII. Domestic Demand Elasticities of Beer

Author	Time period	Method of estimation	Price elasticity	Income elasticity
Niskanen[16]	1934–1954	Simultaneous equations (3-stage least-squares linear)	−0.50[a,b]	−0.33[a,b]
	1934–1954	Ordinary least-squares (linear)	−0.33[a,b]	−0.27[a,b]
Horowitz and Horowitz[20]	Cross-section by state for years 1949–1961	Simultaneous equation; indirect least-squares (linear)	Insignificantly different from zero	Not reported
Hogarty and Elzinga[21]	1956–1959 and by states	Ordinary least-squares (log)	−0.89[a]	0.43[a,b]
Norman[22]	1946–1970	Ordinary least-squares (log)	−0.87[a]	0.35[a,b]
Comanor and Wilson[18]	1947–1964	Ordinary least-squares	−0.56[a] (SR)[c] −1.39 (LR)[c]	−0.18 (SR) −0.46 (LR)

[a] Significantly different from zero at the 0.05 level.
[b] Significantly different from 1 at the 0.05 level.
[c] SR, short run; LR, long run.

a reasonable consensus can be formed. Tables summarizing the results of each study are presented. The price and income elasticities are presented by beverage type, divided into United States and foreign results (except for wine), with beer results in Tables VIII and IX, wine results in Table X, and distilled spirits results in Table XI and XII. The results for cross-price elasticities and advertising elasticities are in Tables XIII and XIV, respectively.

The study closes by examining the effect of price increases on consumption and tax revenues under alternative assumptions of price elasticity of demand.

Comparability across studies is, strictly speaking, unwarranted. The studies differ widely in time period of analysis, country of origin, quality of data used, and econometric technique. However, in order to attempt to establish a consensus on elasticities, comparison across studies is necessary. This, however, is a dangerous technique and should be viewed with a healthy dose of skepticism.

5.1. Price and Income Elasticities

Beer price elasticities in the United States range from an approximately zero estimate by Horowitz and Horowitz to Comanor and Wilson's long-run estimate of −1.39, as seen in Table VIII. Both studies have serious econometric problems and can be safely dismissed. The remaining studies range from −0.33 to −0.89, and all are statistically significant. Norman's estimate of

Table IX. Foreign Demand Elasticities of Beer

Author	Country and time period	Method of estimation	Price elasticity	Income elasticity
Stone[8,a]	U.K. 1920–1948	Ordinary least-squares (log)	−0.73	0.14
Stone[9]	U.K. 1920–1948	Ordinary least-squares (log)	−0.69[d,e]	Not reported
Prest[10]	U.K. 1870–1938	Ordinary least-squares (log)	−0.66[d,e]	0.23[d,e]
Nyberg[28,b]	Finland 1949–1962	Static model Dynamic model	−0.49 +0.003 (SR) +0.01 (LR)	0.22 0.19 (SR) 0.64 (LR)
Walsh and Walsh[12,c]	Ireland 1953–1967	Ordinary least-squares (log and linear)	−0.17	0.79[d]
Lau[3,d]	Canada 1949–1969	Ordinary least-squares (log and linear)	−0.03	0.20
Johnson and Oksanen[14]	Canada 1955–1971	Ordinary least-squares (log and linear)	−0.22[d,e] (SR) −0.38 (LR)	0.40 (SR) 0.06 (LR)
Johnson and Oksanen[15]	Canada 1955–1971	Ordinary least-squares (linear, with error components analysis)	−0.27[d,e] (SR) −0.33 (LR)	0.00[e] (SR) 0.02 (LR)

[a] Stone did not report rest statistics in his first study.
[b] As reported in Lau.[3] Test statistics were not reported.
[c] The price elasticity estimates in these studies are not comparable to the estimates reported in other studies, since the own-price variable is included twice (alone and in the substitute price variable) in the estimating equation.
[d] Significantly different from zero at the 0.05 level.
[e] Significantly different from 1 at the 0.05 level.

−0.87 is clearly biased upward, leaving only Hogarty and Elzinga at the top of the range. The results of Johnson and Oksanen for Canada, which should be close to United States estimates because of similar sociodemographic and cultural characteristics, show significantly inelastic estimates at the lower range of United States estimates. This suggests that beer price elasticity is around −0.3 for the United States and Canada. Results from other countries also suggest inelastic estimates, although not quite as inelastic. An inelastic demand conforms to *a priori* expectations. Wine and distilled spirits are likely poor substitutes for beer, lacking beer's product characteristics (flavor, alcohol content, lightness, and so on).

Income elasticities for beer are consistently quite low but not always statistically significant. The results of Niskanen and Comanor and Wilson suggest that beer is an inferior good, contrary to expectation. In the best study of the lot, Johnson and Oksanen find income to be insignificantly related to consumption. This is disturbing, for although income may have a small effect on consumption, it is certainly expected to be significant. Only Hogarty and

Table X. Demand Elasticities of Wine

Author	Country and period of analysis	Method of estimation	Price elasticity	Income elasticity
Niskanen[16]	U.S. 1934–1954	Simultaneous equation (linear)	−1.59[e]	1.45[e]
		Ordinary least-squares	−0.35	0.80[e]
Comanor and Wilson[18]	U.S. 1947–1964	Ordinary least-squares (log)	−0.68[e,f] (SR) −0.84 (LR)	0.41 (SR) 0.50 (LR)
Stone[9]	U.K. 1920–1938	Ordinary least-squares (imported wine, log)	−1.17[e]	0.98[e]
Nyberg[28,a]	Finland 1949–1962	Static model	−0.83	0.97
		Dynamic model	−0.99 (SR) −3.28 (LR)	0.39 (SR) 1.29 (LR)
Huitfeldt and Jorner[29,b]	Sweden 1963–1968	Fortified wine	−0.9	
		Light wine	−0.9	
Lau[3,c]	Canada 1949–1969	Ordinary least-squares (log and linear)	−1.65[e,f]	−1.43[e,f]
Johnson and Oksanen[14]	Canada 1955–1971	Ordinary least-squares (linear)	−0.50[e,f] (SR) −1.30 (LR)	−0.01[f] (SR) −0.02 (LR)
Johnson and Oksanen[15]	Canada 1955–1971	Ordinary least-squares (linear, with error component analysis)	−0.67[e,f] (SR) −1.78 (LR)	0.04[f] (SR) 0.04 (LR)
Labys[26]	France 1974–1971	Ordinary least-squares (log)	−0.06[f]	−0.15[e,f]
	Italy 1954–1971	Ordinary least-squares (log)	−1.00[e]	0.28[e,f]
	Portugal 1954–1971	Ordinary least-squares (log)	−0.68	0.05[f]
	Spain 1954–1971	Ordinary least-squares (log)	−0.37[e,f]	0.14[e,f]
	F. R. Germany 1954–1971	Ordinary least-squares (log)	−0.38[e,f]	0.51[e,f]
	U.S. (domestic price) 1954–1971	Ordinary least-squares (log)	−0.44	2.35[e,f]
	U.S. (import price) 1954–1971	Ordinary least-squares (log)	−1.65[e]	3.34[e,f]
Miller and Roberts (1972)[d]	Australia 1954–1971		−1.0 to −3.2	
C.C.E. (1972)[d]	Belgium 1954–1971		−1.14	1.81

[a] As reported in Lau[3]. Test statistics were not reported.
[b] As reported in Lidman.[4] Test statistics were not reported.
[c] The price elasticity is not comparable to other price elasticity estimates, since the own-price variable appears twice in the estimating equation.
[d] As reported in Labys.[26] Test statistics were not reported.
[e] Significantly different from zero at the 0.05 significance level.
[f] Significantly different from 1 at the 0.05 significance level.

Table XI. Domestic Demand Elasticities of Spirits

Author	Time period	Method of estimation	Price elasticity	Income elasticity
Niskanen[16]	1934–1954	Simultaneous equation (3-stage least-squares linear)	−2.027[a,b]	0.61[a,b]
		Ordinary least-squares (linear)	−0.93[a]	0.62[a]
Simon[23]	1955–1961	Calculation of arc elasticities[c]	−0.79 (median measure)	—
Wales[24]	1960 (42 states)	Ordinary least-squares (linear)	0.084	1.69[a]
Smith[25]	1970 (45 states)	Ordinary least-squares; seemingly unrelated regressions (linear)	−1.95[a]	1.75[a]
Lidman[4]	1953–1975 (California)	Ordinary least-squares (log and linear)	0.02[b]	1.017[a]
Comanor and Wilson[18]	1947–1964	Ordinary least-squares	−0.25 (SR) −0.30 (LR)	0.18 (SR) 0.21 (LR)

[a] Significantly different from zero at the 0.05 level.
[b] Significantly different from 1 at the 0.05 level.
[c] Simon used a special arc-elasticity formula based on the definition of price elasticity.

Table XII. Foreign Demand Elasticities of Spirits

Author	Country and time period	Method of estimation	Beverage	Price elasticity		Income elasticity	
Stone[8,a]	U.K. 1920–1938	Ordinary least-squares (log)		−0.72		0.54	
Stone[9]	U.K. 1920–1938	Ordinary least-squares (log)		−0.57[f,g]		0.60[f,g]	
Prest[10,b]	U.K. 1870–1938	Ordinary least-squares (log)		−0.57[f]		0.70[f]	
Malmquist[11]	Sweden 1923–1939	Ordinary least-squares (log)		−0.37		0.30	
Nyberg[28,c]	Finland 1949–1962	Static model	Vodka	−0.13		0.42	
			Other spirits	−0.95		1.30	
		Dynamic model	Vodka	−0.60	(SR)	0.25	(SR)
				−2.00	(LR)	0.84	(LR)
			Other spirits	−1.10	(SR)	0.49	(SR)
				−3.65	(LR)	1.62	(LR)

Huitfeld and Jorner[29d]	Sweden 1963–1968		Vodka Other spirits	−0.8 −3.2	
Walsh and Walsh[12e]	Ireland 1953–1967	Ordinary least-squares (log and linear)		−0.64	2.04[f]
Lau[3e]	Canada 1949–1969	Ordinary least-squares (log and linear)		−1.45[f,g]	0.68[f,g]
Johnson and Oksanen[14]	Canada 1955–1971	Ordinary least-squares (linear)		−0.91[f,g] (SR) −1.60 (LR)	0.23[f,g] (SR) 0.40 (LR)
Johnson and Oksanen[15]	Canada 1955–1971	Ordinary least-squares (linear, with variance component analysis)		−1.14[f,g] (SR) −1.77 (LR)	0.11[f,g] (SR) 0.17 (LR)

[a] Stone did not report test statistics in his 1945 study.
[b] Prest also estimated his demand equations in linear form. The price and income elasticities were −0.03 and 0.95, respectively.
[c] As reported in Lau.[3] Test statistics were not reported.
[d] As reported in Lidman.[4] Test statistics were not reported.
[e] The price elasticity estimates reported in these studies are not comparable to the estimates reported in other studies, since the own-price variable is included twice in the estimating equation.
[f] Significantly different from zero at the 0.05 level.
[g] Significantly different from 1 at the 0.05 level.

Table XIII. Estimates of Cross-Price Elasticities[a]

Study	Beer on wine	Wine on beer	Beer on spirits	Spirits on beer	Wine on spirits	Spirits on wine
Malmquist[11]					−0.64 to −1.01	
Walsh and Walsh[12b]			−0.21 to −0.33	0.19 to 0.22		
Lau[3b]		−0.73 to −0.86	1.27	1.00[c]	−0.31 to 0.19	
Johnson and Oksanen[14]	−0.03	0.23[c]	−0.11	−0.13	0.14	0.21[c]
Johnson and Oksanen[15] (signs and significance only)	—	+[c]	—[c]	—	+	+
Niskanen[16]	−0.39	2.55[c]	0.36	0.29	1.38[c]	−0.05
3-stage least-squares						
Ordinary least-squares	−0.44[c]	0.24	0.42	0.65[c]	−0.54	0.41
Lidman[4]				0.28		

[a] The cross elasticities are classified by dependent–independent variable. The first beverage listed is the dependent variable, and the second, the independent variable. For example, the results under "Beer on wine" are taken from regressions of beer on wine.
[b] These studies use relative price variables, e.g., beer price divided by spirit price, in log form. The ratios were not deflated by a consumer price index, and *t*-statistics could not be calculated.
[c] Significantly different from zero at 0.05 level.

Table XIV. Advertising Elasticity Estimates

Study	Product type	Estimate	Comment
Comanor and Wilson[18]	Malt beverages	0.004	Short run
		0.01	Long run
	Wine	0.97[a]	Short run
		1.20	Long run
	Distilled spirits	0.64[a]	Short run
		0.74	Long run
Norman[22]	Beer	−0.07, 0.06[a]	
Grabowski[19]	Alcoholic beverages	−0.012, −0.019	Ordinary least-squares
		−0.008	Two-stage least-squares

[a] Significantly different from zero at 0.05 level.

Elzinga and Norman come up with plausible results. Based on these results, we can make no judgment on the income elasticity for beer in the United States, although it is most likely less than 1.

United States estimates of wine price elasticities range from −0.44 by Labys to −1.59 by Niskanen. None of the results are robust. Labys' result is insignificant, and Niskanen had little faith in his wine demand estimation results. Comanor and Wilson's results are, for the reasons given earlier, highly suspect. Studies of wine consumption in foreign countries offer little additional insight. The short-run and static elasticities from Stone,[9] Nyberg,[28] and Huitfeldt-Jorner[29] suggest a price elasticity of around −0.6. Clearly, the results show an extremely wide variation across all countries. This may be a quite accurate reflection of true conditions, for the role of wine in France and Italy is certainly quite different than in the United States and Canada. We suspect, however, that much of the differences in results within the United States studies and across countries are caused by econometric problems and poor data. As a consequence, we offer no conclusions on price elasticity of wine. For public policy purposes, we will simply assume a unitary price elasticity.

Income elasticity with respect to wine in the United States presents the same diversity in results. Niskanen finds a significant and essentially unitary elasticity; Comanor and Wilson find income unrelated to wine consumption; and Labys finds highly significant and elastic results. With regard to foreign studies, Johnson and Oksanen's results support Comanor and Wilson's implausible results; Labys' finds generally significant and inelastic results; whereas Stone and Nyberg find unitary elastic results. Again, these results may reflect true differences across countries, but we suspect econometric problems to be

of comparable influence. The results are, again, too divergent to draw any conclusions.

United States price elasticities for distilled spirits range from 0.02 by Lidman to −2.027 by Niskanen. None of the low-elasticity studies, those of Lidman, Wales, and Comanor and Wilson, were convincing econometrically, and their price elasticity estimates were all statistically insignificant, an implausible finding. The studies of Smith and Niskanen were the most complete econometrically, and their findings suggest unitary or price-elastic demand. Offering evidence in support of price-elastic demand is Johnson and Oksanen's 1977 study[15] showing both short- and long-run price-elastic estimates. The sociodemographic differences between the United States and Canada are probably close enough to consider Johnson and Oksanen's results valid for the United States. These results suggest an elasticity of around −1.5 for the United States. This is inconsistent with our *a priori* expectation of inelastic demand for spirits based on the extremely high rate of taxation. One possible explanation for this result is that the demand curve for distilled spirits is essentially a straight line, and taxes have pushed prices into the elastic region of the demand curve. Given little variation in price over time, the inelastic region of the demand curve is never seen. This explanation is weakened by the fact that the same argument should apply to beer and wine, but we see little evidence of price elasticity in these beverages.

Studies of distilled spirits in European countries suggest price-inelastic demand in these countries. This conforms to *a priori* expectations. Why demand would be price elastic in the United States and Canada and inelastic in other countries is unknown.

Income elasticities in distilled spirits show no consistent results across studies. Within the United States, significant income elasticities range from inelastic at 0.61 in Niskanen's study to 1.75 and not significantly different from 1 in Smith's study. Johnson and Oksanen find contrasting results, with highly income-inelastic results in both the short and long run. Their 1977 study indicates that income has slight effect on consumption, a rather implausible result for distilled spirits. In short, there is no consensus possible with regard to income elasticity for distilled spirits.

5.2. Cross-Price Elasticities

Cross-price elasticities measure the extent of substitutability or complementarity between products and are of crucial importance for tax policy purposes. If, for example, wine and spirits are substitutable, then tax changes with the intent of reducing consumption or raising revenue must be levied proportionately on each beverage. If not, some consumers will shift to the cheaper substitute beverage, defeating the goals of a tax increase. Unfortunately, there are few good estimates of cross-price elasticities in alcoholic beverages, and those that are available give ambiguous results.

The cross-price elasticities in the studies reviewed are listed in Table XIII.

The Johnson and Oksanen and Niskanen studies are the most thorough econometrically. They are of different time periods and different countries and, therefore, not strictly comparable. However, being the best available, there is little alternative but to compare their results. The major findings in the results are inconsistent across regressions within each study and across both studies. For example, Johnson and Oksanen found beer to be significant as a substitute in the wine equation but wine not to be significant in the beer equation. Niskanen found a similar result for wine and beer and an analogous inconsistency between wine and spirits. In addition, the signs of the elasticities are not consistent between the two studies. In one set of results, Johnson and Oksanen find beer and spirits to be complements, whereas Niskanen's results suggest that they are substitutes. Moreover, the signs of many of the elasticities over all the studies indicate alcoholic beverages to be complements, a rather implausible result for most alcoholic beverages. It is, of course, imprudent to compare results across countries, but given the paucity of results, we risk it. Looking down the results in each column, the most favorable results for substitutability are in regressions of wine consumption on beer price. However, these results are not confirmed by regressions of beer consumption on wine price. The other columns yield mixed results. In general, there is no consistency in findings across studies. These results suggest that there is little substitutability among beer, wine, and distilled spirits.

We can justifiably be suspicious of these results, not because substitutability appears to be absent but because finding substitutability, when it exists, by regression analysis may be very difficult. One problem is simply observing variations in substitutes. The market does not perform the economic experiment that is needed; that is, firms typically do not vary price to see its effects on sales. Moreover, if markets are efficient, we will see even less variation in price and quantity across highly substitutable goods because substitution takes place rapidly at the margin. This influences the competitive price in such a manner that large price and quantity changes are rarely seen. Only a strong shock to an equilibrium between demand and supply will result in a move toward substitution. For example, when fair trade laws were held illegal in California, it only applied initially to spirits and beer. If this condition had remained, then the experimental grounds to test for substitution would have been, in theory, available.

There is also a potentially serious statistical problem of aggregation in prices over an entire industry. If the covariance of prices within industry are negative, aggregation will tend to destroy relative price effects. The reasoning is analogous to that of a virtually riskless portfolio of assets. The portfolio can be riskless even with large changes in individual assets as long as the assets are strongly negatively covariant. To extend the example to prices, relative price differences may explain why one firm's sales decline but may offer no explanation for changes in total industry sales. This aggregation problem can annihilate own-price elasticities in time-series analysis as well as cross-price elasticities. Hence, aggregation problems may have contributed

to the lack of significance of both own-price and cross-price elasticities found in many studies, if prices were negatively covariant.

5.3. Advertising Elasticities

We hypothesized that advertising's main effect on sales was at the intraindustry level and, thus, that total industry sales would be unrelated to advertising. Only three studies included advertising or advertising intensity in their demand equations, and the results are listed in Table XIV. Comanor and Wilson found significant and positive advertising elasticities for wine and distilled spirits but not for malt beverages. In fact, advertising had an approximately zero elasticity for malt beverages. This inconsistency across beverage types was not explained by Comanor and Wilson. It hardly seems possible that advertising would have a pronounced positive relationship with sales in wine and distilled spirits but not in malt beverages. The economic problems in the Comanor and Wilson study are serious enough, as pointed out by Grabowski, to make one highly skeptical of these results. Grabowski found no significant relationship between total alcohol beverage sales and advertising, inconsistent with Comanor and Wilson's findings. Finally, Norman ran two regressions with advertising variables: one found it to be significant, and the other found it insignificant. The main difference was one of sample size. Advertising was significant when run over the period 1946 to 1970, and it was insignificant when run over the period 1953 to 1970. Our attempt to replicate Norman's results found advertising insignificant in both periods. The difference stems from Norman's making errors in his raw data input, the correction of which drastically changes his results.

On the basis of such limited results, strong conclusions are not warranted. However, on the basis of the results to date, advertising elasticity seems insignificant. Hence, such commonly heard proposals as limiting advertising expenditures would have little effect on total alcohol beverage sales, although it would have strong effects elsewhere, if these results are correct.

5.4. Some Tax Policy Implications

The taxation of consumer goods presents a number of issues such as the equity of the tax, consistency of taxation with optimal resource allocation, tax effects on illegal activities, and the uses of tax revenues. Of most direct relevance in terms of this study is the effect of tax changes on consumption, personal expenditures, and government tax revenue. As outlined above, price elasticity of demand for a good indicates the effect of price changes on the quantity demanded and total expenditures. For price-inelastic goods, price increases have less than proportionate decreases in consumption and lead to increases in total expenditures. For price-elastic goods, price increases result in more-than-proportionate decreases in consumption and lead to decreases

in total expenditures. These relationships, plus the effects of price increases on tax revenues, can now be applied to the demand for alcoholic beverages.

Our summary estimates of own-price elasticities for beer, wine, and distilled spirits were -0.3, -1.0, and -1.5, respectively. These are crude at best, particularly for wine, but seem the best available. Many would argue with these estimates on *a priori* grounds, but we will simply take the numbers as given. The effect of a price increase through an increase in tax rates, assuming the full burden of the tax increase falls on consumers, can now be assessed. A 1% tax-induced price increase, assuming all other factors remain constant, would reduce consumption in beer by 0.3%, in wine by 1.0%, and in distilled spirits by 1.5%. Thus, control of consumption by taxation would have the strongest effect on distilled spirits. Since spirits have the highest alcohol content, this control effect has added impact for total alcohol consumption. Changes in total consumer expenditures on each beverage would be an increase in expenditures on beer, no change in expenditures on wine, and a decline in expenditures on distilled spirits. For those concerned with ethical judgments as to how consumers spend their disposable income, these expenditure changes are of importance. For example, if beer drinkers are predominately low-income people, and a tax-induced price increase occurs, all other factors remaining constant, this would lead to increased expenditures on beer. Some may object to low-income groups spending a larger proportion of their income on beer.

The effect of tax rate increases on tax revenue requires the derivation of a tax revenue elasticity equation. Geary[30] derived an equation to calculate tax revenue elasticity as $e_t = 1 + e_p\, t/(P + t)$, where e_t is tax revenue elasticity, e_p is own-price elasticity, t is the unit tax rate, and P is price net of taxes. The tax revenue elasticity depends on own-price elasticity and the percentage of price represented by unit taxes. Since e_p is always negative, a tax increase may increase or decrease tax revenue, depending on the magnitude of $e_p t/(P + t)$. Tax elasticity will always be positive if $e_p t/(P + t)$ is less than 1. By inspection, if taxes are a small percentage of final price, tax revenue elasticity will be positive unless own-price elasticity is extremely large, well beyond the levels found in this study.

It is possible to calculate tax revenue elasticities for beer, wine, and distilled spirits in California using California excise tax rates and some crude estimates of prices in California. These estimates by beverage under alternative own-price elasticities are shown in Table XV. The derivation of the price of each beverage is explained in the footnotes. Since state excise taxes are a small percentage of final price, the tax revenue elasticities, as expected, are positive in each case. Moreover, they are all seen to be close to 1 or of unitary elasticity. The interpretation of these results is that a 1% increase in state excise tax will, to use beer as an example, raise state tax revenue by 0.985%, assuming an own-price elasticity of -0.5. That is, for a 1% increase in tax rates, tax revenues will increase by close to 1% in the case of each beverage, assuming everything else remains constant.

Table XV. Estimates of California Tax Revenue Elasticities

Beverage and excise tax rate	Own-price elasticities					
	−0.25	−0.50	−0.75	−1.00	−1.50	−2.00
Beer[a] ($1.24/barrel)	0.992	0.985	0.977	0.969	0.954	0.931
Wine[b] ($0.01/gallon)	0.999	0.999	0.999	0.999	0.998	0.998
Distilled spirits[c] ($2.00/gallon)	0.985	0.970	0.955	0.940	0.910	0.880

[a] A composite price of beer was estimated by dividing total sales in fiscal year 1974–1975, the most recent year data were available, by total tax-paid withdrawals. Sales data came from IRS corporate tax returns for the malt beverage industry, and total withdrawals from the *Brewers Almanac*, 1977. The price is an average over all beers at the manufacturing level and is assumed valid for California.

[b] A composite price for table wine only was estimated by weighting prices of wine in various categories—less than $1.25, $1.26 to $2.00, $2.01 to $3.50, $3.51 to $5.00, and $5.01 and over—by the percent of sales in each price category as reported in *The Wine Marketing Handbook*, 1978. The price used was $8.48/gallon for 1977. This is an average price estimate for the United States as a whole. Since California taxes on table wine are the lowest in the nation, this may overstate California's average wine price.

[c] A composite price for distilled spirits was estimated by taking the retail price of nine leading brands in California in 1977 and weighting them by the percentage of consumption in each product category as reported in *The Liquor Handbook*, 1978. The price used was $33.21/gallon.

There is clearly room for further work on demand estimation of beer, wine, and distilled spirits. First, there is room for improvement in econometric technique. Second, the quality of the data used is poor in many cases, especially with regard to price indices. Third, of the three beverages, far less is known about the demand for wine. There is a need for additional studies on wine consumption. Fourth, none of the United States' studies have examined the parameters of demand within incidence of drinking groups. There is a need to develop data by drinking groups, for the real issue is what effect changes in the determinants of demand will have on occasional, light, moderate, and heavy drinkers. We hope that this review of the literature will provide some guidance for the direction of subsequent research.

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13

Youth, Alcohol, and Traffic Accidents

Current Status

Richard L. Douglass

Abstract. The interaction of youth, alcohol consumption and driving has been the leading contributor to morbidity and mortality in the United States among persons under 40 for over thirty years. While a considerable literature has evolved, only since 1968 has the literature acquired a truly scientific character; frequently the literature is not widely distributed and found only in technical reports. This contribution reviews the state of knowledge of youthful, alcohol-related traffic accidents based on both the open literature and the research which is found in technical documents. The consequences of youthful alcohol consumption and theories regarding the etiology of traffic accidents which result from drinking by youth, a review of attempts to reduce the magnitude of the problem, and recommendations for research are presented.

1. Introduction

The relationship between alcohol and traffic accidents has been recognized for over a half century as a major source of injury and death in the United States.[1,2] Public attention to this relationship, however, has been inconsistent, despite overwhelming evidence that has been accumulated since the earliest acknowledgment that drinking drivers are at an elevated risk of traffic crash involvement. Youth have been a dominant factor in the larger problem of alcohol-related traffic morbidity and mortality since the 1940s. The progressively increasing availability of automobiles for the recreational use of young people, combined with the consumption of alcohol, has taken on a unique and irrefutable role in youthful society—a role that has become an equal for some, in many respects, to the roles of school, peer group, family, and neighborhood in the expression of youthful independence and social development.[3] And crashes have produced annually growing numbers of fatalities and injuries, many of which have led to permanent disabilities.[3,4] Currently, traffic accidents are responsible for more fatalities among youth than any other cause of death.[4–6] Moreover, as large as the annual number of deaths caused by young drinking drivers is, an even larger number of permanent and disabling injuries result from young drinking drivers.[7]

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In 1978, drivers under 20 were involved in 11,500 crashes with at least one fatality. There were 5,600,000 reported traffic accidents caused by young drivers 15–20 years old.[8] Additionally, an unknown number of youths were seriously injured in these traffic accidents, also caused by drinking drivers. No other single source of mortality approaches traffic accidents among young in terms of sheer numbers, and no other cause of death is as predictably associated as traffic accidents with a single known contributing factor—the interaction of beverage alcohol and a young driver's ability to control an automobile.[9]

Many public and private efforts have been initiated to reduce the number of alcohol-related traffic accidents. These efforts have included educational programs, court and police intervention, and public policy changes.[10] The uniqueness of young drivers, relative to the larger adult population, has been infrequently recognized in the conceptualization of these programs.[4,9] Similarly, the characteristics of youthful drinking practices and the underlying reasons why youth drink and drive are rarely incorporated into programs intended to reduce alcohol-related crashes. Rather, programs based on adult models of behavior, which have little probability of success with youth, are more common.[4,9,10] One reason for the lack of youth-designed programs is a state of knowledge that is largely descriptive. The precursors to youthful drinking or driving are poorly understood in ways that permit meaningful interventions for this specific population.

2. Research into the Causes

The most significant new knowledge on the relationship of alcohol to traffic safety among youth has been the result of applied research conducted since the *1968 Alcohol and Highway Safety Report* from the Secretary of Transportation to the Congress.[11] This report summarized the existing state of knowledge and gave impetus to Federal initiatives during the 1970s.

Every major study in the area identifies youth as being overrepresented in alcohol-related traffic crashes. This finding is entirely consistent despite many variations of measurements and research approaches. Studies of fatalities[5,12–16] are consistent in the finding that between 45 and 60% of all fatal crashes with a young driver are alcohol related. The probability of alcohol involvement increases with the severity of the crash, ranging from property damage only to at last one consequent death. Reviews of this association are also consistent for all levels of crash severity; for all traffic crashes, young drivers are considerably more likely to have been drinking than older drivers. Also, young drivers have more traffic accidents with and without alcohol involvement than older drivers.[17–20] Part of the explanation for the exceptional numbers of young drinking drivers involved in traffic crashes is related to their exposure on the road. Yet, among the youngest drivers, between 16 and 20, those who drink are still more likely to be crash involved after data

are controlled for exposure.[21,22] Young people between 16 and 24 are involved in more fatal, injury-producing, and property damage crashes and more crashes in which alcohol was involved, in a higher proportion than even their high exposure would suggest, than older drivers. The epidemiologic research of the past decade has provided some understanding of the causes and circumstances of the problem.

Young people who have an elevated risk of crash involvement, on the average, have predictable drinking patterns. Smart *et al.*[23] reported that high school students who frequently get drunk and who are heavy drinkers drink away from home and in cars. Other studies confirm the direction of these conclusions. Waller and Warden[24] reported that over half of the males in their study in their teenage years or in their 20s were at increased risk of an alcohol-related crash involvement based on their drinking habits, whereas only 24% of men over 30 were so classified. These authors also reported that very heavy drinking (five or more drinks in 2 hr or less) among high-risk drivers was more than twice as probable with younger male drivers than with men over 30, a difference in drinking pattern that was echoed by Harford and Mills.[25]

The automobile and alcohol consumption are closely associated in the society of the young. A national study of adolescent drinking behavior notes that 40% of all respondents reported occasional drinking while driving or sitting in a parked car at night.[26] Wolfe and Chapman[27] reported that 36% of high school males and 9% of high school females in Washtenaw County, Michigan, had driven after drinking two or more drinks in the 3 months prior to the survey. A 1975 national survey of high school youth found that 32% acknowledged "riding in cars with 'heavily intoxicated drivers' at least once a month." These same students had little understanding of the hazards of driving while intoxicated.[28]

A study of Florida college students in 1976 found that a "significant percentage" of the students reported that they had driven a car either after or while drinking within the previous 2 months.[29] Finally, another study of youth in southeastern Michigan found that by the age of 20, more than half the sampled men drank at least once weekly and drove at least once monthly after drinking.[30]

It has been widely recognized that young drivers become involved in traffic accidents after drinking smaller amounts of alcohol than do older drivers. Even in the Secretary of Transportation's 1968 Report to Congress, it was noted that although an appreciable percentage of teenagers who crash fatally after drinking are very intoxicated, fatally injured young drivers who have been drinking tend, as a group, to have lower blood alcohol concentrations than older drivers who crash similarly.[11] Frequently tested in subsequent research, this conclusion has withstood repeated analyses.

