DRUGS AND DRIVING

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DRUGS AND DRIVING

INTRODUCTION

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This special issue of *Accident Analysis and Prevention* evolved from a symposium on Drugs and Driving presented at the 1975 annual meeting of the U.S. Transportation Research Board, sponsored by the Committee on Road User Characteristics. The impetus for the meeting was the increased frequency of drug use among ever-widening portions of the population. Epidemiological studies indicate that driving occurs routinely during periods when individuals are under the active influence of drugs.

The first five papers in this issue (Gordon, Hurst, Linnoila, Moskowitz and Sharma) reviewed current literature for five classes of drugs regarding the potential driving safety hazards associated with those drugs. The drug groups reviewed were amphetamines, tranquilizers, barbiturates, narcotics and cannabis.

In examining this group of papers, it becomes clear that statements about the effects of drugs in general with regard to driving safety can rarely be made. Rather the papers point to the necessity for evaluating each class of drugs and their individual members. Different drugs affect disparate behavioral mechanisms. Moreover they are likely to be used at different times in differing social contexts with varying motivations and expectations. These latter considerations will differentially influence the likelihood of a particular drug degrading traffic safety.

The papers make apparent that our knowledge is quite limited even in the most frequently studied of these drug classes. Most conspicuous by their absence are epidemiological studies based on samples taken from accident participants and corresponding driving control groups. It was only by the execution of such studies for alcohol, as those by Borkenstein and Haddon, that the extent of the contribution of alcohol to traffic accidents, injuries and fatalities was understood.

Unfortunately, it is far more difficult to obtain adequate samples for drug analyses than for alcohol. Moreover, current laboratory analyses have difficulty in detecting the wide range of possible drugs which may be present.

The review papers reveal the sparseness of relevant data regarding the nature and extent of the behavioral side effects of many drugs. This is especially true of the behavioral effects that are directly relevant to driving, such as the areas of psychomotor performance, and perceptual changes. However, despite the incompleteness of the data base, it appears that at least three out of the five classes of drugs discussed are likely to lead to impairment of driving skills, namely, tranquilizers, barbiturates and cannabis. More definitive statements, which might distinguish between the members of these drug classes and assign an accident probility score to each, must await further research.

The three papers which follow the review papers are representative examples of research currently being conducted on drugs and driving. Smart and Fejer studied licit and illicit drug use, driving patterns and accident rates among a high school student population. Maki and Linnoila examined accident rates as a function of types of prescription drugs used by various categories of patients. Moskowitz, Hulbert and McGlothlin measured the impairment of behavioral skills by marihuana using a driving simulator. A more extensive series of such studies for a greater variety of drugs is clearly necessary.

The issue concludes with two papers—one by Zador, and one by Johnson, Levy and Voas—in which some current countermeasure programs against the drunk driver are evaluated. We have passed well beyond asking whether alcohol is dangerous for driving—we know that it is. However, we do need further information regarding the classes of drivers who drink, their

drinking-driving patterns, the nature of the impairment produced by drinking and how to organize this knowledge into effective countermeasure programs.

In response to the growing social need to deal with the problems of the drinking driver, various countermeasures have already been incorporated into many programs throughout the country. Considerable social resources have been allocated by federal, state and local communities, in an attempt to alleviate the damage resulting from drunk driving. Understandably, those agencies which bear the expense of these programs are interested in determining the effectiveness of the countermeasures, and whether the benefits are worth the cost.

The papers by Zador and by Johnson *et al.* point out the difficulties in choosing effective countermeasure programs, and of measuring the effectiveness of such programs. The papers in this issue are a representative sample of the state of the art of research in drugs and driving.

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INFLUENCE OF NARCOTIC DRUGS ON HIGHWAY SAFETY†

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Abstract—This is a review of available literature relevant to narcotic drug use and driver safety. No studies were found that directly assessed driver capability either in driving simulators or actual driving conditions. There are some studies of illegal users' driving records, including interviews with methadone treated ex-heroin addicts, and laboratory studies of effects of narcotics on skills related to driving. The weight of evidence from available studies indicates that narcotic users do not have driving safety records that differ from age matched individuals in the general population. Furthermore maintenance on methadone (a synthetic narcotic used to treat heroin addicts) does not provide a risk for driving.

A wide variety of substances have been identified as narcotics. The usual standard of comparison is morphine, one of the alkaloids extracted from opium. The comparisons leading to the classification of a substance as a narcotic include manifestation of analgesic and other physiological effects, as well as the manifestation of cross-tolerance and cross-dependence with morphine. In other words, a narcotic drug should, when substituted for morphine, relieve symptoms of withdrawal in morphine tolerant individuals. A narcotic drug also leads to tolerance and physical dependence in its own right [viz. Jaffe, 1970].

Narcotic drugs have important and legitimate medical uses. However, the capacity to provide euphoric and calming experiences to users, and to lead users to develop tolerance and dependence rapidly, has resulted in a variety of epidemic and compulsive usage of certain narcotics, such as morphine and heroin. Recently methadone, a synthetic narcotic (originally developed in Germany during World War II) has come into widespread use in our own country, as well as in others, as part of the treatment for heroin addiction[Dole and Nyswander, 1965]. The addiction liability of methadone was described in detail by Isbell *et al.* [1948]. The major reasons for the use of methadone in treatment in heroin addicts are two of the properties possessed by the drug—first it is longer acting than heroin or morphine; and secondly, it is effective when taken by mouth (heroin and morphine are relatively ineffective by the oral route). Methadone provides a medication which prevents the experience of withdrawal symptoms, and does not lead to the euphoric high states which addicts seek to experience, when taken as prescribed in treatment programs. Methadone is effective for a period of 24–36 hours before a new dosage is necessary. Stabilized methadone patients are given their doses orally, once each day. Each daily dose is taken prior to the time when withdrawal would be experienced. Patients are thus not sufficiently abstinent to experience a euphoric state or to suffer withdrawal. After a period of stabilization on the drug, most patients report that they "feel normal," or "straight." An additional advantage of methadone is that when taken in the manner described, and an individual is stabilized on a given dose, it is possible to maintain the same dose level for a long period of time. In most cases of self-administered use of narcotics the opposite is found—doses can rarely be stabilized.

It is estimated that there are currently about 100,000 individuals maintained on methadone in the United States. In addition, the number of individuals using heroin and other narcotics is estimated at over 250,000.

Prior to discussing the relevance of narcotics to driving performance and safety, an estimate of the number of those likely to be drivers is in order. In 2 studies in which official driving records of known addicts were searched for in the files of the N.Y. State Motor Vehicle Bureau[Babst *et al.*, 1969, 1973], about 25% had records of driving licenses. Extrapolating the percentage found to have licenses to an estimate of 350,000 individuals regularly using narcotics, including legitimate use by methadone patients, leads to a minimum estimate of about 80,000 who

†The preparation of this paper was supported by grant DA 00032 from the National Institute of Drug Abuse.

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drive. Beyond this minimum estimate, one can only guess at the number of those who drive without a license.

It also seems in order to discuss certain relevant facts about the action of narcotics. Narcotic drugs have different effects when they are taken in single doses in non-tolerant individuals, in contrast to the effects after repeated use. Single doses of any narcotic can only be taken in relatively small quantities and may lead to sleepiness, frequently accompanied by mild dizziness, nausea and "mental clouding"[see Beecher, 1957]. Regular narcotics users become tolerant to many of the narcotic effects and must take much larger doses to get the particular effect they seek. Accompanying the development of tolerance is a gradually increasing tendency to develop dependence on the drug. This in part is due to the fact that after a period of use, the user experiences withdrawal symptoms when deprived of the drug. As dependence on the drug increases, the behavior of most users becomes altered to the point where their lives become centered around seeking for and administration of the drug. This has been referred to as narcotic addiction.

Despite the considerable concern that has been devoted to the problem of narcotic addiction, relatively few studies exist which assess the functional capacity of persons who use them. In the past, experimentation was restricted to those confined in hospitals or prisons; and recently with the advent of methadone treatment, it has been possible to study larger numbers of individuals engaged in regular daily pursuits.

The research literature relevant to driving ability and safety consists of two types of study: one, which involves records of accidents and driver offenses, sometimes supplemented by interviews with the drug user, and the other involving studies of skills relevant to driving such as reaction-time, psychomotor performance and vigilance. In addition there is some more general literature in which illegal drugs are implicated as contributing to driving offenses and accidents, but it is difficult to identify the specific findings with respect to narcotics.

General references

Jaffe [1970] provides a very good general presentation of the effects of narcotics from a pharmacological and medical point of view. Uhr and Miller [1962] present theoretical and methodological examples relevant to the evaluation of narcotic effects. Beecher [1959] presents research on the subjective effects of single doses of narcotics. Forkes [1972] presents information relevant to the consideration of the impact of drugs including narcotics on driving skills.

Driving records of narcotics users

Crancer and Quiring [1968], Finkle [1969] and Brownstein *et al.* [1968] present data which tend to show that drug abusers have poorer driving records than the general population, or of non-drug users; and that the use of drugs makes the abuse of alcohol worse. On the other hand, Waller [1965] and Moser *et al.* [1970] have found that drug users (including users of narcotics) do not have accident rates† that exceed those expected in the general population. However data concerning narcotics user is not always clearly differentiated from drug abusers as a whole in these studies.‡

Two studies have specifically studied both heroin and methadone users with respect to driving records. Babst *et al.* [1973] studied the driving records of a sample of 440 methadone patients drawn from the files of the New York State Motor Vehicle Bureau. The records were studied for an average of some 40 months while the patients were heroin addicts, and for some 20 months while they were in methadone treatment. When drivers' ages are taken into account, accident and conviction rates during both periods did not differ from similar rates applicable to New York City drivers drawn from the files at random.

Using an improved methodology including interviews with 1500 methadone patients and over 1000 contrast individuals named by the patients as being drug free, Blumberg and Preusser [1972]

†Moser [1970] found that illegal drug users were no worse as drivers than non-drug users. Waller [1965] found that illegal drug users had higher violation rates than a control sample, but accident rates that did not differ from expectations when properly age adjusted.

‡As an example Waller and Goo [1969] present analyses of accident and citation-types by medical condition including "illegal drug use" but narcotics users are not differentiated from sample of illegal drug users. Waller [1970] points out the need for better analyses of drug effects on driving performance.

found that neither accident records nor driving offenses for the two groups differed. The 1500 patients estimated they drove 18,000 miles annually while they were heroin addicts and these same patients estimated their annual driving to be over 12,000 miles after becoming methadone patients. Patients' estimates of accidents and convictions were corroborated by records drawn from the files of the New York State Motor Vehicle Bureau. Furthermore, there was no evidence that the accident rates of either heroin or methadone users differed from those of average drivers in New York State.

The weight of evidence from the above studies is that narcotics users do not present a driving risk. Blumberg and Preusser [1972] suggest that the heroin user, conscious of the fact that an arrest for a driving offense may lead to discovery of drug use and/or possession, compensates by using greater caution while driving.

Laboratory studies relevant to driving skills

There are practically no studies which have attempted to assess the functional capacity of narcotic addicts. This is undoubtedly due to the fact the self-administered use of narcotics is and has been illegal for some time. Medical use of narcotics is restricted to the treatment of post-operative conditions, or those individuals who suffer from pain as a consequence of chronic illness such as cancer. The effects of single doses of narcotics elevate reaction-time [Wikler, 1965] and the user experiences "mental clouding" [Beecher, 1959]. It is highly unlikely that a person who has received a 10 mg dose of morphine would be motivated to engage in psychomotor activity. In any case, no studies have been found which attempt to evaluate narcotic effects on a dose-response basis.

Frazer *et al.* [1963] administered heroin to addict patients who volunteered for study, and found that chronic administration led patients to show a "pronounced tendency to retreat from all forms of activity and social contacts..." Their conclusion, based on clinical observations and pursuit rotor tests, was "that the depressant effects on activity observed during chronic heroin administration were not due to debility or to psychomotor impairment, but rather suggest a reduced responsiveness of the patient to ambient stimuli."

When methadone treatment was begun in 1965, it was not known how the repeated administration of relatively large doses of a narcotic would effect performance once patients were stabilized on the drug. Since it was presumed that the treatment might be continued for long periods of time, a program of research was initiated by the present writer to examine psychomotor and intellectual functioning of methadone treated individuals, who were stabilized on methadone in doses that averaged 100 mg/day. In most of the studies the individuals studied had been in treatment for 1 year or more. They were outpatients at methadone clinics and were engaged in a wide variety of activities including driving automobiles. The studies conducted to date are summarized below and a fuller presentation of our research is presented in Gordon [1973].

We used the rotary pursuit task [viz. Uhr and Miller, 1962] in our earliest studies with the first methadone patients studied [Gordon *et al.*, 1967]. In these studies performance on the task was observed for periods up to a year, largely to observe consistency in performance as well as to derive comparisons with non-drug using individuals. We also wanted to see if there were any unusual variations in performance as time elapsed after a dose of methadone and before the next dose. Comparisons with non-drug using conrol subjects, and in the same patients over a period of time, did not reveal performance effects that could be ascribed to the taking of methadone.

Later, we began to study reaction-time [Gordon, 1970; Gordon and Appel, 1972]. We compared male and female methadone patients' visual reaction-times, to those of drug free ex-addicts and non-drug using control subjects, and found that reaction-time of methadone patients was either equal to or superior to those of the various control groups, a finding which applied to simple as well as to complex reaction-time experiments. In a later study, we used a test of simple auditory reaction-time and studied patients just before they took their daily medication (i.e. when they were abstinent for 24 hours) as well as 1 hour after they took the medication. Their overall reaction-times did not differ at the 2 time medication points measured; and furthermore comparisons of the patients with appropriate controls confirmed the findings of our early studies of reaction-time reported previously.

More recently, we have been studying vigilance, or the capacity of individuals to sustain their attention to sequences of events (analogous to the task maintaining attention while driving long

stretches of monotonous roadway). In a recently completed set of experiments [Appel and Gordon, 1974], a modified version of the continuous performance task [viz. Uhr and Miller, 1962] was used in which the patients were tested for ability to detect target letters from among a series of letters presented one at a time every two seconds. Three different rates of target presentation were used from 720/hour to 40/hour. We concluded that "for percentage of signals correctly detected, promptness in responding to signals and frequency of overt mistakes, methadone patients responded no differently than controls...." Some evidence of a decrement in performance at the slowest rate was found for employed methadone patients, and we are investigating this further in a planned follow-up study, as well as in a study in which a vigilance task is being used in conjunction with electroencephalographic recordings.

Discussion and conclusion

The purpose of this paper has been to review studies on the effects of narcotic drug use on human performance relevant to automobile driving. No studies were found which directly measure driving ability while individuals were under the effects of narcotics. Two types of study were reviewed, however. In one variety, based on records of users, supplemented in one study by interviews, the weight of evidence indicates that neither heroin addicts nor methadone patients have driving records which differ from individuals in the general population.

A second set of studies, consisting largely of laboratory investigations of skills relevant to driving ability was also reviewed. This latter group of studies included evidence concerning single dose effects (comparable to occasional use) and the effects of chronic use (when the users have developed tolerance and dependence on the drug). It was concluded that single doses of narcotics can have marked effects on performance, such as reaction-time. However, it is not clear whether the depressant effects degrade psychomotor activity directly or lead to a decline in interest in all activity.

Laboratory studies of chronic narcotics users have been limited largely to recent work with methadone maintained individuals as part of their treatment for heroin addiction. The studies reviewed consisted of reaction-time, psychomotor skill and sustained attention. The overall weight of evidence from these studies is that the performance of methadone patients who are well stabilized on the drug, cannot be differentiated from the performance of non-drug using individuals. The laboratory studies thus confirm the driving record studies of methadone patients.

It is concluded that the use of narcotics in and of itself does not present a hazard or exist as a significant factor in automobile driving.

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AMPHETAMINES AND DRIVING BEHAVIOR

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Abstract—Direct evidence concerning the role of amphetamines in highway accidents is scant. Laboratory data indicate that most of the basic skills involved in driving are not adversely affected by amphetamine dosages within the normal clinical range, and may in fact be slightly enhanced. Such enhancement is generally greater in sleep-deprived subjects, but is not limited to states of sleep deprivation. Enhancement has also been reported in subjects whose skills have been degraded by alcohol, although results have not been consistent across performance measures. Although there is some evidence that amphetamines induce overconfidence or increase risk acceptance, the effects reported have been neither so strong nor so consistent as to justify much of the apparent concern. Excessive or prolonged "spree" use is widely recognized to result in abnormal psychological states that are incompatible with safe driving performance, and known amphetamine abusers have been found to be involved in disproportionate numbers of highway accidents. Available epidemiological statistics are inadequate to establish how often such excessive consumption is associated with driving, or in any other way to quantify the total contribution of amphetamine abuse to traffic accidents.

BACKGROUND

Drugs of the amphetamine family are classified, pharmacologically, as sympathomimetic amines. They produce effects on vasomotor functions that are grossly similar to those resulting from stimulation of the sympathetic division of the autonomic nervous system, or from the release of epinephrine (adrenalin) into the blood stream from the adrenal medulla. Among the effects of clinical interest is nasal decongestion, for which the amphetamine (Benzedrine) inhaler enjoyed wide over-the-counter sales in the 1930s and 1940s.

However, amphetamine is also a powerful stimulant of the central nervous system, as are its isomer dexamphetamine and the N-methyl substituted derivative, methamphetamine. Such "central" effects include general activation, appetite depression and euphoria when taken in sufficient dosage. These properties were responsible for a gradual increase in "abuse" of amphetamines by those seeking a temporary mood elevation, or who wished to delay the normal onset of sleep, as in prolonged operation of motor vehicles. Such abuses were responsible for discontinuation of over-the counter "Benzedrine" sales and for continual increases in stringency of the federal regulation of amphetamine production and distribution. Currently, amphetamine and most of its relatives are placed on Schedule II of the 1968 Drug Control Act, a listing that includes the most abuse-prone substances for which legitimate medical purposes are recognized in the U.S.A. (e.g. morphine). Its recognized medical uses are largely limited to narcolepsy, childhood hyperkinesis, and short-term appetite control.

EPIDEMIOLOGICAL DATA

In relating any drug to driving behavior, the primary issue is whether the drug has an effect on accident hazard. We do know that, when consumed in large amounts, amphetamines are capable to producing a confused or paranoid state that common sense tells us is incompatible with safe driving. We do know of specific accident investigations that strongly implicate amphetamines, because of the extremely large amounts consumed by the driver and the abnormal behaviors reported by witnesses. What we do not know, with any degree of accuracy, is the prevalence of such abusive consumption among drivers. What we know even less about is the role of moderate dosages in the normal clinical range, even to the extent of whether or not they actually increase the risk of an accident. The reasons for our ignorance become clear when we consider how one secures epidemiological data on drugs and driving.

To assess a drug's contribution to highway losses, the classical method (since Holcomb's 1938 alcohol study) is to compare the prevalence of the drug in crashed drivers with its prevalence in the driver population at risk. For example, if we test all crashed drivers and find that 20% have

consumed alcohol, we may suspect a causal role for alcohol only if we find it in less than 20% of randomly sampled drivers at similar times and places. The over-representation of alcohol in crashed drivers, particularly in those fatally injured, is thoroughly documented. The imposing body of such epidemiological statistics stands in marked contrast to the fragmentary data concerning other drugs.

With amphetamines, we have very few data even on their simple incidence in highway crashes. There are several reasons why this is so. Consider, first, the screening for drugs in drivers involved in non-fatal crashes. Breath testing won't detect amphetamines. Not everyone can immediately pass urine on request, nor does everyone wish to wait around until he can do so. Many persons, for various reasons, refuse to give blood samples. Non-fatally injured, hospitalized drivers are often given medications *after* crashing but before a blood or urine test is administered. Often, reliance must be placed on self-reported usage, a notoriously unreliable source. Consequently, it is reasonable to conclude that there has never been a representative drug screening in non-fatally crashed drivers.

With alcohol, some of these problems also occur, but due to the convenience of breath-test procedures the incidence of lost data is generally much lower. Another consideration is the relatively high incidence of alcohol in crashed drivers. If 20% of the *tested* drivers have been drinking, we need be only moderately concerned about the bias introduced by a non-random test refusal rate of 5%: The true incidence must be between 19 and 24%. However, if only 1% of the tested drivers are positive for amphetamines, and the test refusal rate is 5%, then the true incidence may be nearly 5 times as great as the observed value. It is easy to see how non-random losses of 10 to 20% of all cases could produce order-of magnitude errors of estimate.