Waller [31] indicated that teenagers were more likely than older drivers to have caused a crash and to have done so at lower blood alcohol concentrations than adults. Zylman[32,33] reported that drivers under 18 have "greatly increased vulnerability" to crash involvement after only one or two drinks. In a study

of nighttime drivers and their alcohol usage, Carlson[21] reported that most of the drivers studied aged 16 to 20 who had been drinking had lower blood alcohol concentrations than older drivers. In Carlson's study, driving at night was independently associated with increased crash involvement. Further, he stated that "the combination of inexperience in drinking and in driving is highly related to crash occurrence." This conclusion was interpreted to include both fatal and nonfatal crashes. Comparable conclusions have been reported by Baker,[7] Byrd,[34] Smart,[35] McIntire,[36] and Shults and Layne[37] for crash involvement and also for drinking and driving arrests.

Another characteristic of alcohol-related traffic accidents is that males are considerably more often involved than females. Typically, research has indicated that alcohol-involved crashes tend to involve a single vehicle and occur at night.[5,11,20,38,39] Douglass *et al.*[40,41] found, however, that between 10:00 p.m. and midnight, female 18- to 20-year-old crash-involved drivers were as likely to have been drinking as males. After midnight, however, young crash-involved males are more likely to have been the driver. In part, this may be because fewer females than males are driving after midnight, and if females are on the road, they are more likely to be passengers than drivers. Associated with this, Waller[31] found that only 25% of the passengers of a drinking driver in a fatal crash were "better risks" with respect to drinking than the driver himself. Many of these studies have also indicated that weekends produce more alcohol-related crashes with young drivers than weekdays, and holidays produce up to twice the number generated during average weekends. The extent to which weekend–weekday, holiday–nonholiday comparisons are explained only as a consequence of increased recreational driving and drinking has not been completely assessed.[42,43] Waller [42] contended that exposure is an inadequate explanation for the overinvolvement of youth in traffic accidents and suggested that the kind of exposure was likely to be as important as the quantity of exposure, a suggestion that has not been thoroughly researched.

A characteristic closely associated with all of the correlates of alcohol and traffic accidents noted thus far is driving at excessive speed or at speeds too great for the road conditions.[14,16,43] This association has been specified repeatedly for youth in studies of arrested drinking drivers and crash-involved drivers. In addition, increased speed in conjunction with drinking is more likely to be associated with younger drivers and with increased crash severity.[19,39,44,45]

A few studies have determined that beer is more likely than wine or liquor to be the alcoholic beverage of choice by youth and, more specifically, by youth involved in traffic crashes. Rooney and Schwartz[46] found that over 65% of a survey of high school seniors in five states reported that their five best friends use beer, and of the respondents themselves, 46% consumed beer weekly. Among drinking drivers, Damkot *et al.*[47] found that younger males considered beer less likely to cause an alcohol-related crash or to be intoxicating than liquor. Douglass and Freedman[48] found that draft beer consumption was indicated, rather than packaged beer, liquor, or wine, to be

associated with an increase in alcohol-involved crashes in Michigan when the age of purchase was lowered in 1972 from 21 to 18.

Many investigations have sought to explain the role of social status, personality, and related factors involved with alcohol-related crashes among youth. Sobel and Underhill[49] and Moses and Burger[50] lend support to the hypothesis that youth, particularly teenage males, reacting to unstable marriages of their parents or to poor relationships with either parent are associated with increased risk of abusive drinking and driving after drinking. A few studies have specifically investigated the social and psychological characteristics of young drivers and associated these factors with drinking and crash involvement. Pelz and Schuman[51] and Schuman *et al.*[52] have reported that alcohol use in traffic accidents and arrests for driving while intoxicated interact with a sense of alienation and feelings of hostility.

Schultz[53] was among the earliest to report that the frequency of young drivers' involvements in alcohol-related crashes was not independent of peer pressures to drink and drive. Kraus *et al.*[54] similarly were among the first to suggest that poor academic performance, early full-time employment outside of school (at or before age 17), and juvenile offense convictions are associated with increased risks of alcohol-related crash involvement. The exact roles of such factors, however, remain to be determined, in that more recent studies have found that adolescents who are likely to drink frequently and to subsequently drive are not necessarily deviant regarding academic performance, school sports involvement, social behavior, liberalism, or impulsivity.[28] Young drivers involved in alcohol-related crashes beyond high school age, however, are more likely on the average to have had prior traffic violation convictions, previous traffic accidents, marital problems; to be unemployed; and to have discontinued formal education beyond high school.[20,55] Young drivers killed in alcohol-related crashes are more likely than others to have lower educational and socioeconomic levels than their cohorts.[56]

To summarize the current state of knowledge of alcohol-related traffic accidents among youth, it appears that for certain youth and at certain times in a young person's life and during or subsequent to certain life events, the likelihood of an alcohol-related accident is greater than would be expected. Thus, the problem of alcohol-related traffic accidents among youth is not entirely a random process but increasingly appears to be predictable.

3. Attempts to Help

Although efforts to reduce the incidence of alcohol-related traffic casualties have been numerous, the majority of these programs have been designed for adults (persons over 21 years of age). These programs have predominantly included mass media, public information projects, special police patrols and law enforcement activities, and court-related identification of problem drinkers and referral to special alcohol schools and treatment or

rehabilitation. Youth-directed efforts have been largely vested in the public schools as part of ongoing driver education or health education classes. Less frequent efforts include public information, law enforcement, and legal measures including the restriction of alcohol availability through minimum legal drinking ages and other related laws.

The largest organized effort during the last decade has been the Department of Transportation's Alcohol Safety Action Projects, some of which had well-developed youth-oriented activities. Although the principal target populations of these projects included drivers over 21, some of the projects included activities aimed at younger drivers. The most frequent type of activity was youth education, and the most frequent outcome was increased understanding of the risks of drinking and driving for young drivers rather than a reduction in alcohol-related casualties.[57] The Alcohol Safety Action Projects were aimed principally at drivers over 21 because this thrust was indicated by the 1968 report on alcohol and highway safety.[11] Throughout the projects, numerous screening tests were used for diagnosis and entrance to court-referred treatment or rehabilitation programs. These tests were basically appropriate for adult problem-drinking drivers.[58] The appropriateness of adult-based diagnostic criteria for youth, however, has been frequently challenged, because most of the itmes in such tests are irrelevant for youth with little experience independent of the family and parental supervision.

Educational programs for youth have been largely public information or public school curricula. Public information efforts have relied on the belief that adolescents depend on television, radio, and parents (in that order) for information about alcohol; peer sources of information, however, are known to become increasingly important as youth progress into high school.[59] Most education programs, either through mass media or the public schools, are implicitly intended to prevent youth from drinking and driving or developing habitual driving after drinking.[60] Infrequently, these programs have had other objectives, such as reinforcing the intervention of a passenger or friend to prevent a drinking youth from driving. Unfortunately, the explicit assumptions and objectives are generally unspecified.[61,62] A recent finding that youth are more concerned about "injuring, crippling, or killing someone else" than other consequences of driving while intoxicated (including killing themselves) holds the potential for developing more sophisticated objectives for public education campaigns than have been developed to date.[63] Public school and public information programs tend to be based on the assumption that knowledge of the possible consequences of drinking and driving will promote nonuse, an assumption that has been challenged by Brotman and Suffet.[64]

One issue that has been addressed in both public information and public school programs is the effectiveness of recovered alcoholics, or youth offenders, as communicators in alcohol education. Clark and Porter[65] reported that the recovered alcoholic is ineffective for youthful audiences. Similar conclusions have been found for other communicators such as entertainment celebrities, race car drivers, and others.[66,67]

Most public information campaigns have been able to increase awareness of drinking and driving situations.[68–71] The potential success of efforts to encourage passengers, friends, and others to prevent a drinker from driving has been insufficiently tested. Although the advisability of this approach in all situations may be questionable because such third party "others" are frequently intoxicated themselves,[31] the potential appropriateness of this approach has been repeatedly suggested.[28,52,72]

The majority of traffic safety education efforts directed at youth have been driver education programs in the public schools. Many curricula developed in recent years have emphasized a variety of conceptual areas such as the responsible use of alcohol, health consequences of drinking and driving, legal consequences of driving while intoxicated, alcohol effects (behaviorally and physiologically), and other less area-specific topics such as "alcohol and food" or alcohol in history. These programs have been directed largely at high school youth[73–76] Some efforts have emerged also in the junior high school, predriving years. The appropriateness of using the driver education or health education classroom as the context for education on drinking and driving has been undisputed by most public educators. The difficulty with these programs, however, is that it is not clear that driver education necessarily reduces any youth's probabilities of crash involvement, with or without alcohol. This belief is based on the hypothesis that such training increases driving exposure and brings more youth into the driving population at earlier ages than would occur without such training.[56,77]

Next to public school programs and public information campaigns, the prevention activities youth are most likely to encounter are those based in the area of law enforcement and court-related treatment, rehabilitation, and education. As indicated above, the majority of these programs have been developed within the Alcohol Safety Action Project system under either Federal or local support, and only a few have made major efforts specifically geared to youth. The direct consequence of an arrest for drinking and driving can include fines, jail sentences (for older youth), license suspension, compulsory treatment, or enrollment in a court-related or court-approved "alcohol school." In addition, insurance premiums frequently increase if youth have arrest records that follow them thereafter, and many families experience a combination of social and financial costs.[4]

The Alcohol Safety Action Projects have had a variety of effects as indicated by their project evaluations. Driving records after attendance in a court-referred school have shown some improvement regarding arrests for drinking and driving as well as for other violations. Short-term, temporary reductions in injury and fatal crash rates have been identified in places with an Alcohol Safety Action Project, and driving-after-drinking exposure has been reduced.[78–82] The experience of these projects, however, suggests that youthful drivers are underrepresented as program participants because older drivers are more likely to be arrested and convicted for drinking and driving than youth. According to the Department of Transportation, the younger

drinking drivers are more likely to be driving while intoxicated, yet less likely to be apprehended than older drivers. If police patrols are missing the younger, highest-risk drinking drivers in enforcement activities, it follows that this youthful target group would be underrepresented in court-referred programs.[57] In addition, Kern *et al.*[83] reported that younger convicted drunken or alcohol-impaired drivers were most likely to drop out of an alcohol education program after they were enrolled, suggesting again that those who needed such programs most were the most likely to drop out. On the other hand, Rosenberg *et al.*[84] reported that treatment outcomes are most successful with clients with less extensive involvement with alcohol, including youth. Thus, it appears that youth are highly appropriate for such activities, and their success is expected to be high if they can be retained for the entire program.

One program, in Utah, involved teenage offenders and their parents in a juvenile court alcohol school program. This approach resulted in increased knowledge among youthful offenders and their parents about alcohol and a decrease in short-term recidivism (rearrests) for drinking and driving. Attitudes regarding alcohol among others in the family were unaffected by the program.[85,86]

4. The Minimum Legal Drinking Age

Public health has relied to a major extent on the use of laws and regulations to effect decreases in morbidity and mortality; however, the use of these mechanisms in the area of alcohol problems, and specifically such problems among youth, has been rare. The control of beverage alcohol, viewed as the agent, in a public health model of prevention, is a predictable public health response to a problem like alcohol-related traffic accidents; yet, unlike most other areas of public health, there has been a long history of resistance to the control of alcohol.[87–89]

An opportunity to test the relationship of alcohol availability to alcohol-related traffic casualties emerged during the last decade; it may force a closer look at alcohol control as a means of prevention, at least for acute problems such as traffic accident morbidity and mortality. In the early 1970s, popular support of lowering of the legal age limits for voting rights in most states and in Congress led to an amendment to the Constitution in 1971 that gave persons aged 18 the right to vote in Federal elections. Within 4 years, more than 30 states and Canadian provinces lowered the local and state voting age to 18, and most also lowered the legal age of purchase of alcoholic beverages, usually from 21 to 18 or 19.[40,41,90–95]

Although the alcohol abuse and traffic safety fields at that time were less developed than they are today, a number of researchers and practitioners were included in the process of lowering the legal drinking ages and attempted to predict the consequences of liberalized alcohol availability for youth. With-

out the research experience of the last decade, however, the fundamental information on which such predictions could be made was unavailable, and the opinions of scientists and scholars were frequently based on little empirical research. Zylman[33] suggested numerous potentially spurious phenomena that would occur that might lead to an unwarranted belief that accidents among youth might increase. Zylman predicted that a more likely result would be increased police enforcement and reporting of youth, giving the appearance of an increase in traffic crashes in the absence of any actual increase.

During the period from 1973 through 1978, a considerable amount of research was generated throughout the United States and Canada dealing with the actual consequences of increased alcohol availability on the frequency of traffic accidents among youth, associated consumption, and alcohol-related problems such as arrests, school problems, and alcoholism treatment. Also, a heated and ongoing political and philosophical debate emerged which often clouded society's attention to the scientific work in progress. The studies of the lower legal drinking ages have provided a major addition to the understanding of increases in alcohol availability and traffic safety which have led to many states' decisions to raise the legal drinking ages back to 19, 20, or 21.

The first reports of the events following the reduction of legal drinking ages were based on little data and generally unscientific interpretations. Although these reports were open to scientific challenge, they did raise the critical issues to be addressed in the more adequate studies that followed. Zylman[96–98] suggested that poorly controlled police reporting practices, increases in the population of young drivers, an overemphasis in the role of drinking in crash causation, and long-term cycles in fatal accident incidence were more likely to explain apparent increases in reported alcohol-related traffic crashes, particularly fatal ones, than the lower drinking ages. His position was that most youth are not involved in excessive drinking or resultant traffic crashes, and police actions were an overreaction to anticipated problems related to lower drinking ages. As Zylman anticipated, the earliest reports of increases in alcohol-related traffic accidents among youth in Michigan were based on official police reports and suggested large increases, over 100%, which added to the political debate and the call for scientific investigation.[99]

The only federally sponsored study to determine if the lowered legal ages increased alcohol-related traffic crashes was conducted by Douglass and associates in Michigan.[40,100–103] This study utilized time-series statistical analyses for young and old drivers involved in traffic accidents during the period between 1968 and 1973. The legal drinking ages in Michigan, Maine, and Vermont were tested. All three of these states lowered the drinking age to 18 for all alcoholic beverages. Control analyses of the same measurements were conducted in New York and Louisiana with long-established 18-year-old drinking ages and in Pennsylvania and Texas where the minimum drinking ages (at that time) remained 21. This study acknowledged the issues suggested by Zylman and others by controlling for potentially biased police

reporting with a conservative and unbiased "surrogate" measure of alcohol involvement in addition to officially reported statistics. Cycles and population growth were also controlled through statistical procedures and the use of age-specific rates in addition to simple frequency data.

Zylman's conclusions that no increase in alcohol-related crash frequencies resulted from the reduced legal purchasing age were challenged, however, and increases were found in Michigan and Maine of "statistical and social significance."[40,102] Minimum net increases of between 10 and 26% for alcohol-related crashes among young drivers directly affected by the lower drinking ages were found in Michigan and Maine and were attributed to the legal changes; an increase of less than 2% was found in Vermont, however, which led these investigators to conclude that all places could not be expected to have the same response to a change in the legal drinking age. Clark[104] found support for the Douglass *et al.* findings with analyses revealing a large increase in drinking and driving arrests among 18- to 20-year-olds in Washtenaw County, Michigan, subsequent to the 18-year-old drinking age. Wolfe and Chapman,[27] in a survey of high school students, also in Washtenaw County, reported that alcohol consumption quantity and frequency increased between 1970 and 1972. Clark *et al.*,[105] in comparative roadside surveys of drivers on the road at night, found a substantial increase in alcohol consumption among drivers in the age group affected by the 18-year-old drinking age.

These studies in Michigan were soon joined by others that produced similar conclusions. Williams *et al.*[106] analyzed data from Michigan and Ontario where the drinking age for all alcoholic beverages was reduced from 21 to 18 and Wisconsin where wine and liquor were reduced to 18 (beer had been legal at 18 prior to the 1972 legislation). These places were compared to three border states including Minnesota, Illinois, and Indiana. The study concluded on the basis of reported statistics among fatal accidents that the legal change produced small, but statistically significant, increases in alcohol-related fatal accidents. A later study by Naor and Nashold[107] found that the change from beer only to statewide availability of all alcoholic beverages at 18 produced no significant increase in alcohol-related fatalities in Wisconsin.

A Massachusetts study[92,108] using time-series analyses with reported alcohol-involvement police data found that drinking-related accidents increased among 18- to 20-year-old drivers,and for fatal accidents, the effect attributed to the lower drinking age was 40%.

In Ontario, Smart *et al.*[109] reported an increase in alcohol consumption of 10% among affected youth following the 1972 change in drinking age to 18. Whitehead *et al.*[95,110] studied all 16- to 20 and 24-year-old drivers in London, Ontario, and found increases in total reportable collisions following the lower drinking age for 16- to 20-year-old drivers, with increases of more than 300% for 18- and 20-year-olds in the first 24 months after the law changed. These analyses resulted in recommendations for increasing the legal drinking age as part of a major effort to prevent alcohol-related traffic accidents subsequent to the lowering of the drinking age and concluded not only that the new law

increased such accidents but also that programmatic efforts to prevent them are futile in the absence of society-wide efforts aimed at drinking habits in general. Warren *et al.*,[111] however, suggested that, based on their analyses of reported alcohol-involved crashes in Alberta, Manitoba, Saskatchewan, and New Brunswick, Canada, the independent effect of the lower drinking ages could be confounded by an earlier change in the legal definition of driving while intoxicated, which reduced the allowable blood alcohol concentration and thereby increased the number of "drinkers" among the crash-involved drivers. The response to these initial studies was a widespread call for increasing the legal drinking age and enforcement of laws governing alcohol sales to youth.[112–114]

A second series of reports and studies emerged after 1975 in Canada and the United States. Smart and Finley[115] reported that one factor in an increase of youthful first admissions to alcoholism treatment programs in Ontario was the lower legal drinking age. Smart and Goodstadt[93] summarized the state of knowledge of the effects of reduced legal alcohol purchasing ages and concluded that traffic accidents usually increased in association with pub or tavern and off-premise consumption of beer by youth. A study conducted in Illinois[116] concluded that the Illinois reduction in 1973 of the minimum age limit for beer and wine from 21 to 19 had contributed to additional fatal accidents. The author reported that 33 fatalities in the first year were directly associated with the legal change, and concomitant increases in property damage and personal injuries were expected.

Follow-up studies in Michigan were conducted in 1977 and 1978.[48,90,91] These studies determined that the increase in alcohol-related accidents as a result of the lower legal drinking age was conservatively estimated to be at least 24% above the level expected for 18- to 20-year-old drivers in the first 4 years subsequent to the legal change. Wholesale increases in such accidents for 16- to 17-year-olds were not found throughout Michigan. At the same time, no deviations from predicted levels were found for drivers aged 21 to 24 or older. Draft beer, rather than wine, bottle beer, or distilled spirits, was determined to be the beverage that was most likely associated with the casualty increase brought about by the lower drinking age. Flora and colleagues[117] analyzed 9 years of Michigan data and concluded that reported alcohol-related fatal crashes with 18- to 20-year-old drivers increased 53% after the legal drinking age changed. These authors reported an increase in the police-reported "had been drinking" rate for fatal-crash-involved young drivers, although the same rates for older drivers remained stable or even decreased. This conclusion was also reported by the Michigan Office of Highway Safety Planning of the Michigan Department of State Police.[118]

Whitehead and colleagues[119–124] concluded that all scientifically conducted studies of states and provinces that found increases in traffic accidents to result from the lower drinking ages were consistent and that these studies validated each other. The basis for Whitehead's comparative analysis on this topic was the scientific design and rigor of the research. He concluded that

additional studies of these states and provinces would further identify the public health impact of these legal changes. Whitehead stated that consideration should be given to raising drinking ages that had been lowered and stopping new initiatives to lower the drinking ages in additional states or provinces.

Rooney and Schwartz[46,125] surveyed high school students in five northeastern states. These authors reported that 42% of seniors in states with 18-year-old drinking ages compared with 45% in states with 20- or 21-year-old limits reported consuming beer at least weekly. The conclusions drawn from their finding was that the minimum purchase age laws had not produced the controlling effects for which they (the laws) were intended. This association, however, failed to use a design that could test the effect of changing the legal purchase age and relied on a static correlation to infer its conclusions. Rooney and Schwartz's conclusions conflict with those of Wolfe.[126] In Wolfe's national roadside breath-testing survey, 14.8% of the 18- to 20-year-old subjects were driving with at least 0.05% blood alcohol content in 18-year-old states compared with 10.8% in 20- or 21-year-old driving age states. Also, Maisto and Rachal[127] reported, on the basis of a national probability sample of youth, systematic differences in adolescent drinking practices between 21- and under 21-year-old drinking states that challenged Rooney and Schwartz. According to Maisto and Rachal:

> the data revealed that higher-drinking-age laws were related to less peer approval of drinking and less perceived peer drinking, less drinking and driving, less accessibility to alcoholic beverages, and less frequent intoxication. These aspects of the data . . . contrast sharply with the Rooney and Schwartz data.

The results of raising the legal drinking age on youth and traffic safety have been tested, to date, in only two studies. Voas and Moulden[128] report that single-vehicle, night-time accidents with a male driver (a measure comparable to Douglass' three-factor surrogate for alcohol-related crashes) decreased significantly after Maine raised the legal drinking age in October, 1977. These data, however, as noted by the authors, were too limited to control for possible confounding factors including seasonal and weather effects or fuel prices.

A more satisfactory test of the effect of raising the legal drinking age has been reported by Wagenaar.[129,130] The work evaluated the first year's experience in Michigan after the legal drinking age was raised in 1978 from 18 to 21 by a constitutional amendment. Wagenaar used a multiple interrupted time-series design, a census of all reported traffic accidents in Michigan between 1972 and 1979, and the Box–Jenkins time-series statistical technique. On the basis of the three-factor surrogate measure,[41] the 18- to 20-year-old drivers in Michigan had 17.7% or 1650 fewer alcohol-related crashes in 1979 than were predicted. On the basis of the officially reported alcohol involvement variable, the net benefit was a remarkable 30.3% reduction. At the same

time, 21- to 45-year-old drivers had more alcohol-related crashes than predicted, and non-alcohol-related crashes were below the predictions, possibly because of the 1979 economic downturn in Michigan.

Scientific research on lower legal drinking ages in most places has concluded that young drivers are more likely to become involved in alcohol-related traffic collisions. Not all places respond to lowering the legal drinking age in the same way. For instance, Douglass[40,102] found significant alcohol-related crash involvement in Michigan and Maine but not in Vermont. The literature also supports the conclusion that beer is more likely than the other beverage categories to be implicated.[48,90]

These findings do not address the basic issues of the difference between youth drinking habits in one state or another as raised by Rooney and Schwartz; differences could exist independently of legal conditions. The question of importance, however, is whether lowering the legal age results in injuries and fatalities in alcohol-related crashes—a question that appears to be answered affirmatively in a variety of studies with complementary methods and analysis approaches, several distinct study sites, and by several independent investigators.

5. Where Do We Go from Here?

The state of knowledge in this area, as reviewed in the discussion above, is generally descriptive. Although we know that certain types of youth in certain kinds of situations and circumstances are more likely than others to be involved in alcohol-related traffic accidents, this knowledge is based entirely on retrospective studies of various designs. The predictive value of this body of information is limited because the variables studied and the designs of the research conducted are neither precise enough nor sensitive enough to accurately identify individuals, in advance, at elevated risk of an alcohol-related crash. Thus, the current scope of appropriate strategies to reduce youthful alcohol-related casualties is limited and is likely to have limited effectiveness. This situation will persist until more research is performed that produces a more sophisticated understanding of the causes and precursors of crash involvement by drinking youth. In the field of alcohol, youth, and highway safety, many questions remain unanswered, and several issues have been virtually untouched by the research community. The list of research that would be useful to the field would be very lengthy; however, a concise list of the most critical of these is discussed here.

The principal justification for support of research in alcohol, youth, and highway safety is the assumption that with scientifically diverse insights, some solutions to the problem will emerge. This applied research perspective in no way diminishes the value of more basic research in the area, which also contributes to the broader body of knowledge of youth behavior and youthful culture, in addition to other research areas. From the applied, problem-ori-

ented perspective, however, it is clear that the basically descriptive nature of the existing literature is inadequate for a concentrated effort to reduce the magnitude of this major health problem of youth. If the goal is to prevent traffic accidents from occurring, then certain research areas emerge as priorities.

The precrash drinking and driving situations that lead to alcohol-related crashes with young drivers have not been adequately researched. Prevention requires an understanding of the precrash circumstances involving youthful drinkers because, particularly with youth, the problem is more likely to be caused by acute situational factors than by long-term developmental problems. Precrash research that is psychological, sociological, or of a broadly social scientific perspective is appropriate. Research questions might include why certain youth drink, how they drink, what they drink, and why they drive after drinking.

A related area of inquiry deals with the motivations and psychological factors that determine drinking practices among youth and that are amenable to educational modification. Although many reports and popular articles suggest cultural reasons for the prevalence of drinking and driving among youth, the actual motivational and other psychological factors determining these behaviors have not been sufficiently studied. Educational programs, if they are to be successful, require substantially more research in this area.

The environment in which youthful drinking and driving take place is not well understood. Part of the environment includes the availability of alcohol to youth; the principal research area in this regard during the last decade has been on the legal drinking age. Several of the states that lowered their legal drinking ages have subsequently raised the legal age because most places that lowered the drinking age experienced increases in alcohol-related traffic accidents among young drivers. The recent increase of age of purchase provides a rare natural experiment of the effects of limiting alcohol availability on a specific kind of morbidity and mortality. The states that have raised their legal drinking ages should be rigorously evaluated with regard to impact on drinking and driving and subsequent crash involvement among youth.

It is likely that by the time youth reach legal drinking age, they have fairly well established attitudes, and perhaps habits, regarding alcohol and drinking. Efforts to modify behavior in order to reduce alcohol-related traffic accidents, therefore, might need to be initiated well before youth are able to legally drive or drink. Attention should be given to young people before they establish drinking and driving habits. Thus, thorough research should seek to understand the attitudes and beliefs about drinking and driving among youth aged 10 through 15.

More than any other group, youth attend to and are influenced by mass media. Media and educational approaches that are effective with young drivers have been modeled on adult themes and messages. New knowledge of the beliefs and perspectives on drinking and driving by youth has not been incorporated into field tests and has not been evaluated for behavioral or

attitudinal impact. This area is of immediate importance because, given the state of practice in alcohol and highway safety, a large proportion of all prevention activities will continue to be media and educationally based. It is clearly essential that we learn how to use these approaches effectively.

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IV

Current Concepts in the Diagnosis of Alcoholism

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Overview

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The concept of diagnosis has importance in medicine because of what it does. Phenomenologically based diagnosis communicates an enormous amount of information succinctly and clearly on which all recipients agree. For example, the two words, pneumococcal pneumonia, communicate with clarity and precision to all medically knowledgeable recipients the world over a series of statistically likely characteristics regarding the following attributes of that diagnosis: etiology, clinical appearance, history of the course of the illness, physical examination findings, natural history both treated and untreated, laboratory findings, x-ray appearance, treatment, prognosis, and inheritability. Thus, the first obligatory characteristic of a valid diagnosis is that it reliably communicate previously agreed on empirically derived information.

The second obligatory characteristic of a valid and practical diagnosis is that it be able to separate the population having the condition in question from other populations defined as not having that condition, by use both of the characteristics and symptoms of that illness and of other characteristics and traits not included within the definition of that illness. Thus, one of the characteristics that might be used to define a "broken leg" is evidence of a disjunction between parts of a bone that is normally continuous when viewed on radiological examination. Associated features not part of the definition but predictably likely to occur in the presense of a "broken leg" include pain and trouble walking. Neither of these are exclusively characteristics of broken legs versus nonbroken legs, but both are more likely to occur in the presence of a broken leg than in its absence. Other examples of associated features might be the history of being an active skier or the presence of some pre-existing calcium-depleting condition. In any case, the concept of the diagnosis tells us things about the patient in addition to his specific diagnosis.

The third attribute of a clinically significant and practical diagnosis is the usefulness it has in predicting future events about that patient. Thus, the diagnosis of pneumococcal pneumonia tells us that this is not a remitting, relapsing disorder, and any future recurrences are not related to this current episode. The diagnosis of rapidly progressing cancer of the head of the pan-

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creas tells us that the patient is not likely to live for any extended period of time no matter what the treatment.

The concept of the diagnosis of alcoholism has evolved steadily during the past 40 years. Leaving aside concepts of alcoholism embracing moral weakness, criminality, feeblemindedness, sin, and debauchery, the use of the label "alcoholism" has shifted based on varying theoretical notions, etiologic assumptions, behavioral and epistemologic concepts, and biochemical and genetic concepts. Further, the breadth of the concept has varied. Alcohol "abuse" may or may not be synonymous with alcohol "dependence" or with "alcoholism." The current public view of alcoholism and of who suffers from alcoholism may not be an accurate perception of the genuine extent of the problem. A much larger net might be cast over our society encompassing substantially greater numbers of alcohol users as being alcohol abusers, alcohol dependent, or alcoholics, depending on one's definition.

Genetic studies point to the probability that severe, recurrent alcoholism requiring frequent hospitalizations for either medical complications or rehabilitation is likely to be genetically based. A defensible theoretical position limits the extent of "genuine" alcoholism to this group of patients and puts aside what are probably the majority of clinical populations currently being defined as alcoholic in rehabilitation programs, detoxification programs, highway assessment programs, or public information programs. If they are not genuine "alcoholics," what are they? Are they "problem drinkers"? Are they alcohol "abusers"? Do they have a "habit" or a conditioned response based on repeated environmental experiences, or are they exhibiting behavior representative or symptomatic of some other psychiatrically defined illness such as antisocial personality or major affective disorder (either depression or mania)? Or might the rest of this population iceberg have a combination of one or more of these factors, genetic, biochemical, symptomatic, behavioral, environmental, or cultural?

We can all agree that no one sets out to become an alcoholic any more than anyone sets out to become a diabetic. But, is that where the similarity between alcoholism and diabetes ends, or is that where the relevance of the medical model actually begins? It is possible that based on a variety of genetic loadings or nonloadings, based on a variety of early life experiences, based on the presence or absence of other psychiatrically definable patterns, based on frequency and extent of contact with the chemical, and based on sociological and cultural permissions or approbations, a variety or even a veritable spectrum of behavioral outcomes and syndromes, possibly all currently collectively defined as alcoholism, may exist.

The crucial and fundamental formulation in the behavioral diagnosis of alcoholism, developed over the past 20 years at Washington University in St. Louis, is the concept that alcoholism, or alcohol abuse, is characterized by the appearance of adverse consequences or problems directly related to the elective use of that substance (alcohol) occurring in multiple areas of the person's life. As summarized and presented by the Washington University

group in their seminal 1972 paper on psychiatric diagnosis, alcoholism involved a problem in two or more of four natural groupings of symptoms which constituted four different life areas.[1] Although not ever labeled as specific life areas, the four groups indeed constituted four areas:

1. Biomedical complications.
2. Control, or the loss of control, of the use of the substance alcohol.
3. Psychosocial complications.
4. Subjective opinions of significant others.

Within these four life groups were 20–25 problems, consequences, or pathological behaviors noted by previous investigators as being common clinical characteristics of individual alcoholics. For example, many of the Jellinek characteristic symptoms are included in the Washington University groupings. The Michigan Alcohol Screening Test, developed by Selzer, used many of the same symptoms and behaviors but chose to score them in instrument fashion rather to cluster them in a traditional medical review of systems fashion as done by the Washington University group. Using this life areas model, and depending on how narrowly or broadly one might define a life area, it is possible to generate four, five, six, or more specific life areas, each of which has an array of problems and pathological behaviors related to the use and effect of alcohol as noted by clinicians for many years.

The entire section on substance abuse diagnosis in the new *Diagnostic and Statistic Manual, Third Edition*, of the American Psychiatric Association, published in 1981, was built on this crucial behavioral concept of multiple problems in multiple life areas as a consequence of the use of this, or some other, elective substance.[2] The authors of DSM-III have further attempted to subdivide alcoholism into two separate categories, alcohol abuse and alcohol dependence. This distinction, here for the first time based on specific behavioral criteria (symptoms) and characteristics, will allow investigators to define their populations in specific and precise ways recognizable to other investigators across time and space for purposes of natural history, follow-up study, differential outcome, and, ultimately, predictive usefulness of these diagnostic distinctions.

Beginning with the Washington University diagnostic formulation as the basis of what became DSM-III, all references to tolerance or the alcohol withdrawal syndrome were removed from group 1 and made to separately constitute the symptoms of "dependence." The remainder of group 1, biomedical complications, and group 2, control issues, were consolidated to form DSM-III's group A, "pattern of pathological alcohol use." The Washington University diagnostic group 3, psychosocial complications, and group 4, subjective opinions of others, were consolidated to form the DSM-III group B, "impairment in social or occupational functioning due to alcohol use." Specific symptoms were added and deleted based on more recent descriptive studies of clinical populations done between 1972 and the formulation of DSM-III. The diagnosis of alcohol abuse for DSM-III is based, then, on the presence

of one symptom, characteristic, problem, or pathological behavior from each of group A, pathological drinking, and group B, psychosocial complications. Alcohol dependence is further diagnosed if one or more symptoms or characteristics of tolerance or withdrawal are present.

Thus, a descriptive diagnosis based on observable or reportable behaviors and not tied to any theoretical constructs of etiology, motivation, genetics, or philosophy of treatment has been achieved. Whether this is an improvement or advancement in the field remains to be determined. Mixing apples and oranges is permitted in a quest for fruit but not in a quest for citrus products or pie fillings. To expect some ultimate diagnostic truth, whether etiologic, genetic, behavioral, descriptive, or biochemical, that in all times and in all places will absolutely separate alcoholics from nonalcoholics is naive. Rather, for our purposes, in late 20th century alcohololology, based on our current level of biological and sociological expertise, this behavioral definition of alcoholism, based on multiple problems in multiple life areas, should provide a stable platform from which the next generation of advances can be made.

Each of the three accompanying papers reviews significant aspects of this concept. Dr. Mandell considers whether there is actually a consistent pattern, an evolution of symptoms, a natural history to alcoholism, as originally proposed by Jellinek and required of a "disease." Further, he reviews the evidence for subgroups of patients with specific characteristics, all subsumed by the term "alcoholism." Dr. Jacobson reviews the spectrum versus unitary positions about alcoholism and then goes on to summarize the variety of ways in which diagnosis may be approached, reviewing the research on assessment instruments with the insights they have provided to our thinking about alcoholism. Drs. Tarter and Ryan organize and present the compelling work that indicates that neuropsychological impairment is a real factor in the natural history of alcoholism and perhaps even in the natural history of social drinking, the elective use of our legal social intoxicant.

Dr. Wallace Mandell in his chapter, "Types and Phases of Alcohol Dependence Illness," presents an excellent review of several definitions of alcoholism developed and presented during the past 30 years by organizations and individuals of influence in the field. Their common use of the crucial concept of life problems is notable. The World Health Organization, for example, required interference with mental facilities or interference with medical or mental health, interpersonal relations, or social or economic functioning. Each of the five positions described were behaviorally derived, based on effects and consequences of alcohol use in the person's life. Additionally, the question of whether alcoholism is a single disease entity, several related disease entities, or a continuum of problems from none to many is reviewed. Dr. Mandell also presents a clear and rewarding summary of many of the basic concepts of Jellinek, valuable in itself as a review of the pioneering work of the father of our field. It highlights the relevance of this work to our current era and shows how much we are still building on his foundation.

Dr. Mandell addresses two fundamental aspects of the disease of alcoholism. Do the proposed signs of alcohol dependence cluster and appear in a developmental sequence; that is, is the pattern of the development of alcoholism consistent over time and across individuals, or are there many patterns for the development of alcoholism? The second question is whether there are consistent subtypes of patient populations who, if adequately defined and subgrouped, would demonstrate differential outcome and differing courses in their illness. Prediction of the differential outcome so clearly present in clinical populations might be possible based on currently available information regarding potential subtyping of patients.

The serious limitations of clinical studies are noted. The fact that patients appearing for treatment of alcohol problems may be very different from the universe of patients with what may or may not be the illness of alcoholism who do not appear for treatment, a sampling bias that may be limiting the usefulness of all work in this field from the biochemical to the sociological. Rarely, if ever, do diabetologists concern themselves with biased sample selection. They merely carefully define the particular set of diabetics that they are investigating as a clinical experimental population.

Reporting diabetes researchers use terms and concepts readily available, but rarely used, by us in the field of alcohol research to more precisely define our populations. Precise descriptions of alcoholic populations, like diabetic populations, might make explicit age of onset, dated either from the development of first problems or the first clinical diagnosis, duration of illness, extent of disability from the illness, type of treatment required to date, complications of the illness, and genetic loading, in addition to the usual epidemiologic characteristics. Thus, a methodological paragraph presenting these characteristics of a study population might be appropriate. An example is herewith presented:

> We studied 30 adult white men with a diagnosis of alcohol dependence using DSM-III criteria, between the ages of 30 and 40, all of whom had an early onset of alcohol problems beginning early in adolescence with a clinical diagnosis possible by age 21, followed by a chronic deteriorating course involving an average of ten specific life complications caused by alcohol use and requiring a minimum of three inpatient detoxification experiences and three inpatient rehabilitation treatment programs. All had had biomedical, legal, marital, and job complications from their alcohol use. All had a positive paternal family history of alcohol dependence extending to at least one first-degree relative and one second-degree relative. All were free of any other psychiatric diagnosis such as major depressive disorder or antisocial personality by DSM-III criteria.

It may be of secondary importance, or even of no importance, whether there is one, two, three, a hundred, or no "alcoholisms" as separate definable diseases. What is of practical importance is the ability to characterize specific conditions in describable, reproducible behavioral terms that communicate clearly what population is being spoken of. If the newly adopted DSM-III

dichotomy of alcohol abuse and alcohol dependence moves the field closer to that clarity of communication, then it is indeed a contribution. If an accumulation of adjectives furthers that communication (such as genetic, simple, primary) and becomes widely accepted and clearly defined, then they too would help in defining subgroups of alcoholics for purposes of prevention, assessment, intervention, treatment, and prognosis.

It has long been apparent that there is no single constellation personality traits in alcoholics. Dr. Mandell reviews five recurrent themes from the literature regarding subtyping of alcoholics. A consistent finding in all of these schemes is that some alcoholics have psychopathic or sociopathic characteristics, and others have depressive or neurotic characteristics. The St. Louis group was most explicit in developing a differential diagnosis among alcoholics that indicated alcoholism in the absence of preexisting psychopathology (primary) as being different from alcoholism that develops subsequent to the onset of some other psychiatric disorder ("secondary").

A review of the work that has attempted to analyze personality traits *per se* in the absence of psychiatric diagnosis demonstrates the confusion in the field. Surveys and questionnaires assemble data without organizing it, confuse epidemiologic characteristics, age, socioeconomic status, education, with consequences in multiple life areas, with a mixture of individual psychological symptoms, evidence of addiction, and genetics, and, often, with a heavy dose of attitudes and opinions as well. In the absence of a specific superior treatment, it is no wonder that diagnostic confusion persists. Dr. Mandell appropriately concludes that treatment programs should institute differential diagnosis and, where appropriate, multiple diagnoses for all chemically dependent individuals.

Dr. George Jacobson, in his excellent review "Detection, Assessment, and Diagnosis of Alcoholism: Current Techniques," addresses the value and real-life consequences of receiving a diagnosis in our society. His appropriate caution is that we, the investigators in the field, have allowed a theoretical concept, that alcoholism is a disease, to become fossilized and axiomatic prematurely. Although the disease concept has served to draw alcoholism out of the shadows of moral weakness, we may soon wish to replace it with a series of illnesses, syndromes, habits, and patterns that in total sum up to the current unitary concept of alcoholism but that individually may only describe one small segment of the current alcoholic population. He suggests that to label because of the final common pathway all avenues as being identical is simplistic. In truth, we all intuitively already accept the probability that there are several different alcoholisms, a genetically based one, a behaviorally conditioned one, a sociologically induced one, a crisis-induced one, and one induced by depression, sociopathy, or loss. Dr. Jacobson has, for some time, helped the field focus on this crucial next step forward at a time when perhaps the field has been resting, or dawdling, on its public relations-induced laurels, i.e., that alcoholism is now at least a marginally socially

acceptable illness. If we are to move alcoholism into the 20th century before the 20th century moves into the 21st century, we must all recognize the validity of his position.

Dr. Jacobson reviews the cumbersome criteria developed by the National Council on Alcoholism to make the diagnosis of alcoholism and accurately assesses the reasons for its lack of acceptance by the wider medical scientific community. Further, he reviews research by himself and others in which portions of the NCA criteria have been applied to large populations within the last 5 years. At exhaustive length and comprehensiveness, he reviews recent work using the National Council on Alcoholism diagnostic criteria, the Michigan Alcohol Screening Test, the McAndrew Scale, and the Mortimer–Filkins Test. Of particular interest is the work of Zung with the MAST, in which, by factor analysis, he derived several orthogonal factors that are remarkably similar to previously described life areas: denial; debilitation, marital discord; work problems; help-seeking; and social discord.

Dr. Jacobson's review of the Alcohol Use Inventory, originally proposed by Wanberg, Horne, and Foster, points up a consistent failing among all of these survey instruments, that is, that they are not used in conjunction with a clinical diagnostic inventory such as that developed and incorporated into the DSM-III. It is hoped that in future studies these sophisticated psychological instruments, which seek to analyze very specific personality traits and correlate these traits with important practical questions such as treatment response, length of outpatient visits, risk of relapse, etc., will be shown to have more utility when used on specific diagnostic subpopulations as predefined by the DSM-III. For example, it may be that when alcoholics who have a preexisting primary affective disorder diagnosis are studied using one or another of these instruments, personality subgroups will be identified that respond differentially to different alcohol rehabilitation programs but that currently such information is lost because the psychological instrument is assessing several diagnostically different alcohol groups.

The finding of neuropsychological impairments among alcoholics and among social drinkers who may or may not be at increased risk for subsequent alcohol problems (as predicted by these impairments) is a significant step forward in our ability to discern subtle changes in normative functioning and correlate those impairments with social behavior. Beyond the field of alcoholism, Dr. Tarter's personal studies and review of other studies has important implications for us all. Drs. Tarter and Ryan ask if there are neuropsychological factors that may predispose certain individuals to consume alcohol excessively, to instinctively attempt to correct some disturbance within the brain. The possible mechanisms of these observed deficits are explored, as are the subsequent effect of overconsumption of alcohol and of sobriety on the recovery of neuropsychological competence. The findings of neuropsychological impairment are subtle but reliably found and require complex tests that assess conceptual, perceptuomotor, and mnestic functions. New

memory efficiency is reduced, rapid motor responses are impaired, and abstraction skills are reduced. Generally, these deficits are mild and subtle, with a wide range of variance among these populations.

Remarkably, applying these same sophisticated tests to nonalcoholic populations has revealed slight but clear evidence that moderate consumption of alcohol beverages may have a deleterious effect on the neuropsychological status of some persons who are not clinically diagnosable as alcoholic. The ways in which alcohol consumption places a person at risk for developing neuropsychological deficits are reviewed clearly and thoroughly. This specific review is must reading for any young investigator setting out on the road to a Nobel prize. The possible pathways by which alcohol consumption may lead to brain effects, none mutually exclusive, aptly summarize the complexity of this field.

The data are reviewed regarding antecedent neuropsychological characteristics among those genetically related to alcoholic patients, various hypotheses of childhood syndromes that might correlate with later alcoholism, and the possibility that alcohol effects on the brain may vary depending on the time of life of the individual consumer. For example, alcohol "consumed" *in utero* or in old age may be far more potent in impairing neuropsychological competence than alcohol consumed in young adulthood. As noted, little is known of the relationship between specific drinking patterns and their neuropsychological test consequences. The answers to the questions how much?, how often?, how long?, how severe?, how permanent?, remain unanswered. From Dr. Tartar's own work, there may be evidence that recovery, with sobriety, may occur in several long slow stages lasting years, although it is clear that after one year of continuous sobriety, alcoholics continue to show some slight impairments on certain tasks.

This review by Tarter and Ryan brings us back to where we began this discussion on the concept of diagnosis in alcoholism. As quoted by Mandell from Pattison, Sobel, and Sobel, as of 1981:

> There is no clear dichotomy between either alcoholics and nonalcoholics, or between prealcoholics and nonalcoholics, even though individuals may have a differing susceptibility to both the use of alcohol and the development of alcohol problems as the result of genetic, physiological, psychological, and sociocultural factors.

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Detection, Assessment, and Diagnosis of Alcoholism
Current Techniques

George R. Jacobson

Abstract. The need for a continuing evolution in methods of detection, assessment, and diagnosis of alcohol abuse and alcohol dependence disorders is emphasized as a necessary step toward altering the stagnating effects of several static and outdated constructs and practices which, despite their humanitarian benefits, may actually be impediments to progress in prevention and treatment. Distinctions are drawn among the purposes and techniques of detection, assessment, and diagnosis; although all three activities are vital, it is the last that must provide a logical basis for understanding causes, courses, and treatment for health problems. Thus far, there has been little genuine progress beyond simply naming the problems we attempt to treat. To illustrate the promising directions that progress might take and to recommend research advances, recent developments in detection, assessment, and diagnosis over the past 5 years are reviewed and critiqued. Special attention is paid to the NCA Diagnostic Criteria and its newest modifications, the Michigan Alcoholism Screening Test and its derivatives, the MacAndrew Scale, the Mortimer–Filkins test, the Essential–Reactive Alcoholism Interview Schedule, and the Alcohol Use Inventory. All of these approaches have demonstrated their utility, but none has yet fulfilled its potential. Their relative advantages are discussed, their flaws are identified, and suggestions are given as to how researchers and clinicians might better use the available tools.

1. Introduction

When invited to prepare a "state of the art" manuscript bringing readers up to date on recent developments in the detection, assessment, and diagnosis of alcoholism, I found that 5 years had passed since a comprehensive review had been published.[1,2] Despite frequent pronouncements about the importance of earlier detection, the need for identification of latent or incipient alcohol problems among women, youth, and ethnic minorities, and the potential value of bringing people to treatment at the earliest possible time, little had been done to develop bold and meaningful innovations in approaches to alcoholism, innovations that could evolutionize, if not revolutionize, our

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entire field. By and large, the diagnosis, assessment, and detection of alcoholism have been a sluggish area of study. Why has this potentially dynamic and enormously important topic failed to keep pace with other areas of research? Two reasons in particular seem especially important. The first has to do with the deterioration of the diagnostic process as a meaningful precursor to treatment into an often empty and sometimes counterproductive and even destructive process of labeling. As Forrest[3] has observed,

> all too frequently . . . diagnosis has unfortunately resulted in an individual being labeled for life, . . . [leading to] a process of social disengagement which these labels both facilitate and maintain. . . . Finding a job, social acceptance, and similar essentials to effective interpersonal relations often become significantly more difficult. . . . While a select few individuals learn to transcend the stigma associated with the label, countless others give up or use the label as a means of validating their lack of personal responsibility. In this respect being labeled an alcoholic, a schizophrenic or whatever amounts to a license to continue the inappropriate or "crazy" behaviors which happen to be associated with the particular label" (pp. 56–57).

Forrest attributes this debasement of the diagnostic process to both the negative effects of labeling and changes in philosophy and treatment approaches in the entire field of mental health. I believe that a far more influential factor is the static nature of the concept of alcoholism itself. There has been much public promotion of alcoholism as a treatable illness, a disease that fits a medical model, one that is predictably progressive and fatal if untreated, with the development of increasing tolerance for alcohol and the eventual loss of control over drinking, accompanied by deterioration in physical, psychosocial, and spiritual status, and for which in most quarters, it is believed that abstinence is the only acceptable treatment goal.

There can be no question that it is better, and closer to the truth, to view alcoholism as a disease amenable to the ministrations of modern medicine and allied health sciences than to view the alcoholic as a sinner, a moral degenerate, a weak-willed person, or a criminal. A problem arises, however, when a construct of alcoholism—a "complex image or idea resulting from a synthesis by the mind"[4]—becomes a stereotype of alcoholism, "fixed or settled in form, hackneyed, conventional."[4] And I believe that that is what has happened. We have traded the old stereotype of alcoholism for a new one, somewhat enlightened and certainly more humanitarian, but a stereotype nonetheless. We have moved, imperceptibly but inexorably, from a rational position to an emotional position regarding alcoholism, and some of us have become dogmatic, intolerant of ambiguity and uncertainty, and have insisted on closure before an appropriate gestalt is warranted.

What I would like to attempt here is not a destruction of the gestalten but merely a temporary disruption of it, a modification, one that will admit the addition of several new components and thereby permit the formation of multiple gestalten, all bearing some resemblance to the initial one but also

allowing for some identifiable differences. To do so, one need only to think in terms of the plural, *alcoholisms*, rather than a singular alcoholism. That simple change, the addition of a single letter, can provide unprecedented opportunity for the research and treatment communities to so broaden their horizons as to permit, if not demand, a flowering of new experimental investigations and the development of new treatment approaches that could radically alter our heretofore dismal statistics on successful treatment outcome. No longer would we have to defend against the observation that type of treatment has no significant effect on outcome or the notion that the effect of treatment *per se* may be nil in terms of conventionally accepted ideas of what constitutes successful recovery.[5-7] Implicit in a construct of *alcoholisms* may be, just to give a few examples of the possibilities opened by such an idea, an alcoholism that is attributable to enzymatic deficiences, or one that develops in response to traumatic personal crisis, or one that conforms to the medical model of disease and may indeed be progressive and fatal if untreated. Such a construct does, of course, stimulate research into multiple etiologies rather than focus on the thus far fruitless search for a single ultimate cause.

A construct of alcoholisms or, alternatively, *alcoholism syndromes*, would encourage if not necessitate a better understanding of prognosis and treatment.[8,9] An illustrative analogy may be drawn by comparing the state of our knowledge regarding the etiology, prognosis, and treatment of pneumonia today[10] with that of a hundred years ago.[11] Nineteenth century medical scientists were in a state of ignorance about pneumonia comparable to our current position regarding alcoholism, yet today we recognize many types of pneumonias, at least 50 known causes, and at least a half dozen treatments. Perhaps we might someday learn to similarly diagnose the alcoholisms, assess their prognoses, and prescribe the most appropriate treatment.

Such a possibility is moved closer to hand by Ewing's recent interview[12] in which he stated that "I do not know how many types of alcoholism there are. However, I think we have reached the stage where we can and should recognize the existence of at least two" (p. 5). Relatedly, another group of researchers had earlier pointed out that there is at least "preliminary evidence indicating that there may be two types of alcoholism in women"[13] and, in later studies,[14] asked "The important question is: Do these findings support the concept of three separate diseases within the rubric of alcoholism?" (p. 530). Although the answer to that question was an equivocal "possibly not," it appeared to me that

> Rather than continue the chimerical pursuit of a "typical alcoholic" or a unitary "alcoholism," it would seem more reasonable and prudent to entertain the idea that there may be several *alcoholisms* which, once detected, assessed, and diagnosed, may be amenable to different treatments (2, p. 16).

That leads us, then, to the central purpose of this chapter: describing and evaluating those developments of the past 5 years relevant to the detec-

tion, assessment, and diagnosis of alcoholisms. *Detection* refers to the process of identifying persons who are "alcoholic" or who have major significant life problems involving the use of alcohol. As such, it involves a binary decision process in response to a dichotomous question—is this person alcoholic?—without regard to whether such a dichotomy clearly exists in objective reality. Although this process may not be particularly useful for understanding or treating alcoholism (in any form), it may nonetheless be a functional necessity in a variety of settings where rapid screening in needed. Most detection approaches tend to be wholly empirical rather than proceeding from hypothesis or theory regarding the etiology of alcoholism, but seem to assume a unitary concept. *Assessment*, on the other hand, may or may not involve a unitary concept but does assume a continuum of severity on one or more dimensions along which the degree of seriousness may vary. There is, then, an underlying assumption about the progressive nature of alcoholism, since such continua usually are anchored in such terms as mild–moderate–severe or early–middle–late. Because of the frequent combining of the detection and assessment processes, these two separable functions are discussed together in this chapter.

As mentioned earlier, *diagnosis* need involve nothing more than a labeling process, or "denoting the name of the disease a person has or is believed to have."[11] But the term is herein reserved to refer to

> the use of scientific and skillful methods to establish *the cause and nature* of a sick person's disease . . . by evaluating the history of the disease process; the signs and symptoms present; laboratory data; special tests. . . . The value of establishing a diagnosis is *to provide a logical basis for treatment and prognosis* (11, emphasis added).

One can see, then, why diagnosis, in the most complete sense of the term, is a direct link in the chain of understanding, prediction, and control that is crucial to improvements in the sciences of prevention and treatment of alcoholisms.

2. Detection and Assessment

Earlier reviews[1,2] focused on a half dozen or so vigorous approaches that warranted special attention. I say vigorous in the sense that (1) they appeared to be widely used in clinical settings or (2) were frequently referred to in the clinical and/or research literature or (3) had simply been around for quite a while. Special attention was paid to such critical issues as validity, reliability, standardization, availability of norms, utility, and applicability in a variety of settings, and with a variety of special populations, costs, time requirements, special training required for administration and interpretation, and related matters of particular concern.

To repeat much the same information here would be redundant. Instead,

I have opted to use this opportunity to discuss new developments in four of the earlier-reviewed detection/assessment techniques which appear to have retained their vigor (which ought not to be confused with their rigor, since that is surprisingly absent in some cases) according to criteria 1 and 2 above. (Criterion 3 above is not adequate justification for inclusion here, only partly because of space limitations.) In addition, two other techniques are discussed here because they represent relatively novel approaches and/or an especially important focus. Some of the detection/assessment techniques reviewed earlier[2] are excluded from consideration here for a variety of reasons, all of which will appear arbitrary or subjective, and so I will not try to defend my decisions.

2.1. The National Council on Alcoholism's Criteria for the Diagnosis of Alcoholism

Nearly 10 years have elapsed since NCA's Criteria Committee published their definitive document on diagnosing alcoholism.[15] Development of the NCA Criteria (CRIT) was stimulated, in part, as a

> response to the *Powell v. Texas* Supreme Court decision that no agreement could be found among medical experts on the "meaning, manifestations, or treatment of alcoholism as a disease." NCA expects that as the new criteria are adopted, the courts will have a basis for reversing that finding (16, p. 5).

The purpose of the CRIT was "to establish a diagnostic system that may be used to promote early detection, and provide uniform nomenclature, while preventing overdiagnosis" (2, p. 131), with the hope that "surely as we advance in this project [of applying and testing the CRIT], we will reach the day when no one will be able to say that we professionals in the alcoholism field disagree about the manifestations of alcoholism" (17, p. 8). It was also expected that

> Our next step will be a validation of these criteria; we're going to mount a study in several hospitals, rating the criteria against independent diagnosis of the same patients by experts. . . . Then, if we can demonstrate their reliability, we can go to a test case and tell the court we do have firm reproducible evidence upon which to make a diagnosis (16, p. 5).

Immediately after publication of the original source document, the CRIT was criticized for its overemphasis on the late stages of the illness,[18] for being an undesirable laundry list of symptoms,[19] and for containing confusing ambiguities and contradictions.[20] On the other hand, it is fair to note that laudatory acceptance of the CRIT was also extended.[18] But 5 years after publication of the CRIT, all of the earlier criticism remained unresolved, little or no progress had been made to clarify issues of major significance, its practical value was still questionable, and its potential was largely unfulfilled. At that time, it was observed that

> A great deal of work needs to be done on the Criteria before any conclusions can be drawn about its validity, reliability, and practical applicability; probably several modifications can be expected during the next few years, and it would be premature to make judgments at this time. Certainly the Criteria represents a major step forward for the medical profession and may indeed prove to be a highly useful and significant addition to the clinician's diagnostic armamentarium . . . (2, p. 137).

But after a decade, the promise of the CRIT still remains largely unfulfilled: large-scale testing of the validity, reliability, and applicability of the CRIT has not been accomplished; the ambiguities, contradictions, and confusion that were noted in 1972 and 1976 still exist today; the CRIT has not been adequately used to promote early detection or provide uniform nomenclature; nor are professionals in agreement over what constitutes alcoholism, and in fact, one group of professionals—psychiatrists—no longer even refers to "alcoholism" (see DSM-III[21]).

Although it may appear that the CRIT is being neglected, that is not entirely true. A highly respectable empirical evaluation study was recently published,[22] detailing the results of applying the CRIT to 120 male alcoholics at three different medical facilities in Germany and to an age-matched comparison group of 80 nonalcoholic male inpatients at a general hospital. The outcome of that study is particularly instructive. When independent diagnoses by physicians were compared to CRIT-based identification of alcoholics, 162 (81%) of the 200 subjects were correctly classified, and 38 subjects (19%) were incorrectly diagnosed. Of crucial importance is the fact that all 38 of the incorrectly classified patients were nonalcoholics, resulting in a 0% rate of false-negative identification and 47.5% rate of false-positive identification. The authors concluded, understatedly, that "in summary, the diagnostic scheme has an unjustifiably high sensitivity" (22, p. 1270).

Rather than go into extensive detail here, I will quickly summarize some of the more salient findings: (1) of the 86 criteria employed in the study, only 11 of them differentiated between alcoholics and nonalcoholics at the 100% level (i.e., none of the nonalcoholics manifested the symptom); (2) the frequency of endorsement of symptoms by the alcoholics ranged from a high of 97% on one criterion (morning drinking) to a low of 0% on 13 separate criteria; (3) five of the criteria were present more often in the nonalcoholics than among the alcoholics, and on 16 other criteria, there were no significant differences in frequency of appearance among alcoholics versus nonalcoholics; (4) overall, 38 of the 86 criteria used in that study did not significantly differentiate between alcoholics and nonalcoholics, and of those remaining 48 which were statistically significant discriminators, "four criteria are sufficient to explain more than 90% of the variance in our sample in respect to the independent criterion. The inclusion of additional criteria increases the explained variance by less than 0.001%" (22, p. 1268). Those four criteria were gross tremor, regressive defense mechanisms, morning drinking, and alcoholic blackouts. (5) When a value of one point is given to each criterion, a

score of 6.5 correctly classifies 99% of the subjects when only the 17 most powerful discriminators are used; a score of 13.5 accurately categorizes 95% of the patients when all 48 discriminators are used; and 95% of the subjects were also correctly dichotomized when only the above-mentioned four criteria were used.

The results of the Ringer *et al.* study immediately became the focus of a controversy, eliciting favorable and unfavorable comments from notable figures.[23–26] All of the commentators uniformly faulted Ringer *et al.* for not realizing that (in American hospitals) anywhere from 20%[23] to 15–40%[24] to 20–50%[26] of general hospital patients have alcohol problems and that physicians are not particularly adept at diagnosing such persons. Far from faulting the CRIT for its dangerously high false-positive rate, one of the commentators[24] turned the tables and assumed that the 47.5% rate was attributable to the Criteria's ability to "pick up early alcoholism very effectively" (p. 1832), and therefore (I infer), those 38 nonalcoholics should not be considered false positives.

When other clinicians[26a] tried to apply the Criteria to a group of 24 VA patients, they found it to be confusing and lacking in clear-cut definitions and decision rules. Their experience led them to conclude that (1) there are too many criteria, and the large number increases the probability that one or more symptoms may occur by chance, (2) the number of criteria should be reduced as a means of making the system more concise, (3) diagnostic-level-3 criteria should be eliminated entirely (which we did in our later studies using a modified Criteria, see below), and (4) "the NCA Criteria do not provide the definitive diagnostic system for alcoholism" (p. 31).

One of the major problems of the CRIT, which has plagued it since its inception, was pointed out by Chafetz[25]: "The limitation [of the Criteria] is that they try to tuck into a medical diagnostic model something that is not a medical configuration" (p. 1834). This fundamental criticism has been repeated in several ways by a variety of voices. An editorial in *Lancet* suggests that "the hunt for a definition of alcoholism should be abandoned as the pursuit of what was never more than an imagined animal: a medical diagnosis was being falsely imposed on behaviours and events of great variability" (27, p. 1087). Rohan believes that

> all attempts to identify "alcoholism" have failed because the concept itself is fundamentally flawed. "Alcoholism" exists in our language and in our minds, but not in the objective world around us. The [NCA Criteria] mark another extended but misdirected effort to identify and diagnose an imaginary entity. . . . The criteria would be of value in identifying characteristic consequences of problematical drinking, and in establishing a continuum of negative consequences, rather than as serving to diagnose the explanatory fiction of "alcoholism" (28, pp. 211,217).

Pattison[29,30] indicates that the CRIT has failed because it is based on a unitary disease model, can be used only for detection rather than diagnosis, and is founded on notions of "progression of symptoms," which recent research

suggests do not conform to empirical observation, and he prefers to attempt differential diagnosis of a variety of alcoholism syndromes. I have recommended abandonment of the chimerical notion of a unitary disease entity of alcoholism in favor of the differential diagnosis of alcoholisms[2] and more recently pointed out that

> some of the individual items within the Criteria may not be valid; the disease concept of alcoholism as a unitary clinical entity implicit in the Criteria may be flawed or inappropriate; but in fact parts or all of the Criteria may be useful and valid for identifying persons with alcoholism syndromes [Pattison's term] other than that classic construct of alcoholism which is embodied in the NCA document (31, p. 985).

It would seem that perhaps Rohan, Pattison, and I are in agreement to some extent, and certainly Pattison[32] and I may be using interchangeable terms.

Pattison[30] has written a comprehensive critique and assessment of the CRIT in which he has pointed out that

> The reasons . . . for the development of the diagnostic criteria are primarily social, political, economic, and legal rather than scientific or therapeutic. The document infers the need for diagnostic criteria to justify the designation of alcoholism as a disease and to justify the diagnosis of specific persons as having said disease. The ultimate ends of this rationale are humanitarian . . . the intent is first and foremost to buttress social legitimation of alcoholism as a disease . . . the criteria items are indeed skewed to justify a "medical diagnosis" of a "disease" (p. 966).

In support of that position, Pattison reviewed a number of studies that documented some of the problems he identified. He cited three studies that suggest that certain of the CRIT items—those involving reports of increasing tolerance for alcohol and measures of escape or symptomatic drinking, for example—may not be reliable[33,34] and that another—elevated blood alcohol concentration found during routine visits to the physician's office—may not be valid.[35] In another study of 70 alcoholics, only five of them had any physical symptoms that matched the CRIT, and only 27 of them had any clinical/medical problems that resulted in their being identified as alcoholics.[36] Four other studies showed that only two of the CRIT's several diagnostic laboratory symptoms were consistently present in a majority of alcoholics, and then only in "severe cases,"[37–40] although a survey of physicians did provide a degree of concensus regarding the face validity of CRIT items.[41] And in a successful attempt to quantify and operationalize the CRIT items, it was found that the Criteria could distinguish between alcoholics and nonalcoholics at an acceptable high level of confidence.[42] By comparison, however, the 5-min self-administering form of the Michigan Alcoholism Screening Test (MAST, see below) was equally successful and had lower rates of false-positive and false-negative identifications.

Based on his survey of the available studies, and his ideological inclinations, Pattison concluded that

> the few studies available do not support the utility of the NCA diagnostic criteria, . . . demonstrate the problems with the validity, reliability, and discriminant power of the criteria items, illustrate the skewed bias toward physical consequences which do not have high diagnostic utility, and reveal that the NCA diagnostic criteria are neither intrinsically accurate nor as accurate as simpler instruments . . . it is narrow in scope and severely limited in applicability, [and other instruments] have more desirable psychometric qualities and are more utilitarian. Finally, more sophisticated methodologies, with the requisite psychometric and conceptual flexibility to deal with the data, have been developed, which render the NCA diagnostic criteria obsolete (30, pp. 977–978).