With fatally-crashed drivers, it is not necessary to obtain consent for blood samples, so there is at least a possibility of securing a relatively unbiased estimate of drug involvement. To my knowledge there is only one published study in which crashed drivers were *routinely* tested for amphetamines: the Davis and Fisk [1966] investigation of fatal single vehicle crashes in Dade County, Florida. Since only 8 of the 179 victims had any drug other than alcohol, and 4 of these 8 had alcohol as well, this does not impute a very strong role to any drug other than alcohol. Unfortunately, the more extensive studies, such as California Highway Patrol [1967], did not screen for amphetamines.

Studies of drugs in DWI citations are also particularly unrevealing for amphetamines. Thus, Finkle's 1969 study is of interest for some types of drugs, since it tabulates drug incidence in drivers cited for DWI who had less than the BAC level that normally accounts for such "drunken" behaviors. Of 700 DWI arrests at BAC $< 0{\cdot}15\%$, there were only 6 that were positive for amphetamines—as opposed to 131 for barbiturates and 37 for other hypnotics and tranquilizers. However, one must ask whether we would *expect* to find amphetamines in drivers who appeared "drunk". Sedative-hypnotics and tranquilizers can mimic the effects of alcohol, or add to the effects of alcohol. Amphetamines are quite different in this regard.

LABORATORY FINDINGS

Without epidemiological data, the best we can do is try to make some inferences based on laboratory studies. The major acute effects of amphetamines, in the normal clinical dose range, can be summarized very swiftly: They don't impair performance, they enhance it! The degree of enhancement is generally greater in fatigued subjects and in simple or repetitive as opposed to complex tasks such as reasoning or I.Q. tests. When subjects are not previously deprived of sleep the effect is usually not a large one. Nevertheless, when any significant effect is found, most of the time it is on the positive side. This has been amply documented in the reviews by Weiss and Laties [1962] and Laties and Weiss [1966]. Driver-related behaviors, such as simple and disjunctive reaction time and various measures of vigilance and psychomotor performance are among those showing positive effects.

The lone negative influence, as it relates to driver behavior, is the tendency sometimes observed for subjects to take greater risks[Hurst, 1962] or to overestimate their performances [Smith and Beecher, 1964; Hurst, Weidner and Radlow, 1967]. However, these influences were small, on the order of 5%. That they might be greater on the road than in the laboratory is a matter for legitimate concern, but at present it is merely a conjecture. It should be

kept in mind that on some tasks, the degree of objective improvement from amphetamines is greater than the effect on self-reported performance, so that the net effect is not one of *over* confidence[Hurst, 1969a].

AMPHETAMINE ABUSERS

In discussing these laboratory studies, I would like to emphasize that one does not study amphetamine *abuse* in the laboratory. One does not usually employ doses outside the normal clinical range, and one does not subject his volunteers to repeated experiences with habit-forming drugs. Nor would there appear to be any crying need for such researches. There is ample documentation, from clinical reports and accident investigations, that amphetamines are dangerous when used excessively, although one does not know how often this occurs. In a retrospective study of 30 psychoactive drug abusers, Smart, Schmidt and Bateman [1969] found that amphetamine abusers had over four times as many accidents as would be expected on the basis of age, sex, mileage and driving experience. Abusers of mixed drugs (amphetamines plus barbiturates, tranquilizers and/or alcohol) had slightly over twice the statistically expected accident frequency. Although based on a small number of subjects (total of 8 involving amphetamines), these results are indicative that amphetamine *abuse* can greatly increase driving hazard.

AMPHETAMINES AND ALCOHOL

We must also consider whether the acute use of amphetamines adds to, or detracts from, the impairment due to beverage alcohol.

Newman and Newman [1956] administered 15 mg of dexamphetamine, 300 mg of caffeine, or no medication to 6 volunteers 45 minutes before they were given alcohol (0·17 g/kg at 20-minute intervals titrated to performance failure). They found no consistent effects of the prior medications on the blood alcohol concentrations at which failure occurred on tests of balance, steadiness or flicker fusion. Hughes and Forney [1964], likewise, found no evidence that dexamphetamine (20 mg) either enhanced performance or antagonized the impairment produced by 0·5 g/kg of alcohol in a series of reading and arithmetical tasks performed under delayed auditory feedback. However, the dexamphetamine was given in divided doses beginning at noon of the day before the test, so that sleep deprivation and acute adaptation to the amphetamine effects were both likely to have occurred. Brown, Hughes and Forney [1966] found that 5 mg of amphetamine had no effect on the pursuit meter performance observed after 0·5 g/kg of alcohol. However, they presented no data to show whether this dose of alcohol by itself actually impaired performance.

Kaplan, Forney and Richards [1966] found that 5 mg of dexamphetamine temporarily alleviated the impairment in reading and arithmetical performance produced by 0·5 g/kg of alcohol under delayed auditory feedback, but that dexamphetamine by itself had a significant positive effect on performance (in contrast to Hughes and Forney, above, with the 2 day dosage protocol). Bernstein, Richards and Hughes [1966] reported that 5 mg of amphetamine largely overcame the optokinetic nystagmus effect produced by alcohol at a mean BAC of 0·05%. Finally, Wilson, Taylor and Nash [1966] reported that amphetamine (15 mg) antagonized the decrement produced by alcohol (1·2 g/kg) in coding, mental addition, and trail-making. It did not antagonize the alcohol effect on digit span, Wonderlic Personnel test, or a series of perceptual-motor tasks.

In summary, it can be concluded from this brief review that amphetamines mitigated alcohol impairment of some but not all of the functions tested. Generally, the effect was not a complete restoration to the sober or placebo level, and the alcohol impairment was particularly evident when comparing results of amphetamine plus alcohol with amphetamine alone. Finally, there was a great deal of concern by some authors about the putative effects of the combination on such functions as "judgment" and "risk-taking". Although such effects were rarely documented, and the data presented actually indicated better performance with the combination than with alcohol alone, some authors were quite ready to condemn the use of amphetamines by persons drinking alcohol. In one study[Hurst, Radlow and Chubb, 1969b] directly addressed to risk taking with real (monetary) stakes, we found a slight increase from alcohol that was not affected by whether or not dexamphetamine was also present. Dexamphetamine by itself had no effect.

CONCLUSIONS

To assess the role of a drug in traffic accidents without adequate epidemiological data requires a good deal of conjecture. If one does not know the role of a drug in accidents, he must consider what he knows about the drug, and what he knows about accidents, and somehow try to put the two together.

1. *Use in normal "clinical" range*

In the earlier days of highway accident research, a great deal of the blame was laid on willful, reckless driver behaviors. More recently, the emphasis seems to have swung toward the driver's built-in limitations as an information processor, and the way in which such limitations may be exascerbated by drugs such as alcohol. Statistics have shown, for example, that contrary to earlier opinion, it is *not* the delighted and devilish "half-drunk" driver who is the major menace: In fatal crashes, it is the *totally drunk* driver who is most over-represented, insofar as one can infer this from BAC levels of crash victims.

Perhaps it should follow, then, that we concern ourselves less about drug effects that may cause some degree of overconfidence, and more about what the drug does to a driver's ability to process and react to information. I do not recommend that amphetamines be doled out indiscriminately to fatigued or intoxicated drivers. A responsible person should plan his trips so that he does not have to drive after drinking or sleep deprivation. Once in a very great while, the circumstances may arise when someone who is sleepy, or has been drinking, suddenly finds that he must drive. In such instances, I do not believe that the use of amphetamine-type stimulants is contraindicated by the known facts. But of course, this matter may be of little practical importance, since it would seem very difficult to legalize use of amphetamines for driving only under such unique circumstances.

2. *Use at high or abusive levels*

This is addressed separately because it is an entirely different thing from the occasional use of normal amounts. In this respect, stimulants like the amphetamines may well be different from all other classes of abuse-prone psychoactive drugs. The difference between moderate and immoderate use is not only extreme: It is a complete difference in kind. There can be no doubt that heavy spree-type amphetamine consumption is hazardous, when it occurs. The only thing we don't know is how often it does occur in drivers, and therefore how much of a problem it is from a public health viewpoint. We badly need more epidemiological data, even if the effort does not go beyond the simple, uncontrolled level of measuring drug concentrations in body fluids of accident-involved drivers.

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TRANQUILIZERS AND DRIVING

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Abstract—The consumption of tranquilizers has increased throughout the 1960s. At present more than 100 million prescriptions of tranquilizers are written annually in the U.S.A. In a Norwegian study diazepam was found in the blood of 18% of people injured in traffic accidents. Other epidemiological studies have demonstrated an increased traffic accident risk to be associated with the use of tranquilizers. The combined use of tranquilizers and alcohol, which is common among patients, increases one's accident risk from that due to either agent alone.

Laboratory studies concerning the effects of tranquilizers on skills related to driving have demonstrated impaired information processing capacity and eye-hand coordination due to these agents. Neuroleptics impair information processing especially at the onset of the treatment whereas the hazards of benzodiazepines become evident during long term treatment. Most of the tranquilizers increase the deleterious effects of alcohol on skills related to driving. Particularly strong is the interaction between diazepam and alcohol.

At present the best countermeasure against accidents caused by tranquilizers seems to be easily available information about the effects of drugs on driving. At the onset of treatment with a neuroleptic or during long term treatment with a high dose of benzodiazepines, one should cease driving.

The term "tranquilizer" often includes three different classes of drugs, the neuroleptics or antipsychotics (e.g. chlorpromazine = Thorazine^{R}), the antidepressants (e.g. imipramin = Tofranil^{R}), and the ataractics, or antianxiety agents (e.g. Diazepam = Valium^{R}). Some of the ataractics are used as hypnotics and sedatives as well. There is overlapping in the use of the three different types of drugs, particularly in the treatment of neurotic patients.

The consumption of psychotropic drugs has increased throughout the 1960s. At present over 50 million prescriptions of diazepam alone are written annually in the U.S.A. [Greenblatt and Shader, 1974]. Two million persons in this country take the drug continuously [Jick, 1974]. If we divide the amount of benzodiazepine tablets consumed in the U.S.A. by the population, we find that the average American ingests annually about 40 tablets. In comparison the respective figure in Sweden is about 30 tablets per person, and in Finland and Norway only slightly less than 20 tablets per person [Idanpaan-Heikkila and Salonen, 1972]. The consumption of diazepam represents more than 50% of the total consumption of tranquilizers in the Scandinavian countries mentioned above [Idanpaan-Heikkila and Salonen, 1972].

Basic difference between the use of alcohol and tranquilizers is the fact that alcohol is often used in order to become intoxicated whereas the tranquilizers should be used under medical control in order to improve health. Regrettably the real situation is not so simple. Several studies reveal that 20–70% of patients use their medication irregularly, in order to alleviate their temporary symptoms, rather than as prescribed [Ayd, 1974; Milner, 1969]. This is by no means a special feature of psychiatric patients.

In some cases the irregular administration of a drug can even be advantageous because one may thus be able to prevent its accumulation in the organism or avoid the development of tolerance. On the other hand, the organism is incapable of adapting to some of the side effects of a drug administered irregularly. Every dose can cause a new process of adaptation and during this adaptation phase 1 may have an increased accident risk [Linnoila, 1973a]. It has also been demonstrated that up to 55% of patients on drugs use alcohol simultaneously in order to relieve their symptoms by this combination more efficiently than by the drug alone [Milner, 1969].

The percentage of drivers under the influence of drugs has been demonstrated to be on the order of 10–20% [Wagner, 1963; Nichols, 1971]. Women seem to use drugs in traffic more often than men [Wagner, 1963]. The majority of the drugs reported have been mild analgetics or sedatives [Wagner, 1963]. These drugs should not cause extra risks in traffic [Linnoila, 1974a].

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There are only a few studies elucidating the relative accident risk of drivers using drugs in road traffic. Furthermore from most of the studies it is impossible to calculate the accident risk caused by a particular drug per se, because the drug users usually have more emotional and social problems than the rest of the population [Nichols, 1971]. These problems as such may increase one's accident risk.

In a Finnish questionnaire study administered to 1000 psychiatric outpatients with age, sex, and living district matched controls, it became evident that the patients using two or more psychotropic drugs had a significantly increased risk of traffic accidents [Maki and Linnoila, in press]. The accident risk was calculated by comparing the two groups on the numbers of accidents per mileage in similar traffic surroundings during two years previous to the study [Maki and Linnoila, in press]. The accident risk tended to increase with increased amount of medication. Heavy medication, however, generally reflects a severe disease. The combined administration of 50 g or more of alcohol with the medication at least once a week further increased one's accident risk [Maki and Linnoila, in press].

In a Norwegian hospital study conducted in the city of Oslo, significant amounts of diazepam were found in the serum of 11% of the people injured in traffic accidents [Bo, Haffner and Langard *et al.*, 1974]. The drug was present in combination with alcohol in another 7% of the victims. There was significantly less diazepam found in the control group which, however, was not perfectly matched. American studies have revealed a 2 to 4 fold increase in accident risk among drug users as compared with nonusers [Nichols, 1971]. None of these studies have differentiated tranquilizers from other drugs studied.

From the facts above one is likely to conclude that tranquilizers may increase a driver's accident risk. Even though some of the agents can cause substantial increase of traffic risk, from an epidemiological point of view, it is less than that caused by alcohol.

Some tranquilizers have a moderate potential for abuse, the benzodiazepines in particular. Because tolerance develops to the desired effects of the drug, the abuser is liable to increase the dose. Some of the side effects of the drug are not diminished in the same amount as the desired ones, and an abuser of tranquilizers obviously is a risky driver.

From a theoretical point of view tranquilizers should also improve some patients as drivers, by calming them in stressful situations and relieving them from excessive anxiety. At present there are no studies available supporting this point of view.

LABORATORY STUDIES

Mental disorders as such impair one's driving ability [Eelkema, Brosseau, Koshnick *et al.*, 1970]. The knowledge of the interaction of these states and drugs on skills related to driving does not exist at present, even at the laboratory level.

The basic mechanism of action of the *neuroleptic drugs* is to alter the chemical communication between the nerve cells in the central nervous system. Because there are different substances acting as transmitters for the communication between the nerve cells in the central nervous system, there are different ways to intervene in a relatively specific manner to affect the way nerve cells interact. The specific effect of a neuroleptic depends on the transmitter which that drug inhibits most. The common neuroleptics used for the treatment of psychotic patients are relatively nonspecific. In other words, they inhibit the effects of several transmitters. In practice this means that, in the beginning of a treatment, we observe side effects, due to both central and peripheral sites of action, e.g. sudden changes of blood pressure and heart rate, impaired vision, etc. Many of these side effects as such are enough to impair one's ability to act appropriately in a driving situation.

The psychomotor skill most affected after the administration of neuroleptics is information processing capacity [Linnoila, 1973a]. This is the case after the acute administration of all neuroleptics studied in our laboratory; chlorpromazine (Thorazine^{R}), thioridazine (Mellaril^{R}), haloperidol (Haldol^{R}), and flupenthixole (Fluanxol^{R}) [Linnoila, 1973a]. The impairment of information processing capacity is severe after all the agents mentioned, and the differences between the individual agents are not significant [Linnoila, 1973a]. Choice reaction performance and eye-hand coordination related to driving remain relatively intact after the administration of these neuroleptics to healthy volunteers in the low doses used for the treatment of neurotic outpatients [Linnoila, 1973a]. Many of the deleterious side effects of neuroleptics are overcome

by the organism due to adaptation after 5 to 6 days of regular drug use [Seppala, in preparation; Saairo, in preparation]. The information processing capacity is also restored during the adaptation and at the end of the period it is no longer significantly impaired [Seppala, in preparation; Saario, in preparation]. Data concerning the effects of the large doses of neuroleptics used for the treatment of psychotic patients on psychomotor skills related to driving do not exist. From the point of view of traffic safety, there are of minor importance since these patients seldom drive.

Neuroleptics are the type of drugs which should be used regularly if driving during the treatment is necessary. One could thus avoid the appearance of new adaptation phases and minimize the deleterious effects on safe driving. In any case, a person under the influence of these agents, no matter how well adapted, is not at the peak of his capabilities and may prove unsafe in unexpected circumstances, e.g. one's sensitivity to reflected light may remain increased due to neuroleptics, rendering night driving dangerous.

Antidepressants in the low doses used for the treatment of neurotic depression do not generally impair one's driving skills[Seppala, Linnoils, Elonen *et al.*, in press]. In some persons the sedative agents like doxepin (Sinequan^{R}) and amitriptylin (Elavil^{R} or Tryptizol^{R}) may cause substantial impairment of information processing capacity[Seppala *et al.*, in press].

The *benzodiazepines* generally cause only mild impairment of psychomotor skills after single doses[Linnoila, 1973a; Linnoila and Mattila, 1973]. In any case, drivers treated with single 10 mg doses of diazepam caused significantly more "accidents" in emergency situations during simulated driving, when another car appeared in front of them from a side road, than drivers treated with placebo capsules filled with inactive sugar[Linnoila and Hakkinen, 1974]. The hazardous feature of the benzodiazepines is the accumulation of the agents and their active metabolites in the organism [Linnoila, Saario and Maki, 1974]. In the long term use of these agents the efficacy is generally lost as the therapeutic effect vanishes, but the side effects increase[Kanto, Iisalo, Lehtinen *et al.*, 1974].

The main active metabolites of the benzodiazepines used as hypnotics, flurazepam (Dalmane^{R}) and nitrazepam (Mogadon^{R}), accumulate in the organism during regular use and may significantly impair one's driving skills on the morning following their use[Saario and Linnoila, in press, a and b]. The impairment of driving skills after the benzodiazepines is mainly observed in complex eye-hand coordination tasks requiring self-assessment of one's skills[Linnoila and Saario, 1974]. However, these agents are so commonly used that even a relatively minor impairment of skills renders benzodiazepines the class of tranquilizers which is commonly associated with accidents[Bo *et al.*, 1974]. The most effective way to use benzodiazepines should be a short term treatment of anxiety, from 7 to 14 days, repeated after a pause of 2 to 3 weeks if necessary. While using benzodiazepines a sensible irregularity of administration may be purposeful. The therapeutic efficacy is retained and the accumulation is prevented. A qualification of this kind of treatment is a good patient-doctor relationship including the knowledge of the effect of the drug on the particular patient.

A sudden cessation of a long-term tranquilizer treatment may also lead to a dangerous situation for driving because the organism has to adapt to a tranquilizer-free state[Le Blanc, 1974].

COMBINED EFFECTS OF ALCOHOL AND TRANQUILIZERS

Alcohol increases the variability of one's psychomotor performance[Perrine, 1974]. After a combination of alcohol and tranquilizers this variability is increased from that after either agent alone[Linnoila, 1973a].

In the beginning of treatment the sedative neuroleptics and antidepressants show additive effects with alcohol[Seppala, in preparation; Saario, in preparation; Seppala *et al.*, in press]. In some cases these additive effects diminish during treatment, in some subjects they remain stable[Seppala *et al.*, in press]. Most benzodiazepines potentiate the hazards of alcohol[Linnoila, 1973a; Linnoila *et al.*, 1974]. This is particularly the case after the simultaneous administration of diazepam or bromazepam and alcohol[Linnoila, 1973a; Saario, in preparation]. Even if diazepam is administered 10 hours before alcohol an additive interaction can be observed[Linnoila, 1973a]. Chlordiazepoxide (Librium^{R}) shows a milder interaction with alcohol than diazepam or bromazepam[Linnoila, 1973a]. The time course of benzodiazepine alcohol interaction after

simultaneous administration is relatively short. The peak effects are seen during the first hour after the ingestion of the agents and after 3 hours the effects are no longer significant[Linnoila and Mattila, 1973]. The maximum effect coincides with the fastest increase of the concentrations of the agents in the blood[Linnoila, Otterstrom and Anttila, 1974]. This phenomenon is due to the distribution of the agents in the body. The brain levels of diazepam peak very early after its administration[Klejn, 1969].

The main limitation of the laboratory studies is their taking place in the laboratory and not in normal traffic. This can not be without effect on the motivation of the subjects, no matter how ingenious the design. Because the subjects in a majority of the studies are healthy volunteers, the drug disease interaction is excluded. Such a laboratory setting may lead to overestimates of the drug effects in real life situations. On the other hand the young subjects often are more capable of compensating for the deleterious effects of drugs on their skills than elderly ones [Linnoila, 1974]. Also a look at the consumption statistics immediately reveals that many relatively healthy people now ingest psychotropic drugs frequently.

So far a reasonable correlation between some psychomotor tests and traffic behavior has been demonstrated [Hakkinen, 1958]. The criterion of traffic behavior was the number of accidents (with mileage and traffic surroundings matched) during a time period [Hakkinen, 1958].

From a forensic point of view the effect of tranquilizers on driving is unfortunate. After the ingestion of alcohol we are able to find a reasonable correlation between the serum concentration of the agent and its effect, whereas after the tranquilizers we are unable to do this [Linnoila *et al.*, 1974], a fact due to the quick appearance of active metabolites in the organism and certain peculiarities of the distribution of the agents [Klejn, 1969]. Also the individual sensitivity to the effects of tranquilizers may vary more than to the effects of alcohol. Therefore strict legislative measures against tranquilizer use and driving are much more difficult to devise than those against drinking and driving.

In order to predict the correlation between a certain blood level of diazepam and driving one must know the time of the last dose, the serum concentration of diazepam, and the serum concentration of N-desmethyldiazepam, the main active metabolite [Linnoila, 1974]. In real life situations the time of the last dose can seldom be objectively verified.

COUNTERMEASURES TO DIMINISH THE TRAFFIC RISKS CAUSED BY TRANQUILIZERS

The ultimate goal of countermeasures is to abolish traffic accidents due to tranquilizers. While allocating resources for countermeasures against traffic accidents one has to remember that the accident risk due to alcohol is more conspicuous than that due to tranquilizers. On the other hand there is a rather large number of accidents where the agents overlap[Bo *et al.*, 1974].

Doctor–patient confidentiality must be respected while figuring out the countermeasures. However, a possibility should be reserved for a doctor to report a patient who, due to his illness or drug use, is obviously a risky driver, and does not obey the instructions of the physician.

Instead of looking mainly at the side effects of drugs on the users, we should also be concerned about the risks of drug use for the society. Because the number of victims in traffic accidents is high, and the age of the victims is relatively low, reduction of the former figure should be one of the main tasks of present preventive medicine. Using Finland as an example it has been statistically demonstrated[Hemminki, Hemminki, Hakulinen *et al.*, 1974] that the total abolition of accidents would provide the male population of that country as many man-years as the total abolition of cancer. Moreover, these man-years would be among the actively working age groups. At its best, the elimination of accidents due to legal drugs other than alcohol could reduce the total number of traffic accidents by 10–20%. There is too little research done to give any assumptions about the role of drugs in occupational accidents, but the role of drugs in this field has to be questioned also.

The best countermeasure at present would be easily available information about drugs at schools, pharmacies, gas stations, open care units and hospitals. This could be supported by campaigns in mass information media. The quality of the information should be such that it reduces unnecessary use of drugs, but does not cause fears among patients needing drugs for their treatment. Medical personnel should be able to inform patients at the individual level, which requires that more attention be paid in teaching clinical pharmacology.

As a driver one should be especially careful at the onset of a drug treatment. In the optimum case one should abandon driving for the first 7 to 10 days of treatment. Then the situation could be evaluated with one's doctor before driving is resumed. This is necessary because the effects of a particular drug on a particular patient are hard to predict without sophisticated methods which are not generally available in out-patient practice.

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MARIHUANA AND DRIVING†

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Abstract—Survey studies have found that marihuana use is increasing and that users frequently drive under its influence. But there is little direct epidemiological evidence to indicate if the presence of marihuana in drivers increases accident probability. However, there is a large body of experimental evidence indicating that marihuana impairs the performance of skills important for driving. Perceptual and attention functions show large decrements under marihuana with a less certain deficit for various tracking functions. Marihuana studies in driving simulators have found the greatest deficit in perceiving and responding to potential dangers from the environment. Simulator studies of risk taking have found no evidence for impairment. Several studies of performance in actual cars have also demonstrated performance decrements but the behavioral functions impaired have not been clarified.

In summary the experimental evidence suggests strongly that marihuana use while driving produces a performance impairment.

Derivatives of the *Cannabis sativa* plant such as marihuana and hashish, are highly potent psychoactive drugs. Recent research surveys [Secretary of the Department of Health, Education and Welfare, 1973; Le Dain Commission, 1972] have reported significant changes in phenomenological, perceptual, cognitive and psycho-motor functions. These are functions which appear important for driving. Moreover, cannabis derivatives are in wide use. By 1972 it was estimated [Secretary of Health, Education and Welfare, 1973] that roughly 15% of the age 11 and over population had used marihuana at least once and perhaps half were current users to some degree. Epidemiological studies by Waller[1974], Smart[1974], and the Le Dain Commission[1972] indicate that the majority of marihuana users have driven under the influence of the drug.

The potency of the drug and its frequency of use in the driving population suggests the need for an examination of the epidemiological and experimental evidence regarding the effects of cannabis upon driving safety. The most helpful epidemiological data would be a comparison of cannabis levels in drivers involved in accidents with cannabis levels in non-accident-involved drivers. Unfortunately, the determination of levels of cannabis and its metabolites in organisms is a difficult procedure, with few laboratories equipped for such determinations. This and the difficulties in obtaining body samples for analysis have to this date prevented drug-driving studies similar to those performed for alcohol by Borkenstein, Crowther, Shumate, Ziel and Zylman[1964] and others. It should be noted that the wide appreciation of the importance of alcohol in traffic safety rested upon such studies.

An alternate method of obtaining estimates of marihuana's role in accident causation has been to compare accident rates of marihuana users with rates for non-users. These studies have produced divergent results. Waller[1965], Waller and Goo[1969], and McGlothlin, Arnold and Rowan[1970] reported accident rates for users as typical of those within the same age range. However, Crancer and Quiring[1968] found above normal accident rates.

In addition to disagreement regarding results, there are difficulties in drawing conclusions from these studies. Drug users may be untypical of selected control groups in many dimensions other than the use of the drug under study. This may well include differential reporting of accidents for which it is well known that many go unreported. Moreover, most illicit drug users tend to be polydrug users. Thus, even if marihuana users were shown to exhibit a higher accident rate, it would be difficult to ascribe the result solely to the use of marihuana. In particular it should be noted that the majority of marihuana users in at least college populations are simultaneously consuming alcohol, a drug already known to increase driving accident probabilities[Waller, 1974].

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Until studies capable of determining marihuana presence in accidents are performed, the major source of information regarding the accident potential of marihuana must come from experimental studies of driving-related performance while under the influence of administered marihuana.

Not all behavioral factors underlying driving performance have been identified, nor do we have adequate measures for such obviously relevant factors as emotion and attitudes. Clearly, however, sensory-motor, perceptual and decision-making functions are major determinants of driving safety and this discussion will focus on these factors.

Motor control appears somewhat impaired by marihuana. Kiplinger, Manno, Rodda and Forney[1971] reported decrements in hand and body steadiness. Moskowitz, Sharma and Schapero[1972] reported impairment on phoria and duction tests of ocular motor control. However, the reported decrements were so small that loss of motor control of vehicles appears unlikely.

Tracking of complex functions is a more relevant task for driving. In an extensive study of human operator tracking characteristics employing a single-axis compensatory tracking task, Reid, Ibrahim, Miller and Hansteen[1973] found only a small increase in random output under 88 mcg delta-9 THC/kg B.W. while a lower dose of 21 mcg had no effect. Neither the phase nor amplitude function showed any deficits. However, an as yet unpublished study by Moskowitz, Ziedman and Sharma examined compensatory tracking using a critical tracking test technique under a 200 mcg THC/kg B.W. dose. This technique has the tracking task become more difficult and unstable until a point is reached when all subjects fail to compensate adequately for the forcing function. The instability at the fail point becomes a measure of the subject's performance. Marihuana significantly decreased the performance of subjects under this task with the degree of impairment approximately midway between subjects under 0·075 and 0·15% blood alcohol levels. Moreover, there have been reports[Manno, Kiplinger, Bennett, Haine and Forney, 1970; Manno, Kiplinger, Scholz and Forney, 1971; Kiplinger, Manno, Rodda and Forney, 1971; Kielholz, Goldberg, Hobi, Ladewig, Reggiani and Richer, 1972, 1973; Roth, Tinklenberg, Whitaker, Darley, Kopell and Hollister, 1973] of performance decrements on pursuit tracking tasks beginning at 5 mcg THC/kg B.W. The greater sensitivity of pursuit tracking to marihuana may be related to the greater perceptual demands of such tasks in contrast to compensatory tracking. It should be noted that sensory-motor tasks involve both perceptual and motor demands and deficits can be due to impairment of either or both aspects.

A variety of studies have examined the effects of marihuana on sensory-perceptual skills. In general, studies of simple sensory functions with anticipated stimuli which have low demands for information analysis demonstrate no performance decrement. There is no marihuana effect upon visual brightness threshold[Caldwell, Myers, Domino and Mirriam, 1969], depth perception[Clark and Nakashima, 1968], dark adaptation or visual acuity[Moskowitz, Sharma and Schapero, 1972]. On the contrary, in agreement with some subjective reports of increased sensory sensitivity under marihuana, Hill, Goodwin, Schwin and Powell[1974], found that thresholds of awareness and pain levels for electrical stimulation were lowered and Schwin, Hill, Goodwin and Powell[1974] found critical flicker fusion thresholds to increase.

There is considerable evidence of a performance decrement under marihuana when the subject is faced with stimuli which demand constant attention, which appear at random, unexpected intervals, or which require additional central processing such as storage and retrieval. Large performance decrements under marihuana have been found for conditions which require continuous attention and a stable perceptual framework, as in autokinesis[Sharma and Moskowitz, 1972]. Also, detection of intermittent random signals in either or both central and peripheral vision is impaired[Casswell and Marks, 1973; Moskowitz, Sharma and McGlothlin, 1972] as is recognition of previously presented material[Abel, 1970, 1971].

Sustained attention or vigilance is a perceptual function affected by marihuana. Sharma and Moskowitiz[1973] examined performance of an observing task which required detection of randomly presented signals. The signal was the double jump of clocklike movement of lights which normally made single jumps. Initial performance was impaired and continued to decline over the one-hour experimental session. There was more than a 100% increase in errors by the end of the session under a dose of 200 mcg THC per kg B.W. A further study by Moskowitz and Sharma (unpublished) demonstrates through use of eye-movement recording that the errors

occur at some fairly central point in the information processing system since eye fixations continued to correctly follow the lights whether a single step or the double step signal occurred. This finding supports the suggestion that the marihuana deficit is in central processing of information or attention allocation rather than earlier stages of sensory transduction or transmission.

There have been two studies applying signal detection theory analysis to performance under marihuana. Moskowitz and McGlothlin[1974] examined auditory functions and Le Dain[1972] examined visual functions. Both found decrements in d', demonstrating a deficit in perceptual sensitivity independent of a change of criterion. To a lesser extent, both studies found some evidence for criterion change.

This review of experimental studies has found strong evidence for decrements in perceptual tasks and in sensory motor tasks which have important perceptual elements. No evidence was found for sensory decrements per se nor much evidence for motor impairment in tasks without perceptual demands. An analysis of the proximal causes of drug-related accidents is required to determine the significance of these decrements in attention, signal detection and vigilance for driving performance. Since such data is not yet available, we shall examine the nature of deficits found under marihuana in driving simulators and driving tests, which are the closest approximations we have to the actual demands of driving.

There are three simulator studies which have been concerned primarily with evaluating the effects of marihuana on the perceptual and tracking aspects of driving. The earliest by Crancer, Dille, Delay, Wallace and Haykins[1969] will not be discussed as there are objections to elements of the experimental design[Kalant, 1969] and questions regarding the actual dose administered[Manno, Kiplinger, Scholz and Forney, 1971].

A second simulator study by Rafaelsen, Bech, Christiansen, Christrup, Nyboe and Rafaelsen[1973] required the subject to simultaneously perform a tracking task while monitoring light signals. Response measures were the latency in responding with the brake to stop light signals and with the accelerator to start lights, the number of gear changes and the speed. Three orally-ingested doses of marihuana of 8, 12 and 16 mg delta-9 THC/kg B.W. were compared with 70 grams of alcohol.

The two larger marihuana doses and the alcohol dose produced changes in the latency of response to the signal lights. Neither drug affected the mean speed. Alcohol had a small but significant effect on the number of gear changes.

Marihuana primarily produced a failure or delay in responding to signals from the environment rather than affecting the car control or tracking aspects.

A third simulator study by Moskowitz, Hulbert and McGlothlin[1973] utilized a car mounted on a chassis dynamometer facing a screen on which was projected a film of a 31-mile drive. Speed was controlled through the use of the brake and accelerator. The driver also tracked a varying lateral shift of the scene which followed the contours of the road. In addition, appropriate responses were required to four lights which appeared intermittantly in the peripheral visual field.

Subjects received marihuana containing 0, 50, 100 and 200 mcg delta-9 THC/kg B.W. which they smoked. None of the measures derived from manipulation of the steering wheel, accelerator or brake showed any decrement under marihuana. However, the response to the peripheral light signal detection task showed an increasing delay which was linearly related to the drug dose. Moreover, the number of incorrect responses indicating misinterpretation of the signal increased significantly, reaching a maximum of 70% greater errors at the highest dose.

Thus the simulator studies examining the perceptual aspects of driving showed a deficit in attention or perception. Tracking and car control exhibited little impairment.

Further findings are available from actual car-driving studies. A pilot project[anonymous 1972] by the North Carolina Highway Safety Research Center failed to find any performance decrement under "a relatively high" dose of THC. A study by the Le Dain Commission[1972] which required driving 6·6 miles through narrow lanes, with both forward and backward maneuvering being performed, used smoked doses of 21 and 88 mcg delta-9 THC/kg B.W. Observors both in the car and on the course were unable to perceive any impairment, but there was a statistically significant increase in the number of overturned cones for the high marihuana dose only.

The most extensive study involving subjects in cars both on a closed driving course and in

actual city traffic was performed by Klonoff[1974] who used doses of 4·9 and 8·4 milligrams delta-9 THC. Performance decrements on a series of quite stringent closed-course driving tasks were found at the higher dose primarily. Driving performance on the city streets were scored by observers. Of the eleven dimensions used for scoring performance, marihuana appeared to significantly affect judgement, care and concentration. What was most surprising in the study was the great variation in performance among subjects under the drug treatment with some subjects appearing to improve their performance.

A conservative conclusion from simulator, laboratory and car driving studies suggests that marihuana can impair aspects of the tracking and car control components of driving. However, such decrement is likely to arise in conditions demanding considerable maneuvering skill or which also involve demands on the perceptual functions. On the other hand, monitoring the environment for potential dangers and signals clearly is impared at fairly low marihuana levels and in situations which do not have extraordinary demands for information processing.

In an attempt to understand how marihuana impairs the monitoring functions required for driving, Moskowitz, Ziedman and Sharma [unpublished] are currently examining the effects of marihuana upon eye movements while subjects observe driving scenes in a simulator. It is clear that marihuana affects the patterns of observation. Thus, subjects under marihuana spend less time on each single fixation extracting the information in that visual glance. Rather they increase the total number of fixations per unit time, flitting from one fixation to another. If requested to maintain a fixation on a given item by the experimenter, they have great difficulty although placebo subjects normally have no problem with this task.

Another dimension of significance for driving performance is decision making. This is an area in which quantitative experimental data are difficult to acquire. Subjective reports suggest that marihuana in many situations has a tranquilizing rather than a stimulating effect. The Le Dain report[1972] suggested that little evidence exists for the notion that marihuana increases aggressiveness. A study by Pliner, Cappell and Miles[1972] had observers compare the mood of subjects who had smoked marihuana with those under alcohol. Marihuana subjects were rated as less aggressive, less anxious, with less concentration or vigor and more fatigued.

There have been two simulator studies of the effect of marihuana upon risk taking. Dott[1972] examined risk acceptance in a passing task using a closed loop simulator with an optical projection from model cars on two belts simulating two road lanes. The subject's task was to pass the car in his lane before a car approached in the passing lane. Occasionally they were required to abort a passing maneuver or to attempt a rapid completion when a red light signal was present.

Twelve subjects were tested under 0, 11·25 and 22·50 mg delta-9 THC treatments as well as a non-smoking condition. The major finding was that when a warning signal occurred, subjects under marihuana were more likely to take the safer approach and aborted the attempt. In general they attempted fewer passes and had no greater likelihood of having an accident. However, measures of the time they took to reach a decision when the opportunity for a pass occurred increased greatly under marihuana. Finally, no differences appeared in the path traversed in the passing maneuver under the different doses. The author concludes that marihuana produces decreased willingness to take a risk. The increased decision time occurred in situations which were not of an emergency nature. There was no change in decision time when the warning signal demanded emergency response. Apparently, the increased demand for attention from the emergency overcame the marihuana deficit as exhibited in decision-making in the non-emergency situation.

Another examination of risk taking under marihuana undertaken by Ellingstad, McFarling and Struckman[1973] reported similar results. Thus, the data from examining risk taking do not suggest any increased risky behavior under marihuana.

In conclusion, there is consistency between the experimental reports from more abstract laboratory tasks and results from driving simulators and car driving tests. Perceptual functions of importance for driving are clearly and greatly impaired and would be expected to interfere with the ability of drivers to monitor the environment for important signals and potential dangers. To some degree tracking aspects of driving would also be affected by the impairment of the perceptual functions necessary for their control. The motor aspect of tracking is less likely to be affected as motor performance per se appears less affected by marihuana. No evidence was found that emotional or attitudinal changes under marihuana would be likely to lead to increased risk taking in the driving situation.

The evidence presented for a decrement in driving performance skills would be expected to lead to some increase in accident probability, a supposition supported by reports from experienced users of self-awareness of impairment. A more definitive estimate of the increased accident rate will require further understanding of the character of the performance deficits associated with marihuana, a drug about which our knowledge is still limited. Additional knowledge is required regarding the interactions between alcohol and marihuana since epidemiological evidence indicates their frequent joint use. The few experimental studies combining these two drugs have suggested additional impairment is to be anticipated.

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BARBITURATES AND DRIVING

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Abstract—Barbiturates are general nervous system depressants which are commonly used as sedatives and hypnotics. Approximately 4% of the adult population, 12% of high school students and 19% of college students have reported using barbiturates. The lack of controlled studies comparing barbiturate involvement in traffic accidents and in the at-risk population has restricted any conclusive interpretations which can be made about the causal relationship between barbiturates and traffic accidents. Barbiturate incidence in traffic accident involvement varies from 2 to 9%. The variance in numbers represents different methods in data collection, different techniques in identifying barbiturates in body fluids and the differences in the populations sampled. Laboratory studies have found barbiturates at moderate doses to degrade driving skills. Motor skills performance, perceptual and tracking task performance and vehicle-handling test performance are impaired under barbiturates. This impairment is further degraded by the combined use of alcohol and barbiturates beyond that found under either drug alone. It is clear that barbiturates are dangerous for driving and its effects are likely to produce impairment on those components of driving necessary for safe operation of a motor vehicle.

In this paper the relationship between the use of barbiturates and driving will be considered. Barbiturates are among the most widely used mood-modifying drugs [Devenyi and Wilson, 1971]. It has been estimated that approximately 3·62 billion 100 mg dosage units (362,000 kg) of barbiturates are available for legal use. Of these about 2·26 billion 100 mg dosage units (226,000 kg) are dispensed through prescriptions or other medical uses [McGlothlin, 1973]. Illegal barbiturate sales for 1970 have been placed around 1·6 billion 100 mg dosage units (160,000 kg).

Barbiturates are derivatives of barbituric acid and produce a nonspecific general depression of the nervous system, ranging from mild sedation to coma. The depression is a function of the type of barbiturate, dose, route of administration, degree of excitability and possible tolerance due to previous use of the drug.