In my analysis[31] of Pattison's review, I supported many of his observations and opinions, but I am more optimistic than he regarding the applicability of the CRIT, suggesting that at least parts of the Criteria may be useful, but some modifications must first be made. With that idea in mind, I will briefly summarize some of the research done since 1976 in which my colleagues and I have applied portions of the CRIT in a variety of settings to well over 1000 persons, including motorists arrested for driving while intoxicated,[43,44] hospitalized alcoholics,[45] and unemployed welfare recipients.[46,47] In all of our empirical validation studies of the CRIT, we focused primarily on the psychological, behavioral, and attitudinal symptoms from Track II because (1) we agree with Dr. Seixas that it was never intended for the entire CRIT to be applied as a diagnostic test, (2) most of our test sites had neither physicians nor clinical laboratory facilities available, (3) *being* an alcoholic—as opposed to *having* alcoholism—obviates the need for a medical/disease model and requires a focus on behavior, and (4) behavioral symptoms usually occur long before any of the physical consequences of alcohol abuse are likely to develop.

For our first study,[43,44] we selected the 35 CRIT items that could be operationalized as observable, reportable behaviors and trained our interviewers to elicit relevant information on the basis of a structured CRIT-based interview schedule. We included items A.1 (a,b,c,d) and A.3 in Track I, all three of the major symptoms in Track II, and all 27 minor symptoms in Track II (see original source document[15] for specifics).* The interview was individually administered to 205 male and 11 female motorists who had been arrested for driving while intoxicated (DWI) between November 1, 1976 and March 31, 1977 and who were processed at any one of 12 disparate urban and rural sites throughout Wisconsin. Most of these DWI offenders were also administered a standard version of the MAST (see below) and another structured interview then in use in Wisconsin (Traits of Alcoholic Scale, TAS), and nearly 40% of them were also administered a form of the Mac Andrew Scale (MAC, see below).

* Copies of the CRIT format listing the 35 symptoms, their diagnostic values, and our instructions for using the format are available from the Ralph G. Connor Alcohol Research Reference Files (CAARRF), Center of Alcohol Studies, Rutgers University, New Brunswick, New Jersey 08903.

Interviewers had been instructed to identify as alcoholic any DWI offender who manifested any one of the diagnostic-level-1 symptoms ("classical, definite, obligatory" according to the NCA Criteria Committee) or any three of the diagnostic-level-2 symptoms (the Criteria Committee indicates only that "several" must be present for the diagnosis of alcoholism). As a result, 22.7% of our sample expressed two or more level-1 symptoms, and 39.4% manifested one such symptom; 31.5% acknowledged five or more level-2 items; 23.6% acknowledged three or four of the symptoms; and 44.9% endorsed two or fewer of the level-2 criteria. The most frequently observed diagnostic-level-1 symptom was item 2 in Track II: "drinking despite strong, identified social contraindications (job loss for intoxication, marriage disruption because of drinking, arrest for intoxication, driving while intoxicated)," in which we specified that all four constituent behaviors must be present in order for the criterion to be considered fulfilled.* Our CRIT-based detection procedures were significantly correlated with the results of our other three dichotomous measures (alcoholic–not alcoholic, as determined by specified cutoff scores), with coefficients ranging from 0.266 to 0.575 (all P values ≤ 0.01), and rates of agreement ranged from 39% to 76%. Among our interviewers, 79% of them said they were "confident" or "very confident" about the accuracy of their CRIT-based decisions, but they also noted that subjects being interviewed were "more angry, defensive, fearful, threatened, and less calm, relaxed, cooperative."

Although a number of problems were identified (see 31,43,44 for details), the results of this study allowed us to conclude that with adequate training in interviewing and behavioral observation, nonmedical field personnel could be taught to use specified portions of the CRIT for identifying some alcoholics with a reasonable degree of confidence. We also pointed out that although coefficients of concurrent validity and levels of agreement between the CRIT and other binary detection procedures seem reasonably high, the CRIT should still be viewed as an equivocal instrument in need of further validation studies. We also advised the Criteria Committee of a need to clarify the meaning of "social contraindications" to the use of alcohol and "blatant indiscriminate use of alcohol" in the light of sociocultural and contextual complexes within which drinking may occur. And we encouraged the modification of the all-or-none concept of alcoholism as a unitary disease entity, on which the CRIT is based, and the elimination of the dichotomous alcoholic–nonalcoholic decision-making process in favor of a differential diagnostic approach that would more closely correspond to the reality of multiple alcoholism syndromes or alcoholisms. In the meantime, our 35-item CRIT-based interview schedule is now in use throughout Wisconsin for all convicted DWI offenders, and we

* Compare Ringer *et al.*,[22] who required only one of the four component behaviors for fulfillment of this criterion. Also, they required only two level-2 symptoms for positive diagnosis versus our requirement of three. These two factors could account for some of their reported 47.5% false-positive identifications.

expect that within a year or so we will have enough data to present the results of further studies with this instrument.

Because the MAST, MAC, and TAS used in our first study do not possess all of the desirable psychometric properties of valid and reliable detectors of "alcoholism," we sought another measure against which we could assess the concurrent and discriminant validity of our modified CRIT. In the course of conducting a larger study of biochemical variables in alcoholism,[45] we settled on the ratio of α-amino-*n*-butyric acid to leucine in plasma (the plasma A/L ratio, see below) as an objective criterion. We administered our CRIT-based interview (along with MAST and MAC) to 37 men and 17 women who had been admitted to De Paul Rehabilitation Hospital between January 9, 1978 and February 24, 1978. Subjects had no previous history of inpatient treatment for alcoholism, and all were diagnosed by physicians as alcohol addiction ($N = 50$), habitual excessive alcohol use ($N = 3$), or other/unspecified alcoholism ($N = 1$), according to the criteria of the then-current DSM-II of the American Psychiatric Association. Mean age of the patients was 36.24 ± 11.61 years, self-reported length of problem-drinking history averaged 56.25 ± 36.4 months, and patients described themselves as daily drinkers ($N = 38$), binge drinkers ($N = 3$), weekend drinkers ($N = 3$), or mixed-pattern drinkers ($N = 10$). Blood specimens for determining the plasma A/L ratio were drawn within 24 hr after admission to the hospital, and all other measures were administered 3–7 days later.

Number of CRIT symptoms present was not significantly correlated with A/L ratio, and when point-biserial correlation techniques were applied, only five of the CRIT symptoms were significantly (2-tailed $P \leq 0.05$) related to A/L ratios: patient's subjective complaint of loss of control of alcohol consumption ($r_{pbis} = 0.31$); repeated conscious attempts at abstinence ($r_{pbis} = 0.26$); shifting from one alcoholic beverage to another ($r_{pbis} = 0.30$); frequent change of residence for poorly defined reasons ($r_{pbis} = -0.24$); and psychological symptoms consistent with permanent organic brain damage ($r_{pbis} = 0.24$). We concluded that these results reflected poorly on the 35 behaviorally oriented CRIT items, but it is equally likely that A/L ratio is of questionable validity, and we determined that additional validation studies were necessary before we could unequivocally accept or reject the NCA Criteria.

An opportunity for further testing arose when we attempted to develop a screening technique for detection of alcoholism among welfare recipients in Milwaukee County. Our subjects represented approximately 10% of 2996 male and female welfare recipients ($N = 309$) being screened for participation in CETA activities between March 1, 1978 and September 30, 1978. Because complete data are reported elsewhere,[46] only a summary of the basic procedures and findings is discussed here. We made some further modification in the Criteria (calling our new instrument the MODCRIT) and assigned scoring weights of 5 points to the diagnostic-level-1 items and 1 point to the level-2 symptoms. The revised MODCRIT was administered as an individual interview by our specially trained and experienced interviewers, who had been

instructed to identify (and subsequently refer for further evaluation and/or treatment) as "alcoholic" any subject who scored 5 or more points on the instrument. A standard version of the MAST was also administered to each participant after voluntary informed consent had been obtained.

Nearly one-third of our sample was identified as alcoholic according to our scoring of the MODCRIT: 31.9% scored 5 points or higher (at least one level-1 symptom or at least five level-2 symptoms), 2.9% scored 4 points, 6.1% scored 3 points, 4.9% scored 2 points, 8.1% scored 1 point, and 45.6% scored 0. Assuming MAST and MODCRIT scores to be continuous variables (even though their decision processes are essentially dichotomous), the two measures were highly correlated ($r = 0.81$, $P < 0.001$), and persons identified as alcoholic by both measures simultaneously endorsed a mean of 8.8 ± 4.9 of the NCA symptoms versus only 1.4 ± 2.9 for all other participants ($P < 0.001$). Those subjects identified as alcoholic on the MODCRIT manifested an average of 1.6 ± 1.6 level-1 symptoms, 5.3 ± 2.7 level-2 items, and 1.9 ± 1.6 of the few level-3 symptoms we had retained (although the level-3 items were not part of our scoring system), and all of these values were significantly ($P < 0.001$) higher than those for other participants. To a certain extent, then, we believed we had demonstrated a fair level of concurrent validity for the MODCRIT, but our subsequent item analyses indicated that only 14 of the 35 items displayed significant discriminant validity (one-tailed P values ranged from < 0.05 to < 0.0001). Although our overall classification error had been only 14% (6.5% false negatives and 7.5% false positives), it was obvious that our MODCRIT needed further work.

A subsequent follow-up grant allowed us to pursue the further development of an appropriate screening technique, results of which are reported elsewhere.[47] Additional revisions of our MODCRIT yielded the MODCRIT-II, using only those 14 earlier-identified statistically significant discriminators, which we cast in the format of simple, descriptive, declarative statements which were read aloud by our examiners to small groups of subjects who then checked appropriate boxes on individual answer sheets. The MAST was also administered in a standard format. Subjects were 500 male and female welfare recipients in Milwaukee County who were being processed to determine their eligibility for CETA programs. Informed voluntary consent was obtained before testing was begun.

Applying our previously developed weighting system for Criteria items, the MODCRIT-II identified 33% of the sample as "alcoholic" (i.e., scores of 5 or higher). Considering MODCRIT-II and MAST scores as continuous variables, the Pearson correlation coefficient was 0.655 ($P < 0.001$); for dichotomous decisions of alcoholic–nonalcoholic, the ϕ coefficient was 0.493 ($P < 0.001$) with a MAST cutoff score of 5 and 0.587 ($P < 0.001$) when the MAST cutoff score was raised to 12. Overall rates of agreement between the CRIT-based and MAST-based decisions ranged between 72.4% and 82.4%, with the rate of agreement increasing as the MAST score moved toward 12. We also found a statistically significant ($P < 0.001$) correlation between scores on

MODCRIT-II and a quantity/frequency measure of alcohol consumption.[48] We also determined that the three most powerful discriminators for use among this special population were blatant indiscriminate use of alcohol, patient's subjective complaints of loss of control over alcohol consumption, and spouse (or other family members) complaints about drinking behaviors. The ϕ coefficients for these three items were, respectively, 0.670, 0.523, and 0.519, and nearly 80% of the alcoholic sample was correctly identified by the combination of these items. Our MODCRIT-II also correctly identified as alcoholic 69.6% of those participants who later indicated that they had had previous treatment for alcoholism (as well as identifying 87.5% of the self-reported drug abusers in the sample).

Interestingly, there was a 72% overlap between MAST and MODCRIT-II in terms of the symptomatic behaviors described by the two measures, and on that account, perhaps Pattison[30] is correct in inferring that simpler instruments may be just as accurate as the NCA Criteria. On the whole, however, I would disagree with Pattison about the CRIT being obsolete. Quite the contrary, it is my firm conviction that the value of the NCA Criteria has not yet been adequately investigated. We have demonstrated to some extent the potential of portions of the CRIT to identify alcoholic individuals in a variety of settings and under differing circumstances, and it is possible that future research may prove the Criteria to be even more useful. True, it does not possess all of the psychometric properties of a valid, reliable, well-standardized instrument, but I suspect that additional validation studies could reduce or eliminate many of the problems. For example, I would predict that appropriate factor-analytic studies of the CRIT could lead to an effective means of selectively applying specific portions of the Criteria to the identification and definition of a variety of alcoholism syndromes in the near future. But first we need to overcome the obstacle of the stereotyped notion of a single, unitary alcoholism, which also creates problems by forcing us into a binary-choice position of "alcoholism, yes or no?"

2.2. Michigan Alcoholism Screening Test

Soon after Dr. Melvin Selzer's Michigan Alcoholism Screening Test (MAST) was published,[49] it became one of the most widely used detection instruments in the country and even received favorable publicity in a Chicago newspaper.[50] When the MAST was initially reviewed,[1,2] it was pointed out that the test had the advantages of being quick (approximately 10 min for the initial 25-item version), easy to score (no special training required to sum the point values for each item; items are weighted 0, 1, 2, or 5 points each; scores of 0–3, no alcohol problem; 4, possible/suspected problems; 5 or higher, alcoholism), and suitable for individual, group, or self-administration.

The high level of face validity was viewed as both an advantage and a disadvantage, and questions were raised regarding the weighting of some specific items (e.g., having attended an AA meeting was weighted 5, but

having experienced DTs was weighted 2),* and the unacceptably high rates of false-positive identifications (possibly as high as 33%; see 50a). Another reviewer also raised the issue of increasing the cutoff score to lower the false-positive rate,[51] a third reviewer commented on the matter of high face validity by pointing out that "the MAST . . . appears to work wonders at detecting those who admit drinking a good deal" (52, p. 354), and a fourth reviewer commented that the MAST is

> most effective in identifying persons who define themselves as alcoholic . . . it is questionable whether the MAST, or any screening instruments that rely on the patient to provide valid information, will detect alcoholics denying their condition or those who have not fully confronted the implication of their behavior" (53, p. 1248).

In the 10 years since its initial publication, and especially during the 5 years since it was last reviewed, the MAST—in one or another of its three versions—has been used to screen for alcoholism among college students,[54] unemployed welfare recipients,[46,47] DWI offenders,[43,55–59] and patients in general hospitals,[60,61] VA hospitals,[62] and psychiatric hospitals[59,63] as well as being used with alcoholics and their spouses[64–66] and other groups.[36] Most of these studies have been empirical, descriptive, noncritical research projects whose data tend to support the practical utility of the MAST without questioning its validity and reliability, and as such they have greatly extended the popularity of that detection instrument. In the course of these studies and others, derivatives of the MAST, such as the brief MAST (BMAST,[67] comprising only ten items), the short MAST (SMAST,[68] composed of 13 items), and two self-administered versions (SAAST,[60–62] one containing 25 items and the other having 35 items) have been demonstrated to correlate fairly well with the original version of the test.

Questions had been raised regarding the validity of the MAST for use with women, because the original normative and standardization samples[49,68] had been composed almost entirely of men. One of the cardinal values of psychometrics asserts that the more one's sample deviates from the composition of the normative population, the greater the reduction of the test's validity, and certainly the MAST appears to have some traditionally male-oriented items (e.g., physical fights, trouble at work, job loss, arrests for drunken behavior and DWI). Selzer and his colleagues[66] have responded to that criticism by comparing the response patterns of 123 male and 80 female alcoholics and reporting that total scores did not significantly differentiate between the sexes. On seven of the items, however, men responded affirmatively much more often than women ($P < 0.01$), and the authors offered some alternative wording for two of the items which were not earlier suspected of being gender-specific.

* In 1980, however, some modifications in weighting were made, such that having experienced DTs is valued at 5 points, arrests for DWI or other drunk behaviors are weighted 2 points for each occurrence, etc. (See 71 for other modifications.)

The more general issue of the validity and reliability of self-report data was raised several years ago when it was suggested that

> under controlled conditions the MAST [could] be administered in the first person to the proband and in the third person to a spouse or family member (e.g., Has he gotten into fights when drinking?), using the coefficient of agreement as an index of validity/reliability (2, p. 281).

Morse and Swenson[69] found a 90% rate of agreement using the SAAST with a group of known alcoholics and their spouses, which speaks well for their version of the test. When McAuley and colleagues[64] administered the MAST to 75 psychiatric patients and one member of each patient's family, 39 patients and 29 family members identified the proband as an alcoholic. Patient and informant scores agreed in 76% of the cases (44% of whom were alcoholic) and disagreed in 24% of the cases (including 78% of patients who had identified themselves as alcoholic). In 60% of the cases where both the proband and the informant had identified the patient as alcoholic, the physician had also diagnosed alcoholism; physician diagnosis and proband score agreed 67% of the time, and physician and informant agreed in 80% of the cases.

The results of the latter study do not reflect as well on the MAST's validity as do those of Morse and Swenson,[69] primarily because of a consistent problem of false-positive identifications, which is much more likely to occur in populations where alcoholism has not already been diagnosed (e.g., Morse and Swenson's sample was drawn from an alcoholism-treatment unit). Thus, for example, when 76 adults on an inpatient psychiatric unit were administered the MAST, 29 patients (38%) scored more than 4 points, but only 17 of them had a primary or secondary diagnosis of alcoholism on their chart, indicating a possible false-positive rate of 59% (although the authors concluded that the diagnosing physicians were in error and the MAST was correct). In an interesting variation, those authors then substituted the word "drugs" for "alcohol" and readministered the MAST, and 29 patients scored more than four points. None of these patients had primary or secondary charted diagnoses of drug abuse, but it was later determined that 15 of the patients should have been so diagnosed, leaving a false-positive rate of 48%.

Unusually high rates of false-positive identifications may also have been a problem in a study conducted four years ago in New Zealand.[59] The author found a 0% false-negative rate among 100 hospitalized alcoholics and MAST scores of 5 or higher among 24% of 100 psychiatric inpatients without diagnoses of alcoholism, 82.9% of 70 DWI offenders, and 10.9% of social drinkers. To reduce the rate of false-positive identifications, the author "suggests that MAST scores in the range of 4 to 10 could be classified as 'indicative of problem drinking, but not necessarily alcoholic.' . . . This potential for discriminating alcoholics from other problem drinkers invites further investigation" (pp. 379–380), and indeed it does, as we found to be the case in our own studies (see below).

When a random sample of medical charts from a population of 1002

general patients at a large midwestern medical center[61] were checked for previously noted symptoms similar to those of the CRIT, over- and under-detection by the SAAST was found to be only a minor problem. But at a southern VA hospital, the problem was of a greater magnitude.[62] There, Page administered items from the MAST and SAAST to 147 males being admitted for the first time and later checked their medical records for clinicians' ratings of signs and symptoms of alcoholism. Clinicians identified 28 patients as "problem drinkers," but only 16 of them had scored in that range of the test, yielding a false-negative rate of 57%, although the false-positive rate was an acceptably low 8%. Current use of a revised 34-item SAAST (in which all items are weighted 1 point, and scores of 7–9 indicate "possible alcoholism," and scores of 10 or more "probable alcoholism") now in use at the Mayo Clinic and associated hospitals is described by Swenson and Hurt.[70] They find that the test "saves valuable professional time without compromising significant information" (p. 169), that it is an effective screening device, and that administering it to the spouse provides "the most valid source of objective data concerning the patient's drinking patterns" (p. 169).

This range of variation in user-reported rates of misclassification based on test results may be attributable to differences in both content and method of administration among the many studies of the MAST, SMAST, and SAAST. Skinner and Charalampous[57] investigated differences in alcoholism-detection rates by three alcoholism counselors when three sets of data were available from 30 probationers who had been arrested for DWI: a self-administered MAST whose results were not known to the counselors until later, a counselor-administered MAST, and the counselor's contextual interview. Subjects were to be classified as social drinkers, borderline, or problem drinkers, and λ was the statistic used to calculate the strength of association between compared outcomes.

When self-administered and counselor-administered MAST results were compared, there was a 30% disagreement rate ($\lambda = 0.53$), for which the authors found four explanatory circumstances. For counselor-administered MAST versus counselor's interview, λ was 0.47, and in 40% of the cases, the counselors overrode the MAST results, and three explanatory notions were invoked. When self-administered MAST and counselor's interview were compared, the outcome was worse: 47% of the participants were classified differently by counselors and test scores ($\lambda = 0.20$). This study is of great importance since, as the authors point out, it is

> directed [against] the idea that interpretive work either can or should be eliminated . . . [since] the MAST is not so much administered as it is negotiated. . . . Diagnostic classification is not so much identified as it is established. . . . The score on a screening test screens out the specific sense of the answers . . ." (57, p. 121).

Readers who are considering using the MAST should, therefore, carefully weigh the advantages and disadvantages of the time and cost savings of self-

administration against the wealth of significant and useful information available through interaction with patients/clients achieved by interview administration of the test.

Misclassification by the MAST was also a problem for us when we used it in conjunction with the CRIT and MODCRIT in our work with DWI offenders[43,44] and welfare recipients.[46,47] One group of 196 DWIs had a mean MAST score of 14.27 (±10.57), and the second group of 169 offenders had a mean of 14.34 (±10.49). In the first group, 89.3% of the subjects had scores of 5 or higher, whereas 88.8% of the second group scored at or above that cutoff point. When subjects in the second group were individually interviewed on our 35-item CRIT, correlation between the two measures ranged between 0.45 and 0.484 ($P < 0.001$), but only 55.1% to 62.1% of the sample could actually be classified as alcoholic according to NCA guidelines (depending on how one interprets the meaning of "several" in reference to minor and diagnostic-level-2 symptoms). Assuming the validity of the CRIT, MAST-identified false-positive classification rates varied between approximately 27% and 34%. With that possibility in mind, I would strongly support Brown's[59] recommendation of using scores of 4–10 as indicative of "probable alcoholics" and scores in excess of 10 as "positives."

With our two welfare samples ($N = 809$, MAST = 12.14 ± 14.25), we encountered similar problems of overidentification by the MAST when our MODCRIT and MODCRIT-II were viewed as the valid indicators of alcoholism. In our first sample ($N = 309$), the MAST clearly discriminated between alcoholics (that 36.57% of the sample with scores of 5 or higher on MAST and MODCRIT) and nonalcoholics (scores of less than 5 on both measures), since mean MAST scores were, respectively, 25.7 ± 14.2 and 6.25 ± 9.5 ($P < 0.0005$). For the entire sample, scores on the MAST and number of CRIT symptoms present were correlated at $r = 0.82$ ($P < 0.0001$). However, 53.6% of the participants had MAST scores of 5 or higher, although only 31.9% could be classified as alcoholic on the MODCRIT, yielding a potential false-positive rate of 21.7% but a very favorable 1.65% false-negative rate. Because of our concern that the MAST may unintentionally discriminate against the poor and unemployed (e.g., arrests are more likely to occur; the use of money to buy alcohol is more likely to be perceived as a problem and to provoke complaints from family members), we decided that a higher cutoff score was needed, and a reanalysis of our MAST data indicated that a cutoff score of 12 yielded a much more acceptable false-positive rate of 7.5% while raising the false-negative rate to only 6.5%.

In our second study ($N = 500$), we first applied a 5-point cutoff score on the MAST and found a 23.4% false-positive rate and a 4.2% false-negative rate when MODCRIT-II scores were considered valid. Raising the MAST cutoff to 12 points yielded 5.2% false positives and 12.4% false negatives, and although this is far from perfect, we consider these rates to be within the range of acceptability.

Continuous scores on the two instruments were again significantly cor-

related ($r = 0.655$, $P < 0.0001$), and it was determined by content analysis that there was a 72% overlap in item similarity. Analyses of individual MAST items using ϕ coefficients for dichotomous data (binary decision for alcoholic–nonalcoholic) indicated that the five most discriminating MAST items were previous help for drinking ($\phi = 0.63$, misclassification rate = 13.4%), previous arrests for drunken behavior ($\phi = 0.605$, misclassification rate = 14.5%), hospitalization because of drinking ($\phi = 0.59$, misclassification rate = 14.6%), neglect of obligations, family, or work for two or more consecutive days because of drinking ($\phi = 0.567$, misclassification rate = 15.4%), and having attended an AA meeting ($\phi = 0.564$, misclassification rate = 16.1%). Further research will be necessary, of course, to determine the extent to which these results can be generalized to other populations.

Several other studies reveal the heretofore unexamined potential of the MAST to go beyond the limitations of a detection instrument to become a true diagnostic instrument. When Zung and Charalampous[55] conducted an item analysis of the MAST results obtained from 200 DWI offenders, their data suggested that

> if alcohol dependency is a unitary characteristic, MAST items [that] reflect this characteristic should be endorsed more frequently with increases in the overall test score. Conversely, items [that] do not correlate highly with the overall score may be extraneous or *may imply more than one relevant dimension of alcohol dependence* (p. 130, emphasis added).

They identified two general aspects of MAST content, one dealing with specific, objective alcohol-related events and consequences (e.g., fighting, arrests) and the other involving self-evaluation (e.g., not being a normal drinker).

In a subsequent study, Zung[56] implied that there might exist several different and identifiable alcoholism syndromes which might be discerned by the MAST but which were occluded by consideration of only the global score, thereby limiting the potential value and meaning of the test. He consequently submitted to factor analysis the MAST results obtained from 1000 DWI offenders, resulting in the identification of four factors for the entire sample: help-seeking (use of rehabilitative services), discord (violation of law, belligerence, marital problems), alienation (unstable employment, loss of friends, symptoms of addiction), and denial (evaluation of normality of drinking and control over drinking, as identified earlier[55]).

For the alcoholics (that 54% of the sample with MAST scores of 5 or higher), six factors were identified: denial (as for the total sample), debilitation (help-seeking, neglect of responsibility, health problems), marital discord (complaints from, disagreements with, spouse), work problems (problems at work, job loss), help-seeking (other than hospitalization for drinking-related problems), and social discord (arrests, belligerence, loss of friends). Although there are some problems inherent in the nature of the test, Zung's work represents an innovative and imaginative use of a simple screening instrument whose value may have been underestimated. For one thing, the factors are

orthogonal; i.e., they are independent, and, therefore, alcoholiclike problems in one area of life are not predictive of problems in other areas. This characteristic permits the possibility of constructing an "alcoholism profile" whose components do not overlap, thus providing an opportunity for delineation and assessment of particular life problems for which specific treatment can then be prescribed. And one can still retain the speed and simplicity of administration and scoring that have made the MAST so popular.

Although somewhat speculative, Zung's initial work with the MAST represents a major advance for that instrument from the level of a pedestrian screening method to a multidimensional diagnostic technique with major implications for the referral, treatment, and assessment processes. One must keep in mind, of course, problems regarding possible flaws in the factor-analytic results as well as the issue of a cutoff score of 5 points permitting the inclusion of so many false-positive identifications in Zung's sample as to vitiate his assumptions. One must wonder how the statistically derived factors might change if the samples included only those persons scoring above 10 or 12 points as Brown[59] and I[43,44,46,47] have suggested. Indeed, Dr. Selzer himself has given this matter some consideration:

> there are difficulties, to the effect that the MAST may be too sensitive. One of the early complaints was that too many subjects were scoring in the alcoholic range, that is 5 or more points. . . . There are a number of possible explanations, . . . and I suspect all are partially applicable. The MAST is somewhat too sensitive and may misclassify a few social drinkers as alcoholics. Respondents do not clearly understand some questions and provide the wrong answers. There are more alcoholics in the adult male population than was previously realized (7, pp. 52–53).

He went on to point out that one must not forget that the MAST is a screening instrument. "However, with each point total above 5, the diagnosis becomes more of a certainty" (71, p. 53), and, therefore, I infer that both the method of administration and the cutoff scores are matters of legitimate concern.

Both of these concerns have been vigorously pursued in a host of recent research[72–76] (B. J. Zung, unpublished data, 1979) during the past several years. In one such study, Zung[72] rigorously applied sophisticated statistical analyses to assess the psychometric properties of the complete MAST and the 10- and 13-item brief versions.[67,68] Using the individual interview method of administration, he gathered data from two groups of DWI offenders and assessed the discriminative validity, internal consistency, predictive validity, and predictive efficiency of the individual items within the tests and the full test *per se*. He found that (1) discriminative validity of most of the items of the full test is highly significant (P values of <0.01 to <0.001), but as many as seven of the items (generally, those dealing with medical complications, hospitalization, and other extreme consequences of alcohol abuse) may not be relevant to a population of DWI offenders. Coefficients of internal consistency (the extent to which individual items are related to the entire test) for the 25-item

test were equally significant, with the exception, again, of those items dealing with alcohol-related illness and hospitalization, suggesting that the MAST is not unidimensional. The standard error of measurement for the full test yielded such a broad confidence interval that identifying a specific cutoff score for the alcoholic–nonalcoholic dichotomy will result in high rates of false-positive and false-negative identifications. (2) For the 10-item BMAST, only the question dealing with hospitalization failed to significantly discriminate between problem and nonproblem drinkers; scores on the brief and full tests were highly correlated, but the conventional cutoff score of 6 points yielded false-positive rates ranging from 0% to 50% and false-negative rates as high as 43%. Overall misclassification rates were 34% and 37% for the two groups, and obviously, the cutoff score for this version of the BMAST needs to be reexamined. (3) For the 13-question SMAST, again, only the hospitalization item was not significantly discriminative; coefficients of internal consistency were acceptably high, and the correlations between SMAST and MAST scores were positive and significant ($P < 0.001$), indicating that the former adequately represents and predicts the latter. However, when the conventional 3-point cutoff score was applied, false-positive rates varied from 2% to 43%, and false-negative rates ranged between 24% and 29%, with an overall classification error of 25% for the two groups of subjects combined.

Zung[72] concluded that, at least for DWI populations, the BMAST "performs somewhat better than chance, but is generally unimpressive in its overall 'hit rate' with the MAST as the criterion" (p. 852), and the SMAST fared little better: "75% of the [SMAST] classifications were compatible with the MAST classifications. Although substantial, this level of agreement, in my opinion, is not sufficient to justify [its use] in place of the MAST" (p. 854). He recommends either discontinuing the use of both brief versions or researching the validity of altered cutoff scores. Nevertheless, his overall conclusions supported his earlier impression of the multidimensional nature of the MAST, but his inferences became more specific, and he suggested that psychologically dependent alcohol abusers "can have alcohol-related problems in discrete areas of living while remaining relatively problem-free in other areas" (p. 857), and in samples dominated by such persons, the MAST would indeed appear to be multidimensional. On the other hand, where physically dependent alcohol abusers dominate the samples, a unidimensional appearance could be expected to emerge.

The specificity of such a multidimensional hypothesis was further studied by Skinner[77] who used a self-administered version of the original MAST with 418 male and female alcoholics and drug addicts and submitted the results to item analysis and factor analysis. Overall classification error consisted of only a 7% false-negative rate, and the item analysis revealed that only the question about liver pathology failed as a discriminator. The factor analysis identified a principal component—apparently a "general alcoholism" factor—which accounted for 41.3% of the total variance, suggesting that the MAST

is largely unidimensional when applied to a population of alcoholics and other drug abusers (cf. Zung's results with DWI offenders). Other factors were also identified: recognition of alcohol problem by self and others (similar to Zung's denial factor); legal, work, and social problems (a mixture of Zung's discord and alienation factors); help-seeking (nearly identical to Zung's factor of the same name); marital–family difficulties (similar to Zung's discord factor); and a one-item factor called liver pathology.

Thus, this study suggests that the MAST may not have great utility as a differential diagnostic technique. Skinner did note, however, that the MAST is misused as simply a detection instrument. For one thing,

> the use of an alcoholic–nonalcoholic diagnosis is an oversimplification, . . . [and] the use of a specific cutoff point for assigning an individual to an "alcoholic" versus "nonalcoholic" group is wasteful of information since reliable differentiations can be made within each group. . . . It is argued that the MAST is best interpreted as an instrument for ordering individuals along a continuum according to their degree of alcohol involvement (p. 843).

Thus, if not useful as a diagnostic tool, the MAST has at least proceeded from being a detection instrument to being an assessment device, as I had suggested in 1976.[2] Skinner also noted that for this purpose the MAST can be validly scored by summing the number of positive (alcoholic) responses rather than by using the designated weighting system. Furthermore, Skinner's report contains much useful information about the relationship of MAST scores to personality characteristics, sociodemographic variables, response style, and cognitive and intellectual performance, and readers are urged to fully appreciate the value of his study.