Barbiturate classification is generally made according to the duration of action: ultra-short, intermediate, and long acting. The ultra-short acting ones such as thiopental are used as general anesthesia and are not abused. The intermediate ones, such as pentobarbital, secobarbital and amobarbital, are the forms most widely prescribed as sleeping pills and sedatives. These are the most often abused group. The long acting barbiturates such as phenobarbital are used as sedatives or hypnotics and as anti-convulsants. This group is only occasionally abused.

One national survey of barbiturate use in 1970 found 4·4% of the population between 18 and 74 years to be using barbiturates [McGlothlin, 1973]. A 1973 survey and a 1970 survey found approximately 12% of high school students to be using barbiturates [cited in NIDA, Recent Surveys of Non-medical Drug Use, 1974]. Recent surveys of college students by Holroyd and Kahn [1974] and by Goode [1972] reported barbiturate use to be 17 and 19% respectively.

This paper reviews the relationship between barbiturate use and traffic accidents as assessed by epidemiological studies and reviews the literature regarding the effects of barbiturates on driving-related skills. There are methodological problems in collecting data and assaying barbiturate blood levels in the at-risk population. Consequently, it has not been possible to make direct comparison of barbiturate blood levels in operators involved in traffic accidents and in the at-risk population. The lack of such a comparison severely limits the accuracy of estimates of driving accident probabilities under barbiturates.

Indirect methods of estimating the interaction of barbiturate use and driving accidents include studies of driving records of drug users, and the incidence of barbiturate involvement in traffic accidents.

Smart, Schmidt and Bateman [1969] interviewed 6 patients using barbiturates, and

compared the accident involvement of the 6 subjects with the "expected" involvement rate. They concluded from their observations that fewer traffic accidents were indicated in the barbiturate users. These findings should be interpreted with caution since the N is small.

In 1968 Crancer and Quiring [1968] compared crash and citation rates of 302 drivers with a control group matched for sex and age. Users of depressants and stimulants had 57% more accidents than did the control group. The authors did not report the percentage of barbiturates in this drug category. However, barbiturate use in the population using depressants is high.

Investigations of single car crashes involving fatal injuries have frequently found barbiturates in drivers. A California Highway Patrol study [1967] estimates that 9% of 772 victims of single vehicle crashes showed the presence of barbiturates or tranquilizers. Another recent study [Turk, McBay and Hudson, 1974] found 2·5% of the single car crash operators to demonstrate a positive incidence of barbiturates.

Briglia [1966] estimated the incidence of barbiturates in multiple car crashes to be about 9·3%. The investigator indicated that more extensive testing probably would have revealed a greater drug involvement. Sunshine [1956] and Sunshine, Hodnett and Hall [1968] found that a smaller percentage, about 3–4%, of fatal crash victims showed the presence of barbiturates. Konkle [1969] and Kaye [1970] both found that only 1% of fatal traffic crashes showed barbiturate involvement. Perrine, Waller and Harris [1970] found only 2% of fatally injured persons revealed the presence of barbiturates.

These studies demonstrate the variance in estimates of barbiturate involvement in traffic accidents. Sampling different populations, collecting data at various times of day, and varying laboratory techniques for identifying barbiturates in the body fluids may account for the differences. Interpretation of these studies is uncertain as the amount of barbiturate use in the at-risk population is not known and the issue will remain unclear until more controlled studies are conducted.

If barbiturate involvement in traffic accidents does approach the 9% figure reported by Briglia and the California Highway Patrol, then barbiturates are clearly over-represented in the driving-accident population. It should also be noted that barbiturates are generally taken as sedatives and sleeping pills at night; and hence the at-risk driving population should contain a lower proportion of persons under the influence of barbiturates than that in the total population of drivers.

Barbiturate intoxication, similar to alcohol intoxication, is often accompanied by impaired thinking, lack of emotional control, aggressive behavior, motor incoordination, and drowsiness. These effects make barbiturates potentially dangerous in terms of driving. In addition to surveys of traffic fatalities and crashes, barbiturate effects on driving-related skills also have been investigated in laboratory studies. Motor control, perception and information processing play substantial roles in driving. The following discussion considers the effects of barbiturates on these functions.

Dickins, Lader and Steinberg [1965] report that 300 mg cyclobarbitone, an intermediate acting barbiturate, degrades motor control as measured by the frequency of taps in a finger-tapping task. Kornetsky, Vates and Kessler [1959] and Goldstein, Searle and Schimke [1960] found the frequency of taps to be reduced under 200 mg secobarbital, also an intermediate acting barbiturate. The latter authors found a hand-steadiness task to be affected also. This task required subjects to insert a metal stylus into a metal ring with the arm extended. The score was the total number of contacts of the stylus with the ring.

Simple reaction time also shows deterioration under barbiturates. Goodnow, *et al.* [1951] tested subjects 4 hours after ingestion of 100 mg pentobarbital and reported that the speed of reacting to an auditory stimulus was slowed. Simple visual reaction time is slowed under barbiturates [Goldstein, Searle and Schimke, 1960] and complex reaction time requiring discrimination ability is also slowed.

Tasks requiring visual-motor coordination performance have shown impairment under barbiturates. Dickins, Lader and Steinberg [1965] asked subjects to track dots in a rotating metal disk. They were instructed to hit as many dots as possible with a metal stylus. A dose of 300 mg cyclobarbitone greatly reduced the performance scores at 40, 60, 80 and 100 minutes after ingestion. Goldstein, Searle and Schimke [1960] with a task requiring processing of low amounts of information, showed barbiturates to affect arithmetic computation. Goodnow, *et al.* [1951]

reported slowing of response time in a task requiring naming the opposite of common words by barbiturates. Digit symbol substitution tests and symbol copying tests are also degraded by barbiturates [Kornetsky, Vates and Kessler, 1959].

A variety of studies have investigated the effects of barbiturates on oculomotor functions. It is reported that nystagmus is induced under low doses of barbiturates [Bergman, Nathanson and Bender, 1962] and low doses also interfere with eye vergence. Westheimer and Rashbass [1961] found that 390 mg of sodium amytal reduces the amplitude of the fusional range with a recession of the near point of convergence. Accommodation measurements were not affected.

Rashbass [1959] found that 100 mg of pentothal (thiopental) abolished smooth pursuing movements with a series of eye movement jerks occurring at each refixation of the target. This is supported by the recent work of Norris [1970] who reported that 100 mg of phenobarbitone suppressed smooth tracking eye movement. Saccadic and vestibular stimulated movements were not affected.

Psychomotor functions requiring division of attention and tracking are sensitive to barbiturate effects. These functions are clearly important for automobile driving. McKenzie and Elliott [1965] tested subjects on a flying simulator under 200 mg secobarbital. Subjects were required to monitor simultaneously four instrument displays, which varied independently in direction and rate. The subject's task was to keep the indicators "on target" by manipulating the proper controls (stick, rudder pedals and a throttle quadrant control). Testing occurred 10 hours after ingestion and lasted 12 hours with a 15-second rest periods following each minute of operation. The results indicated that multiple pursuit tracking is degraded at testing 10 hours after ingestion and continues to show degraded performance for the remaining 12 hours.

A number of vehicle-handling tests have also been carried out under barbiturates. Betts, Clayton and Mackay [1972] examined the effects of 150 mg sodium amytal on three vehicle handling tasks. These were a weaving test, a parking test and a gap estimation test requiring subjects to estimate the gap between two traffic cones through which they could drive. Fifty men and 50 women were subjects. The men increased their failures in gap estimations under sodium amytal. The women decreased their distances from the curb in the parking test but increased their successes in gap estimation.

Simulated driving skills have also shown impairment under barbiturates. Loomis and West [1957] tested eight subjects in a driving simulator at various time intervals, from 1 to 6 hours, after ingestion of 100 mg secobarbital. They measured the subject's response times to braking to the appearance of a red light, and to the release of foot pressure on an accelerator pedal at the appearance of an amber light. A steering score was also taken by measuring the cumulative time during which the subject was not "centered on the road". Secobarbital produced a significant decrease in performance on all measures. These tests are sensitive to the barbiturate-induced changes because they represent the demands of driving in that they require the subject to perform multiple acts simultaneously.

Doenicke [1962] testing 25 subjects under 200 mg butabarbital, found impairment up to 24 hours after ingestion. Kielholz [1967, 1969] gave subjects 200 mg phenobarbital, required them to drive over a test course 2 hours after ingestion, and found significantly impaired driving ability.

Clearly, barbiturates degrade skills which are components of driving. Reaction times increase and performance of a variety of skilled tasks are impaired. This indicates that driving may be severely impaired under barbiturates.

The deleterious effect of alcohol on driving ability has been documented in the literature. There is evidence that the combined use of alcohol and barbiturates produces greater hazard for driving than the single use of either drug.

Devenyi and Wilson [1971] reported that 70% of drug-abusing alcoholics and 10% of all alcoholics admitted to the Addiction Research Foundation Unit in Ontario, Canada has used barbiturates. Since the intoxicating effects of barbiturates are similar to those of alcohol, alcoholics often use alcohol together and interchangeably with barbiturates [Glatt, 1962]. A 1969 study [Finkle] in Santa Clara county in California found that of some 10,000 routine investigations of drinking drivers, 1·4% were under the influence of barbiturates. This represented 67% of all drugs identified in the drivers.

In a recent survey of traffic accidents Turk, McBay and Hudson [1974] found 3% of pedestrian fatalities had both alcohol and barbiturate involvement. Two and a half percent of the

cases showed the presence of alcohol and barbiturates in operators involved in fatal single automobile crashes.

In the Kielholz, Goldberg and Im Obersteg studies [1967, 1969] cited earlier a 0·08% blood alcohol level increased the impairment in driving found under barbiturates. Gupta and Kofoed [1966] also found the combination of alcohol and barbiturates to further impair driving ability beyond the levels of impairment under either drug. This is in contrast to Betts, Clayton and Mackay [1970] who found no interaction between a combination of about 0·05% blood alcohol and barbiturates in the performance of parking tests, gap estimation tests and a weaving task. The latter lack of an effect may have been due to the method of administration of the drugs. The barbiturate was consumed in 30 mg doses over a period of 6 hours, after which alcohol was given, and then 1 hour elapsed before testing. It is possible that the effect of the barbiturate had disappeared at the time of peak blood alcohol level.

The consensus is that the effects of alcohol are enhanced by barbiturates [Joyce, Edgecombe, and Kennard, 1959; Smith, 1966]. It has also been established that the disappearance of barbiturates from the blood is slowed by alcohol [Melville, Jordan and Douglas, 1966]. This would tend to prolong the behavioral effects of the combined use of the drugs.

Although both simple additive and synergistic models have been advanced [Forney and Hughes, 1969], the interactive mechanisms of alcohol and barbiturates are not known. The laboratory evidence relating to barbiturate use and driving suggests that it is unsafe to drive under barbiturate influence. The effects appear at moderate doses and appear to be long-lasting. The epidemiological evidence is unclear in implicating barbiturates in traffic accidents due to methodological problems. However, the increased deterioration in driving skills under barbiturates, alcohol or under a combination of alcohol and barbiturates indicates that driving should be avoided under their influence.

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DRUG USE AND DRIVING RISK AMONG HIGH SCHOOL STUDENTS

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Abstract—No studies have yet been made of high school drug users which determine the frequency of accidents and the frequency of drug related accidents, with comparisons of driving exposure while under various drug effects. The present study attempts all of these tasks.

The study population was 1538 upper level high school students in Toronto, chosen at random. Anonymous questionnaires of known validity were used to collect information about drug use, accidents, violations, drug related accidents and violations, and numbers of drug-driving occasions.

Of the 1538 students, 710 had driven in the past year. About 15% reported an accident and 20% a driving offence. Users of all drugs more often reported accidents than non-users but the results were statistically significant for tobacco, marihuana, opiates, speed, LSD and other hallucinogens. Only 2·7% had an alcohol-influenced accident and 2·0% a drug-influenced accident. Exposure to drinking and driving was far more common than drug use and driving (56% of students compared to 1 to 6%). When exposure to drug related driving occasions are considered LSD, tranquillizers and stimulants are the most dangerous drugs and they are more dangerous than alcohol. The infrequent use of drugs makes their total effect on accidents small compared to alcohol.

Research on drugs and driving is typically of several types as described by Smart [1974a] and includes: (1) laboratory, simulator and closed course studies of the impairing effects of various drugs and combinations; (2) surveys of the prevalence of drug use in driving populations; (3) studies of drug use among various persons involved in accidents; and (4) studies of accident rates among drug using or drug abusing populations. Several reviews of research in these areas have been completed lately [Nichols, 1971; Smart, 1947b] and the work done by these reviews need not be duplicated here. It is probably sufficient to note that both reviews indicated that there were considerable gaps in knowledge of the epidemiological aspects. The review by Smart [1947b] pointed out the need for studies of hallucinogens (including cannabis), opiates, and stimulants in driving risk. Because easy roadside screening methods for these drugs have not been developed, much research in this area must be epidemiological rather than experimental or pharmacological and it must depend largely on self-report. This paper reports a study of self-reported accident and offence involvement related to drug use for a large sample of high school students.

Several studies have been made of accident and offence rates among drug users such as heroin addicts and cannabis users [e.g. Moser *et al.*, 1972; Waller, 1971; Waller and Goo, 1969; Smart, 1974a] with rather inconsistent results. Apparently no study has attempted to determine the frequency of accidents occurring under the effects of various drugs, using an epidemiological method with high school students.

The present study focuses on high school students as one of the heaviest drug using groups [Berg, 1970; Mercer and Smart, 1974]. The general aim was to determine the frequency of accidents for users and non-users and the frequency of drug-related accidents. High school students have not had much driving experience and hence drug effects on their driving may be greater than with more experienced groups. A further purpose was to make some comparison of driving exposure while under various drug effects. A recent study by Smart [1974c] indicated that cannabis users had far fewer accidents under cannabis use than under alcohol. However, when exposure in terms of driving occasions was taken into account this large difference almost disappeared. In this study a variety of drugs such as alcohol, tobacco, hallucinogens, opiates, cannabis, etc. were investigated for both exposure and accident relationships.

METHOD

This study was part of a larger survey of drug use among Toronto high school students which has been conducted every 2 years from 1968 [Smart and Fejer, 1974].

Sample

The sample involved some 1538 Grade 11 and 13 students chosen at random by a method established in previous surveys in 1968, 1970 and 1972. In general, classes were chosen at random from 20% of the high school districts also chosen at random until 120 students of each sex were obtained from each grade in each district. In 1974, this method had to be modified in some schools because individualized programs meant that classes could not be sampled as in the past. In these cases school principals were asked to designate a group of students equivalent in grade and program to those sampled in 1972.

Only grade 11 and 13 students completed the section of the questionnaire on driving and drug use because so few students in earlier grades would be licenced to drive. In all, some 710 students held a drivers licence and their responses provide the basic data for this study.

Data collection

Data were collected by means of an anonymous questionnaire administered in a classroom situation. All students required parental permission to participate and these permissions were available in a variety of languages. About 69% of all eligible students in the selected classes participated and most of those not participating were lost because permission forms were not taken home or returned by students.

The questionnaire included questions on the use of 12 drugs, knowledge and attitudes about drugs, drug education programs and the relationship between driving and drug use. Some of these questions had been included in earlier studies, especially those on drug use, in the 1970 and 1972 studies.

Considerable validity and reliability information has been accumulated for these and similar drug use questions. Much of this has been summarized in an earlier report [Whitehead and Smart, 1972] and only a few details are important here. In one study, the anonymous self-report method was compared with a group method of asking about "others" behaviour and similar results were obtained. In another study non-existent drugs were enquired about and few claimed to have used them. Later, a 1972 study used a lie scale and found that few students exceeded the cut off point and that those who did had little effect upon the rates of drug use.

RESULTS

The driving sample

On the 1538 students 710 or 46·9% had driven at least once in the past year. About 67% of the 710 were males. It should be noted that this is a young and inexperienced driving sample. Those licenced were mostly 18 years of age or older. Most (84%) had held a licence for 2 years or less and only 7·5% for 3 years or more. Driving exposure in terms of miles driven was low (e.g. 59% had driven as much as 5000 miles and only 9% had driven more than 30,000). Only 15% reported one or more car accidents in the past year and 20·2% reported a driving offence (i.e. exclusive of parking offences). These figures are not very different than expected from a study of Ontario drivers of the same age [Ontario DOT, 1969] although an exact comparison is difficult because all accidents, not just reported accidents were enquired about and because basal data related to a period five earlier than that for the present study.

Accidents and driving offences among drug users

Table 1 shows the percentage of various drug users and non-users reporting accidents. It can be seen that users of all drugs more often report accidents than do non-users. However, the differences are statistically significant ($p < 0{\cdot}001$) only for the following drugs: tobacco, marihuana, opiates, speed, LSD and other hallucinogens. The largest differences are for users of opiates, speed, LSD and other hallucinogens where the rate is three to four times as high for users as non-users.

Similar results can be seen in Table 2 for driving offences. Again, all types of users have higher rates than non-users but the results are statistically significant only for tobacco, marihuana, stimulants and other hallucinogens. The largest differences are for stimulants and other hallucinogens. Some of these results are based on relatively few driver users ($n = 15$, and 6 for solvents and glue) and they should be cautiously interpreted.

Table 1. Number and per cent with car accidents

DRUG	Non-Users		Users		Chi Square	P <
	Total Number	% With Accident	Total Number	% With Accident		
Tobacco	426	11.5	284	20.8	10.65	.001
Alcohol	52	11.5	658	15.5	.04	.80
Marihuana	439	11.4	271	21.4	13.81	.001
Glue	704	15.1	6	33.3	.43	.50
Solvents	695	15.0	15	26.7	.78	.30
Barbiturates	579	14.0	131	20.6	3.13	.05
Opiates	685	14.2	25	44.0	14.41	.001
Speed	693	14.1	17	58.8	22.33	.001
Stimulants	670	14.6	40	25.0	2.39	.10
Tranquillizers	634	14.0	76	25.0	5.50	.02
LSD	681	13.8	29	48.3	23.02	.001
Other Hallucinogens	658	13.4	52	38.5	21.61	.001

Table 2. Number and per cent of users and non-users of drugs with driving offences

DRUG	Non-Users		Users		Chi Square	P <
	Total Number	% With Offence	Total Number	% With Offence		
Tobacco	426	15.8	284	27.5	15.03	.001
Alcohol	52	13.5	658	20.4	1.14	.20
Marihuana	439	15.5	271	27.7	14.72	.001
Glue	704	20.0	6	33.3	.08	.70
Solvents	695	20.0	15	26.7	.09	.70
Barbiturates	579	20.4	131	19.1	.48	.30
Opiates	685	19.6	25	36.0	.55	.30
Speed	693	19.9	17	29.4	1.61	.20
Stimulants	670	19.1	40	37.5	6.83	.001
Tranquillizers	634	19.6	76	25.0	.93	.70
LSD	681	19.7	29	31.0	1.57	.20
Other Hallucinogens	658	18.4	52	42.3	15.68	.001

Percentage of drivers having accidents while under the influence of various drugs

As expected, small proportions of drivers were involved in accidents under various drug effects. Table 3 shows the proportions of drivers ($n = 710$) and of users reporting drug-influenced accidents. Only 2·7% of all drivers have had an accident under the influence of alcohol and this exceeds the total for all other drugs combined (2·0%) each of which is of minor importance

Table 3. Number and per cent of drivers having accidents under various drug effects

DRUG	Number of Drivers With Accidents	(n=710) % of Drivers With Accident	% of Users With Accident
Alcohol	19	2.7	2.7
LSD	2	.1	3.4
MDA or other Hallucinogens	3	.3	1.9
Tranquillizers	5	.7	3.9
Sleeping Pills	2	.3	.7
Speed	0	0	0
Stimulants	4	.6	5.0

separately considered (less than 1% of drivers). However, when those with accidents are expressed as a proportion of users, alcohol appears one of the *less* dangerous drugs. Users of drugs such as LSD, tranquillizers and stimulants more often report accidents under their influence than do drinkers of alcohol. Data on the percentage of drivers having accidents under cannabis use have not been included because of an error in the presentation of the question on cannabis.

Questions were also asked about the frequency of driving under different types of drug use. The data are shown in Table 4 and indicate something of relative exposure to various drug use (other than cannabis) and driving situations. Clearly, exposure to drinking accidents is far greater as about 56% of all drivers drink and drive. However, only 1·3 to 6·1% of drivers report driving after drug use. Only 1 to 2% of drivers report drinking and driving after drug use as often as 3 times but about 34% report drinking and driving this often. It would appear from Table 4 that drinking and driving is 9–60 times as common as any type of drug use and driving. Reported accidents under all drugs are far more common than those under alcohol taking exposure into account (except speed).

Accidents under all drug effects are nearly as common as those under alcohol (14–19) but exposure in terms of driving occasions is only 28% of the drinking-driving occasions. Driving is most common under the effects of tranquillizers although drinking and driving is still 9 times as common.

Table 4. Reported frequency of drug use and driving (Total Sample n = 710)

DRUG	Number of Times							Total Drug Use Driving Occasions*
	0	1	2	3	4	5+	Blank	
Alcohol	42.7%	12.4%	9.4%	4.6%	4.5%	24.8	1.4%	1278
LSD	96.6%	1.0%	0	.4%	.1%	.6%	1.3%	48
MDA	96.5%	1.0%	.6%	.1%	0	.6%	1.3%	57
Tranquillizers	92.5%	2.5%	1.5%	.6%	.1%	1.4%	1.3%	132
Sleeping Pills	96.1%	1.3%	.6%	.3%	.1%	.4%	1.3%	48
Speed	96.8%	.8%	.3%	.0%	.1%	.1%	1.8%	21
Stimulants	95.1%	1.4%	.8%	.3%	.3%	.4%	1.7%	57

* Taking 5+ as 7 times in each case.

Percentage of drivers involved in accidents as passengers because of various drug effects

Table 5 shows the proportions of all grade 11 and 13 students who reported involvement in accidents because of various drug effects in the drivers. About 10% of students were involved in an alcohol related accident. All other drugs individually involved fewer than 1·4% of students and the total for all drugs is less than for alcohol.

Table 5. Number and per cent of grade 11 and 13 students involved in accidents as passengers because of various drug effects

DRUG	Number of Students	Per Cent of Grade 11 & 13 Students (n = 1538)
Alcohol	158	10.3
LSD	16	1.0
MDA	13	.8
Tranquillizers	21	1.4
Sleeping Pills	6	.4
Speed	12	.8
Stimulants	16	1.0

DISCUSSION

Non-users of all drugs less frequently report accidents than do users. The significant differences are for users of tobacco, marihuana, opiates, speed, LSD and other hallucinogens. Few students report having an accident while under the influence of any drug enquired about and the drug most commonly mentioned was alcohol. When the data are expressed as a percentage of drivers alcohol is far more commonly mentioned, but when they are a percentage of users of a given drug it is not. At that point the most dangerous drugs appear to be LSD, tranquillizers and stimulants. It was also found that drinking-driving occasions are far more numerous than drug-influenced driving occasions. When the exposure levels (drug-driving occasions) are taken into account tranquillizers and stimulants appear more dangerous than does alcohol.

In the present study it appears that drugs such as hallucinogens, tranquillizers and stimulants are relatively (compared to alcohol) unimportant in accidents of high school students. However, this may be because of their infrequent use; such drugs involved only 5 to 8% of students as users. Any social or legislative changes which resulted in increased drug use would be likely to lead to higher accident rates for them. Expressed as a problem for users they appear substantial but they are a relatively trivial problem for the general population because of their infrequent use. Two or three times as many users of LSD, other hallucinogens or tranquillizers report accidents under their influence than do users of alcohol under alcohol influence.

A number of methodological considerations should be kept in mind for this study. Although this study involved a large sample of drivers, users of some drugs, e.g. glue, solvents, were not numerous. This is partly because drugs such as glue are more commonly used by younger, probably unlicenced students [Smart, Fejer and White, 1970] and because the use of drugs such as solvents and hallucinogens has been decreasing in this population for several years. It might also be noted that the data are based on self-report, albeit on a questionnaire with some validity. At present, the questions raised in this study could probably not be investigated without self-report data. Total reported accident involvement is not greatly different than expected on the basis of a recent Ontario study of the general driving population, thus lending some evidence to the findings.

It is also worth noting that some drugs such as cannabis are frequently used with alcohol. An earlier study with college students [Smart, 1974c] showed that about half of all cannabis accidents also involve drinking. Unfortunately, few relevant studies have been made with which to compare the present findings. No study has been made of high school students who would be both inexperienced drug users and inexperienced drivers. Moser *et al.* [1972] found no accident rate difference for drug users and non-users but theirs was a prison sample. Studies of cannabis users and accident rates has always been somewhat contradictory [Smart 1974a] and no previous study has looked at such a variety of drugs separately.

It would be of interest to repeat this study with a larger sample of students, perhaps including college students as well. In general, the findings tend to confirm the earlier study by Smart [1974c] which showed that college student cannabis users, had infrequent accidents under cannabis (compared to alcohol) but that the difference was far less important and almost non-existent when cannabis-driving occasions were taken into account. Until further data are available it seems possible to conclude that most drug use is rarely a driving risk for whole populations but a much larger risk given the small number of drug and driving occasions. It would be of interest in later studies to determine the actual number of miles driven under the effects of various drugs, although this may be a difficult task. Later studies should also include a variety of drug using populations such as college students and adults.

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TRAFFIC ACCIDENT RATES AMONG FINNISH OUT-PATIENTS

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Abstract—A questionnaire was administered to 765 rheumatoid arthritic, 715 tuberculous, and 1050 psychiatric outpatients concerning their use of alcohol and drugs, driving habits, and traffic accident involvement. The same questionnaire was administered to 587 controls. The driving populations of all groups were matched as to their age, and living district. The main finding of the study was that, as the traffic exposure was controlled, the non-drug treated patients were not involved in accidents more often than the controls. In the psychiatric out-patient group, drug use was linked with an increased accident rate. Heavy use of alcohol was linked with increased traffic exposure in the present study. The combined use of alcohol and drugs tended to increase one's involvement in accidents.

INTRODUCTION

Early questionnaire studies have demonstrated a moderate increase in road traffic accident rate among drug users [Nichols, 1971]. The only drug-specific epidemiological study conducted so far, demonstrated diazepam to be present in the blood of 18% of traffic accident victims admitted to hospitals. This figure was higher than expected from the consumption statistics [Bo, Haffner, Langard *et al.* 1974]. Because the study excluded accidents without human injuries severe enough to warrant hospitalization, a majority of road traffic accidents were excluded from the data. The present study was conducted among three out-patients groups generally receiving drugs for prolonged period of time. The issues of interest were: 1. The driving habits of chronically ill out-patients; 2. The use of drugs and alcohol among out-patients; 3. The relation between alcohol and drug use, and driving characteristics.

MATERIAL AND METHODS

The subjects were 765 rheumatoid arthritic, 715 tuberculous, and 1050 psychiatric out-patients treated by socient-supported facilities throughout Finland, which are used exclusively for the treatment of these disorders. The samples were randomly selected from patients attending the clinics. The overall return rate of the questionnaires was above 70%. However, some of the out-patient clinics which originally agreed to cooperate did not do so. When the units not giving the questionnaires to their patients at all were dropped from the material, the return rate of the patient groups was above 90%.

The control group was matched with the patient groups as to age range, which was between 18 and 70 years, and living-district. The control group was sampled immediately after the patient groups and it did not include subjects from districts where the open care units did not cooperate. The size of the control group was 587 persons. They were chosen from the censuses of the counties having the out-patient clinics. The ages of the subjects were within the age range during which one is allowed to drive a car in Finland. All data were collected during 1 January–31 October, 1973. For the variables included in this study see below.

Variables included in the questionnaire

For this study, reasons for driving were categorized as either recreation, going to work, or chauffeur, where more than half of the individual's driving could be attributed to one of these categories.

Alcohol use was reported by subjects in terms of the amount usually consumed at a drinking session. They indicated the kind of alcoholic beverage and the amount, expressed in lay measures

such as glasses or standard size bottles. From these data then were calculated grams of absolute alcohol ingested during a typical drinking session.

Drug use was indicated as the number of drugs used concurrently on a daily basis.

Subjects were asked for the number of traffic accidents in which they had been involved as drivers during the 2 years previous to the study.

RESULTS

Characteristics of the subjects

Even though the total groups differed from each other in many respects as expected from the epidemiology of the diseases, subjects possessing driver's licenses proved to be relatively similar; i.e. within all groups the majority were men, aged 30–49 years. Males represented 70% of the drivers in the control group, 89% of the drivers in both the psychiatric and rheumatoid arthritis groups. Generally the social status of the drivers [Rauhala, 1966] was similar in the patient and control groups. A significant exception was that 33% of the drivers in the psychiatric group were of the lowest social class whereas the respective figure in the control group was 13% ($p < 0{\cdot}01$).

Driving characteristics

Significantly fewer subjects of the rheumatoid arthritic and psychiatric groups possessed a driver's license that those of the control group. The tuberculous group did not differ from controls in this respect (Table 1).

The drivers of the rheumatoid arthritic and tuberculous groups drove significantly more often under 10,000 km annually than the drivers of the control group (Table 2). Patient groups reported more often than the control group that the type of route mainly used was a highway (Table 3).

The main reason for driving was difficult to decide in many cases. Many subjects indicated two categories as equally important motivations; therefore the percentage figures total more than 100%. The main difference between the control and patient groups was the small number of chauffeurs in the patient groups and the large amount of recreational driving among rheumatoid arthritic and psychiatric groups. This finding was statistically significant (Table 4). The number of traffic accidents during 2 years preceding the study did not differ significantly between the tuberculous and control groups. The rheumatoid arthritic group was involved in accidents less often than the control group, and the psychiatric group was involved in accidents more often than the control group (Table 5).

Table 1. Possession of driver's license

Driver's License	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
n	587	765	715	1050
Percentage with	58[a12]	37[a1]	59	29[a2]
Percentage without	42	63	41	71
Total	100	100	100	100

a_{12} = Statistically significant difference at the level of 1%

Table 2. Traffic exposure

Kilometers Driven Annually	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
n	326	229	378	239
	Percent	Percent	Percent	Percent
0	4 } 38[a12]	9 } 47[a1]	4 } 29	12 } 52[a2]
1 - 9,999	34 }	38 }	25 }	40 }
10,000 - 19,999	26	27	23	25
> 20,000	36	26	48	23
Total	100	100	100	100

a_{12} = Statistically significant difference at the level of 1%

Table 3. Road types used

	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
n	328	319	378	297
	Percent	Percent	Percent	Percent
Street or Local Road	37	29	21	22
Highway	$63^{a}123$	$71^{a}1$	$79^{a}2$	$78^{a}3$
Total	100	100	100	100

$^{a}123$ = Statistically significant difference at the level of 1%

Table 4. Motivation of driving

	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
n	332	229	358	
	Percent	Percent	Percent	Percent
Recreation	$55^{a}12$	$62^{a}1$	51	$68^{a}2$
Going to Work	$52^{a}3$	45	56	$43^{a}3$
Chauffeur	$35^{a}456$	$16^{a}4$	$18^{a}5$	$11^{a}6$

$^{a}123$ = Statistically significant difference at the 5% level

$^{a}456$ = Statistically significant difference at the 1% level

Table 5. Accident involvement

	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
n	332	208	258	236
	Percent	Percent	Percent	Percent
Involved	$35^{a}12$	$23^{a}1$	32	$41^{a}2$
Not Involved	65	77	68	59

$^{a}12$ = Statistically significant difference at the 1% level

Characteristics of drug use

The use of prescribed drugs was high in all patient groups. Seventy-eight percent of the rheumatoid arthritic group had used drugs regularly for several years previous to the study. In the tuberculous group the respective figure was 82% even though the duration of drug treatment was generally shorter than that in the rheumatoid arthritic group. In the psychiatric group 71% of the patients used continuous medication. All these figures are significantly ($p < 0{\cdot}01$) higher than the 41% of the control group using some kind of prescribed drugs (Table 6).

Characteristics of alcohol use

The rheumatoid arthritic subjects used alcohol significantly ($p < 0{\cdot}01$) less often than the control group and also drank significantly ($p < 0{\cdot}01$) less alcohol per drinking session than the control group. A similar tendency was evident in comparing the tuberculous groups with the control group (Table 7).

Alcohol, drugs, and driving interactions

In all groups, subjects using more alcohol during drinking sessions also drove more and were more often involved in accidents. If the exposure factor was eliminated, alcohol use per drinking session did not significantly affect accident involvement per kilometers driven in the tuberculous and psychiatric groups. However, in the control and rheumatoid arthritic groups alcohol use was positively correlated with accident involvement (Table 8).

The significant features of the accident involvement of the groups were that despite the low exposure of the psychiatric group's nondrinkers, they were significantly more often involved in

Table 6. Characteristics of drug use of rheumatoid arthritic, tuberculous, psychiatric patients having driver's license

	Control Group	Rheumatoid Arthritic Group	Tuberculous Group	Psychiatric Group
The Use of Prescribed Drugs				
n	284	223	319	252
	Percent	Percent	Percent	Percent
Do Not Use Drugs	59[a]123	8[a]1	16[a]2	9[a]3
Uses 1 - 2 Drugs	36 } 41	55 } 92	55 } 84	54 } 91
Uses 3 or More Drugs	5	37	29	37
Total	100	100	100	100
The Frequency of Drug Use				
n	92	156	247	190
	Percent	Percent	Percent	Percent
Uses Every Week	49[a]456	22[a]4	15[a]5	18[a]6
Uses Every Day	51	78	85	82
Total	100	100	100	100
The Regularity of Drug Use				
n	111	165	280	202
	Percent	Percent	Percent	Percent
Uses Regularly	78	81	93	73
Do Not Use Regularily	22	19	7	27
Total	100	100	100	100
The Duration of Drug Use				
n	94	164	223	196
	Percent	Percent	Percent	Percent
- 999 Days	67	49[a]7	40[a]8	53[a]78
1,000 - 2,999 Days	33	34	34	34
3,000 -	--	17	26	13
Total	100	100	100	100

[a]12345678 = Statistically significant difference at the level of 1%

Table 7. Characteristics of alcohol use among rheumatoid arthritic, tuberculous and psychiatric patients having driver's licenses

	Control Group	Rheumatoid Arthritic Group	Tuberculous Group	Psychiatric Group
Type of Alcohol				
n	328	234	379	264
	Percent	Percent	Percent	Percent
Nondrinkers	15 [a]123	33 [a]1	29 [a]2	27 [a]3
Use Only Beer or Wine	16	18	17	25
Use Only Hard Liquor	17	23	18	18
Use Both Types	52 [a]456	26 [a]4	36 [a]5	31 [a]6
Total	100	100	100	100
Amount of Alcohol Per One Drinking Session				
n	279	154	258	190
	Percent	Percent	Percent	Percent
1-49 G Per One Drinking Session	33 [a]7	50 [a]7	36	33
≥ 50 - G Per One Drinking Session	67	50	64	67
Total	100	100	100	100
The Frequency of Drinking				
n	275	154	260	191
	Percent	Percent	Percent	Percent
Seldom (1 Time a Month)	43 [b]1[a]8	60 [a]8	56 [b]1	46
Moderately Often (1-3)	36	27	30	25
Often (3 times or more)	21	13	13	29
Total	100	100	100	100

[a]12345678 = Statistically significant at the level of 1%
[b]1 = Statistically significant at the level of 5%

Table 8. Exposure controlled accident involvement as a function of the use of alcohol during a drinking session

G of alcohol	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
0	0.07	0.05	0.09	0.17
1 - 49	0.10	0.10	0.10	0.12
> 50	0.13	0.11	0.08	0.19

Number of Accidents/5,000 kilometers

accidents than the control group's nondrinkers (Table 8). Also the psychiatric group's drinkers were involved in accidents per kilometers more often than the heavy drinkers of the control group (Table 8). Within the control and tuberculous groups, the heavy use of alcohol was significantly ($p < 0{\cdot}01$) more often linked with a chauffeur's license than with other reasons for driving.

Drug use by rheumatoid arthritic subjects seemed to be linked with fewer kilometers driven annually as compared with the control group. The significantly lower number of accidents in the group of rheumatoid arthritic patients using 1 to 2 drugs, as compared with the respective figure of the control group as absolute number, was not real if the number of accidents was divided with the number of kilometers driven (Table 9). This finding turned out to be due to different exposure (Table 9). In the psychiatric group, the drug users drove only a few thousand kilometers annually and still they were often involved in accidents (Table 9). Within all the patient groups, the nondrug subjects had similar accident involvement per kilometers driven as the nondrug control group (Table 9).

If the accident rates per kilometers driven of the drug and drug + alcohol-using psychiatric patients are compared with those of nondrug-using control subjects, as the high number of accidents among the medicated psychiatric patients becomes evident (Tables 8 and 9).

DISCUSSION

All questionnaire studies about drinking and driving have had severe limitations [Nichols, 1971]. There have been no studies to answer this question: How much of observed impairment among the patient groups was due to the disease, and how much was due to its treatment or the interaction of these factors?

The present study was undertaken to elucidate the above mentioned relationships. Through the comparison of the nondrug patient groups with controls, the role of the diseases as such on accident rates was revealed. This proved to be insignificant. However, it should be kept in mind that heavy medication generally reflected a severe disease, and the role of the severity of the disease in impairing skills related to driving remained somewhat obscure.

The reliability of the present results is believed to be good, due to the high percentage of the subjects returning the questionnaires, and the strict confidentiality of the data. In no phase of the study were the questionnaires labeled with the names of the patients nor with any symbols referring to them. The health care personnel treating the patients did not have access to information from the individual data sheets because they were sealed in envelopes by patients themselves.

The relatively low number of driver's licenses among the rheumatoid and psychiatric patient groups might be due to three factors: 1. The disease *per se* impaired the person so severely that

Table 9. Exposure controlled accident involvement as a function of the regular use of drugs

No of drugs	Control Group	Rheumatoid Arthritis Group	Tuberculous Group	Psychiatric Group
0	0.08	0.10	0.07	0.10
1 - 2	0.08	0.07	0.09	0.20
3	0.05	0.09	0.10	0.22

Number of Accidents/5,000 kilometers

he or she was not willing to drive. 2. Income was so low due to the disease that the person could not afford driving. 3. The health care personnel had recommended discontinuing driving due to the disease or police had confiscated the license for the same reason. In the rheumatoid arthritic group, the first possibility was probably most common, because a majority of the patients in Finland were elderly women having severe disease [Rimon 1969]. On the other hand, in the psychiatric group, the social status of many subjects was such that they could not afford driving.

Despite the preselection of license holders in the rheumatoid arthritic and psychiatric groups, as reflected in the relatively low numbers in those groups, they also drove less than those in the control group. This probably reflected an impairment either due to the disease or its treatment. The low number of kilometers driven annually meant that the rheumatoid arthritic and psychiatric outpatients were more inexperienced in the present traffic conditions than the tuberculous and control subjects. This fact as such might increase their traffic accident risk. On the other hand, the rheumatoid arthritic and psychiatric outpatients drove more often for recreation than the control group. Thus they often had the option of choosing the time of driving with the possibility of avoiding difficult traffic conditions.

Use of alcohol

The smallest portion of abstainers was found among the control group. In all groups, a heavy use of alcohol per drinking session was linked with increased driving except in the psychiatric group, where the effect of heavy alcohol use on driving seemed to be bimodal. The psychiatric non drinkers were involved in accidents per kilometers driven more often than the respective control subjects. Within the tuberculous group, the heavy use of alcohol was linked more often with accidents than the nonuse of it. This seemingly increased accident rate was, however, mainly due to increased exposure to traffic.

Use of drugs

A surprising finding was that 41% of controls used some kind of medication. As to the correlation between *drug use* and accident rates, the most important finding was that none of the *nondrug-using* patient groups had higher accident rates than that in the respective control group. One the other hand, in the psychiatric group, subjects using one or more drugs were involved in more accidents than the nondrugged patients. The psychotropic drugs may be a significant contributing factor to traffic accidents.

In the rheumatoid arthritic and tuberculous groups, the use of drugs did not increase accident rates. This may be due to the antirheumatic treatment frequently being acetylsalicylic acid, which seemed not to impair one's driving related performance in the laboratory [Linnoila *et al.*, 1974]. Isoniazid, the most common antituberculous agent in Finland, has been demonstrated to impair one's driving skills in a simulator study [Linnoila and Mattila, 1973]. This effect was not evident in the present study, and it is probably true only during the acute phase of drug administration.