In a followup study, Zung and Ross[76] used individual interview administration of the 24-item MAST with 182 acutely disturbed psychiatric inpatients. Comparison of MAST detection rates with charted diagnoses indicated a 58% misclassification rate, almost entirely false positives. Factor-analytic statistical evaluations of the data from these patients yielded a "general alcoholic impairment" factor which accounted for 49% of the total variance, although three other minor and independent factors were also identified. On the whole, however, it would appear that for this population also, the MAST is most applicable as a unidimensional detection or assessment procedure.

Continuing this line of research, Zung[74] used a self-administered 25-item MAST with 87 male and 153 female psychiatric outpatients and found only 20% concurrence between detection of alcoholics by the test and those identified by clinical diagnosis. Two factor analyses both identified a dominant "general alcoholic impairment" factor which accounted for the largest proportions of the variance, but again, four minor factors also emerged: work problems, help-seeking, discord, and self-identification with alcoholism (earlier labeled denial). Thus, the apparently multidimensional nature of the MAST changes to one of primarily a unidimensional general alcoholism measure,

depending on the type of population it is applied to and the technique of factor analysis used. Zung is confident that the MAST can continue to be used with certainty as a detection technique and possibly even as an assessment procedure. The likelihood of the MAST evolving as a truly valid and reliable differential diagnostic tool is limited, according to Zung, although Skinner disagrees and has faulted Zung's work for its flawed factor analysis.

It is interesting to note that, *vis à vis* our earlier studies of welfare recipients, Zung[74] reported highly significant ($P < 0.001$) inverse correlations of total MAST scores, help-seeking, and discord with income. These data support my earlier concern that some of the MAST items may discriminate against the poor and unemployed. One must also consider that perhaps the construct of alcoholism on which the MAST is based may itself discriminate against such persons.

One may fairly summarize the state of the art regarding the MAST in the following manner. (1) It is indeed an inexpensive and rapid detection procedure of imperfect but reasonable validity and reliability and will probably remain in widespread use for years to come. (2) Although self-reports may be suspect, the MAST can compensate for that shortcoming by being administered to a spouse or other appropriate informant. (3) Classification errors may still be a significant problem, particularly regarding false-positive identifications and especially if subjects are of low socioeconomic status. (4) The MAST may possess the psychometric properties of a multidimensional diagnostic instrument, although the data are far from clear, and the nature of the population to which it is applied and the technique of factor analysis from which it is derived still occlude that issue. (5) The test may be useful as an assessment device suitable for ranking respondents along a continuum of severity of general alcoholic deterioration or involvement.

2.3. Mac Andrew Scale

Second only to the MAST in widespread use as an alcoholism detection instrument and object of research, the Mac Andrew Alcoholism Scale* (MAC) is a 49-item self-administered true–false questionnaire empirically derived from the Minnesota Multiphasic Personality Inventory (MMPI[78]) and has been publicly available for 15 years.[79] Of the eight alcohol and drug abuse scales (see 78 for details) derived from the MMPI, only the MAC has been so widely accepted and applied,[80–92] although occasionally rejected[93] as well.

In initial reviews of the MAC,[1,2] I was favorably impressed by the total absence of face validity, which rendered the scale virtually invulnerable to faking, the low misclassification rates (8.5% false negative, 10% false positive),

* Mac Andrew[88] points out that because two studies have shown that test scores of alcoholics are indistinguishable from the scores of nonalcoholic substance abusers,[94,95] he does not consider his scale to be solely or even primarily a detector of alcoholism, and hence, he uses the acronym MAC rather than ALC in reference to the test.

its ease of administration, and the notion that whatever it was in fact measuring (i.e., it is not a direct measure of alcoholism *per se*) appeared to remain stable over intervals of up to a year. At the same time, however, I noted the lack of validity data for women and other special populations, the unresolved controversy over where the cutoff score should be set (Mac Andrew recommended 24 points, but other researchers[2] sometimes achieved better discrimination with cutoff scores ranging from 23 to 27), the time required for administration (because the MAC at that time was always administered as part of the complete 566-item MMPI, 1½ hr were usually needed for completion), and other problems and disadvantages.

And how is the MAC viewed today? Still imperfect, to be sure, but some of the questions and issues that plagued the scale when it was 10 years old have been resolved in time for its 15th birthday. For one thing, the validity and reliability of the MAC for off-scale administration appears to have been reasonably established, and it may no longer be necessary to administer the full MMPI in order to obtain acceptable MAC scores, thereby reducing administration time by more than 90%. Approximately 4–5 weeks after self-administration of the off-scale MAC, Burg[80] gave the complete MMPI to 38 of the same 50 DWI offenders (34 men and four women). A test–retest reliability coefficient of 0.89 was found, and an obtained off-scale score of 24 was estimated to have a true-score value of 23.87 (± 2.56 at $P < 0.05$). Mac Andrew[88] himself administered the full MMPI and the independent (off-scale) MAC at 1 to 2-week intervals to alcoholic male inpatients at a VA hospital, male DWI offenders, and male college students and found test–retest correlations of 0.81, 0.73, and 0.80, respectively. These values are within the accepted range of reliability coefficients for MMPI test–retest correlations, even though MAC-scale scores were consistently lower than on-scale scores. Only the DWI offenders had significantly ($P < 0.05$) lower MAC scores (1.02 points) than MMPI MAC scores, but that difference was well within the range of average changes (1.33–1.49 points) for MMPI test–retest differences. Moreover, the misclassification rate of the MAC was only 13%, versus 11% for the MMPI-embedded scale. This loss of 2% in accuracy is nugatory in light of the enormous saving of time, and I encourage other researchers to follow Mac Andrew's and Burg's example.

Relatedly, it has been customary to consider invalid any MAC scores that were accompanied by MMPI F-scale scores of 16 or higher because of the atypical, unusual, or confused nature of response patterns associated with such F-scale elevations. It now appears likely, however, that that convention can be abandoned without loss of confidence in the validity of MAC scores. Apfeldorf and Hunley[81] and Mac Andrew[88] have both demonstrated no loss of discriminative power when the F-scale is eliminated from consideration, although Mac Andrew recommends retaining the MMPI L-scale (15 items) as an indicator of deliberate attempts to falsify, and he uses a cutoff score of 9 points as a basis for rejecting MAC-scale data. One can very easily, then, abstract the 49 MAC items and the 15 L-scale questions and type up a 64-

item self-administering true–false questionnaire which requires only 15 min for testing and scoring. We have, in fact, followed this procedure in our own studies of DWI offenders[43,44] and found low but significant ($P < 0.01$) correlations between MAC and CRIT and MAST, and our highest rate of detection agreement was between MAC and MAST (72%).

Issues regarding the validity, reliability, and applicability or utility of the MAC have to some degree been favorably resolved. Indeed, major critical reviews of alcoholism scales in general[51] and MMPI-derived alcoholism scales in particular[83,85] provide general agreement that the MAC can consistently differentiate alcoholic outpatients from nonalcoholic psychiatric outpatients, alcoholic and nonalcoholic DWI offenders, alcoholic inpatients and nonalcoholic psychiatric inpatients, alcoholics and nonalcoholic "normals," and can even predict which college students will eventually become alcoholic.

What has not been resolved, however, is the question of construct validity: what does the MAC measure? That is, granted that it identifies alcoholics, what is (are) the construct(s) underlying the scale? Mac Andrew's own factor-analytic study (in 2) revealed 13 factors, such as claims to be outgoing and socially competent, low sexual preoccupation, religiosity and guilt, and so on. High scores on the MAC have also been found among "uninhibited, sociable people who appear to use repression and religion in controlling rebellious, delinquent impulses" (83, p. 1545). It has also been shown (erroneously, according to Clopton[83]) that the MAC may be incapable of discriminating between alcoholics and criminals, and so perhaps it taps a "criminality" or "delinquency" dimension. And because it does not adequately differentiate alcoholics and heroin addicts, the MAC may be assessing a general characteristic of all addictions, substance abuses, and chemical dependencies.[94,95]

That this last observation is a substantive one can be inferred from the caution with which Mac Andrew has studiously avoided referring to his derivation as an alcoholism scale, instead consistently calling it only the MAC scale. It would seem, then, that the scale is gradually evolving into the MAC that the author may have initially intended, and his own recent work is certainly headed in that direction. In summarizing some of the current literature, Mac Andrew[87] noted that MAC scores of male alcoholics and male abusers of other substances were very similar, stable over time, usually unrelated to duration of history of abuse, unaffected by treatment, and predictive of future substance abuse. When he subsequently administered the MAC Scale to four groups of young (ages 16–22) male subjects (42 alcohol-related multiple offenders, 49 alcohol-related first offenders, 48 psychiatric outpatients without history of substance abuse, and 79 college students), the abusers were consistently differentiated from the psychiatric patients and students with an 82.1% accuracy rate. More importantly, however, the MAC scores of these subjects were virtually identical to those of corresponding groups of subjects in Mac Andrew's initial standardization and validation samples despite the age differences. Thus, the MAC may have additional

application as a predictor of later alcohol and/or other substance-abuse problems (although Mac Andrew encourages us to move beyond the confines of MMPI items to develop new and better predictive techniques).

Mac Andrew[89] later evaluated the outcomes of 24 earlier studies based on use of the MAC. One study had investigated 52 polydrug abusers and 48 heroin addicts, another project had involved 100 heroin addicts, and all of the others had focused on alcoholics. Two important conclusions emerged. The MAC's classificatory accuracy rate ranged between 80% and 100%, and the mean scores of alcoholics, polydrug abusers, and heroin addicts were indistinguishable. Mac Andrew then went back to his original standardization data, judiciously reanalyzed the complete MMPI response patterns for the four groups of true and false negatives and positives, and identified two characterological types of substance abusers. The typology appeared to remain consistent regardless of age, was equally discernible among alcoholic outpatients and nonalcoholic psychiatric outpatients, and became even more marked when false negatives and false positives were removed from the sample. The more prevalent of the two types, occurring in approximately 85% of the sample, is called *primary substance abusers*, which Mac Andrew assumes to be in the nature of a character disorder. The remaining 15% are labeled *secondary substance abusers*. Mac Andrew views his scale as tapping a construct referred to as sociopathy or "secondary psychopathy," and further descriptive information should be soon available.[96]

When Rathus, Fox, and Ortins[97] administered an abbreviated 20-item MAC to 1672 high school students and allowed them to respond anonymously, they also collected information on alcohol and other drug use (marijuana, cocaine, heroin, psychedelics, tranquilizers, amphetamines) and delinquent and criminal behaviors (theft with and without force, carrying a weapon, fighting with and without weapons, destruction of property, etc.). The MAC scale scores were positively and significantly (P values ranged from <0.05 to <0.001) related to 17 of the 20 behaviors, such that it

> is a significant indicator of behaviors that may seem as "thrill-seeking" or hedonistic among adolescents. . . . It is not uniquely sensitive to alcohol-related behaviors. It is sensitive to [possibly exaggerated] self-reports of soft drug abuse, hard drug abuse, theft, property destruction, and violence. . . . For suburban high school students the scale is clearly sensitive to a pattern of behavior of which drinking is only a part (pp. 582–583).

Clearly, more research is needed to elucidate this new aspect of Mac Andrew's perhaps misunderstood scale.

Meanwhile, Burke and Marcus[82] reported that 85% of alcoholics, 94% of polydrug abusers (alcohol plus one or more other drugs), and 63% of alcoholic schizophrenics were correctly identified by MAC scores of 24 or higher, for an overall "hit rate" of 83%. At the same time, 69% of drug abusers and 86% of drug-abusing schizophrenics were also correctly identified. Comparison of alcoholics' and drug abusers' mean MAC scores showed no significant dif-

ference, and the authors concluded that the scale seems to be measuring "a general addictive propensity," which is by now a familiar and not unexpected result.

And because the MAC is becoming more readily "believed to measure characteristics common to users of a wide variety of chemicals," (91, p. 906) it should not be surprising that Willis, Wehler, and Rush found that male alcoholics who smoke 20–50 cigarettes per day had significantly ($P < 0.01$) higher MAC scores than their nonsmoking counterparts. Another new and unconventional application of the MAC was reported by Friedrich and Loftsgard,[65] who noted that elevated test scores among the wives of alcoholics were significant predictors of suicide attempts, prior counseling, and drug use (primarily minor tranquilizers) for those women. Thus, as we by now might be able to infer, the MAC "seems to reflect acting-out in general, rather than alcoholism in particular" (p. 785).

Other studies of conventional applications of the MAC have proliferated, and further review would be redundant; suffice it to say that most of them are supportive of the value of the scale for identification of alcoholics. For pragmatic purposes of users of the MAC, however, one additional report must be mentioned. Regression analysis of full MMPI response patterns and MAC-scale scores in particular led Clopton and Klein[84] to recommend optimal cutoff scores of 25 for males and 27 for females (cf. Mac Andrew's recommendation of 24 for men) when attempting to discriminate between alcoholics and psychiatric patients.

By way of summarizing, recent research on the Mac Andrew Scale recommends it as a reasonably valid and reliable means of detecting alcoholism, and it may be particularly useful in settings requiring a quick, simple, self-administered technique with no face validity. The 49 items should be combined with the 15-item MMPI L-scale for added confidence regarding veracity of responding. Universally accepted cutoff points are not known, but useful recommendations are available. Because its content is irrelevant to any construct or theory of alcoholism, I would not recommend use of the MAC in isolation if any meaningful identification of alcohol abuse is required (e.g., disposition of a DWI offender's driving privileges, court testimony). Several studies strongly indicate that the MAC has matured beyond the point of being simply a detection tool, and its application to identification of substance abuse and chemical dependency in general is becoming more widely accepted. Its potential for tapping some more profound underlying constructs, such as acting-out tendencies, psychopathic or sociopathic character, and similar concepts, may soon be more fully explored.

2.4. Mortimer–Filkins Test

My particularly high regard for this test is largely attributable to the elegant, conscientious, and extensive manner in which it was standardized, tested for validity and reliability, corrected and revised, and adapted to suit

the needs of its users.[1,2,44,98,99] As described elsewhere in detail,[2,98] the Mortimer–Filkins Test is a 58-item forced-choice questionnaire and a 30-min structured interview designed to be used with ancillary information. It has excellent construct validity, has demonstrated classificatory accuracy as high as 98.5%, may be useful for differential assessment of areas of conflict or mental health problems (e.g., marital problems, depression), and permits the development of individualized treatment plans based on outcome. Initially developed specifically for identification of problem drinkers among DWI offenders, it has the flexibility and potential to be adapted to other uses. Published in 1971 and revised slightly in 1973 (cutoff scores and scoring formats were changed), this test has a solid 10-year history of useful application throughout the country.

It is, therefore, disappointing to report that neither Dr. R.G. Mortimer (personal communication, May 30, 1980) nor I could identify more than one instance of significant research during the past five years. Mushill and Struckman-Johnson[100] studied more than 9900 DWI offenders in Texas and South Dakota and convincingly established that for first-time offenders, the Mortimer–Filkins is an excellent predictor of the probability of future DWI arrests.

Dr. Mortimer has anecdotally reported that in the past decade tens of thousands of persons passing through state and local DWI and ASAP (Alcohol Safety Action Programs) units have been screened with the Mortimer–Filkins, and "the general feedback that we have received is that the test is operating satisfactorily" (99, p. 60). Some agencies have used the questionnaire without the interview in the interest of saving time, but to do so compromises the validity of the procedure and obviously sacrifices much information that is vital for differential treatment planning. Some minor problems have been reported in the scoring process, and Dr. Mortimer has provided a practical solution.[99]

I certainly join Dr. Mortimer in his position[99] that

> the test has potential applications other than in DWI programs. For example, there is little reason to believe that the test would not be found effective in an industrial environment. In addition, research should be done to determine how well the test can be used for the purpose of treatment planning and not just as a [detection] tool (p. 61).

3. Diagnosis

Advancing from the nominal and ordinal levels of data provided by detection and assessment techniques, one moves hopefully toward the point of applying science and technology to understanding the nature and causes of our patients' illnesses and problems and ultimately establishing logical bases for treatment, prognosis, and evaluation of the impact of our interventions. Seven years ago, I identified, described, and evaluated two technologically attractive vehicles for advancing toward our goals. Disappointingly little

movement has been made during that period, and the two brief sections that follow reflect the dearth of research in what I had believed were vital areas of concern.

3.1. Essential-Reactive Alcoholism Dimension

Rudie and McGaughran's[101] Essential-Reactive Alcoholism (ERA) interview schedule is 22 years old, and I am feeling like the only guest at the birthday party. Am I being unreasonably stubborn in attempting to uphold the virtues of their technique? Perhaps so, in which case I shall be brief.

Details are provided elsewhere,[2,101] so it is sufficient to describe the ERA as a structured-interview format comprising 55 scorable items and 14 "fillers" (which can, in practice, be eliminated), which a trained interviewer and cooperative patient can complete in 20–30 min. Eight specific subscales provide information regarding dimensions of personality behavior, alcohol and drug use, social interactions, and other variables hypothesized as being relevant to psychoanalytic constructs of the etiology of alcoholism. Basically, two alcoholisms are proposed (although the ERA describes a continuum rather than a dichotomy, such that the two syndromes are distinctly recognizable only toward the extremes of the distribution): The *essential* alcoholic bears a strong resemblance to the archetypal sociopath but appears to be similar to what one would call the primary alcoholic. Contrastingly, the *reactive* alcoholic is one who is relatively well adjusted in most life areas but becomes an alcohol abuser in response to some real or perceived crisis. The validity of this construct is still being debated[102–104] and thus far can not be unequivocally accepted or rejected.

Be that as it may, several studies (reviewed earlier in 2) have partially validated, clarified, and extended the ERA, and the 2-, 6-, and 12-month outcomes of the experimental-treatment programs established by Treffert *et al.*[105] have established the value of matching treatment characteristics to diagnostically determined patient characteristics. Since that flawed but valuable study was reported in 1974, however, no further ERA research has become publicly available as far as I know. However, results of our unpublished ERA-based study (G.R. Jacobson, T.G. Sternbach, A. Wallace, R. Brethauer, and B. Clark, unpublished data) indicate that deficits in the ability to establish and maintain long-term goals (represented in subscale 3 of the ERA as "persistent application to reality tasks") are related to multiple readmissions of detoxification, and such information may be relevant to development of special treatment plans. We have also conducted ERA interviews with 30 female alcoholic inpatients along with extensive data on several dimensions of personality, perceptual and cognitive functioning, self-concept, and related variables, by means of which we expect to elucidate the meaning of the essential-reactive construct (G.R. Jacobson, J. Riedel, and S. Ryba, unpublished data).

Thus far, we have determined that the wording and scoring of the ERA,

particularly in the subscales on economic and emotional dependency, are distinctly sexist, and we are in the process of developing some necessary modifications. In the meantime, it is hoped that other researchers will join us in our attempts to develop and exploit the full potential of this diagnostic method.

3.2. Alcohol Use Inventory

As it was represented as the *sine qua non* of multivariate differential diagnostic techniques when first reviewed,[1,2] I praised the Alcohol Use Inventory (AUI), congratulated the authors[106] for their standardization and validation techniques and sampling, skillful use of factor analysis, development and advocacy of a multiple-syndrome theory of alcoholism, and generally inferred that their tool could evolutionize the diagnosis and treatment of a variety of alcohol-abuse syndromes. Although mildly disappointed 7 years later, I remain optimistic.

The AUI is composed of 147 items in a forced-choice self-administered questionnaire. Scores can be plotted on a profile sheet to provide a graphic representation of the patient's syndrome. Three conceptually distinct domains—styles of alcohol use, negative consequences of drinking, and perceived benefits of alcohol use—are represented in 16 relatively narrow primary scales, five broader second-order dimensions, and a general alcoholism dimension. The authors have gone to great lengths to fully explicate the exact nature of the scales of the AUI and the way they might relate to specific treatment strategies. For example, elevated scores on scale 7—drink to change mood—suggests that

> The therapy process needs to help the patient to relieve anxiety and depression, to explore the feelings and experiences that precede the anxiety and depression, and learn ways to develop more productive behavior in dealing with stress. Methods such as relaxation therapy . . . might also be valuable for persons scoring high on scale 7" (106, p. 522).

In addition to their notion that "This individualized description of patients can lead to a more personalized approach to treatment planning" (p. 541), the authors describe some very useful research applications of the AUI, such as the determination of projected treatment costs over a 3-year period for particular profiles. To what extent has such potential been fulfilled during the past 7 years? A small but growing body of experimental and applied studies is being reported in the literature. For example, a group of Wisconsin researchers and clinicians[107] have developed a computerized system that allows patients to use an interactive terminal which administers, scores, plots, and stores the AUI for diagnostic and evaluation purposes. While Skinner[77] was performing his factor-analytic study of the MAST, he noted a correlation

of 0.83 between that test and the general alcoholism dimension of the AUI, which Zung[74] also referred to in support of his position.

The general alcoholism dimension was also used by Silberstein and Parsons[108] to demonstrate the relationship of severity of alcoholism problems to neuropsychological impairment among women. Chalmers and his colleagues[109] administered the AUI to a group of problem drinkers, then substituted the words "smoke" and "overeat" for each reference to drinking and administered the respective scales to groups of self-defined problem smokers and overeaters who had presented themselves for treatment. Comparisons of scores and profiles indicate that all three groups are similar in their psychosocial motivation for abuse, overeaters and drinkers are similar in style of consumption, and smokers and drinkers similarly perceive social and mental benefits associated with their excesses. Other similarities and differences are discussed, and the authors speculate on theories of substance abuse and implications of their data for differential treatment of the three classes of problems.

We[110] have recently completed a study in which we found scores of AUI scale 4 (obsessive–compulsive drinking) to be positively and significantly (P values of < 0.05 and < 0.01 for two groups) correlated with number of outpatient sessions and significantly ($P < 0.05$) predictive of treatment-dropout status. We also found AUI scale 4 to be positively and significantly ($P < 0.01$ for three of our four groups) correlated with the psychasthenia scale of the MMPI (a putative measure of obsessive–compulsive personality traits). Currently, we are using off-scale (independent of the context of the test) administration of the AUI scale 4 to predict inpatient-treatment dropouts (G.R. Jacobson and B. Michalec, unpublished data).

It is thus encouraging to note that the AUI is stimulating at least this modicum of experimental and applied research, since there are still many aspects of the tool that remain speculative and largely untested. Research is needed to test the hypotheses underlying the multivariate syndromes construct of alcoholism, to develop and test hypotheses regarding differential treatment outcomes for various typologies or profiles, and so on. We have just begun to scratch the surface of the marvelously complex and varied opportunities for clinical research becoming available to us in the AUI.

4. Epilogue

This brings us up to date, then, on the most relevant research of the past 7 years pertaining to the continuing development and application of those four detection and assessment techniques, and the two diagnostic tools, that appeared to me to have the greatest influence in the field of alcoholism. I say influence in the sense of actual and potential impact, in terms of current utility and future value, in terms of capacity for generating meaningful and inter-

esting research, in terms of provoking thoughtful and productive controversy. I have, of course, exercised a good deal of selectivity and bias in my choice of representative tools and techniques, in weighing the relative merits of current research for inclusion and exclusion, in deciding how much detail to reveal and conceal, and in determining which of the inevitable flaws to emphasize and which to disregard.

Additional materials I would have included, had I had the luxury of more space and time, are briefly summarized below in the hope that other researchers will follow up on these potentially valuable leads.

1. Several of the biochemical detection techniques, including the plasma α-amino-*n*-butyric and acid-to-leucine ratio, serum γ-glutamyl transpeptidase, and mean corpuscular volume, because they are new, controversial, quick, can not be faked, and can be used for identifying alcoholics and for monitoring alcohol consumption.[14,111–113]
2. The Adolescent Alcohol Involvement Scale, because it is the only technique I know of to have been developed specifically for this special population, it appears to be well constructed and standardized; it has successfully gone through several necessary steps for assessing its validity and reliability, its factor analysis was reasonable, the authors' stated goals and purposes seem rational, and data collected from nearly 4000 adolescents are being analyzed and should make a clear statement about the validity, reliability, and meaningfulness of the scale.[114,115]
3. The Missouri Alcohol Severity Scale of the Alcohol History Form, because I may have underestimated it 7 years ago. Some new data are becoming available that indicate that the scale may also be sensitive to treatment-induced changes, and results are now accessible from nearly 6000 consecutive admissions to public facilities.[33,116]
4. *The Diagnostic and Statistical Manual* (Third edition) of the American Psychiatric Association and its two most relevant precursors reduces ambiguity and confusion, encourages uniformity of nomenclature, is new and controversial, insurance companies respond to it, and, like it or not, it is becoming a fact of life.[21,117]

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15

Types and Phases of Alcohol Dependence Illness

Wallace Mandell

Abstract. The published data based on retrospective recall support the position that there is a characteristic developmental sequence of clinical signs and symptoms in alcohol dependence illness. These symptoms cluster in three sequential temporal phases: psychological dependence, physiological dependence, and neurological disorganization. Individuals may discontinue alcohol consumption during any phase and are not doomed to complete the sequence. Attempts to develop subtypes of alcohol-dependent individuals are generally based on inadequate samples from nonrepresentative treatment centers, many of whose patients are not alcohol dependent. The proposed essential-reactive, primary-affective, and life-style typologies have very weak support. Studies repeatedly confirm sociopathic, depressed, and anxious subgroups of alcohol dependent patients. However, such classifications have not been able to encompass more than one-half of the treatment populations studied. These concurrent symptom types are more prevalent in publicly operated treatment facilities and are predictive of recovery.

1. Definition of Alcoholism

There are four current definitions of alcoholism. The first and most commonly accepted definition is provided by the World Health Organization[1]:

> Alcoholics are those excessive drinkers whose dependence upon alcohol has attained such a degree that it shows a noticeable mental disturbance or an interference with their bodily and mental health, their interpersonal relations, and their smooth social and economic functioning; or [those] who show the prodromal signs of such developments.

Within this definition, the expert committee on health distinguished two subtypes, "alcohol addicts" and habitual excessive "symptomatic drinkers," the latter group composed of nonaddicted individuals who produce social, economic, or health costs because of their drinking patterns.

Several significant authorities have recently taken a second position that alcoholism is a unitary disease. Mann,[2] Gitlow,[3] Madsen,[4] and Johnson[5] all

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hold that there is a single disease entity, alcoholism, identifiable by its history, symptoms, and signs, which form a recognizable pattern. Although obscure on this point, the criteria of the National Council on Alcoholism[6] for the diagnosis of alcoholism can be interpreted as implying that there is a single disease entity.

Pattison, Sobell, and Sobell[7] have taken a third position:

> There is no single entity which can be defined as alcoholism. Alcoholism is not a thing but a collection of various symptoms and behaviors that collectively comprise different types of syndromes. There appear to be broad arrays of life situations dependent upon psychological disposition, past learning, sociocultural influences, and physiological states that lead to inappropriate use of alcohol, which in turn leads to deleterious physical, psychological and social consequences for the individual. . . . [Further,] there is no clear dichotomy between either alcoholics and non-alcoholics or between prealcoholics and non-prealcoholics, even though individuals may have a differing susceptability to both the use of alcohol and the development of alcohol problems as the result of genetic, physiological, psychological, and sociocultural factors.

A fourth very popular position was articulated by Jellinek in his 1960 book,[8] when he defined five species of alcoholism: *alpha,* the continued use of alcohol to relieve bodily or emotional pain contrary to social rules about occasion and amount of consumption; *beta,* heavy drinking producing ill health and social problems without addiction; *gamma,* loss of control over the amount consumed with characteristic increased tissue tolerance to alcohol, adaptive cell metabolism, withdrawal symptoms, and craving; *delta,* a variation of *gamma* in which the patient does not lose control of the amount consumed on an occasion but rather cannot abstain from using alcohol continuously; *epsilon,* a periodic version of *gamma.*

Jellinek was also specific (9, p. 674) in declaring that the disease concept attaches only to alcohol addicts who, after several years of excessive drinking, display the phenomenon of "loss of control" over alcohol intake. Loss of control was later defined as the inability to take only one or two drinks of alcohol.[8] The other group, habitual excessive drinkers, were considered as displaying symptoms of psychological or social difficulties that are temporarily relieved by excessive drinking. According to Jellinek,

> the "loss of control" is a disease condition *per se* which results from a process that superimposes itself upon those abnormal psychological conditions of which excessive drinking is a symptom. Not all excessive drinkers, even after many years of habitual drinking, develop "loss of control."

A fifth position was taken by the Committee on Alcohol-Related Disabilities of the World Health Organization published in 1977.[10] The Committee endorsed the term "alcohol dependence syndrome" to clarify their position that alcohol addiction is one of a family of dependence disorders. The term "syndrome" emphasizes the position that a number of phenomena tend to

cluster with sufficient frequency to constitute a recognizable occurrence. All elements will not always be present with the same magnitude or relative magnitudes, nor will all elements invariably be present.

On the basis of the earlier work of Edwards and Gross,[11] the Committee proposed the following as the essential elements of the syndrome: (1) a narrowing (regularity) in the repertoire of drinking behavior; (2) salience of drink-seeking behavior; (3) increased tolerance to alcohol; (4) repeated withdrawal symptoms; (5) repeated relief or avoidance of withdrawal symptoms by further drinking; (6) subjective awareness of a compulsion to drink; (7) reinstatement of the syndrome after abstinence.

In Table I, it can be seen that the constructs proposed by the Committee can be considered as labels for some of the specific signs reported by Jellinek as characteristic of the disease alcoholism.[8]

Table I. Comparison of Jellinek Phases and Symptoms of Alcohol Addiction and the 1977 WHO Alcohol Dependence Constructs

Jellinek[8]	WHO[10]
0. Prealcoholic phase	
1. Increased tolerance	Increased tolerance
2. Response to alcohol as a needed drug	Narrowing of repertoire of drinking behavior
I. Prodromal phase	
1. Blackouts (palimpsests)	
2. Surreptitious drinking	
3. Preoccupation with alcohol	Subjective awareness
4. Avid drinking	Narrowing of repertoire
5. Guilt feelings about drinking	Subjective awareness
6. Avoid reference to alcohol	Subjective awareness
7. Frequent palimpsests	
II. Crucial phase	
8. Loss of control	Narrowing of repertoire
9. Rationalize drinking behavior	
10. Social pressures	
11. Grandiose behavior	Narrowing of repertoire
12. Marked aggressive behavior	
13. Persistent remorse	Subjective awareness
14. Periods of total abstinence	Subjective awareness
15. Changing the pattern of drinking	Subjective awareness
16. Drop friends	Subjective awareness of compulsion to drink
17. Quit job	
18. Behavior becomes alcohol centered	Narrowing of repertoire
19. Loss of outside interests	Narrowing of repertoire
20. Reinterpretation of interpersonal relations	
21. Marked self pity	Subjective awareness

(Continued)

Table I. *(Continued)*

Jellinek[8]	WHO[10]
22. Geographic escape	Subjective awareness
23. Change in family habits	
24. Unreasonable resentments	
25. Protect supply	Subjective awareness
26. Neglect of proper nutrition	
27. First hospitalization	
28. Decreased sexual desire	
29. Alcoholic jealousy	
30. Regular morning drinking	Avoidance of withdrawal by drinking
III. Chronic phase	
31. Prolonged intoxications	Reinstatement of the syndrome after abstinence
32. Ethical deterioration	
33. Impairment of thinking	
34. Alcoholic psychosis	
35. Drink with persons below social level	
36. Recourse to "technical products"	Avoidance of withdrawal by drink
37. Loss of tolerance	
38. Indefinable fears	
39. Persistent tremors	
40. Psychomotor disinhibition	
41. Obsessive drinking	Narrowing of repertoire
42. Vague religious desires	
43. Rationalization system fails	

Edwards and Gross[11] imply in their clinical descriptions the position that Jellinek states explicitly (9, p. 676). The "phases" and the sequences of symptoms within the phases are characteristic of the great majority of alcohol addicts and represent what may be called the average trend.