Among the heavy drinkers from both the control group and the psychiatric patients using 1–2 drugs, about *half* had been involved in accidents during the two years prior to the study. This high figure suggests that heavy use of alcohol with or without the use of other drugs, increases the accident risk factor.

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MARIHUANA: EFFECTS ON SIMULATED DRIVING PERFORMANCE†

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Abstract—Performance of 23 male subjects was tested under smoked marihuana treatments on 4 occasions in a complex driving simulator. Doses were 0, 50, 100 and 200 micrograms delta-9 tetrahydrocannabinol per kilogram bodyweight.

The simulator uses a car mounted on a chassis dynamometer facing a filmed scene subtending 160 degrees. Twenty-five performance measures are derived based on steering wheel, brake and accelerator pad usage as well as speed and tracking. The simulator also incorporates a visual search-and-recognition task based on random appearance of lights in the periphery.

There is little evidence for a significant effect of marihuana upon car control and tracking. None of the 25 car control-tracking scores was significantly changed in either mean or variance by the treatments. However, there was a clear, statistically significant decrement in performance of the search-and-recognition task. Marihuana produced increased errors in recognition of the lights and delayed response times to their appearance.

The results suggest the prime locus of marihuana impairment of driving performance as being in the interference with perceptual processes involved in data acquisition necessary for safe control of the vehicle.

Two previous studies have measured the effects of cannabis and alcohol on simulated driving performance, with conflicting results regarding the effects of marihuana [Crancer, Dille, Delay *et al.*, 1969; Rafaelson, Bech, Christiansen *et al.*, 1973]. Crancer, Dille, Delay *et al.* [1969] utilized a simulator in which the subject manipulated steering, acceleration, braking and turn signals in response to a filmed driving presentation. The accelerator depression controlled the speedometer reading, but otherwise the subject's action did not affect the filmed presentation. The smoked marihuana treatment was reported to contain 22 mg delta-9 THC, although subsequent analyses indicate that the actual THC content may have been substantially less [Manno, Kiplinger, Scholz *et al.*, 1971]. The alcohol dose was intended to produce a blood alcohol concentration (BAC) of 0·10%. In comparison to a no-treatment control, alcohol significantly increased errors on 4 of the 5 measures, whereas marihuana only increased speedometer errors, i.e. failure to maintain the speedometer reading within prescribed limits.

Rafaelsen, Bech, Christiansen *et al.* [1973] employed a simulator projecting a moving landscape which was responsive to steering and accelerator changes. The car mock-up was also equipped with red and green lights above the windshield. Placebo treatments were compared with 70 g of ethanol, and oral hashish doses containing 8, 12 and 16 mg delta-9 THC. Both alcohol and hashish increased the latency of stop and start responses to the red and green lights, and impairment resulting from hashish was dose-dependent. The effect of the alcohol dose fell between that for the 2 higher hashish treatments.

The present study was designed to shed further light on the effect of marihuana on simulated driving performance using a more complex simulator than the cited studies.

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METHOD

Subjects

Subjects were 24 male college students between 21 and 32 years of age, with a mean of 24. They had volunteered for a paid experiment without prior knowledge it involved drugs. Applicants were excluded if (1) they reported less than 10 marihuana experiences, (2) they were currently using marihuana more than three times per week or had an extensive history of other drug use, (3) did not have a driver's license and a minimum of 2 years driving experience or (4) gave evidence of emotional or health abnormalities at the initial interview.

The experimental design required 24 subjects to be exposed to 4 treatments in 6 replications of a 4×4 Latin square. One subject did not complete the experiment, so the results are for 23 subjects. Before participating in the experiment, subjects were fully informed regarding the treatments, procedures and possible health consequences. They signed consent forms conforming to HEW requirements for protection of human subjects.

Apparatus

The experiment utilized a driving simulator which has an actual car mounted on a chassis dynamometer facing a 20-foot wide cylindrical screen. The screen subtends a 160 degree view from the driver's eye position. A film photographed from a car during approximately 31 miles of travel is projected on the screen. The subject can proceed over this journey at his own rate since the speed of the film projector is controlled by the subject's use of the brake and accelerator. The subject is required to manipulate the steering wheel in an appropriate fashion as the projected scene moves laterally to follow the contours of the road. The movement is produced by a heading rotation of the projector which is controlled in part by an input from a paper tape recorder. A tape record of the angular movements of the road is synchronized with the film presentation. The final angle of display is a joint function of the inputs from the tape recorder and the subject's use of the steering wheel.

Data produced by transducers which measure the steering wheel, accelerator and brake positions are recorded on analog tape for subsequent computer analysis. These measures are transformed into 25 performance scores designed to describe aspects of driver control and tracking behavior.

The study utilized three films, a training drive and 2 test drives. The 2 test films contained about 85% of common footage. Film sections occurred in different sequences in the 2 films to minimize driver boredom. The study restricted its analysis to 36 segments of the drive which occurred in both films, so that all subjects were examined for the same driving segments on all sessions. These segments, called "events" in the computer output, occupied about 50% of the driving time. They represented a sample of all the driving conditions found on the film.

For the 36 segments the following 25 performance scores are examined:

7 speed scores (speed at beginning of segment, speed at end of segment, minimum speed, maximum speed, average speed in film frames per time unit, average speed in miles per hour, and number of speed reversals of at least 5 mph)

6 accelerator scores (number of reversals of 2% of total possible travel, number of reversals of 5% of possible travel, average accelerator position, maximum position, time to first accelerator let-up of 3%, and time to first complete accelerator let-up)

3 brake scores (maximum brake pressure, time to first brake use, and time from accelerator let-up to first brake pressure)

5 steering wheel scores (average position, average rate of change, number of reversals of 5 degrees, number of reversals of 10 degrees, and number of reversals of 15 degrees)

4 tracking scores (length of path of the car ride, ratio of path driven to minimum possible path, average difference between steering wheel heading, and the heading of the real car path, and maximum difference between steering wheel heading and the heading of the real car path).

In addition, subjects were required to respond to a visual subsidiary task. The purpose of the subsidiary task is to present the driver with a demand for information processing similar to that found in actual driving where attention is divided between a tracking task, and a search and recognition task of the environment. Since the film has less than usual demands for visual search

for possible dangers, the subsidiary task serves to increase the necessity for joint information processing.

The subsidiary task required 1 of 4 lever responses to 4 corresponding light signals. The signals appeared in 2 boxes mounted near the sun visor of the car, approximately 13 inches in front of the subject, 15 degrees to each side and 12 degrees above the eyes. Each box contained an amber and a green lamp. Turn signal levers were mounted on both sides of the steering wheel. When the signal was presented, the subject was required to move the lever on the same side as the light—down for green and up for amber. The light was extinguished by the correct response,or after the lapse of 10 seconds without a correct response. Fifty light presentations were made during the 36 drive segments and were keyed to occur at the same point in the drive on the 2 test films. The measures recorded were (1) number of incorrect responses, and (2) reaction time for correct responses, regardless of whether or not it was preceded by an incorrect response.

Light presentations were keyed to the film frames and always occurred at exactly the same point in the drive. This ensured that they appeared to all subjects under the same conditions of joint information processing with stimuli from the road scene. A wide variety of representative situations on the film were selected for the subsidiary task presentations. Generally, reaction times were greatest at those points which required the greatest attention to the road scene.

Treatments

The 4×4 Latin square experimental design required 4 test days for each subject. The placebo and active marihuana treatments were administered by 2 cigarettes of approximately 1/2 gram each, composed of marihuana containing 1·4% delta-9 tetrahydrocannabinol (THC) and/or detoxified marihuana.

The delivered doses were 0, 50, 100 and 200 micrograms delta-9 THC per kilogram bodyweight (kg B.W.). The marihuana cigarettes were prepared by a person having no contact with the subjects. All persons in contact with the subjects were unaware of the dosage level, and the experiment was a double blind study.

The smoking administration was standardized by requesting subjects to inhale, hold the smoke for 15 seconds and exhale. Inhalations occurred at 35-second intervals, and 20 minutes was allowed for consumption of the two cigarettes. A glass holder was utilized to permit the cigarette butts to be fully smoked.

Procedure

Subjects participated in one training and four experimental test sessions. The treatment sessions for an individual subject occurred at weekly intervals, and at the same time of day.

At the training session subjects were instructed in the car handling procedures, given practice on the subsidiary task alone, and then drove the simulator vehicle over a filmed road scene for approximately 20 minutes. The road scenes used in the training session were not included in the test films. The training driving included the presentation of the visual subsidiary task.

On experimental test days subjects were requested to first relax in a comfortable chain for 15 minutes. Their pulse rates were then taken, followed by the experimental treatment. Smoking required 20 minutes, after which the post-smoking pulse rate was taken. Subjects immediately entered the driving simulator and drove the 31 mile drive which required between 45 and 70 minutes depending on the speed of driving. Pulse rates were again taken after completion of the simulator run.

Table 1 presents the pulse rates for the 4 treatments and shows the characteristic cannabis-induced increments. The explanation for the pulse rate increase following the placebo administration is unknown. It may result from residual active ingredients in the post-extracted

Table1. Mean pulse rate before and after smoking marihuana

	PLACEBO	MARIHUANA DOSE mcg. delta-9 THC/Kg. B.W.		
	0	50	100	200
Before Smoking	76.7	79.8	79.0	79.3
After Smoking	87.6	101.4	106.3	111.1
After Driving	74.0	83.0	83.4	87.0

material, anxiety about the treatment or failure to adequately control for subject activity.

The results for the car control and tracking aspects of the driving simulator are summarized in Tables 2 and 3. Table 2 presents the mean performance scores and Table 3 the mean within-subject variability on these scores. This latter is of considerable importance for driving since inconsistency of response can represent a potential threat to safety.

Examination of the mean and variability scores for the 25 response measures fails to reveal any consistent trend. This impression was supported by the statistical analysis. Data were analyzed both by an analysis of variance (computer program BMD 05V, Health Sciences Computing Facility, UCLA, described in Dixon, [1970]) and by paired comparisons between the placebo and other treatment scores using Student's t test. None of the analyses of variance were statistically significant for treatment effects. Of the 150 paired comparisons only 7 or 4·7% were statistically significant for treatment effects. Such a finding for *post hoc* comparisons would be expected by chance. Thus, the data provide no evidence that marihuana significantly affects the car control performance as measured by the driving simulator.

Table 2. Mean performance scores for 36 segments (events) of the driving simulator runs under 4 marihuana treatments (mcg. delta-9 THC/kg. B.W.)

	PLACEBO 0	50	100	200
Speed at the Beginning of the Event (MPH)	37.211	36.877	37.220	36.050
Speed at the End of the Event (MPH)	37.314	36.652	37.018	36.225
Minimum Speed During the Event (MPH)	32.630	31.926	32.354	31.379
Maximum Speed During the Event (MPH)	41.722	41.484	41.678	40.724
Speed Revs of 5 MPH Per 25 Film Frames	0.052	0.067	0.047	0.053
Average Speed During the Event (MPH)	37.515	36.797	37.221	36.127
Average Speed During the Event (Film Frames/Sec)	25.359	24.273	24.980	23.422
Acc. Revs of 2 Percent per 25 Film Frames	0.132	0.156	0.170	0.156
Acc. Revs of 5 Percent per 25 Film Frames	0.048	0.056	0.059	0.058
Time To 1st Complete Acc. Let-up (Secs)	1.435	1.505	1.580	1.777
Average Acc. Position (Percent Depressed)	5.228	4.879	5.025	4.805
Time to 1st Acc. Let-up of 3 Percent (Secs)	1.519	1.601	1.730	1.607
Maximum Position of Acc. (Percent Depressed)	8.547	8.503	8.636	8.231
Time From Acc. Let-up to 1st Brk Prs. (Sec)	-0.070	-0.077	-0.164	-0.199
Time to 1st Br. Prs. From Start of Evt. (Sec)	0.178	0.360	0.171	0.323
Maximum Amount of Brk Prs. (Percent of Maximum)	2.874	4.963	3.496	2.476
Average Position of the Steering Wheel (Degrees)	-18.086	-17.344	-16.176	-19.600
Average Rate of Chg. of Steering Wheel (Degree/Sec)	163.381	159.653	161.372	159.088
Steer Revs. of 5 Degrees per 25 Film Frames	0.445	0.437	0.482	0.417
Steer Revs of 10 Degrees per 25 Film Frames	0.235	0.237	0.235	0.236
Steer Revs of 15 Degrees per 25 Film Frames	0.162	0.174	0.168	0.168
Len. of Pth. of Car in Event (Eq. Film Frames)	317.184	319.689	330.608	320.088
Ratio of Eq. Film Frames to Real Film Frames	1.050	1.048	1.046	1.054
Average Dif. Between Steering and Steering Comp. (Degrees)	23.182	22.157	22.666	25.394
Maximum Dif. Between Steering and Steering Comp. (Degrees)	51.878	52.996	53.973	57.573

Table 3. Mean within subject standard deviation of performance measures for 36 segments (events) of the driving simulator runs under 4 marihuana treatments (mcg. delta-9 THC/kg. B.W.)

	PLACEBO 0	50	100	200
Speed at the Beginning of the Event (MPH)	7.368	7.391	7.545	7.567
Speed at the End of the Event (MPH)	7.423	7.562	7.858	7.824
Minimum Speed During the Event (MPH)	7.481	7.476	7.789	7.638
Maximum Speed During the Event (MPH)	7.385	7.395	7.810	7.600
Speed Revs of 5 MPH Per 25 Film Frames	0.080	0.104	0.080	0.087
Average Speed During the Event (MPH)	7.890	7.447	8.256	7.397
Average Speed During the Event (Film Frames/Sec)	5.697	5.355	7.713	4.810
Acc. Revs of 2 Percent per 25 Film Frames	0.128	0.154	0.180	0.151
Acc. Revs of 5 Percent per 25 Film Frames	0.076	0.084	0.091	0.088
Time to 1st Complete Acc. Let-up (Secs)	3.135	3.531	3.749	3.965
Average Acc. Position (Percent Depressed)	2.364	2.314	2.351	2.269
Time to 1st Acc. Let-up of 3 Percent (Secs)	3.096	3.380	3.804	3.732
Maximum Position of Acc. (Percent Depressed)	3.232	3.424	3.086	3.167
Time From Acc. Let-up to 1st Brk Prs. (Sec)	1.388	1.256	0.963	1.400
Time to 1st Br. Prs. From Start of Evt. (Sec)	0.891	1.135	0.716	1.206
Maximum Amount of Brk Prs. (Percent of Maximum)	11.913	13.657	10.105	9.506
Average Position of the Steering Wheel (Degrees)	15.859	16.301	17.553	16.266
Average Rate of Chg. of Steering Wheel (Degree/Sec)	14.854	12.041	13.707	10.935
Steer Revs. of 5 Degrees per 25 Film Frames	0.419	0.406	0.409	0.362
Steer Revs of 10 Degrees per 25 Film Frames	0.249	0.251	0.240	0.251
Steer Revs of 15 Degrees per 25 Film Frames	0.188	0.210	0.194	0.196
Len. of Pth. of Car in Event (Eq. Film Frames)	243.338	238.214	243.298	238.594
Ratio of Eq. Film Frames to Real Film Frames	0.119	0.115	0.133	0.094
Average Dif. Between Steering and Steering Comp. (Degrees)	7.971	9.000	9.036	9.579
Maximum Dif. Between Steering and Steering Comp. (Degrees)	33.067	33.315	34.285	35.931

The mean reaction times and within-subject standard deviations for the subsidiary task light presentations are shown in Tables 4 and 5. These data are for all 23 subjects. The data in both tables are presented for various response categories: (1) all responses including omissions counted as 10 second responses, (2) all responses excluding omissions, (3) only those responses which were initially correct, and (4) only those responses which were initially wrong and then corrected. There was only 1 incorrect response in the entire experiment which was not corrected, and it was dropped from the analysis. Table 3 also presents the number of omissions and wrong responses.

Table 4. Mean reaction time to the subsidiary task under marihuana (sec)

	MARIHUANA DOSE: mcg. delta-9 THC/kg. B.W.				STATISTICAL SIGNIFICANCE LEVEL
	PLACEBO 0	50	100	200	
All Responses Including Omissions	1.0601	1.1160	1.1727	1.1829	.05
All Responses Excluding Omissions	1.0460	1.1017	1.1510	1.1620	.01
Initially Correct Responses	1.0261	1.0573	1.1220	1.1194	.01
Initially Incorrect Responses	1.3354	1.5007	1.3965	1.5064	Not tested due to unequal subject numbers
Omissions	2	2	3	3	Not tested due to small number in the category
Number of Initially Incorrect Responses	73	110	90	124	.05

Table 5. Mean within subject standard deviation on the subsidiary task (sec)

	MARIHUANA DOSE: mcg. delta-9 THC/kg. B.W.				STATISTICAL SIGNIFICANCE LEVEL
	PLACEBO 0	50	100	200	
All Responses Including Omissions	.3755	.5065	.5315	.5715	Not significant
All Responses Excluding Omissions	.3238	.4387	.4649	.4767	Not significant
Initially Correct Responses	.2698	.3296	.4302	.3771	Not significant
Initially Incorrect Responses	.1976	.5380	.3214	.4135	Not significant

The data suggest a dose-related impairment of reaction times to the subsidiary task. For the category of all responses including omissions, the 3 active drug treatments produced increases of mean reaction times of 5·3, 10·6 and 11·6%. Relative changes in the category of all responses excluding omissions were 5·3, 10·0 and 11·1%. Initially correct responses had increased reaction times of 3·0, 10·3 and 9·1%. All these changes were statistically significant.

While Table 5 shows a trend toward increasing within-subject reaction time variability as a function of marihuana dose, this did not prove to be statistically significant.

DISCUSSION

The results of the present experiment are consistent with those found in 2 earlier studies of the effect of cannabis on driving simulator performance. Both reported the impairment of a perceptual or attentional task which was performed while attending to the steering or compensatory tracking requirement. None reported a decrement in the latter task. The only significant marihuana effect found by the Crancer group was an increase in speedometer errors, i.e. a decrease in the sustained attention necessary to maintain the speedometer reading within the prescribed limits. The increase in the response times to the red and green lights found by Rafaelsen, Bech and Christiansen *et al.* [1973] is quite similar to the results of the present study.

It appears highly probable that these cannabis-induced deficits are primarily related to attention or perception and not to an impairment of motor responsiveness. First, several laboratory studies have found that cannabis has little or no effect on the speed of simple motor behavior. Second, the relatively long reaction times shown in Table 6 are indicative of perceptual rather than motor impairment. Third, a recent laboratory experiment found marihuana markedly reduced the probability of perceiving briefly presented peripheral visual stimuli, but did not alter the reaction time for those stimuli which were perceived [Moskowitz, Sharma and McGlothlin, 1972].

Table 6. Distribution of reaction times to the visual subsidiary task under marihuana

MARIHUANA DOSE: mcg. delta-9 THC/Kg. B.W.	SECONDS										
	<.50	.5-1.0	1.0-1.5	1.5-2.0	2.0-2.5	2.5-3.0	3.0-3.5	3.5-4.0	4.0-4.5	4.5-5.0	>5.0
Placebo	0	646	404	64	9	1	1	1	2	0	5
50	8	608	361	80	16	11	4	4	2	1	7
100	2	587	396	82	27	11	7	4	1	1	11
200	6	559	404	86	38	14	5	6	1	0	9

The fact that the present study did not find an impairment of car handling or tracking does not, of course, eliminate this aspect as a possible driving hazard related to marihuana consumption. An earlier study of alcohol effects on this simulator also failed to show significant impairment on these measures, although it did indicate a significant increase in within-subject variability[Moskowitz, 1971]. Both the LeDain Commission [1972] and Klonoff[1974] studies of closed course driving did show an impairment in tracking. A laboratory study of pursuit meter tracking found marihuana doses as low as 12·5 mcg delta-9 THC/kg B.W. produced significant impairment[Kiplinger, Manno, Rodda *et al.*, 1971]. However, another study of compensatory tracking found only minimal decrements resulting from doses up to 88 mcg delta-9 THC/kg B.W.[Reid, Ibrahim, Miller *et al.*, 1973]. Of course, all of these tracking tasks involve perceptual as well as motor skills, so perceptual or attentional decrements may be the primary source of the observed impairment. Overall, the available evidence from the present and other relevant studies appears to indicate that any marihuana-related impairment of driving ability is more likely to be associated with perception and attention deficits than with the motor skills involved in car handling.

A confident assessment of the degree of the effects of marihuana on driving safety will require the integration of data from laboratory studies with that from epidemiological investigations of the role of the drug in traffic accidents. The latter will likely have to await the development of practical means of detecting the presence of marihuana in the body fluids.

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STATISTICAL EVALUATION OF THE EFFECTIVENESS OF "ALCOHOL SAFETY ACTION PROJECTS"

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Abstract—The U.S. Department of Transportation's "Alcohol Safety Action Programs" were evaluated by comparing motor vehicle crash fatalities in a number of communities with such programs with fatalities in similar communities without programs. No evidence of program effectiveness was found.

1. INTRODUCTION

Excessive use of alcohol is involved in more than one-half of all motor-vehicle related fatalities in the United States [Department of Transportation, 1968]. In 1969 the Department of Transportation initiated a comprehensive program intended to reduce alcohol-related highway deaths and injuries [Burkhat, Crancer and Voas, 1972]. The most ambitious component of this program consists of Alcohol Safety Action Projects, "ASAPs", conducted at the community level.

The major goals of ASAPs are threefold [Department of Transportation, 1974]:

"1. To demonstrate the feasibility and practicality of a systems approach for dealing with the drinking-driving problem, and further, *to demonstrate that this approach can save lives*" (Emphasis added).

"2. To evaluate the individual countermeasures within the limits permitted by the simultaneous application of a number of different countermeasures at the same site."

"3. To catalyze each state into action to improve its safety program in the area of alcohol safety."

On-site ASAP activity is said to be composed of four distinct types of countermeasures integrated into a whole by the local program management. These are: (1) increased law enforcement, (2) stepped-up court presentence investigation to isolate problem drinkers, (3) rehabilitation of problem drinkers and (4) public information and education (Department of Transportation, 1974).

In toto, 35 local ASAPs have been started at an average cost of more than $2 million each, a total allocation of $78 million [Department of Transportation, 1974]. These figures indicate that ASAPs are expensive countermeasures, and it is essential that they be subjected to rigorous scientific evaluation to determine the appropriateness of the resource allocations involved.

The first batch of 8 ASAPs was started at the beginning of 1971. It is these 8 and the second batch of 20 ASAPs which started in early 1972 that are being considered in this paper. The ASAP in Vermont was started in August, 1971 and another 6 were started in the middle of 1972—these ASAPs are outside the scope of this study. The Vermont ASAP was excluded because this study employs annual data, and the other 6 ASAPs were excluded because of the shortness of time they were in force. All 7 ASAPs that are not in this study were excluded also from the Department of Transportation [1974] evaluation.

As planned by the Department of Transportation (DOT) the active duration of an ASAP is 3 years. Additional half-year allowances were made for each project for both start-up preparation and post-project evaluation of results [Department of Transportation, 1974].

The purpose of this paper is to examine statistical evidence concerning the effectiveness of ASAPs in reducing overall motor vehicle fatalities in the ASAP program areas.

Year-to-year variations in fatality statistics are compared between groups of areas with ASAPs and groups of similar areas without ASAPs selected on the basis of the comparability of factors such as geographic location, population size, population density and population growth rate.

2. METHODOLOGY

A number of complex and incompletely understood factors are related to motor vehicle fatality rates. In illustration, levels of economic activity [Joksch and Wuerdemann, 1973], vehicle

weight distributions [Campbell and Reinfurt, 1973], applicability of federal motor vehicle safety standards [Joksch and Wuerdemann, 1973; Griffin, 1973], drinking-driving patterns of drivers and demographic trends all may vary from year-to-year and even locale-to-locale, and all these factors are associated with motor vehicle fatality rates. Moreover, it has been well documented that changes in motor vehicle fatality rates are sometimes mistakenly attributed to programs that in fact have no demonstrable effect [Robertson, Rich and Ross, 1973]. In addition, data collected without attention to minimal scientific requirements have also led to invalid conclusions [Williams, *et al.*, 1974; Williams and Robertson, 1974].

Because of considerations of these types, an evaluation of the results of social experimentation must satisfy certain criteria if it purports to show scientifically that those results were in fact produced by the measure in question [Campbell, 1969; Haddon, Suchman and Klein, 1964]. A scientifically valid demonstration that a change in fatality statistics was due to a certain countermeasure should include the following minimal requirements:

1. The use of comparison areas that did not have the countermeasure;
2. A demonstration that before the start of the countermeasure the comparison areas were in fact comparable to the experimental areas (i.e. areas subjected to the countermeasure) as regards year-to-year fluctuations in fatalities;
3. A demonstration that after the start of the countermeasure, a unidirectional statistically significant change took place among the experimental areas but not among the comparison areas; and
4. A demonstration that the data employed were appropriately and similarly collected in the comparison and experimental areas, and that this remained the case throughout the period of evaluation.

Design

The design employed in this study evaluates ASAP effectiveness by comparing year-to-year variations in fatality statistics between groups of areas with ASAPs and comparison groups of areas without ASAPs. ASAPs that were initiated at different times are examined separately.

ASAPs operate in whole states, rural counties, counties that include the whole or a part of a metropolitan area and in individual cities. For the purpose of this study, the 28 ASAP areas that are included in this study were examined in 13 groups—5 groups were formed from the 8 ASAPs that began in 1971 and eight groups were formed from the 20 ASAPs that began in 1972. The composition of these groups is outlined below, more detail is given in Appendix A.† Cities with ASAPs with populations over 100,000 were grouped by population size and population growth rate. Six groups consisting of cities were formed. Two midwestern counties containing large metropolitan areas were placed in the same group. Two counties covered by the ASAP in Maine were combined into one group with the statewide ASAP in New Hampshire since these counties border on New Hampshire. One group consists of a statewide ASAP, and the remaining four groups consist of 1 or more counties from the same state.

Because of the diversity of ASAP areas, no single criterion could be invoked in choosing the comparison areas, and each of the 28 ASAPs included in this study was considered individually. In the case of whole states and rural counties major consideration was given to geographic proximity and population density. For a county that is part of a metropolitan area, its relation to the metropolitan area was also considered. For instance, while Suffolk Co., N.Y. is adjacent to Nassau Co., N.Y., Suffolk is separated by Nassau from the city of New York. Therefore, Westchester Co., N.Y. was preferred to Suffolk Co. as a comparison area to Nassau Co. since Westchester Co. borders on the city of New York as does Nassau Co. For counties that include a complete metropolitan area, metropolitan counties of approximately similar sizes were chosen from the same general geographic area of the U.S. In the case of cities, population growth rates were considered a factor in establishing the comparison groups in addition to the population size at the time of the 1970 census.

Thus, the design employed in this study is briefly summarized as follows.

The 8 ASAPs that began operation in 1971 were placed in 5 groups. On average, 72% of

†Tables A3 and A6.

the 5·2 million people who live in areas in which these ASAPs operate are included in these groups.† No fatality data were readily available for areas left out of the study.

The twenty ASAPs that began operation in 1972 were placed in 8 groups. On average, 94% of 10·3 million people who live in areas in which these ASAPs operate are included in 1 of these groups.‡ No fatality data were readily available for areas left out of the study.

Individual comparison areas were chosen for each of these 13 groups, employing criteria based on 1 or more of the following factors: geographical location, population size, population density and population growth rate.

A more detailed description of the 13 groups of ASAP and comparison areas is given in Appendix A.

Statistical model

The basic statistic used in this analysis was a proportion, calculated by dividing the number of fatalities in each ASAP area in a year by the combined number of fatalities in the ASAP and comparison areas for that year. This proportion would not change systematically from year to year if changes in the ASAP and comparison area fatalities were comparable. For example, if an ASAP area such as the County of Nassau, N.Y. had a 10% decrease in fatalities from 1 year to the next and its comparison county, Westchester, N.Y. also had a 10% decrease, Nassau's proportion of the fatalities in both counties would be the same in both years. However, if Nassau's fatalities decreased while Westchester's remained the same or if Westchester's fatalities increased while Nassau's remained the same, Nassau's proportion of the fatalities in both counties would decrease.

Nonrandom (statistically significant) year-to-year variation in the ASAP proportions of the total fatalities in ASAP and comparison areas prior to the introduction of ASAPs or subsequent to the introduction of ASAPs would simply indicate that the comparison areas were not comparable with the ASAP areas. The presence of statistically significant reductions in the proportions of ASAP area fatalities coincident with the existence of the program would be strong evidence of program effectiveness. The absence of any significant change in the proportions of fatalities in ASAP areas during the time period that included several years preceding the program and 1 or more years following the introduction of the program would indicate both that the comparison areas were comparable to ASAP areas prior to the start of ASAPs and that ASAPs were apparently ineffective over a short term in reducing fatalities.

Year-to-year fluctuations in the fatality proportions about the pre- and post-ASAP averages of these proportions and the difference between the average fatality proportions during the baseline and operational periods were tested for the presence of nonrandom (statistically significant) values separately for the 5 study and comparison areas that include the 1971 ASAPs and for the eight study and comparison areas that include the 1972 ASAPs. The minimum change between the average fatality proportion during the period when the ASAPs were operational that would be detected as nonrandom 90% of the times when similar statistical tests are used was also determined.

These tests were performed using two different methods. With the first method transforms of the fatality proportions, chosen to make the data suitable for such analyses, were fitted by an additive model. The transformed fatality proportions were decomposed in this model as the sums of four terms: a grand mean, a main year effect, a main comparison group effect and an error term. In this model the main year effect for a particular year represents the excess of the average transformed fatality proportions for that year over the grand mean of these proportions. Similarly, the main comparison group effect for a particular group represents the excess of the average transformed fatality proportions for that comparison group over the grand mean of these proportions. With this method only the systematic components of the year-to-year changes in the transformed fatality proportions, that is the main year effects, were analyzed. With the second method the fatality proportions were analyzed directly and nonsystematic ASAP effects, that is, any ASAP effects dissimilar among the different study and comparison areas, could also be detected. The more complicated first method is also more powerful and is capable of detecting

†Group-by-group inclusion percentages are given in the last column of Table A1 in Appendix A.
‡Inclusion percentages group-by-group are in Table A2 of Appendix A.

smaller magnitude changes as statistically significant than the second method. The details of these methods are described in Appendix B.

3. RESULTS

Yearly variations in the proportions of ASAP area fatalities are graphed in Fig. 1 for the 5 groups including the first batch of 8 ASAP areas that became operational in 1971 and in Fig. 2 for the 8 groups including the second batch of 20 ASAP areas that became operational in 1972. Both sets of graphs include the proportions for the 3 years preceding the start of ASAP operations. The numerical values of these proportions are given in Table 1 for the 1971 ASAPs and in Table 2 for the 1972 ASAPs.

A visual inspection of the year-to-year fluctuations in the proportions of ASAP area fatalities reveals no systematic reduction in those proportions either for the first or for the second batch of ASAP areas during the years they were in effect.

The lowest proportions of ASAP area fatalities occurred in the first batch of 5 groups, that include the 1971 ASAPs, in 1971 for the first group, in 1968 for the second and third groups and in 1969 for the fourth and fifth groups. The study period includes 3 pre-ASAP years and 2 years during which ASAPs were in operation. Hence, unless ASAPs increased the numbers of fatalities, the chance, in the absence of any favorable ASAP effect, that the minimum would occur during a year in which ASAPs were in operation is 2 in 5 (0·40) for each of the 5 groups. Thus, the minimum could be expected to occur in 2 out of the 5 groups either during 1971 or during 1972, instead of the just 1 observed occurrence in 1971.

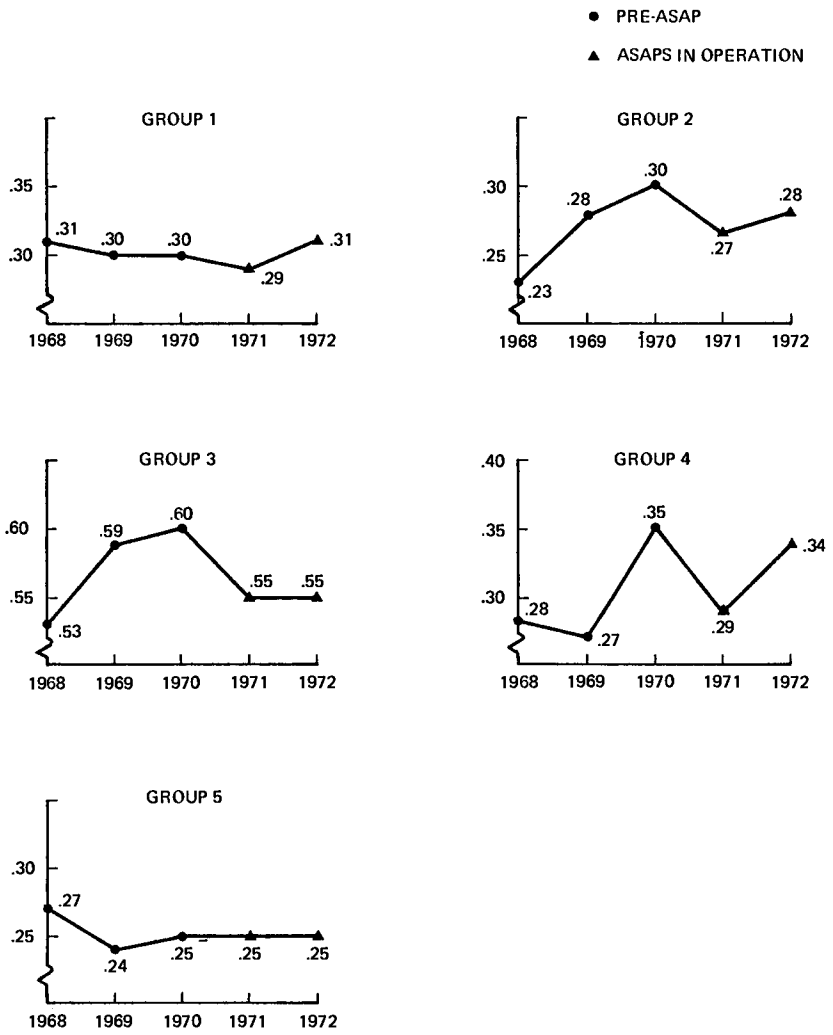

Fig. 1. Annual variations in the proportions of fatalities in ASAP areas—1971 ASAPs

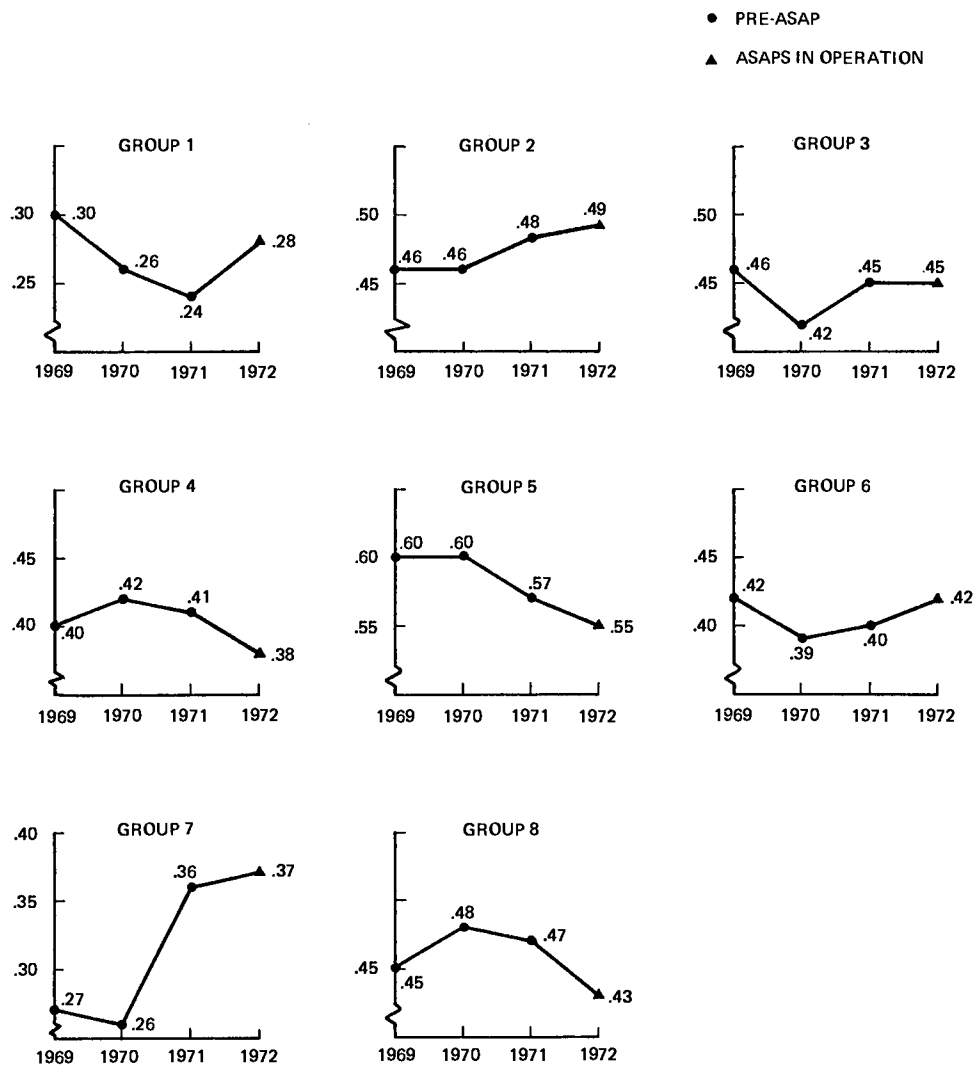

Fig. 2. Annual variations in the proportions of fatalities in ASAP areas—1972 ASAPs

Table 1. Annual proportions of fatalities in areas with ASAPs that began in 1971

	Year				
Group	1968	1969	1970	1971	1972
	Pre ASAP			During ASAP	
1	.31	.30	.30	.29	.31
2	.23	.28	.30	.27	.28
3	.53	.59	.60	.55	.55
4	.28	.27	.35	.29	.34
5	.27	.24	.25	.25	.25
All	.321	.339	.350	.329	.339

The lowest proportions of ASAP area fatalities were reached in the second batch of 8 groups, that include the 1972 ASAPs, in 1970 for the second, third, sixth and seventh groups, in 1971 for the first group and in 1972 for the fourth, fifth and eighth groups. The study period includes three pre-ASAP years and 1 year during which the ASAPs operated. Reasoning as above, the chance that the lowest proportion would occur in 1972, the year during which ASAPs were functioning, is 1 in 4 for any of the groups, and so the expected number of minima reached during 1972 in this batch of 8 groups would be 2. In fact, the minimum was reached in 1972 for 3

Table 2. Annual proportions of fatalities areas with ASAPs that began in 1972

Group	Year			
	1969	1970	1971	1972
	Pre ASAP			During ASAP
1	.30	.26	.24	.28
2	.46	.46	.48	.49
3	.46	.42	.45	.45
4	.40	.42	.41	.38
5	.60	.60	.57	.55
6	.42	.39	.40	.42
7	.27	.26	.36	.37
8	.45	.48	.47	.43
All	.440	.429	.438	.437

among the 8 groups. It will be shown below that both departures from the expected values are results that could have commonly occurred due to chance fluctuations in these proportions.

Two different groups of statistical tests were carried out on each of the 2 groups of ASAP and comparison area fatality figures. The first group of tests to examine the comparability of ASAP and comparison areas in regard to the year-to-year fluctuations in the number of ASAP and comparison area fatalities, and the second group of tests to decide whether the changes in the proportions of ASAP area fatalities between the pre-ASAP baseline period and the periods during which ASAPs operated were in excess of what could have been observed due to chance. The changes in the proportions of ASAP area fatalities following the start of ASAPs that would result in a 90% probability of detection with the use of the statistical tests employed were also calculated. The details of the methods used are given in Appendix B.