This brief review has illustrated the wide diversity of positions as to the nature of alcoholism, ranging from a unitary illness model to a view of it as a collection of syndromes. A major source of difficulty seems to be that most researchers in the alcoholism field do not organize their data around specific hypotheses that derive from these fundamental conceptions. As a result, the data relevant to specific hypotheses are scattered.

The present chapter brings together the evidence relevant to two fundamental conceptions of alcoholism as an illness. The first question addressed is whether the proposed signs of alcohol dependence cluster and appear in a developmental sequence. The second question is whether there are, within

the group of people treated as alcoholism patients, subtypes who have common symptoms that are relevant to the course and outcome of alcohol dependence illness.

There have been over 125 years of effort invested in attempting to describe types of alcoholics and alcoholism.[12] Most clinician–researchers have agreed that there are common signs and symptoms observable among alcoholics. They have also agreed that some of the symptoms appear in a developmental sequence over a course of years. However, because all of the symptoms do not appear regularly in all alcoholic patients, it did not seem possible to chart the course of the illness. This difficulty made it unclear whether alcoholism was a single illness. If alcoholism showed a definite time sequence in ordering of signs and symptoms, it would be possible to investigate etiologic relationships by demonstrating conditional probabilities, e.g., that symptoms occur conditioned on the appearance of earlier symptoms. This area of research is critical in establishing the course of the illness and exposing potential points and modalities of intervention.

2. Methods

2.1. Methodological Issues in Studies of Phases of Alcohol Dependence

The reader should be alert to major methodological weaknesses that characterize the studies to be discussed. Perhaps most critical is the peculiar nature of the samples studied. The subjects are almost all individuals who have come voluntarily to a facility or organization that treats alcohol problems. They are thus self-defined to some extent as alcoholics. Often, it is not possible to determine from the criteria for entry into the study if they are indeed addicts or dependent rather than simply having alcohol-related problems for which they seek relief in a hospital. They have, at least to some extent, accepted a cultural view of themselves as being disabled and requiring help in order to overcome their disability. Many have failed to achieve or maintain desirable social roles and status. They have also picked up culturally influenced stereotypes of alcohol effects and alcoholics. It is to be expected that their recollection of events in their life history will tend to incorporate these cultural images.

Problems of recall may be considerable as subjects are asked to report on events that have occurred as much as 30 years earlier. The problem of recall is intensified by the fact that data may be obtained a few days after detoxification. There is now substantial evidence that there is a high rate of at least temporary memory deficit for 30 or more days after detoxification.

In all but one study, the questions are presented in a fixed order. This may influence the order in which symptoms are remembered by subjects.

The wording of the questions is very influential in determining the variation in response. Questions may not be specific as to whether the first

experience of a phenomenon, its frequent occurrence, or regular occurrence is being requested. In addition, terms such as blackout and loss of control require definition and illustration for many subjects. These definitions may influence whether the subjects recognize the phenomenon. Many alcoholics have been shown to be influenced by environmental cues, external loci of control. Several studies have used group questioning procedures. This technique may introduce a suggestion effect. Goodwin *et al.*[13] found that some subjects misinterpreted questions even when they were presented by interviewers. In fact, they describe "blackouts" as an elusive phenomenon, with patients changing their mind after hearing descriptions of others' experiences.

There is also a statistical problem in organizing the data. The usual procedure, following Jellinek, is to average the reported ages of all subjects who experienced a phenomenon. Some items have only a small proportion of subjects reporting that item. The average, based on this small sample, is then used as the equivalent of an average based on the total sample. In addition, the variance or standard deviation of ages for each phenomenon are not reported. Actually, a phenomenon may occur over a time period rather than as a single event, so that a recalled age really lies within a range. Very small differences in average age, i.e., 1 or 2 months, are used to order events. Only if samples studied were very large would this be reasonable. In fact, most studies are of very small samples. Thus, the common interpretations of ordering in several studies constitute an overinterpretation of data.

Finally, many phenomena associated with alcohol dependence may be general to the population. Failure to use control groups leaves this issue unresolved.

2.2. Developmental Sequence of Alcohol Dependence Symptoms

In 1946, E. M. Jellinek applied a statistical method to information provided by 98 members of Alcoholics Anonymous.[13a] Each subject was asked to record the age at which he had experienced each of 36 symptoms. Jellinek then calculated the average age for each symptom and placed them in chronological order on this basis. Some additional symptoms were later added by Jellinek.[9] The 43 symptoms in the final list were presented as describing the markers of the course of the disease.[9]

Jellinek was specific in noting that not all alcoholics experienced all of these 43 symptoms, nor did they always occur in the same order. Jellinek's purpose was to chart the course of alcohol addiction in contrast to symptoms that might be associated more frequently with habitual excessive use of alcohol. The "phases" and the sequences of symptoms within the phases are characteristic, however, of the great majority of alcohol addicts and represent what he called the average trend. Jellinek believed that the phases varied in duration according to individual characteristics and environmental factors. Jellinek described a prodromal phase marked by alcoholic palimpsests, or amnesia, often called "blackouts" at low levels of alcohol, and two phases of

addiction: the first, called crucial, marked by loss of control of the quantity consumed; the second, called chronic, marked by prolonged intoxication lasting several days.

Trice and Wahl suggested that Jellinek's serialization of the symptoms might mask significant clustering of groups of symptoms and that the typical sequence, based on averages, might not be typical.[14] To examine this, they obtained recalled dates of onset of 14 symptoms selected to represent early, middle, and late stages. The 252 subjects included 133 nonaffiliates of Alcoholics Anonymous from the Mendota State Hospital and 119 affiliates from several Alcoholics Anonymous groups in Wisconsin. The AA affiliates included twice as many white collar workers, earning one-third more income, who were more than twice as likely to be married (70% vs. 30%), and who had some college education (25% vs. 13%). Alcoholics Anonymous members also reported having experienced more symptoms.

The average age at which each symptom occurred in each population and in the 1946 Jellinek study were compared. The Wisconsin group had each of their symptoms appear 4 to 6 years later than Jellinek's AA group. However, there is almost a perfect rank order correlation between the average age of appearance of each of the symptoms in the Wisconsin group and Jellinek's 1946 AA group. The only variation was a reversal in the Wisconsin AA group between appearance of protecting supply of alcohol and first convulsions.

For each case individually, each of the 14 symptoms was examined to determine if it appeared earlier, the same year, or later than every other symptom. There was evident clustering of symptoms as follows: (1) first daytime intoxication, prolonged bouts, and morning drinking; (2) convulsions, tremors, and protecting supply; (3) getting drunk on less and loss of control; (4) loss of control and daytime bouts.

Glatt, as part of a larger inquiry,[15] obtained information about 18 symptoms included in the Jellinek questionnaire. Drinking histories were obtained from 192 male patients at Warlingham Park Hospital in England. The subjects were described as 71% middle class, 42% married, with an average age of 44.7 years.

Glatt calculated average age of occurrence for each symptom. The ages reported were strikingly similar to the Wisconsin data, the average age of each symptom occurring 4 to 6 years later than in the Jellinek study. There were five items that occurred at different points from the Jellinek sequence. "Loss of control" occurred after "sneaking drinks." Other items out of sequence were "friends walking out," "early morning drinking," "alibi for excess," and "periods of abstinence." The average ages of occurrence of these symptoms are within an 18-month interval. Considering the 6.9 year standard deviation for each of these average ages reported by Jellinek[8] and by Glatt,[15] the stability of sequence is impressive.

Glatt noted that part of the variability in his data was produced by a small group of men over 50 who were late starters. These men went through life as moderate drinkers and then became excessive drinkers as a result of

severe emotional stress such as loss of wife. For comparison purposes, Glatt obtained information from 80 male middle-class moderate drinkers, mean age 40 years, a majority of whom were professional workers in mental health facilities. Decrease in tolerance was reported by 16.3%, amnesia by 10%, needing to drink more to get the same effect by 8.8%, early morning drinking by 7.5%, and solitary drinking by 3.8%. This finding supports Jellinek's observation that some symptoms of alcohol dependence appear in nonhospitalized populations. Some of these individuals may be undiagnosed cases of alcohol dependence. On the other hand, some symptomatic behavior may not be unique to alcohol-dependent individuals.

Glatt also reported that 77 English female alcoholics experienced the same symptoms and stages as their male counterparts, but 5 to 7 years later in life.

Goodwin, Crane, and Guze[13] examined 120 randomly selected patients at the Malcolm Bliss Mental Health Center and The St. Louis Detoxification Center. Patients with brain syndromes or psychiatric disorders other than alcoholism were excluded. Criteria for the diagnosis of alcoholism included having symptoms in at least three out of the following five groups: (1) tremors, delerium tremens, cirrhosis, impotence, blackout, binges; (2) daily drinking, 54 oz of whiskey consumption per week, evasiveness about amount; (3) unable to stop, drinking deliberately limited to certain circumstances, drinking before breakfast; (4) fighting associated with drinking; (5) felt drinking too much, family objected, others object, lost friends, felt guilty.

The sample was 85% male, 71% white, mean age 44, 22% still married; two-thirds had not completed high school, and 75% belonged to the lowest social classes, IV and V on the Hollingshead scale.

Twelve symptoms of alcoholism were placed in order by mean age of onset. The only symptom that varied from the Jellinek order was blackouts which had occurred in 64% of the subjects at the average age of 35 compared to Jellinek's reported age of 30.2 years.

Goodwin *et al.*[13] suggest that the difference may have resulted from differences in populations that were studied, since 43% of their sample had never belonged to AA, whereas 100% of Jellinek's sample had been AA members. It seems more likely that the discrepancy was caused by a difference in the method of eliciting information, i.e., questionnaire versus a group interview.

An attempt to replicate the Goodwin *et al.* study was carried out by Curlee,[16] who examined 100 patients voluntarily hospitalized in the alcoholism treatment unit at the Indianapolis VA Hospital. Using the Goodwin *et al.* method, interviews were conducted with groups of three to seven patients by an alcoholism counselor who explained the meaning of the terms "blackout" and "loss of control" (defined as inability to stop after one or two drinks). Each patient indicated the age at which the symptom was first experienced. Subjects were male, 89% white, mean age 45; 39% were divorced or separated; 55% had completed or gone beyond high school. The definition of "blackout" provided to the St. Louis patients was "amnesia for any part of a drinking

episode and the history of the patient." Of the 32 subjects who had experienced delerium tremens, half had experienced DTs before or during the same year as their first blackout. Almost half of the group had been hospitalized prior to experiencing a blackout, and 49% had experienced head injuries. The average age at which symptoms appeared corresponds closely to those reported by Jellinek and Goodwin.

Park[17] used the drinking history, gathered by questionnaire, of 806 Finnish alcoholics drawn from Alcoholics Anonymous clinics and alcoholism work homes throughout Finland. Of these, 684 males had experienced "loss of control," Jellinek's critical marker of addiction. Further analyses were based on this latter sample. Twenty-eight of Jellinek's 43 descriptive items from the three phases were selected as unambiguous and appropriate to the Finnish culture. Park examined the probability of a given experience, *A*, following another experience, *B*. A probability of 1.0 means *A* is highly likely to follow *B*. A probability of less than 0.50 means that *B* is more likely to follow *A*. The expected Jellinek order of symptoms occurred in 78% of the 228 comparisons. With reinterpretation of some relevant items, there was a slight improvement so that using the Jellinek sequencing would be incorrect in only 19% of comparisons between any two items. Chance ordering would produce a rate of 50% incorrect decisions. This 2½-fold improvement over chance did not inspire Park with confidence. He set about to empirically reorder the sequence of symptoms based on the Finnish sample to improve ability to predict the sequence of symptoms.

It became clear to Park that the first occurrence of certain items such as tremors should be replaced by the age of frequent occurrence of the item. After this change, the empirically derived reordering supports the place of the three critical markers for phases—blackouts, loss of control, and benders—but redistributes many other events into different phases than those that Jellinek found. In the empirical ordering, only three items reversed in 281 relevant probabilities of which phase or within-phase sequence they should occur.

Park concluded that Jellinek's symptoms within phases do not correspond with the Finnish data, and perhaps not to other samples of American alcoholics, without refinement. However, the empirical ordering of phases and symptoms within phases by Park is remarkably exact and theoretically reproducible. This suggests that developmental experiences in alcoholism are indeed serially ordered. Kirivanta,[18] as reported by Park,[17] was able to create a Thurstone paired comparison scale for 13 of the 34 items taken from this data set, confirming that another statistical method also shows serial ordering of experiences.

Park believed that a strong order of occurrence could be observed, but for only a small number of characteristics. He believed that this ordering would be even clearer when alcoholics were subdivided into more homogeneous groups. Park's findings support the Jellinek hypothesis. However, Park's study needs replication on an American sample.

Park and Whitehead,[19] in 1973, examined data from 148 male alcoholics in Massachusetts of whom 67 were prison inmates, 51 were outpatients of three alcoholism clinics, 16 were patients in a VA hospital, and 14 were residents of a halfway house. The American alcoholics filled out a questionnaire about 63 experiences associated with alcoholism using Jellinek's phrasing. The mean age of the subjects was 43.7 years, and 77% of the subjects were in socioeconomic classes IV and V.

Using the method previously reported by Park, the responses were ordered in such a way as to minimize inconsistencies in chronological ordering. This order was compared with Jellinek's ordering of 25 responses of 98 Alcoholics Anonymous members. A substantial Spearman rank order correlation (ρ) was computed as 0.74 between the two lists. A correlation was also computed for ages of frequent occurrence of symptoms from the Finnish data.[17] Jellinek's reported sequence of symptoms yield a correlation of 0.64. A rank order correlation was also calculated for a longer list of first and frequent occurrence of symptoms using the data from the 148 Massachusetts alcoholics and 806 Finnish alcoholics. The ρ correlation between the two orderings was 0.70.

Park and Whitehead concluded that there is a main sequential ordering in the development of alcoholic experiences which is repeated in two culturally different settings and is also in conformity with Jellinek's theoretical formulation. They suggest that there is indeed an invariant order among certain crucial experiences of alcoholics, although a wide variation exists among others.[19]

In an attempt to determine which symptoms were more vulnerable to cultural influence, a principal factor analysis was performed for the Finnish data. The first four factors extracted from the Finnish data set accounted for 63% of the communalities and 43.5% of the explained variance. These dimensions were subjected to a varimax rotation to clarify their structure and are identified as economic problems, family problems, social problems, and core symptoms. The core symptoms included sneak drinking, anticipatory drinking, physical craving, tremors, fear of dependence, and greater amounts of alcohol needed to produce effect. Other familiar items, including benders, blackouts, and morning drinking, would have been part of this dimension except for a technical restriction that prevented their inclusion in the analysis. This finding supports the concept that there is a core of physiological signs and symptoms of alcohol dependence that varies independently (orthogonally) from the appearance of economic, social, and family problems. The factor analysis was repeated for the Massachusetts sample, and the same factor structure was found, corroborating the initial analysis.

Park and Whitehead ordered the developmental sequence of symptoms within each of the four dimensions. This could be accomplished in each sample. The Spearman ρ rank order correlation between dimensions is almost unity in the American and Finnish samples except for economic problems dimension where it was 0.83. These correlations greatly exceed the correlation

obtained on the total list of 31 items which was 0.55. Using an ingenious empirical method of estimating the probability of obtaining these correlations, Park and Whitehead were able to determine that the observed correlations could not be attributed to chance. The finding that the subjects go through the experiences on each dimension in the same order does not mean they do so at the same pace. The time between symptoms is yet to be determined. Park and Whitehead were not able to establish an order in which the dimensions precede each other.[19]

Continuing the search for a stable order of symptoms, Orford and Hawker[20] reported a study of 59 consecutive admissions to a male London halfway house. Using a standardized interview, they obtained information about age of first occurrence of symptoms. A statistical test based on the trinomial theorem, which takes into account cases in which both events occur in the same year, was used to determine if the sequence of events is predictable. The test examines each item of higher mean reported age of first occurrence with the succeeding item of lower mean age of occurrence to determine whether the sequence is statistically significant.

Orford and Hawker found "drinking became a problem" and "loss of control" to be reliable indicators of a first stage of alcohol dependence. "Tremor," "morning drinking," and "amnesia" were indicators of a middle stage. A third stage was indicated by a cluster of symptoms including visual hallucinations, auditory hallucination, delerium tremens, and hospitalization. A still later stage was marked by attendance at Alcoholics Anonymous meetings and crude spirit drinking.

Orford and Hawker hypothesized that the physical dependence cluster followed a psychological dependence cluster by 3 to 4 years in their sample. "Loss of control" was associated with the psychological cluster in terms of mean age of occurrence and most frequently occurred before the physical dependence cluster. However, many individuals reported this item as occurring in the same year. This suggested that the term "loss of control" is used by some patients to refer to psychological dependence and by others to refer to physical sensations related to withdrawal. The third cluster contained elements of central nervous system damage or psychosis which occurred in slightly less than one-half of the Orford–Hawker sample.

A different approach to examining sequence of symptoms is found in the work of Cahalan and Clark.[21] Cahalan and Clark report on changes in problem drinking over a 4-year span using data collected on the first wave by interview and on the second wave by mail questionnaire or interview. Subjects were initially selected from a random probability sample of white males aged 21 through 59 living in San Francisco. Approximately 80% of the sample (N = 786) were interviewed on the first wave. There was also an 80% completion rate for the second wave sample 4 years later, yielding a final N of 615. The greatest loss in the second interview seemed to be among the unmarried and manual workers. The authors note that it is possible that those not reinterviewed may have developed alcohol-related problems, but they do not believe

this to be the case. Of the 69 individuals who reported at least one item of loss of control at time 1, 73% reported also having other drinking problems. Of those respondents who did not report loss of control, only 20% reported a drinking problem. On the other hand, of 160 respondents who reported at least one minimum-severity drinking problem, 31% also had "loss of control" compared to 4% of those who did not report having a problem. This difference is statistically significant. This can be interpreted as supporting the position that "loss of control" is a factor that is associated with an increased risk factor of 8 for having another problem. Clark and Cahalan correctly point out that many individuals (110 subjects) who have a current drinking problem would be conceptually marginal to the scheme. However, no author has ever proposed that all alcohol-related problems are related to or stem from loss of control.

A comparison of data from the two waves of the study shows that 4 years later, many symptoms disappear. However, individuals who have some problem at time 1 tend to have a problem, albeit a different one, at time 2. Thus, the continuity of specific problems is low, but the continued involvement in an alcohol problem is the rule. In fact, 76% of those who have symptomatic drinking and 78% of those who show loss of control have one or more drinking problems at time 2. Similarly, 62% of those showing symptomatic drinking and 68% of those showing loss of control at time 1 have heavy alcohol intake 4 years later.

Clark and Cahalan urge the position that alcohol problems have a large situational component which bears on the problem drinker's behavior, because the same problem is not present 4 years later. Their data do not exclude the possibility that individuals do become psychologically and physiologically dependent in an orderly fashion. Their data can, in fact, be interpreted to mean that 78% of alcohol-dependent individuals are at great risk of developing social problems and central nervous system illnesses 4 years later.

A new effort to improve the research methodology in this area is represented by the work of Chick and Duffy.[22] Chick and Duffy interviewed 38 consecutive male admissions to an alcoholism treatment unit who were aged 65 or below and who, 1 week after admission, exhibited no clinical evidence of memory impairment or dementia. Twenty-seven (71%) claimed they were alcoholic, and all had been diagnosed to be alcohol dependent. Twenty-three items of information were obtained by asking patients to order shuffled cards into early, middle, or late sets. Each card had on it an item already reported as having been experienced by the patient. Interrater reliability for two observers was determined, and it ranged for each item from 92% to 100% agreement. One-week test–retest reliabilities ranged from 0.34 to 1.0, with a median value of 0.75. The ordering of items was shown to be nonrandom in this sample. Each ordering of events of the 38 individuals was correlated with the groups' modal ordering. These correlations ranged in value from -0.56 to 1.00. The median value of correlations was 0.50. Considering the fact that

29% of the sample did not believe they were alcoholic and that some retest reliabilities were as low as 0.34, this order of magnitude is quite acceptable. It seems reasonable to accept the finding that the symptoms can be divided into early-, middle-, and late-appearing subgroups.

The loss of control items, "unable to keep a limit to drinking" and "difficulty in preventing drunkenness," appear early in the set, as do items of salience: "spending more time drinking," "missing main meals because of drinking," and "needing more companions." The late symptom set contained items related to withdrawal, "trembling," "morning nausea," "sweats," "morning drinks," "panics," and "hallucinations."

Five of 13 patients who reported atypical sequences of symptoms were controlled drinkers and may be part of the group who believed that they were not alcoholics. Thus, with the most sophisticated methodology yet applied, the phases of alcohol dependence reappear, and their sequential order is affirmed.

In summary, all studies support the position that, at least within Western societies, there is a recognizable developmental sequence of alcohol dependence symptoms. This developmental sequence can best be recognized when cultural and social responses to alcohol dependence are winnowed out of the many signs that have been proposed as characteristic of alcohol dependence.

Unfortunately, our knowledge of the chronology of the alcohol dependence sequence rests on less than 1000 poorly defined cases wherein patients were asked different questions under varying circumstances. Four reports present enough information to allow a combined estimate of the developmental sequence of alcohol symptoms. These data are presented in Table II. The symptoms are presented and ordered according to the weighted average age of occurrence across all samples. Interpretation of this table is facilitated by study of Table III in which symptoms are presented along with the interval in years between their development and morning drinking, taken as the critical marker of physiologic dependence.

Morning drinking was chosen for this purpose for two reasons:

1. Morning drinking is associated with the end of the psychological dependence phase, i.e., reduction of behavioral repertoire so that drinking goes on at all waking times, and the beginning of physiological dependence, because alcohol is necessary to fend off withdrawal symptoms produced by decrease in blood alcohol concentrations during the night.
2. In almost no western culture is morning drinking considered acceptable.

Using these calculations, it is possible to estimate intervals between appearance of symptoms. The interval between loss of control and morning drinking is 1 year; that between morning drinking and tremors is 2½ years. The period from loss of control to hospitalization is about 7 years on the

Table II. Average of Appearance of Alcohol Dependence Symptoms

	Average age all studies	Percent reporting	Number of observations	Jellinek[13a] av. age (N = 98)	Glatt[15] av. age (N = 192)	Goodwin[13] av. age (N = 100)	Orford[20] av. age (N = 59)
Getting drunk	19.7	100.0	290	18.8	20.1	—	—
Sex self-conscious	25.8	46.9	98	25.8	—	—	—
Weekend drunk	27.6	78.8	198	27.2	—	28.0	—
Extra behavior	27.6	78.6	98	27.6	—	—	—
Sneak drink	29.5	75.5	290	25.9	31.9	—	—
Indif. qual.	30.0	85.7	98	30.0	—	—	—
Blackout	30.2	79.3	449	25.2	30.1	35.0	32.6
Lose work	30.4	91.8	98	30.4	—	—	—
Midweek drunk	30.4	79.6	98	30.4	—	—	—
Family disapproval	30.5	96.9	98	30.5	—	—	—
Lose advance	30.6	57.1	98	30.6	—	—	—
Daytime drinking	31.0	86.7	98	31.0	—	—	—
Antisocial behavior	31.3	61.2	98	31.3	—		—
Loss of control	31.8	90.2	349	27.6	34.4	—	30.3
Escape environment	32.0	64.3	98	32.0	—	—	—

Remorse	32.2	92.9	98	32.2	—	—	—
Lose friends	32.5	51.0	290	29.7	34.5	—	—
Morning drinking	32.9	88.6	449	29.9	35.3	31.0	32.8
Fears	32.9	73.5	98	32.9	—	—	—
Benders	33.1	77.2	390	31.8	34.9	31.0	—
Lose job	33.1	68.1	257	30.9	—	34.0	34.2
Solitary drinking	33.3	79.3	290	31.2	34.3	—	—
Rationalization	33.7	79.0	290	29.2	36.1	—	—
Change pattern	34.2	53.4	290	32.7	35.6	—	—
H_2O-wagon	34.3	71.5	390	30.7	36.2	34.0	—
Tremors	34.6	83.1	449	32.7	37.2	33.0	—
Resentment	34.8	66.2	290	33.1	35.7	—	—
Seek psych. adv.	35.1	47.1	157	35.0	—	—	35.2
Protect supply	35.5	62.8	290	32.5	37.4	—	—
Sedatives	35.5	61.2	98	35.5	—	—	—
Religious	35.7	61.2	98	35.7	—	—	—
Seek med. adv.	37.8	64.1	290	35.8	39.3	—	—
Hospitalization	38.1	71.9	449	36.8	40.2	37.0	36.3
Admit inability	39.5	92.9	98	39.5	—	—	—
Admit defeat	39.7	85.5	290	38.1	40.7	—	—
Reach lowest	41.3	95.5	290	40.7	41.7	—	—

Table III. Interval in Years between Appearance of Alcohol Dependence Symptoms

Psychological dependence			Physiological dependence			Central nervous system disorganization		
Symptom	Av. age	Diff[a]	Symptom	Av. age	Diff[a]	Symptom	Av. age	Diff[a]
Weekend drunks	27.6	−5.3	Morning drinking	32.9	0	Tremors	34.6	1.7
Sneaking drinks	29.5	−3.4	Fears	32.9	0	Protect supply	35.5	2.6
Blackouts	30.2	−2.7	Benders	33.1	.2	Seek med. advice	37.8	4.8
Loss of control	31.8	−1.1	Periodic abstinence	34.3	1.3			
			Tremors	34.6	1.7	Hospitalization	38.1	5.9

[a] Difference in years between appearance of alcohol dependence symptoms and morning drinking.

average. The short period between loss of control and the development of neurological symptoms might explain why more than half of all alcohol-dependent people arrive at treatment programs before the age of 40.

Given the clear direction of the findings in every study, it is of some interest to identify the source of confusion for the many interpreters in the field. The confusion seems to arise from the several domains of events that are of concern to policy makers and are embodied in the definition of alcoholism given by the WHO Expert Committee on Mental Health, Alcoholism Subcommittee, 1952. This definition included social damage events such as job loss and arrests and treatment events such as hospitalization.[1] This confusion has in part been redressed by the 1977 report of the Expert Committee which recognizes the alcohol dependence syndrome and that the occurrence of social damage, and to some extent physical health damage, among alcohol-dependent people is a result of cultural attitudes and political policy.

What is apparent from Table II is the lack of quantitative information about symptoms that have been hypothesized by the WHO report[10] to be markers of the alcohol dependence syndrome. The inevitable conclusion is that the basic research that might support the position of the expert committee is missing. It may be prudent at this time to confine discussions of the signs of dependence to the published data (to prevent a new confusion in the field) until additional information can be gathered.

3. Types of Alcoholics

Eight reviews of studies of the "alcoholic" personality have concluded that research has failed to establish the existence of any single constellation of personality traits in alcoholics that would predispose a person to alcoholism. The most recent reviewer, Barnes,[23] concluded that alcoholics present a fairly common personality pattern when they arrive for treatment but acknowledged that the evidence for a prealcoholic personality is limited to reports from one group of researchers. Most research on the clinical alcoholic personality has been successful in characterizing how alcoholics are different from normal subjects or psychiatric patients, but it has not satisfied those clinicians who have been impressed with the subgroupings of personality within alcoholism treatment populations.

The following review is organized around five themes in the research literature about subdivision of alcoholics into types as follows: (1) essential versus reactive alcoholics; (2) primary versus affective disorder alcoholics; (3) psychiatric syndrome groups: depressed versus neurotic versus psychopathic alcoholics; (4) clustered personality trait type alcoholics; (5) successful and unsuccessful "life style" alcoholics.

In order to shorten the discussion, the methodological inadequacies that plague these studies is here summarized.

3.1. Methodological Considerations in Studies of Types of Alcoholics

The ultimate task of a study of a proposed typology is to determine whether each type can be described and identified with unique markers that reliably differentiate it from other types. To do this adequately, there must be operational definitions of signs and symptoms of the type, a reliable method of data gathering, validity of the measurement procedure, and evidence that the typing makes a difference in the natural or treated outcome of the condition.

Most of the studies of alcoholic types do not begin with clearly defined types that can be reliably identified in the clinical population that seeks treatment for alcoholism. This failure results from lack of operational definition of the characteristics that define the types. It is undetermined whether it will be possible to replicate assignment of individual cases into the types at other clinical facilities. Most authors do not include critical information used to define the patient as alcohol dependent, for example, tolerance of alcohol. When included, tolerance is often described simply as high or decreasing. Similarly, no data are reported about signs of withdrawal, frequency, quantity, and duration of alcohol consumption, or the age of first appearance or regular appearance of dependence symptoms. It is therefore extremely difficult to determine if the patients are alcohol dependent and at what phase of illness they might be.

The reliability of data used in the studies of types of alcoholism is generally not examined. There is evidence in the research literature that patients in alcoholism treatment programs provide different information about their current and past behaviors at different stages of treatment.[24] Information obtained by different interviewers from the same patients has also been shown to vary.[13] Thus, reliability of information must be determined and taken into account when data are interpreted.

The validity of the information is also often questionable. Most studies do not report the length of time from detoxification of patients before information is obtained. In most studies, data are collected as early as 1 week after detoxification. Several studies have found that many patients have substantial deficits in memory. Thus, recall of age of occurrence of symptoms is in doubt. Postsedative withdrawal periods are also marked by depression, sleeplessness, and irritability, all of which will influence personality descriptions, whether by interviewer or in self-description inventory. These well-known facts are not taken into account, so that validity, i.e., the measurement of that which was intended for measurement, is in doubt. Thus, the observed patient traits may be preexisting, concurrent, or resultant conditions of alcohol dependence.

Perhaps because of the lack of access to general population samples, subjects are almost always selected from alcoholism treatment programs or state hospitals. Pattison, Coe, and Doers[25] and Smart, Schmidt, and Moss[26] have shown that the populations served in different agencies are different in

social class and other characteristics such as debilitation or competence. Information about intelligence or other illnesses such as traumatic injuries that might affect brain function are not included. Even though age and sex distribution of subjects is usually provided, it is not entered into the factorial or multivariate analyses of data. As a result, it is not possible to determine whether patterning of symptoms is uniquely related to alcohol dependence or whether it is a function of age-related biological phenomena.

3.2. Essential and Reactive Alcoholism

In 1937, Knight, at the Menninger Foundation, proposed that two types of alcoholics could be distinguished.[27] The "essential" alcoholic was characterized as emotionally dependent, pleasure seeking, irresponsible, insincere, unreliable, and unable to establish long-term goals or intimate relations with others. Knight described the "reactive" alcoholic as having quite different, perhaps opposite, traits, notably, considerable compulsivity. In addition to these personality traits, the reactive class of alcoholics would be more likely to be better educated, of higher occupation, have a later onset of problem drinking, and a better prognosis.

Dale and Ebaugh,[28] in experimenting with disulfiram as a method of treatment for alcoholics, used the essential–reactive distinction. They interpreted essential alcoholism to be an expression of neurosis characterized by overly dependent, sensitive, and insecure traits. Reactive alcoholics were described as showing depressive features clinically and, in the face of stressful situations, regression to alcoholism. Reactive alcoholic patients had fewer relapses than essential alcoholics.

Rudie and McGaughran added to Knight's description the notion that the essential alcoholic shows a general culture combativeness and is relatively free of anxiety and guilt.[29] They believed that the essential alcoholic's personality could be subsumed under the terms "psychopathic" or "sociopathic." The reactive alcoholic was described as having more defenses and intellectualizations. Using these ideas, they created a 69-item instrument with a retest reliability of 0.66. This instrument did correlate with education but not with age. The instrument was administered to 56 patients at the Moose Lake State Hospital. The essential group had fewer defenses, came from more disorganized families, and had more marital difficulties and more psychopathic adjustment.

Sugarman, Albahary, and Reilly[30] administered the Rudie and McGaughran Questionnaire to 118 voluntary male patients consecutively admitted to the New Jersey Neuropsychiatric Institute. A Phillips and Zigler social competence score was also calculated for each patient. A moderate but significant relationship ($r = 0.45$) between the scales was found.

Levine and Zigler[31] studied 60 patients who were hospitalized for less than 3 months in the Westhaven, Montrose, and Bronx VA hospitals and the

Connecticut Valley and Fairfield Hills hospitals. Twenty subjects were psychiatric patients (17 depressives) with the primary diagnosis of alcoholism; 20 were psychiatric patients without psychosis; 20 were medical or surgical patients without psychiatric symptomatology and screened for alcoholism. Patients were selected in each group as being high or low on prehospitalization social competence as measured by the Phillips–Zigler social competence index.

The groups were compared on the Rudie–McGaughran Essential Reactive Alcoholism Scale. Low-competence patients in all diagnostic categories obtained higher scores. Alcoholics obtained higher scores than psychiatric patients, who in turn had scores higher than medical patients. A further analysis was carried out with social competence items deleted from the essential–reactive scale. Again, the social competence scale and the essential–reactive scale were significantly correlated for each group. A large, significant correlation between age and the essential reactive scale was also found among alcoholics ($r = 0.52$) and all subjects ($r = -0.33$; $P < 0.01$). Levine and Zigler concluded that the essential–reactive scale measures social competence and maturity as represented by age and is inadequate for defining two types of alcoholics other than along a dimension of social competence–maturity.

In a confirmatory study, Blum and Levine[32] examined the relationship between depression, life events, and social competence and the essential–reactive alcoholic typology in 28 middle-aged male alcoholics hospitalized in the West Haven VA Hospital for more than 3 months. As in the preceding study, the essential–reactive scale is correlated (0.59) with the social competence index. Retrospective reports of depression measured by the Zung and Beck scales were not statistically significant in association with the essential–reactive scale scores.

At this point, it is safe to conclude that there is no support for the utility of an essential–reactive typology as defined by the Rudie and McGaughran scale.

3.3. Primary and Affective Disorder Alcoholism

A second approach to developing a typology of alcoholics was based on the observations by Pitts and Winokur[33] that there is a close relationship between alcoholism and affective disorder. Of particular interest was the observation that as many as 50% of female alcoholics exhibit symptoms of other psychiatric entities.

Schuckit *et al.*[34] interviewed 70 consecutive female admissions to a private psychiatric hospital and the Malcolm Bliss Mental Health Center Alcoholism Unit. If the patient had depressive symptoms before onset of alcoholism or during a sustained period of abstinence, it was considered primary. Moderate depression during alcohol use was not considered primary. Patients with any history of schizophrenia during their illness were grouped separately. Simi-

larly, patients with hysteria, obsessive–compulsive neurosis, and other personality disorders were also grouped separately. Thirty-nine women were classified as primary alcoholics. Nineteen were classified as having an affective disorder, six as sociopathic, and the rest with a variety of conditions. The rates of psychiatric illness in relatives of both types of alcoholics were determined by interview to be identical. The authors were convinced that there were different types of psychiatric illness in the two groups of relatives, which led them to conclude that the differentiation of primary and affective alcoholics was useful.

Winokur *et al.*[35] interviewed 259 patients admitted to the Renard Psychiatric Hospital and the Malcolm Bliss Mental Health Center Alcoholism Unit. Most patients' families were contacted for confirmation of information about symptoms in the proband. Seventy-three percent of the men and 61% of the women were diagnosed as being primary alcoholics. Five percent of the men were diagnosed as affective disorder alcoholics, compared to 25% of the women. Twenty percent of the men were diagnosed as having a personality disorder, mainly sociopathy, in contrast to 7% of the women.

Winokur *et al.*[36] studied the 259 alcoholic probands and obtained a systematic family history of psychiatric illness from 507 first-degree relatives who were interviewed. Familial depression was found to be statistically significant more often in relatives of depressed alcoholics than of primary alcoholics. Familial alcoholism was also seen more often in families of depressed alcoholics than in families of primary alcoholics. Similar comparisons with sociopathic alcoholics were not statistically different. Among males, the sociopathic group has more sociopathic male relatives than either the primary or depressed alcoholic group. The segregation of these diagnoses in families of the three types of probands was construed by the authors as support for the hypothesis that these are separate conditions. The data were not able to establish whether this segregation is the result of cultural or familial phenomena.

The support for the primary–affective typology is not strong. In both Winokur *et al.* reports,[35,36] the sum of risks for alcoholism and depression in first-degree relatives of primary alcoholic, depressed alcoholic, and sociopathic alcoholic groups are almost identical. The absence of large differences, the lack of reliability and validity of data about diagnoses, and the lack of evidence relating types to cause and outcome suggest that at the present time this distinction may only be considered as a very tentative hypothesis.

3.4. Psychiatric Syndrome Groupings of Alcoholic Patients

Another approach to classifying alcoholics into subgroups has been through factor analysis of Minnesota Multiphasic Psychological Inventory scores obtained from alcoholic patients. The original work in this area derived from the efforts of M. A. Brown[37] at Elgin State Hospital to classify 80 consecutively admitted male alcoholics into psychiatric syndrome types. The MMPI profiles

were sorted independently by three raters into either a neurotic or psychopathic group. There were 67 cases (84%) of complete agreement. In the final group, there were 34 neurotic and 33 psychopathic alcoholics. When neurotic alcoholic profiles were compared with other neurotics, there was great similarity, and similarity between psychopathic alcoholics and other psychopaths.

Goss and Morosko[38] analyzed data from 200 male consecutive admissions to the outpatient service of the Texas Research Institute of Mental Sciences Alcoholic Treatment Unit. These patients were administered the MMPI on the first day at the Institute. The sample had a mean age of 41.9, IQ of 111.6, and education of 11.3 years. The MMPI scores were transformed to *T* scores. A psychopathy–depression elevated *T*-score pattern appeared in 42% of the patients, whereas a hypochondriasis–hysteria pattern appeared in 23% of the patients. Within these two groups, there was a 12% overlap of individuals who showed both patterns.

The psychopathic, depressed, and neurotic patterns again appeared in a study by Goldstein and Linden[39] who obtained MMPI profiles from 513 behaviorally identified male alcoholics tested within 5 days of admission to a state hospital. The sample was probably of low-SES chronic alcoholics. The responses of 239 subjects were used in a clustering procedure, and four patient types were identified, which included 45% of the standardization sample. The process was replicated in another sample of 251 cases. The same four types emerged and included 42% of the sample.

The authors described the types as follows: (1) an emotionally unstable personality with a trait of poorly controlled anger resulting in "temper tantrums," usually diagnosed as psychopathic; (2) psychoneurotic, either of an anxious or reactive depression character associated with a great number of somatic complaints and suicidal ideation; (3) psychopathic, mixed type, with a long history of chronic alcoholism interspersed with acute episodes; (4) excessive user of alcohol with addiction to drugs and the use of nonbeverage alcohol but able to stop using for long periods of time and lead normal lives.

Mogar *et al.*[40] selected a random sample of 101 men and 100 women who had been administered the MMPI at Mendocino State Hospital. Sixty-two percent of the men had elevations of two scales above *T* scores of 70. Most frequently occurring profiles were identified by a clinician. All profiles were then sorted into mutually exclusive and exhaustive subtypes.

Four groups were identified among males: (1) psychopathic, mania, social introversion, labeled the passive–aggressive type; (2) depression psychasthenic introversion, labeled depressive–compulsive; (3) schizophrenia elevations, labeled schizoid–prepsychotic; (4) hypochondriasis and hysteria elevations, labeled passive–dependent, constituting 15% of cases. Of the passive–aggressive type, 78% had only one elevated *T* score above 70, which was depression.

Among females, the patterns were: (1) psychopathic mania, social introversion, and femininity, labeled normal–manic; (2) psychopathic, psychasthenic, social introversion, and femininity; (3) hysterical, hypochondriasis,

and femininity, labeled hysterical; (4) psychopathic, mania, and masculinity, labeled psychopathic; (5) depression, psychasthenia, introversion, and masculinity, labeled passive–aggressive. In this study, the psychopathic and depressive patterns again emerge as dominant in the symptomatology of hospitalized alcoholics.

Bean and Karasievich[41] performed a cluster analysis on the MMPI scores of 80 men admitted to the alcoholism treatment unit of the VA Center at Temple who completed a 60-day inpatient stay. About one-half of the patients could be identified as belonging to one of four clusters of symptoms. The clusters were quite similar in pattern to those previously reported. Proportion of patients in each category is reported here for the analysis done at 60 days of sobriety. Psychotic patients comprised 6% of the sample and were characterized by disorganized thinking and distrust. Patients labeled as latent schizophrenic comprised 18% of the sample and were described as demonstrating bodily preoccupation, anxiety, and compulsiveness. Neurotic patients comprised 26% of the sample and were characterized by somatic complaints, depression, and hysteria. Psychopathic patients comprised 50% of the patients and were characterized as antisocial, acting out, and impulsive.

The MMPI studies consistently find that alcohol treatment program patients can be described as portraying one of four psychiatric syndromes: psychopathy, depression, hypochondriacal–hysterical syndrome, or a psychotic syndrome. The studies also consistently report that about one-half of hospitalized patients are not classifiable within one of these types.

Related to the MMPI studies are two recent attempts to characterize alcoholism program patients using psychiatric constructs or syndromes. Whitelock *et al.*[42] administered an 80-item symptoms-of-alcohol-abuse questionnaire along with the 373-item form of the MMPI to 136 newly admitted male patients in a state psychiatric hospital. All patients were prescreened for at least moderate alcohol use, although most patients were diagnosed as having some problem related to alcohol. The patients were mostly younger men, 98 under the age of 49, 121 were Anglo, 87 had at least one previous hospitalization, 46 were married, and 55 had never been married. A 38-item abuse scale was developed on the basis of a factor analysis, and severity of abuse scores were computed for each subject. Scores were *Q* clustered on two independent subsamples of 68. A mean MMPI profile was then computed for each of four types: (1) elevated psychopathic score; (2) elevated depression, schizophrenic, and psychopathic scale scores; (3) elevated psychopathic, depression, and psychasthenic scale scores; and (4) depression score alone.

After transforming the Goldstein and Linden distribution of scores to their population distribution, Whitelock *et al.* compared the two typologies. The resemblance of the first three patterns, psychopathic, depressed schizophrenic psychopathic, and psychopathic, depressed psychoasthenic, was striking.

Severity of alcohol abuse covaried significantly with the profiles. The depressed and psychopathic patients reported fewest alcoholism symptoms.

Patients with schizophrenic psychopathic pattern and psychopathic, psychasthenic depression pattern showed most symptoms.

Skinner *et al.*[43] examined 282 white male patients consecutively admitted to the Minnesota State Hospital for inpatient treatment of acute alcoholism with the Differential Personality Inventory (DPI) and the MMPI. The mean age at admission was 44.9 years. The population was divided into three samples of 94 subjects, and a *Q*-technique factor analysis was conducted on the DPI items in each group. Final types were replicated in each sample. Eight common types emerged from the analysis. The first two bipolar dimensions accounted for 85 patients, and the other six dimensions described 53 patients. The first four types, derived from these dimensions, are as follows: (1) a defensive, repressed, and shallow affect group; (2) an irritable, panicky, labile mood group; (3) a hypochondriacal group complaining of somatic symptoms and health concerns; and (4) a rebellious, socially deviant, impulsive, and desocialized group recognizable as psychopathic.

The authors were able to sort hospitalized alcoholic patients into coherent and recognizable subgroups using psychiatric symptomatology. However, the first type, defensive, shallow affect, included 37% of the sample, whereas all eight types could only classify 56% of the alcoholic sample.

The factorial studies of the MMPI have repeatedly demonstrated that a sizable portion of individuals treated in state hospitals for alcohol problems can be consistently categorized, during their stay, into types using familiar psychiatric descriptors. The types that emerge consistently include, as the largest group, a predominantly psychopathic type, a depressed type, and complex types involving combinations of elevated scores for depression and psychopathy, hypochondriosis and hysteria, or, occasionally, schizophrenia and psychopathy.

None of the typologies, based on symptoms of mental illness, has been able to adequately encompass much more than one-half of the alcoholism patients. Alcohol dependence illness seems to occur in association with many psychiatric syndromes in state hospital populations. The data suggest that psychopaths and depressed individuals may be more prone to develop alcohol dependence. Alternatively, it may be that these personality types, having developed alcohol dependence, are more likely to end up in a state hospital.

3.5. Personality Trait Groupings of Alcoholics

In light of the finding that many, if not most, alcoholic patients do not fit into psychiatric categories, examination of general personality traits may be helpful in classifying alcoholics. There have been two attempts to divide alcohol patients into subtypes using general personality trait tests.

Lawlis and Rubin[44] administered the 16 P.F. Questionnaire to 100 consecutively admitted white patients (22 women) of the alcoholic ward at Benton State Hospital. The study was replicated on 80 subjects at the same hospital and on 84 alcoholics in treatment at the Wisconsin Division of Vocational

Rehabilitation. The first cluster type seems to represent a highly anxious group of individuals, expressing anxiety by withdrawing, whereas a second group expresses high anxiety outwardly through aggression. A third, less clearly interpretable group seems to deal with anxiety by following rules.

Nerviano[45] examined 366 detoxified veterans at the VA Hospital in Lexington, Kentucky with a primary diagnosis of chronic alcoholism. The men were free of physical and cerebral impairment, with a mean age of 44 years, a mean education of 11 years, and a mean estimated IQ of 107. The patients, for the most part, were lower socioeconomic level whites.

Typological analysis using a clustering procedure was tested on successive halves of the sample. The final typology was created on the entire sample, yielding seven Jackson Personality Research Form profile types which classified 49% of the total sample. The seven types were as follows: (1) highly pervasive impulse control, a moderate degree of social ascendencey, moderately low anxiety, and some extroversion; (2) lack of impulse control; (3) moderately impulsive, defendant, and emotionally independent, but highly anxious; (4) submissive, defendant; (5) highly anxious and introverted; (6) low social ascendency, low dependency, low defendency; (7) socially ascendent, low in anxiety, and extroverted.

Nerviano suggested that most of these types correspond to recognized clinical syndromes.

The failure to develop a typology that includes more than half of the patients identified as alcoholics by hospital admission criteria, using general personality traits, suggests that a substantial part of the alcoholic population in state hospitals is there neither for personality reasons nor for processes basic to alcohol dependence illness. They seem to be in the hospital because of social, economic, or familial problems that are independent of alcohol dependence illness.

This proposition has been examined more directly in studies of life styles, attitudes, and habit patterns of alcoholism patients.

3.6. Life Style, Attitudes, and Habits of Alcoholics

The most extensive study of habits and attitudes of alcoholic patients has been carried out by Horn and Wanberg using a drinking history questionnaire created for the purpose along with several other domains of data. Horn and Wanberg[46] administered 69 selected questions to 2300 patients admitted to the Fort Logan Mental Health Center, a hospital for the treatment of the emotionally disturbed in the Denver metropolitan area. These 2300 patients were 82% male, 43% married, 27% with more than 12 years education, 81% white; 51% had previous treatment for alcoholism, and 8% were of the lowest socioeconomic stratum. At first, 41 items were factor analyzed. A varimax rotation yielded 13 factors. The first factor included variables such as cannot stop after one or two drinks in the morning, drinks every day, intermittent sobriety, frequent blackouts, not much eating while drinking, miss work while

drinking, shakes after drinking, had delerium tremens, fears after drinking, sleep better when drinking, drinking getting worse. This severity of alcoholism factor was independent of other factors associated with admission for alcoholism. Only 30% of the hospital population had experienced the extreme condition which included DTs. Inability to stop after one or two drinks was present in 78% of the population.

Other factors emerging from the analysis include an assortment of variables that describe drinking style and motivation to drink as follows: married beer drinker who wishes to feel superior; wino; binge drinker; fixed time of day drinker; person who drinks to improve performance; social drinker; home drinker, female latecomer to intoxication. Individuals with any of these drinking styles can develop any degree of alcohol dependence illness.

Horn and Wanberg[47] also factor analyzed childhood and family history data from the population just described. Seven factors emerged from the analysis as follows: (1) played hookey, trouble with the police, bad temper, gambled a lot, drank heavily; (2) disliked school, played hookey, unable to keep up in sports, bad grades, disliked teachers, did not belong to a church; (3) major illness, unable to keep up in sports, unaffiliated with a church; (4) parents died, no mental illness in the family, stuttered, belonged to a church; (5) parental drinking problem, nail biting, stuttering, kept up in sports, liked teachers; (6) didn't get along with family, mental illness in the family, nightmares, nail biting, stuttering, punished severely, disliked teachers, bad temper; (7) major illness, nightmares, at least one close friend, belonged to a church or club.

The factors suggest that the childhood and family histories of many state hospital alcoholic patients correspond with those described in the literature on psychopathic, anxious, and depressed patients—e.g., parental loss and illness—and neurotic patients—i.e., interpersonally maladjusted. Horn *et al.*[48] reanalyzed their data and were able to distinguish alcoholics who accepted their addiction and those who did not. These dimensions were independent of the various life style patterns which were as follows: sociopathic–gregarious, drinking–marital problems, despairing–worry, anxiety–hypochondriacal pattern, and socially withdrawn.

Horn and Wanberg repeated their earlier analysis of the same data set but first divided it into male and female groups. The same primary factors were descriptive of both groups.

The authors summarized the status of research in this area in their statement that a substantial amount of reliable variation in drinking symptoms factors is not explained by background, current condition, or self-evaluation. They do not go on to consider the possibility, which is concordant with their analyses, that alcohol dependence can occur in all types of individuals, only a fraction of whom can be classified with recognizable psychiatric syndromes.

Pokorny *et al.*[49] examined 201 men referred to the alcoholism service of the VA hospital at Houston. Information obtained included a symptoms rating scale, background and demographic information, four sobriety ratings, the

Iowa Scale of Preoccupation with Alcohol and Index of Trouble Due to Drinking. A varimax factor analysis yielded 11 factors labeled as follows: (1) loss of control over drinking, (2) low socioeconomic level, (3) psychological dilapidation, (4) duration of alcoholism, (5) social isolation, (6) marital–emotional disruption, (7) multiple hospitalization, (8) severity of alcoholism, (9) aggressive–outgoing, (10) elation, (11) steady worker. Comparison of 21 successful outcome patients with 34 nonsuccesses indicated that patients with marital disruptions did better. This reflects the incentive for recovery provided by an upset wife.

A study reported by Partington and Johnson[50] is most important because it is one of the few to be carried out in an outpatient setting and therefore included a broader range of personalities and life styles than can be found in state hospital patients.

Partington and Johnson examined 186 males, mostly first admissions to the Alcohol and Drug Addiction Research Foundation Outpatient Clinic in London, Ontario during 1965. The subjects reported a mean of 8.2 years of problem drinking and were judged alcoholic by several clinicians, but no criteria for this judgment were reported. The subjects were of lower to middle class; 60% were self-referred; their average age was 41; the subjects on average had a tenth grade education; 60% were employed; and about one-half were married.

Responses to the 318-item Differential Personality Inventory were scored on 18 dimensions for samples of 90 and 96 subjects, factor analyzed, and rotated to a simple orthogonal structure. A profile cluster analysis produced five distinctive personality types which were examined in relation to performance, life history, and clinical characteristics.

An antisocial group, 20% of the sample, was found to be relatively young with a poor employment record, poor accommodations, and very serious consequences from drinking. They had little interest in the treatment process.

A group with impulse control problems included about 19% of the sample and was characterized as conforming to social norms but having a tendency to lose emotional and cognitive control. Though this group was intelligent, they had not been successful maritally and were not suffering serious consequences of alcohol use. They seemed to have the best record of abstinence and were interested in treatment.

A neurotic hypochondriacal group made up 10% of the sample. They were characterized as concerned about their health, older, less verbal, and less intelligent. They were stable workers, married, and drank more steadily and had little understanding of alcohol but were less ready to live without it.

A normative group included 23% of the total sample and were characterized as stable, healthy, with good education and few serious consequences of drinking. They were normative in social orientation, presenting a good picture of themselves. However, their drinking was uncontrolled.

Another group of normals included 28% of the sample. These individuals

had few neurotic symptoms and were not cognitively or emotionally upset. They were more satisfied with their social lives. However, their life style included frequent drinking episodes and consumption of large quantities of alcohol.

Chandler *et al.*[51] administered a questionnaire to 246 male members of AA and 92 upper-class private patients. The questionnaire covered drinking history and complications, personal characteristics, and a short version of the Maudsley Personality Inventory. The upper-class group were older, had fewer social consequences or health consequences, e.g., DTs (14% vs. 58%), and had intact marriages. Lower-class alcoholics had higher neuroticism scores, a measure of manifest anxiety, an earlier age of onset of alcoholism, and more adverse consequences from drinking. However, there was no significant difference between classes on the one available measure of addiction, "severe morning shakes." These findings support the position that social circumstances may influence only the concomitants and consequences of alcohol dependence rather than alcohol dependence itself.

Evenson *et al.*[52] administered an Alcohol History Form generating 157 variables to 1395 inpatients admitted to eight alcoholism treatment facilities in Missouri. The 1023 patients who completed the form had a mean age of 45.6 years, 84% being between 30 and 59 years of age; 13.7% were females, and 22% also used other drugs. A varimax factor analysis was completed for the sample. A *Q*-factor analysis was completed on a random sample of 146 patients' responses on 118 variables. The first three clusters or types accounted for 144 of the 146 subjects. The first type included younger, married, employed individuals likely to be living with their primary family. The second type included older individuals who were unemployed, living alone, and who had experienced greater deterioration including blackouts, benders, nightmares, and were likely to be drinking continuously. The third group was mostly female, living with relatives, tending to blame their spouses for their drinking, and were clinic rather than AA participants. These groupings seem to reflect the large differences in life style associated with economic and family situations found in clinical populations.

Finney and Moos[53] studied patients from five heterogeneous residential treatment programs. The sample consisted of 387 persons who had been successfully followed after completing treatment in either a Salvation Army Program, a 28-day hospital-based program, a county-funded halfway house, a private 28-day milieu therapy program, or a private 2-week aversion program.

Sociodemographic, social psychological, and social environmental information was collected. The Engleman and Fu method of cluster analysis was used, which aggregates all cases into one or another cluster based on greatest similarity. A final set of eight clusters was produced. In all, 87% of the sample could be assigned to a type. Four broad groupings emerged, based on availability of social resources and social competence. For example, the low social

competence, low resources group was unmarried, unstable, uneducated, with lower social functioning, little feeling of psychological wellbeing, unassertive, with several heavy drinkers in their family. The high social competence and high resources group described their drinking problems as not severe and had little physical impairment, few arrests, and a high income. As would be expected, this typology had a positive and significant relationship with outcome variables. However, the typology accounted for only 13% of the outcome variance among those patients who could be followed.

Methodological weaknesses in the studies of social and economic concomitants of alcohol dependence do not allow firm conclusions at this time. However, the trend of the studies suggests that socially competent individuals with economic and familial resources suffer fewer consequences of alcohol dependence and have better prognoses. Members of the upper class, people who are married and employed, suffer fewer consequences and have better prognoses than lower class, young, antisocial, poor workers or older unemployed persons. A concerned and upset wife is apparently a significant factor. In outpatient alcoholism program populations, large numbers of individuals without indications of psychiatric illness appear. Those manifesting anxiety are more likely to control their drinking than the calmer heavy drinkers and the group of drinkers who have lost control.

4. Conclusions

Many writers and researchers have invested a great deal of energy and thought in order to develop a classification system for types of alcoholics. They are responding to an important clinical need to develop a descriptive system that can promote better research on the problem of matching patients and treatment. Several significant advances have been made using cross-sectional research methods. However, almost nothing has been produced that provides evidence on the relationship between proposed typologies of alcoholics and etiology, course, and outcome of the alcohol dependence condition itself in contrast to concurrent psychiatric conditions.

The retrospective studies consistently support the view that there is a consistent sequence of symptoms in the development of alcohol dependence illness. This sequence can be reliably divided into phases or stages, each of which has major markers. The phases are psychological dependence, physiological dependence, and central nervous system disorganization or damage. Individuals are able to stop or control consumption of alcohol at any stage and arrest the progression of symptoms.

A major source of confusion in research arises from the fact that a large portion of patients in alcoholism treatment programs are not alcohol dependent. The confusion could be resolved by distinguishing among alcoholism treatment program patients on the basis of whether they are alcohol depen-

dent or excessive drinkers who are causing a social problem which brings them to seek professional help. Prognosis, treatment, and outcome for these two groups are clearly different.

Those patients who show signs of alcohol dependence can be divided into phase of dependence groups by using the symptoms of psychological dependence, physiological dependence, or central nervous system damage presented at time of admission. There is no research evidence on this matter, but clinical experience suggests that treatment plans and outcome will be different for individuals in different phases.

Research on subtyping the alcohol-dependent patient is rudimentary and generally of very poor quality. However, the general outlines of what will probably be confirmed may be discerned.

The essential reactive distinction has been shown to be an expression of an underlying dimension of social competence and maturity. Apparently, all levels of maturity and social competence can be found among alcoholics. Division into types based on competence is useful insofar as it predicts likelihood of control of dependence. Social competence is correlated with severity of central nervous system damage. However, a cause-and-effect relationship has not been established. Some evidence suggests that maturity and competence seem to help the individual avoid deterioration after alcohol dependence has set in.

Several attempts have been made to classify alcoholics into psychiatric subgroups. The state hospital alcoholic population seems to be composed almost equally of individuals who can be classified into psychiatric categories by either interview or MMPI examination and those who can not. The largest part of the classifiable group are psychopathic. This group of psychopathic alcoholics seems to include those individuals who in the Schuckit and Winokur studies are classified as primary alcoholics. However, several studies have found it necessary to create subtypes of psychopaths according to whether they were prone to tantrums and mania or prone to depression. The next largest psychiatric grouping observed in all studies includes depressed patients, followed by, depending on the facility, neurotic patients who were highly anxious or hypochondriacal–hysterical.

The classification of alcohol-dependent individuals by concurrent psychiatric syndromes has an as yet untested promise of utility in treatment planning. It is a misleading typology, however, insofar as it implies that all alcohol–dependent individuals are psychiatrically ill. Half of state hospital patients seem to be there only because of social, economic, or family problems associated with habitual excessive use of alcohol. Neither psychiatric symptomatology nor social consequences predict for either group the level of alcohol dependence illness. Perhaps it would be better to say that the level of alcohol dependence illness is independent of other social, economic, and familial problems in patients who volunteer for alcoholism treatment.

As might be expected, alcohol-dependent individuals with psychiatric conditions have family and childhood backgrounds that correspond with what

most textbooks describe as precursor conditions for mental illness. But such backgrounds did not predict the severity of alcohol dependence symptoms.

The information available from one outpatient program and one hospital that treated employed individuals suggests that many fewer of their patients have concurrent psychological disorders or psychiatric syndromes. For these more normal populations, personality traits have some small relationship to prognosis. Life style type and social support type also are important factors in predicting course of the illness.

These findings suggest that many public alcoholism treatment programs must often treat two conditions, alcohol dependence illness and a concurrent but independent psychological disorder. In such cases, the recommendation is that alcoholism dependence illness be described in terms of the alcohol dependence sequence, and the psychological disorder in its own appropriate nosology. In the few studies in which it has been examined, the evidence points to the psychological problems interfering with the bringing of the dependence illness under control. The evidence also suggests that the social, economic, and family response to the alcohol dependence illness is a significant factor in bringing it under control.

The summary recommendation is that alcoholism treatment program patients be given dual diagnoses when evidence exists for the presence of concurrent conditions with the presenting condition primary in the classification, e.g., alcohol dependence illness and psychopathy. This will lead to more effective treatment planning.

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16

Neuropsychology of Alcoholism
Etiology, Phenomenology, Process, and Outcome

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Abstract. A number of empirical and conceptual issues are addressed in an effort to explain the diversity of neuropsychological deficits demonstrated by chronic alcoholics. In addition to consumption characteristics and the neurotoxic effects of ethanol, evidence is marshalled to implicate nutritional deficiency, hepatic disease, congeners in the beverage, and cognitive regression as also being contributory to the manifest impairments. Moreover, predrinking disturbances are considered that may be responsible in part for the neuropsychological deficits observed in chronic alcoholics.

Our understanding of the neuropsychological concomitants of alcoholism can be increased by the adoption of a life-span approach to alcohol effects, localizing the system or region of maximal cerebral damage and relating these findings to treatment intervention. The extent to which adaptive capacity in alcoholics and social drinkers is predicted by neuropsychological test performance is of utmost importance, especially since nonalcoholic social drinkers also demonstrate a number of impairments.

1. Introduction

Central nervous system disturbance, both as a cause and a consequence of excessive alcohol consumption, is of vital importance to researchers and clinicians concerned with the prevention and treatment of alcoholism. Do, for example, certain predisposed individuals consume alcoholic beverages in order to correct, modify, or compensate for a chemical or physiological disturbance within the brain? Does long-term use of alcohol reduce the capacity of the brain to mediate cognitive and motivational processes? If so, how? Answers to questions such as these are crucial to understanding the person's ability to meet the everyday demands for personal and social adjustment. As the structure of society becomes progressively more complex, and the skills required for successful adjustment increase, the relationship between alcohol and brain function will assume ever greater importance. Thus, the presence

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of even subtle brain damage may significantly interfere with personal, social, and vocational adjustment.

It is evident that neuropsychological research has implications for understanding the etiology of alcoholism as well as for elucidating the pattern and consequence of disturbed cerebral capacity. During the past few decades, alcohol researchers have demonstrated that the integrity of the central nervous system is affected by interactions among genetic, nutritional, physiological, and biochemical factors associated with the consumption of alcoholic beverages. In this chapter, we critically review the current status of knowledge of the neuropsychological impact of alcohol use and abuse and consider neuropsychological factors that may predispose certain individuals to consume alcohol excessively. In addition, we discuss the course of neuropsychological changes in the context of the mechanisms and processes underlying the observed deficits and examine the relationship between sobriety or controlled drinking and recovery of neuropsychological competence.

2. Phenomenology

2.1. Neuropsychological Characteristics of Alcoholics

Although chronic alcoholics usually score in the "average" or "bright average" range on standardized IQ tests,[1–4] they tend to perform in the "impaired" range on neuropsychological test batteries such as the Halstead–Reitan and Luria–Nebraska. Because validation studies have repeatedly demonstrated that these test batteries reliably discriminate brain-damaged individuals from those without such damage (for review see 5,6), a poor performance on these tests is considered diagnostic of brain dysfunction. Evidence that the integrity of the central nervous system is disrupted in alcoholics has been provided by a number of investigators.[7–9] For example, Fitzhugh and his colleagues[2] reported that alcoholics obtained a Halstead–Reitain Impairment Index score that was midway between that of normals and acutely brain-damaged adults, whereas Miller and Orr,[10] in a more recent study, found that the Impairment Index of their alcoholic sample was not significantly different from that of a brain-damaged comparison group. Similar results have been obtained by Chmielewski and Golden,[11] who found that on the Luria–Nebraska Battery, detoxified alcoholics earned significantly higher Pathognomonic Scale scores than nonalcoholic control subjects.

A detailed analysis of alcoholics' performance on these neuropsychological test batteries reveals no evidence of global intellectual deterioration. When evaluated 2 or 3 weeks after the beginning of detoxification, the typical alcoholic shows normal language skills[12] and performs normally on simple perceptual tasks requiring only pattern recognition.[13–15] In contrast, detoxified alcoholics almost invariably perform more poorly than control subjects on complex tests that assess conceptual, perceptuomotor, and mnestic functions.

That is, impairments are most likely to occur when unfamiliar information must be mentally manipulated or when motor responses must be made rapidly.

Numerous studies have demonstrated that the ability to reason abstractly, test hypotheses, and formulate strategies for solving problems is most frequently disrupted by long-term alcohol abuse. Abstraction deficits have appeared on several different types of tests, including the Halstead–Reitan Category Test,[2,10,16] the Wisconsin Card-Sorting Test,[15,17] Raven's Progressive Matrices,[16,18] and Levine's Hypothesis-Testing Task.[19,20] Detailed qualitative analyses of alcoholics' behavior on these tests have demonstrated that the ability to generate or identify various concepts remains intact,[21,22] whereas the capacity to test hypotheses and persist with a particular problem-solving strategy or rule is significantly impaired. For example, Tarter[17] administered the Wisconsin Card-Sorting Test to alcoholics who had been drinking for more than 10 years and found that these subjects were more likely than controls to shift prematurely to a different (incorrect) sorting principle and were less likely to use feedback about incorrect responses to modify their behavior. Although these problem-solving deficits are most apparent when the stimulus elements are visuospatial in nature,[23] several investigators have reported that alcoholics also have difficulty on verbal abstraction tests such as the Shipley–Hartford Test.[24,25]

Deficits are also evident on tests that do not require sophisticated reasoning skills. For example, alcoholics have a great deal of difficulty searching a stimulus array for a particular visual target[26] and perform more poorly than nonalcoholics on motor tasks that demand rapid responding[27,28] and finger or manual dexterity.[29] Given these types of deficits, it is not surprising to find most alcoholics impaired on a variety of complex perceptuomotor tests including digit–symbol substitution tests,[30,31] embedded figures tests,[32,33] the Maze-Tracing Speed Test,[15] and the Trailmaking Test.[10,34] An examination of the demand characteristics of these tests reveals that all require the subject to first scan a visual array and then make some sort of motor response rapidly—two skills that seem particularly vulnerable to the effects of long-term alcohol abuse.

The ability to learn new information and retain it for more than a few minutes also appears to be disrupted in detoxified alcoholics, although this reduction in memory efficiency is relatively subtle when compared with impairments in conceptual and perceptual processes. Most researchers agree that alcoholics perform normally on standardized clinical memory tests such as the Benton Visual Retention Test or the Wechsler Memory Scale (e.g., 35–37). It is only when alcoholics are administered more difficult information-processing tests, originally developed in the experimental psychology laboratory, that mnestic deficits become detectable. For example, Mohs and his colleagues[38] used the memory-scanning paradigm of Sternberg[39] and found that alcoholics processed information in short-term memory more slowly than comparable nonalcoholic controls, and Ryan and associates[31,35,40] noted a sig-

nificant reduction in short-term memory capacity when they tested alcoholics on a variant of the Brown–Peterson distractor task.[41] These short-term memory deficits may occur because alcoholics tend to rely on relatively ineffectual mnemonics. As studies on paired-associate learning bave demonstrated,[42] the typical alcoholic is more likely to use a rote rehearsal strategy and repeat the to-be-remembered words to himself rather than employ a more sophisticated, semantically based, "mental elaboration" strategy whereby the to-be-remembered items are embedded in meaningful sentences.

In summary, it is evident that alcoholics do indeed manifest neuropsychological impairments, for they perform poorly on a variety of tasks that are sensitive to CNS dysfunction. It is also clear that within this population of subjects, a wide range of impairment exists. Although most alcoholics show signs of mild deficit, there are some who perform as poorly as neurological patients who have suffered extensive, diffuse brain damage,[43] whereas others show no impairment whatsoever. In one investigation, Wilkinson and Carlen[8] found that 20% of their alcoholic sample referred for neuropsychological testing presented insufficient evidence to implicate an organic deficit. Likewise, Goldstein and Shelly,[44] in another large-scale study, reported that almost one-quarter of their sample could not be diagnosed as brain-damaged on the basis of neuropsychological test results. The nature and extent of deficit in any particular subject is probably determined by multiple factors, including those associated directly with alcohol consumption (e.g., the amount of ethanol consumed per drinking occasion),[45] medical complications (e.g., the degree of severe liver dysfunction),[46,47] nutritional deficiencies (e.g., B_1 avitaminosis),[48] and perhaps the presence of genetic anomalies (e.g., genetically determined abnormality of a thiamine-dependent enzyme).[49,50]

2.2. Neuropsychological Competence of Nonalcoholic Social Drinkers

In many of the studies previously discussed, the performance of alcoholics was compared with that of nonalcoholic social drinkers on the assumption that this latter group is "normal." However, results from several recent investigations have seriously questioned the validity of that assumption. Parker and her colleagues[51–53] have attempted to map the relationship between social drinking practices and cognitive competence by administering both a neuropsychological test battery and a detailed drinking habits questionnaire to groups of nonalcoholic social drinkers. Surprisingly, they found that the lifetime quantity of alcohol consumed was unrelated to the performance of these subjects on cognitive tests. In contrast, low but statistically significant negative correlations were found between the amount of alcohol consumed per drinking episode and performance on the Wisconsin Card-Sorting Test, the Category Test, and the Shipley–Hartford Test, and this was true of both heavy and moderate social drinkers. Moreover, these subtle changes in cognitive functioning are not limited to older alcoholics, for Parker *et al.*[53] have noted a similar inverse relationship between amount of alcohol

consumed per drinking event and the performance of college students (mean age 22.6) on the Shipley–Hartford Test. Although the magnitude of the deficits in social drinkers tends to be relatively small, it is evident from studies such as these that moderate consumption of alcoholic beverages may deleteriously affect the neuropsychological status of persons who are not clinically diagnosed as alcoholic.

2.3. Brain–Behavior Correlates

Significant correlations have been observed between measures of brain atrophy and neuropsychological performance.[54,55] Bergman and his associates[56] have observed correlations between neuropsychological test scores and ventricular and sulcal width, and Cala and colleagues[4] have also reported that both the degree of cortical atrophy and performance on Wechsler Adult Intelligence Scale (WAIS) subtests such as Block Design, Digit Symbol, and Object Assembly were associated with the duration of drinking. A significant relationship between various morphological indices derived from computerized tomography (CT) scans and performance on the WAIS and the Halstead–Reitan Battery has also been observed by Wilkinson and Carlen.[8] In all of these studies, the correlation coefficients have tended to be rather low (though statistically significant), indicating that the severity of brain pathology is a poor predictor of psychological capacity—perhaps because psychological functions are not represented in the brain in a point-to-point fashion.[57] Although two groups of researchers[58,59] have suggested that neuropsychological test scores provide a better index of cerebral dysfunction in alcoholics than CT scan measures, it remains to be determined which of these measures is the better predictor of adaptive functioning in a natural setting, outside the laboratory.

2.4. Localization of Cerebral Pathology

To explain the distinctive pattern of cognitive deficit that is seen in detoxified alcoholics, theorists with a "structural" orientation have advanced two competing neuropsychological hypotheses.

1. Structures in the right hemisphere are more vulnerable to the neurotoxic effects of ethanol and its metabolites than are structures in the left hemisphere.
2. Structures in the anterior–basal (frontal–diencephalic) region of the brain are more vulnerable to damage resulting from chronic alcohol abuse than are other cortical and subcortical structures.

2.4.1. Right Hemisphere Hypothesis. Because alcoholics are impaired on a wide range of visuoperceptual tasks, yet perform normally on most verbal tasks, several researchers have speculated that neuropsychological changes are lateralized primarily to the right hemisphere.[60,61] Unfortunately,

the neuropsychological data are not consonant with the neuroradiological findings. Although it is generally agreed that most alcoholics show more cortical and subcortical atrophy than do age-matched nonalcoholic controls,[8,59] there is no evidence that one hemisphere is more affected than the other. It is likely, as several writers have recently pointed out,[62,63] that alcoholics perform so poorly on "right hemisphere" tasks because these sorts of tasks (e.g., scanning a complex visual array as rapidly as possible to locate a "hidden" geometric design) require skills that are less well practiced than those required by the typical "left hemisphere" task (e.g., defining words or answering general information questions). That is, visuoperceptual tasks tend to be more difficult than verbal tasks, and as a consequence, they are more vulnerable to any generalized reduction in mental efficiency. Poor performance on a battery of so-called right hemisphere tests may not signal right hemisphere pathology; rather, it may be an early indicator of bilaterally distributed cerebral damage.

2.4.2. Anterior–Basal Hypothesis. To account for the fact that alcoholics show significant impairments on tests measuring motor regulation,[64] visual scanning,[26] hypothesis testing,[19] and learning and memory,[40] Tarter[23,65,66] has postulated that chronic alcohol abuse produces damage to a functionally and structurally integrated neurophysiological system. This network of nuclei and fiber tracts, located within frontal, limbic, and diencephalic regions of the brain,[67] is considered to be critical for programming and regulating complex patterns of behavior.[68] Both animal and human studies have demonstrated that lesions within various parts of this system will produce many of the same deficits seen in detoxified alcoholics, including impairments on tasks requiring the modulation of motor responses,[69] visual searching,[70] problem solving,[71] or learning.[72] Additional support for this hypothesis has come from a number of neuropathological and neuroradiological studies which have indicated that when alcoholics show evidence of brain damage, it appears most prominently (but not exclusively) in frontal[4,73] and diencephalic[55,74] regions of the brain.

Despite the many behavioral similarities between alcoholics and neurological patients with anterior–basal lesions, we are reluctant to conclude that alcohol and its metabolites selectively disrupt the integrity of this frontal/limbic/diencephalic system. As Ryan and Butters[63] have pointed out, the deficits characteristic of these two patient populations are not unique to them but can be found in neurological patients with moderately large lesions anywhere in the brain[57,75] as a result of the rich network of interconnections between this anterior–basal system and other cortical and subcortical structures.[68] Consequently, it is not possible to ascribe psychological functions to a single locus within the brain solely on the basis of neuropsychological test results. Given the fact that cortical atrophy is not limited to a single region but is found in all areas of the brain,[8,54,76,77] it is most parsimonious to conclude that alcoholism eventuates in neuropathological changes diffusely distributed throughout the brain.

3. Etiology of Neuropsychological Deficit

There are two separate problems concerning the etiology of the neuropsychological deficits in alcoholics. The first pertains to those factors associated with the consumption of alcoholic beverages that directly and indirectly produce central nervous system dysfunction. The second pertains to neuropsychological disturbances that may be extant prior to the onset of drinking and thus involve either inherited or acquired cerebral disorders.

3.1. Alcoholic Beverage-Induced Neuropsychological Impairment

The consumption of alcoholic beverages places the person at risk for manifesting neuropsychological deficits via a number of possible mechanisms.

1. The ethanol in alcoholic beverages is neurotoxic, and the brain damage it produces is expressed as a set of cognitive deficits.
2. Ethanol causes liver damage, and the resulting metabolic derangement, rather than the action of ethanol *per se*, is primarily responsible for neuropsychological impairments.
3. Consumption of alcoholic beverages interferes with the intake, absorption, and utilization of essential vitamins and leads to nutritional imbalances which, in turn, are responsible for the behavioral deficits.
4. The congeners in alcoholic beverages have direct neurotoxic effects.
5. Nonbiological factors, such as a regression on the part of the alcoholic to rely on more concrete information-processing strategies, contribute to the severity of the cognitive deficit.

3.1.1. Ethanol Neurotoxicity. The empirical support for a direct neurotoxic effect comes entirely from animal experimentation in which optimal control over nutritional and metabolic factors can be maintained. For example, Freund and his colleagues examined the relationship between alcohol consumption and learning efficiency by feeding mice and rats nutritionally adequate liquid diets containing 35% ethanol for periods ranging from 3[78] to 9 months.[79] Approximately 2 weeks after alcohol feeding was discontinued, animals were tested in a shuttle box avoidance learning situation. It was found that ethanol-consuming animals learned more slowly than pair-fed sucrose-consuming controls, and the severity of this deficit was positively correlated with the duration of alcohol consumption.[80] Moreover, this learning impairment appears permanent, for ethanol-consuming animals tested after a $4^1/_2$-month period of "sobriety" performed more poorly than age-matched controls.[80] At the neuronal level, investigators have found a decrease in the number of dendritic spines and a reduction in the density of dendritic trees in ethanol-consuming mice.[81] Although these changes are seen in a number

of areas of the brain, they are most apparent in the hippocampus and dentate gyrus.[81]

It is apparent from this research that even when nutritional factors are controlled, brain damage can be induced by ethanol or its metabolites. This latter point must be emphasized, inasmuch as the toxic agent may be acetaldehyde[82] rather than alcohol. Although its exact mode of action remains unknown, it is possible that ethanol affects brain tissue primarily by inhibiting protein synthesis.[83,84]

3.1.2. Role of Liver Disease. Disturbed liver functioning in the form of steatosis (fatty liver), parenchymal inflammation (hepatitis), and cirrhosis frequently accompanies chronic alcoholism. A number of investigators have suggested that hepatic encephalopathy, as an acute complication of cirrhosis, may exist in a low-grade chronic form that causes impairments in neuropsychological capacity.[85] In one recent test of that hypothesis, Gilberstadt and his colleagues[46] administered the WAIS, the Trailmaking Test, the Speed of Writing Test, and a reaction time test to groups of alcoholics with and without cirrhosis. Although verbal capacities were found to be preserved in both groups, the cirrhotic subjects performed more poorly on the Block Design, Digit Symbol, Speed of Writing, and reaction time tests than did the noncirrhotic alcoholics and earned significantly lower performance IQ scores. These differences in performance could not be attributed to alcohol consumption *per se*, for both groups of subjects had similar drinking histories. However, the fact that statistically reliable correlations were found between a number of these behavioral measures and several indices of liver dysfunction, including serum albumin level and fasting venous ammonia level, strongly suggests that alcohol-induced liver disease can have a profound effect on intellectual functioning.

The relationship between liver disease and neuropsychological test performance has been observed by a number of other investigators. Smith and Smith[47] found that a group of cirrhotic alcoholics performed more poorly than a group of noncirrhotic alcoholics who, in turn, earned lower scores than a group of nonalcoholic control subjects. Of particular interest is a study by Rehnstrom *et al.*[86] They administered a series of cognitive tests to a group of cirrhotic alcoholics and found that those patients who had been treated with a portosystemic shunt procedure showed significantly less impairment than cirrhotics whose liver disease was not treated with this surgical procedure. Similar results obtained by Rikkers and associates[87] suggest that the intellectual changes associated with liver disease are not permanent but may reverse or reduce following a surgically induced improvement in liver function.

3.1.3. Nutritional Status. Liver functioning and nutrient metabolism are synergistically related processes, so that a disturbance in one invariably produces changes in the other. As a consequence, it is not practicable to delineate the relationship between liver pathology and neuropsychological impairment without taking into consideration the possibility that alterations

in nutrient metabolism produced by liver disease may be directly responsible for the cognitive deficits seen in cirrhotic alcoholics. Because the liver normally serves as a storage depot for vitamins, and because it regulates processes concerned with the absorption and utilization of vitamins, any derangement in liver function may produce significant nutritional deficiencies. For example, animal studies by Sorrel *et al.*[88] have demonstrated that perfusion of the liver with alcohol triggers the release of stored vitamins. Similarly, Leevy and associates[89] found reduced blood levels of B-complex vitamins in 49% of their sample of alcoholics with cirrhosis and in 44% of their alcoholics with fatty liver. In contrast, only 32% of the alcoholics with normal liver functions showed this alteration of B vitamin levels. These data indicate that as liver functions become more seriously compromised, there is an accompanying reduction in this important vitamin complex.

Although liver disease plays an important role in the development of nutritional disturbances, an inadequate diet is the most obvious cause of subclinical vitamin deficiencies in alcoholics. Unfortunately, it is virtually impossible to obtain reliable indices of nutritional intake from these subjects. Many alcoholics, particularly those who have developed chronic gastritis,[90] consume nutritionally adequate diets only sporadically, relying instead on alcoholic beverages to supply them with necessary calories. Yet, even if alcoholics consume a nutritionally adequate diet, they may still manifest vitamin deficiencies because of their inability to absorb sufficient nutrients from food.[91,92] Direct measurement of circulating levels of vitamins in the bloodstream does not completely resolve this problem, because those values typically reflect only very recent diet, and provide no information about the efficiency with which these nutrients are being absorbed and utilized. There is presently no objective measure for determining prior history of nutritional functioning in the now healthy alcoholic.

Despite the many methodological problems involved in assessing the relationship between nutrition and behavior, a number of investigators have demonstrated that B avitaminosis can produce neuropsychological and neuropathological changes in both humans[93] and animals.[94] The best known example of this is provided by the Wernicke–Korsakoff syndrome, a neurological disorder associated with lesions in the mammillary bodies and medial dorsal nucleus of the thalamus[95] and characterized by a profound anterograde amnesia, visuoperceptual and abstract-reasoning impairments, and changes in olfactory acuity and affect.[96,97] Although this syndrome is most frequently found in malnourished alcoholics, it is by no means limited to that group, having also been identified in nonalcoholic patients with a history of malnutrition produced by pernicious vomiting,[98] gastric carcinoma,[99] or starvation.[100] Similar learning and memory deficits have recently been induced in nonhuman primates by feeding rhesus monkeys thiamine-deficient diets for several months.[101] To what extent nutritional deficiency accounts for the neuropsychological deficits seen in alcoholics who have not developed Korsakoff's

syndrome remains unknown, though preliminary work by Guthrie and Elliot[102] suggests that alcoholics with clinical evidence of malnutrition tend to show more neuropsychological deficits than those who are not malnourished.

3.1.4. Congeners. The various additives, flavors, and impurities in alcoholic beverages are collectively known as congeners, and there is now sufficient empirical evidence to indicate that such substances contribute to the acute effects of beverage alcohol on the central nervous system of man. For example, Murphree and Price[103] examined electroencephalographic changes induced by three types of distilled beverages having low (vodka), moderate (bourbon), and high (synthetic "superbourbon") concentrations of congeners and found that those beverages having large amounts of congeners produced greater and longer-lasting depressant effects than beverages having very small amounts. They hypothesized that these congener-induced changes may affect the severity of hangover symptoms and may also be responsible for some of the histopathological damage found in the brains of chronic alcoholics. Unfortunately, this latter hypothesis has not been tested systematically, for no investigator has yet examined whether alcoholics who have consumed a specific type of beverage (e.g., beer, wine, whiskey) exclusively over a long period of time will show a unique pattern of neuropsychological and neuropathological change.

3.1.5. Nonbiological Factors. The cardinal assumption made in any neuropsychological evaluation is that the performance decrements that appear on testing are primarily a function of structural damage within the central nervous system. However, that assumption may not be completely accurate, since it is now known that factors other than cerebral pathology can deleteriously affect performance on neuropsychological tests. For example, affective illnesses such as depression may seriously impair learning, memory, and problem-solving capacity.[104,105] Given the prevalence of depressive symptomatology in heavy drinkers,[106] it would not be surprising to find that depression has also contributed to the pattern of deficits seen in chronic alcoholics, although this hypothesis has not yet been tested in a systematic fashion.

In a recent review article, Ryan and Butters[63] have described another nonbiological factor that may be partly responsible for the failure of the typical detoxified alcoholic to perform normally on learning, memory, and problem-solving tasks. They have speculated that as a result of thousands of episodes of intoxication, the alcoholic has acquired a set of information-processing strategies that are less efficient than those ordinarily used by nonalcoholic adults. Evidence supporting this view has come from a number of sources. For example, it is known that the efficiency with which social drinkers learn new information is significantly reduced during a single episode of intoxication[107,108] and that a qualitatively similar change in learning is found in detoxified alcoholics.[42] Although it is likely that the neuropharmacological action of ethanol and/or its metabolites is directly responsible for how intoxicated individuals process information, this same pharmacological mechanism may not be solely responsible for the similar information-processing changes

that are seen in alcoholics who have been detoxified for several weeks. Ryan and Butters suggest that the frequently inebriated individual may become so habituated to using these less effectual information-processing strategies, perhaps because less mental effort is required,[109] that he generalizes and begins to use them in other situations, even when he is not intoxicated. This is a very intriguing hypothesis; unfortunately, the absence of systematic empirical research renders it speculative at the present time.

3.2. Antecedent Neuropsychological Characteristics

It is now well established that children and first-degree relatives of alcoholics have a greater risk of becoming alcoholic than the general population.[110,111] Efforts to identify genetic markers have been largely unsuccessful from studies of blood typing, color blindness, chromosome analyses, and other biochemical assays. Recent attempts have, however, tentatively implicated certain neurobehavioral characteristics in alcoholics and their relatives that support the hypothesis of disturbed neurological organization. The behavioral disorders described below may thus constitute markers for an inherited predisposition to become alcoholic.

One such marker has been reported by Lipscomb, Carpenter, and Nathan.[112] They measured static ataxia (body sway at rest) in a group of young adults (mean age 20.7) and found that those subjects whose first-degree relatives had a history of alcoholism tended to sway more than those who had no alcoholic relatives. The degree of body sway was not related to the subject's history of drinking or current drinking practices. Since this was not a prospective study, it is impossible to determine whether those who showed the greatest static ataxia are more likely to be diagnosed as alcoholics 5 or 10 years hence. Nevertheless, these results suggest that the motor impairments often seen in alcoholics[64,113] may to some extent be innately determined characteristics of the individual which are present prior to the onset of heavy alcohol consumption.

Another avenue of research has been pursued by Tarter,[66,114] who hypothesized that a childhood history of hyperactivity and/or minimal brain dysfunction may be an etiologic factor that increases the risk of future alcoholism. In one study (114), alcoholics were categorized as primary or secondary drinkers on the basis of a drinking history questionnaire. Individuals were considered to be primary alcoholics if they reported (1) increased alcohol tolerance, (2) withdrawal symptoms, (3) positive psychic effect (euphoria, relaxation) after the first drinking experience, (4) positive psychic effect after the first drink following a period of abstinence, (5) absence of a history of social drinking, (6) abnormal drinking prior to age 40, (7) personal and interpersonal problems from alcohol prior to age 40, and (8) loss of control. Those who did not report at least six of these characteristics were assigned to the secondary alcoholic group. Both groups of subjects were administered a checklist of symptoms associated with minimal brain dysfunction, and it

was found that the primary alcoholics endorsed significantly more symptoms of hyperactivity and minimal brain dysfunction than the secondary alcoholics. Secondary alcoholics, in turn, earned scores that were similar to those obtained by nonalcoholic control subjects. The primary group had a higher familial incidence of alcoholism than the secondary group and showed less evidence of psychopathology on the Minnesota Multiphasic Personality Inventory.

These findings indicate that primary alcoholism may represent a genetic variant of which hyperactivity is a putative behavioral precursor. In support of this conclusion, Tarter *et al.*[114] also observed that the primary alcoholics had higher scores on the MMPI Hypomania Scale and also scored significantly higher on the MacAndrews Alcoholism Scale. These basic findings have been replicated in a more recent series of studies.[115] Again, primary alcoholics were found to be more impulsive, psychosocially immature, and extroverted and had a more extensive history of drinking. Because perceptual and cognitive capacities did not differentiate between these two groups, it is likely that the critical characteristics are related to the control and intensity of behavior rather than to higher-order cognitive competence.

Other investigators have also reported an association between hyperactivity and alcohol abuse. Blouin, Bornstein, and Trites[116] compared hyperactive children with controls who were not hyperactive but had school adjustment problems. At a 5-year follow-up, it was found that the hyperactives consumed more alcoholic beverages than the controls. Data presented in several other studies[117–119] also indicate increased incidence of hyperactivity in persons with drug and alcohol abuse problems. Goodwin and associates[120] have reported that alcoholics reared away from their biological parents had more symptoms of hyperactivity as youngsters than did a control group. Familial studies conducted by Cantwell[121] and Morrison and Stewart[122] have also revealed an association between childhood hyperactivity and parental alcoholism. These investigations suggest that there may be a common genetic basis for both alcoholism and hyperactivity, with the latter occurring early in childhood and serving as a predisposing risk factor for alcoholism in adolescence and early adulthood.

4. Process

4.1. Life-Span Effects on Neuropsychological Capacity

Because the development and maintenance of psychological functioning is continuously occurring throughout life, it is important to know if the relationship between alcohol intake and intellectual impairment is more pronounced at one age than at another. Unfortunately, evaluating the life-span effects of alcohol on neuropsychological capacity is a difficult task because age and consumption variables are inextricably confounded. Children, for example, either do not drink or drink only occasionally, whereas adult social

drinkers typically engage in highly idiosyncratic patterns of alcohol consumption—patterns that are determined to a very large extent by peer influences, situational factors, and cultural background. Despite the many methodological problems inherent in this area, a series of recent investigations has examined interactions between alcohol and cognitive competence at three critical points on the life-span: prenatal development, young adulthood, and old age.

4.1.1. Prenatal Effects. The teratogenic effects of alcohol have now been well established. As few as two to three drinks per day have been found to place the offspring at risk for psychological, physical, and central nervous system disturbances in what has been described as the *fetal alcohol syndrome* (FAS).[123] The severity of the physical anomalies and other disturbances appears to be related to the quantity of alcohol consumed by the gestating woman, although other metabolic factors and toxic agents may also contribute to the pathology. The behavioral and neurological sequelae are variable and extensive. Mental retardation, learning difficulties, attentional problems, and hyperactivity have all been reported.[123] Other indices of disturbed CNS functioning such as body tremors and reduced vigor have also been described.[124] Over 50% of FAS children exhibit hypotonia, and about 80% are microcephalic.[125] Learning an operant response is slower,[123] and developmental milestones are often delayed.[126]

There is thus substantial evidence that alcohol consumption during pregnancy is related to morphological, psychological, and behavioral disturbances in the offspring. Since not all children are affected, one must conclude that drinking during pregnancy places the child "at risk." Whether a particular child will actually manifest this syndrome is probably a function of constitutional vulnerability in the developing fetus, maternal drinking, and other maternal characteristics (e.g., nutritional deficiency, nicotine and caffeine ingestion, environmental stress). Although the actual mechanism responsible for the appearance of this disorder in affected individuals remains uncertain, it is clear that exposure to alcohol during the fetal stage of development may significantly impair neuropsychological competence during childhood.

4.1.2. Young Adulthood. The neuropsychological capacity of drinkers between the ages of 20 and 40 has been assessed in a number of recent studies. Blusewicz and his colleagues[9] administered the Halstead–Reitan Neuropsychological Battery to a group of young alcoholics (mean age 33) and to groups of nonalcoholic control subjects and found that the alcoholics performed more like older controls (mean age 71) than like younger controls (mean age 31). Other investigators have reported that young alcoholics exhibit both clinical and neuroradiological signs of pathology. For example, Lee and associates[59] examined neuropsychological and CT scan data from a group of alcoholics under the age of 35 and found evidence of intellectual deficits in 59% of their sample and evidence of cerebral atrophy in 49%. Similar findings have been reported by Hill and Mikhael[58] who found that alcoholics with a mean age of 35 years performed more poorly than nonalcoholic controls on a number of tests measuring abstraction and visuospatial ability. From these investiga-

tions, it can be concluded that neuropsychological deficits are extant in even young alcoholics.

4.1.3. Old Age. The vulnerability of the aging brain to alcohol has not been studied systematically, and so it is not currently possible to determine whether people who begin drinking late in life show the same sorts of impairments that are found in individuals who began drinking early in life. It is clear, however, that the severity of alcohol-related intellectual changes is greater in elderly subjects. For example, Horvath[43] studied a sample of 1100 patients and found that approximately 9% of them showed evidence of dementia. He noted that the incidence of dementia increased with age and was three times more common in alcoholic women than men. Those patients with an alcoholic dementia manifested a heterogeneous set of symptoms, including amnesia for recent events (95% of cases), affective disturbance (81%), inappropriate behavior (85%), disorientation in time (76%) and place (61%), dyspraxia (56%), confabulation (38%), dysphasia (33%), and perseveration (25%). This pattern of deficits differs from that seen in the patient with Korsakoff's syndrome and from that seen in younger alcoholics. Of particular interest is the observation that those who suffered from dementia consumed more alcohol per drinking occasion and had a longer history of excessive drinking than those not so afflicted.

4.2. Consumption Factors

Little is known of the relationship between drinking patterns and neuropsychological performance. Do binge drinkers, for example, manifest a more or less severe pattern of deficit than steady or continuous drinkers? This sort of question has not yet been addressed by researchers in any systematic way. However, two features that have been examined are duration of alcoholism and quantity consumed per given unit of time. Although precise dose–response relationships have not been established, the available evidence nonetheless suggests that the degree of neuropsychological impairment is somehow related to the quantity of alcohol consumed. For example, scores on neuropsychological tests have been found to be correlated with the duration of alcoholism,[17] the amount of alcohol consumed during the previous year,[45] and the quantity consumed during a typical drinking occasion.[51,52] Very recent work by Parker and her colleagues suggests that the association between consumption and performance is not unique to alcoholics but can be seen in young social drinkers as well.

5. Outcome

It is clear from our review that chronic alcohol abuse usually eventuates in neuropsychological deficit. Are these impairments permanent sequelae of drinking, or will intellectual capacity recover over time? This issue is of vital

interest to mental health specialists working with alcoholics because new modes of treatment might have to be developed if the cerebral changes induced by a history of alcohol abuse rendered the individual incapable of responding to the demands of traditional psychotherapeutic intervention. Another related issue of major importance pertains to the degree to which the brain is capable of utilizing its residual capacity for developing alternative coping strategies.

5.1. Neuropsychological Recovery

Neuropsychological improvement has been reported on numerous occasions (for reviews, see 62,63,66). Tarter[66] theorized that recovery is a multistage process that involves a reversal of deficits caused originally by a number of different mechanisms. He has hypothesized that the first phase of recovery is the dramatic improvement associated with detoxification. Subsequent improvement occurs after about 6 months and then again after about 1 year. In discussing the various processes underlying these changes, Tarter speculated that a restabilization of nutritional and biochemical status is responsible for the improvement that occurs approximately 6 months after the beginning of sobriety. Tentative support for this notion is provided by data reported by Guthrie and Elliot.[102] Improvements in liver functioning may be another factor contributing to recovery at this stage, although this possibility has not been empirically verified. There is, however, evidence that suggests that alcohol-induced liver disease can deleteriously affect cerebral integrity and in turn impair neuropsychological performance even after sobriety has been achieved.[46]

Continued recovery of capacity has been noted in subjects who have been abstinent for a year or more. The mechanisms underlying recovery at this stage are unknown but may in part be the result of synaptogenesis.[127] Improvement may also follow from the relearning of cognitive strategies that were discontinued during the period of heavy drinking.[63] Maximizing the use of remaining brain tissue may be another mechanism responsible for recovery of function in abstinent alcoholics. Nevertheless, despite the improvements that do occur, the neuropsychological test scores of these individuals never become "normal," that is, never reach the level of a group of nonalcoholic control subjects.[128–130] Whether or not the impairments are completely reversed after several years of sobriety has not been determined, although it is clear that after 1 year of continuous sobriety, alcoholics show impairments on certain tasks. The deficits observed at that time appear most frequently on tests of memory, abstraction, and visuospatial capacity.[130,131]

5.2. Treatment Outcome

Only a handful of studies have examined the relationship between neuropsychological functioning and treatment outcome. One such study[132] found

that the alcoholic who responded best to insight forms of therapy was most adept psychologically, whereas the more concrete alcoholic, who was less able to think abstractly, benefited most from directive forms of intervention. In contrast, a number of other studies have not found a clear association between performance on neuropsychological tests and treatment outcome.[133] One reason for the failure of neuropsychological tests to predict outcome may be that these tests are more sensitive to cognitive, rather than motivational, variables. In addition, it may not be possible, given the current stage of knowledge regarding the content and processes of therapy, to relate such global dimensions of behavior change to more discrete components of brain function. Although at first glance it would appear that the potential to profit from therapy would be related to neuropsychological competency, as yet there is no substantial evidence that this is indeed the case.

In conclusion, neuropsychological capacity does not appear to be related to treatment outcome, although it seems reasonable to speculate that performance on such measures may, along with other variables (e.g., motivation, personality), increase our ability to predict which patients will benefit most from particular treatment methods. The research on neuropsychological recovery strongly indicates that improvement occurs over time but is not complete. Some tentative evidence exists that indicates that recovery is a multistage process that is tied to a number of different biological, and possibly psychological, processes.

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