These tests showed that the additive components of the year-to-year fluctuations in the fatality proportions about their pre-ASAP and post-ASAP averages in each of the groups were no larger than would be expected on the basis of the assumption of random fluctuations in these proportions. As shown in Table 3 (tests T1 and T2), deviations from the pre-ASAP and post-ASAP averages equal to or larger than those observed for the comparison groups that include the 1971 or 1972 ASAPs could be present due to chance alone in more than 10% or more than 50% of the instances, respectively, when similar analyses are performed.

Table 3. Summary of the results of statistical tests for the validation of the comparison group design and the detection of changes in the proportions of ASAP area fatalities between the baseline and the operational periods for the comparison groups that include the 1971 and 1972 ASAPs

Hypothesis	Test	First Year of ASAP Operation	Distribution of Test Statistics	Value of Test Statistics	Significance	Result
Constant year effect in transformed fatality proportions during baseline and operational periods	T1	1971	$F_{3,16}$	2.40	≥ 0.10	Accepted
	T_2	1972	$F_{2,21}$	0.45	≥ 0.50	Accepted
No change in transformed fatality proportions between baseline and operational periods	T_3	1971	$F_{1,19}$	0.13	≥ 0.50	Accepted
	T_4	1972	$F_{1,23}$	0.24	≥ 0.50	Accepted
No comparison group x year interaction in transformed fatality proportions during study period	T_5	1971	χ^2_{16}	11.18	≥ 0.80	Accepted
	T_6	1972	χ^2_{21}	27.79	≥ 0.10	Accepted
Fatality proportions within comparison groups are constant during baseline and operational periods	T_7	1971	χ^2_{15}	14.54	≥ 0.40	Accepted
	T_8	1972	χ^2_{16}	22.26	≥ 0.10	Accepted
No change in fatality proportions within comparison groups between baseline and operational periods	T9	1971	χ^2_5	1.19	≥ 0.90	Accepted
	T10	1972	χ^2_8	10.12	≥ 0.20	Accepted

These tests also showed that the additive components of the changes from the baseline period to the operational period in the fatality proportions were no larger than would be expected on the basis of the assumption of random fluctuations in these proportions. In fact, baseline to operational period changes equal to or larger than those observed for either the comparison groups that include the 1971 ASAPs or for those that include the 1972 ASAPs could be present due to chance alone is more than half the instances when similar analyses are performed (Tests T3 and T4 in Table 3).

No violations of the year-to-year proportionality of the numbers of fatalities that occurred during the whole study period in the matched pairs of ASAP and comparison areas could be detected by statistical tests either when these pairs were examined individually without the use of the additive model. In fact, as shown in Table 3 (Tests T5 and T6), deviations from the average fatality proportions for these pairs matching or exceeding those observed for the 2 sets of comparison groups could be present due to chance alone in more than 80% or more than 10% of the instances, respectively, when similar analyses are performed.

Deviations in the fatality proportions from their average values for the baseline and for the operational years within each comparison group were found to be no larger than would be expected under the assumption that they are random fluctuations. In fact, as shown in Table 3 (Tests T7 and T8), deviations comparable to those observed could be present due to chance alone in more than 40 or more than 10% of the instances, respectively, when similar analyses are performed.

The changes in the proportions of fatalities in ASAP areas within each comparison group from the baseline average to the average during the operational period were again no larger than could be expected due to chance fluctuations under the assumption of randomness. As shown in Table 4 (Tests T9 and T10) the chances in similar analyses of deviations having magnitudes comparable to those observed exceed 90 or 20% respectively.

The 2 sets of statistical tests (T3, T4 and T9, T10 for the comparison groups that include the 1971 and the 1972 ASAPs, respectively) revealed no significant changes in the proportions of ASAP area fatalities between the baseline and the operational periods. Following the procedure described in Appendix B, it was possible to estimate the magnitudes of reductions that these tests would have detected as significant 90% of the times when similar analyses are performed. With the first method such a minimal detectable difference is a reduction of about 10% for the comparison groups that include the 1971 ASAPs. With the second method the minimal detectable difference is about 12%. For the comparison groups that include the 1971 ASAPs the minimal reduction in fatality proportions that would be detected 9 out of 10 times when similar tests are performed is about 5% with the first method and about 9% with the second method. Real changes smaller than those specified could not be detected as statistically significant by either of these methods.

Thus, there is no evidence of a decline in the total number of fatalities in any of the communities examined that could be attributed to ASAP programs.

4. DISCUSSION

The National Highway Traffic Safety Administration (NHTSA) claimed, "... that a small but significant reduction in fatalities has occurred which can be attributed to ASAPs at those 8 projects with 2 full years of operations..." [Department of Transportation, 1974]. The present study of the overall impact of ASAPs finds no support for these claimed reductions of fatalities in ASAP areas. Furthermore, examinaion of NHTSA'S alleged demonstration of a reduction in fatalities attributable to the ASAPs shows that it is fallacious.

NHTSA analyzed the fatal crash data for the areas where the 8 1971 ASAPs operated using a "time series analysis of covariance" (see Appendix C for details). No significant reduction in the total number of fatal crashes from the baseline to operational period was found by NHTSA in this analysis (Department of Transportation, 1974). On the other hand, the same analysis revealed the presence of a significant "... interaction between the change from baseline to operational period and the number of nighttime as compared to daytime crashes...." In other words, there was a significant change as measured by NHTSA's model in the relation between daytime and nighttime fatal crashes that followed the introduction of ASAPs. Since the probability that alcohol is involved in a fatal crash is much higher during nighttime than during

daytime [Department of Transportation, 1968], and since the observed interaction corresponds to a decrease in the number of nighttime fatal crashes in comparison with daytime fatal crashes, NHTSA incorrectly concluded that the ASAPs were in fact responsible for the observed interaction and, therefore, that ASAPs reduced the number of nighttime fatal crashes.

The NHTSA demonstration that ASAPs reduced the number of nighttime fatal crashes in comparison with the number of daytime fatal crashes fails to satisfy one of the minimal requirements for a scientifically valid demonstration that an observed change is in fact due to a countermeasure under evaluation. Namely, no convincing evidence was induced to rule out the possibility of alternative explanation. NHTSA failed to show the absence of a similar interaction for areas that did not have ASAPs, and therefore, the decrease in the number of fatal nighttime crashes in comparison to the number of fatal daytime crashes could have been part of some regional or national trend not related to ASAPs.

The hypothesis that there are no year-to-year changes in the proportions of nighttime fatal crashes either in areas with 1971 ASAPs or in comparison areas comprising 20 states, for which data were given in the cited NHTSA study, was tested by means of the procedure described in Appendix B. For the period 1968–1971—this period includes 3 baseline years and the first of the 2 years of the ASAP operation—the hypothesis that nighttime fatal crashes represent a fixed proportion of all fatal crashes could not be rejected. However, after 1972 was included in the analysis, the hypothesis that the proportion of nighttime fatalities is constant was rejected. Further analysis of the data revealed that this decrease in the proportion of nighttime fatal crashes was equally present *both* in the ASAP and in the comparison areas (see Appendix C for details). It is therefore not justified to conclude that ASAPs were responsible for the changed relationship between nighttime and daytime fatal crashes; and, in fact, the fact that they occurred in the areas without ASAPs forces the opposite conclusion, namely, that the ASAPs can *not* have been responsible. Although no definitive explanation can be offered for the nationally observed shifts in the proportion of nighttime fatal crashes, it is interesting that these shifts appear to parallel changes in the industrial production index published by the Department of Commerce. Parallelism between overall fatality rates and the production index have been known (Joksch and Wuerdemann, 1973), but the author of this paper is unaware of any study on the parallelism between the proportion of nighttime fatal crashes and the production index.

Having erroneously concluded that the 1971 ASAPs were effective, NHTSA proceeded to estimate the number of all fatalities eliminated by the first batch of 8 ASAPs as follows: the number of deaths in ASAP areas was extrapolated from the baseline period to the operational period of the basis of various trends. An upper bound for these claimed savings (179 fatalities during 1971 and 1972 combined) was obtained by transferring the trend from non-ASAP areas within the same state to ASAP areas and comparing the result with the number of fatalities actually found in the ASAP areas. A lower bound for these claimed savings (64 fatalities during 1971 and 1972 combined) was obtained when the actual number of ASAP area fatalities was compared to a projection based on the trend in fatalities in urban areas whose population exceeds 250,000.

In the absence of a valid scientific demonstration that shows that ASAPs were in fact responsible for decreases in the number of fatal crashes, it is pointless to estimate the magnitudes of these claimed savings. It may be worth noting that no significant change in either the frequency of all fatal crashes or in the frequency of nighttime fatal crashes in ASAP areas could be found relative to these crash frequencies in the 20 state comparison areas by means of the commonly used χ^2 test (for details, see Appendix C).

The present study is based on the analysis of all motor vehicle fatalities in ASAP and comparison areas, both before and during such programs. It is known that single vehicle crashes involve the use of alcohol more often than multiple vehicle crashes and, similarly, that crashes occurring at night involve the use of alcohol more often than crashes occurring at daytime [Haddon and Bradess, 1959; McCarroll and Haddon, 1962; Department of Transportation, 1968]. It is thus conceivable that comparison of some ASAP areas and properly chosen comparison areas using 1 or more of these more sensitive indicators of alcohol-involvement in motor vehicle fatalities would show that some ASAPs had some small effect in reducing some types of motor vehicle fatalities, but no evidence of reductions in overall fatalities or in nighttime fatalities as the result of the Alcohol Safety Action Program has been found in the research reported here.

In view of the results of the several detailed analyses presented in this paper, and in the absence of any evidence of an overall reduction in fatalities, it is only possible to conclude scientifically that ASAPs, as large scale social programs, have been ineffective.

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APPENDIX A

ASAP operations began in 8 states in January, 1971 over areas whose combined total population is about 5·2 million [Webster's New Geographical Dictionary, 1972]. Three among these 8 ASAPs are included in this study in their entirety. Portions of the areas in which the remaining 5 ASAPs are operational were excluded from this study because of lack of readily available fatality data either for those areas or for suitable comparison areas. For instance, the Denver ASAP includes 3 counties (Adams, Arapahoe and Jefferson) that are adjacent to Denver County, but only Denver County was included in this study. The combined population of areas with ASAPs that are included in this study amount to 72% of the combined population covered by the 8 ASAPs that began in 1971. Table A1 presents the list of areas included and excluded from the study.

ASAP operations began in 20 states in January, 1972 over areas whose combined total population is about 10·3 million [Webster's New Geographical Dictionary, 1972]. Thirteen among these 20 ASAPs are entirely included in this study. Portions of the areas over which the remaining 7 ASAPs are operational were excluded from the study for reasons similar to those stated above. Only 6% of the combined population of areas with ASAPs that began in 1972 were excluded from this study. Table A2 presents the list of areas included and excluded from the study.

The 8 1971 ASAPs were consolidated into 5 groups. For instance, the first group contains the cities of Denver, Colo., Seattle, Wash., and Portland, Ore. The average population size of the 3 cities is 477,000. The smallest among them, Portland, has a population of 384,000, while the largest, Seattle, Wash., has a population of 531,000. On the average, the population of these cities remained unchanged during 1960–1970. Specifically, the population of Seattle declined 5% and the population of Denver increased 4%. Eight cities were chosen as comparison cities for this group. They are: Akron, Oh.; Atlanta, Ga.; Long Beach, Calif.; Milwaukee, Wisc.; Newark, N.J.; Norfolk, Va.; Oakland, Calif.; and San Francisco, Calif. The average population size and decline rate over the same period for the comparison group are 474,000 and 2% respectively. The population sizes for cities in the comparison group contain all cities for which data were available and which had comparable population sizes and population growth rates. Group 2 consists of two cities while the remaining three groups consist of one or more counties from the same site. The definitions of the 5 groups of matched ASAP and comparison areas are given in Table A3. The entry under comments in Table A3 indicates the matching criteria employed. Table A4 contains the fatality data and the population data for the 5 ASAP and matched comparison areas. The numbers of fatalities are given for years 1968–1972. All city fatality data were taken from published sources [Accident Facts, 1969, 1971, 1973].

Table A1. Areas included in study with 1971 ASAPs[1]

State	Name of ASAP	ASAP Areas[3] included in study	ASAP Areas[3] excluded from study	population size[2] (in thousands, 1970 census)	% of population included in study[1]
Colorado	Denver	Denver	Counties of Adams, Arapaho and Jefferson	1,096	47
Michigan	Washtenaw Co.	Washtenaw Co.	-	234	100
New Mexico	Albuquerque	Albuquerque	Bernalillo Co. outside Albuquerque	316	77
New York	Nassau Co.	Nassau Co.	-	1,429	100
North Carolina	Mecklenburg Co.	Charlotte	Mecklenburg Co. outside Charlotte	355	68
Oregon	Portland & Eugene	Portland	Eugene	460	83
Washington	Seattle	Seattle	King Co. outside Seattle	1,157	46
Wisconsin	Marathon & Sheboygan Counties	Marathon & Sheboygan Counties	-	194	100
Combined	N.A.	N.A.	N.A.	5,241	72

(1) The Vermont ASAP began operations in August 1971 and was therefore excluded from this analysis.

(2) Webster's New Geographical Dictionary (1969, 1972).

(3) Department of Transportation (1974).

The sources of county fatality data are given in Table A5. In addition to the total population sizes the average population growth rates, Table A4 also contains the range of population sizes and growth rates, wherever applicable, for the 5 groups of ASAP and comparison areas [Webster's New Geographical Dictionary, 1969, 1972].

The 20 1972 ASAPs were consolidated into 8 groups. The first 4 among these 8 groups consist of cities with populations in excess of 100,000. Group 5 contains 2 large midwestern metropolitan areas. Groups 6 and 8 consist of a separate state each except that 2 counties with ASAPs in Maine that are adjacent to the state of New Hampsire were merged with New Hampshire into 1 group. Group 7 is a county located in a metropolitan area. Comparison areas were chosen for these groups on the basis described above. The definitions of the 8 ASAP and matched comparison groups are given in Table A6. The specific criteria employed in selecting the comparison areas are given under the heading "Comments" in Table A6. Since the state of New Hampshire borders on states that either have ASAPs themselves or are geographically different from New Hampshire, it was decided to match individually each county from New Hampshire with a county from the states of New York or Vermont on the basis of population density and growth rate. The other 2 counties from Maine

Table A2. Areas included in study with 1972 ASAPs

State	Name of ASAP	ASAP areas[2] included in study	ASAP areas[2] excluded from study	population size[1] (in thousands, 1970 census)	% of population included in study[1]
Arizona	Phoenix	Phoenix	-	582	100
Arkansas	Pulaski County	Little Rock	Pulaski County outside of Little Rock	287	46
Florida	Hillsborough County	Tampa	Hillsborough County outside of Tampa	490	57
Georgia	Columbus	Columbus	Muscogee County outside of Columbus	167	93
Indiana	Indianapolis	Marion County	-	794	100
Kansas	Wichita	Wichita	Sedgwick County outside of Wichita	351	79
Louisiana	New Orleans	New Orleans	-	593	100
Maine	Cumberland and York Counties	Cumberland and York Counties	-	305	100
Maryland	Baltimore	Baltimore, City of	Areas of Baltimore and Anne Arundel Counties inside the Beltway outside of Baltimore City	582[3]	100

Table A2. (Continued)

State	Name of ASAP	ASAP areas[2] included in study	ASAP areas[2] excluded from study	population size[1] (in thousands, 1970 census)	% of population included in study[1]
Massachusetts	Boston	Boston	-	641	100
Minnesota	Hennepin County	Hennepin County	-	960	100
Missouri	Kansas City	Kansas City	-	507	100
Nebraska	Lincoln	Lincoln	-	150	100
New Hampshire	New Hampshire	New Hampshire	-	738	100
Ohio	Cincinnati	Cincinnati	-	452	100
Oklahoma	Oklahoma City	Oklahoma City	-	369	100
South Carolina	Richland County	Columbia	Richland County outside of Columbia	234	49
South Dakota	South Dakota	South Dakota	-	666	100
Texas	San Antonio	San Antonio	-	654	100
Virginia	Fairfax County	Fairfax County	-	455	100

AREAS INCLUDED IN STUDY WITH 1972 ASAPS

State	Name of ASAP	ASAP areas[2] included in study	ASAP areas[2] excluded from study	population size[1] (in thousands, 1970 census)	% of population included in study[1]
Combined	N.A.	N.A.	N.A.	10,301	94

(1) Webster's New Geographical Dictionary, (1969, 1972)

(2) Department of Transportation (1974)

(3) Baltimore City data used

Table A3. Groups of areas with 1971 ASAPs in study and matched groups of comparison areas

Group	Areas with ASAPs in Study	Comparison Areas	Comments
1	Denver, Colorado; Portland, Oregon Seattle, Washington	Akron, Ohio; Atlanta, Georgia; Long Beach, California; Milwaukee, Wisconsin; Newark, New Jersey; Norfolk, Virginia; Oakland, California; San Francisco, California.	Matched on basis of population size and growth rate of population from 1960 to 1970.
2	Albuquerque, New Mexico Charlotte, North Carolina	Tucson, Arizona; Corpus Christi, Texas; El Paso, Texas; St. Petersburg, Florida; Toledo, Ohio.	Same as for Group 1
3	Nassau County, New York	Westchester County, New York	Westchester adjacent to Nassau, both adjacent to New York City and both have had similar population growth rates from 1960 to 1970
4	Washtenaw County	Ingham, Kalamazoo, Saginaw Counties, Michigan	Matched on the basis of population density
5	Marathon and Sheboygan Counties, Wisconsin	Adams, Clark, Lincoln, Portage, Price, Shawano, Taylor, Waupaco, Wood Counties, Wisconsin for Marathon and Manitowac and Racine Counties, Wisconsin for Sheboygan	Marathon and its nine comparison counties are located in central Wisconsin; Racine, Manitowac and Sheboygan counties border Lake Michigan

that together with New Hampshire form Group 8 were treated similarly. The resultant population densities for ASAP and comparison areas in Group 8 are about 94 and 104 per square mile respectively.

Fatality data for the years 1969–1972, population sizes and population growth rates for 1972 ASAP and comparison groups are given in Table A7. Except for Boston, all city and state fatality data were taken from published sources [Accident Facts, 1971, 1972]. The sources for county fatality data and for fatality data for Boston are given in Table A5. Population data were taken from Webster's New Geographical Dictionary [1969, 1972].

APPENDIX B

The number of fatalities occurring during a year in any geographic area was considered a sample value from a Poisson population.

Table A4. Population and fatality data for groups of areas with 1971 ASAPs and matched comparison groups

Group		Number of Fatalities (2)					Population size (1) (in 000's)				Ratio of 1970 to 1960 (1) population size		
		1968	1969	1970	1971	1972	Total	Min.	Avg.	Max.	Min.	Avg.	Max.
1	ASAP	261	226	210	187	205	1,427	381	476	531	.95	1.00	1.04
	Comparison	577	535	495	455	457	616	275	452	717	.94	.98	1.04
2	ASAP	71	88	103	94	96	485	241	242	245	1.19	1.20	1.21
	Comparison	234	222	241	253	245	1,390	205	278	384	1.16	1.20	1.23
3	ASAP	145	196	173	174	161	1,429	-(3)	-	-	-	1.10	-
	Comparison	131	135	114	143	133	894	-	-	-	-	1.10	-
4	ASAP	64	57	78	67	69	234	-	-	-	-	1.36	-
	Comparison	165	153	143	167	137	683	-	-	-	1.15	1.19	1.24
5	ASAP	57	53	47	46	57	194	-	-	-	1.09	1.11	1.13
	Comparison	157	164	143	141	173	531	-	-	-	.96	1.11	1.23
Total	ASAP	598	620	611	568	588	3,769	-	-	-	-	1.08	-
	Comparison	1,264	1,209	1,136	1,159	1,145	7,114	-	-	-	-	1.08	-

(1) Webster's New Geographical Dictionary (1969, 1972)

(2) Fatality data sources are given in Table A5

(3) Not applicable

Table A5. Sources of fatality data for counties in study and for the city of Boston

Indiana	Computer Tape of Fatal Crashes. Division of Motor Vehicles, Department of Traffic, State of Indiana.
Maine	Highway Fatal Accident Fatalities By County, 1967-1973, Maine State Police Planning and Research.
Maryland	Private Communication from Sgt. Harvey, Maryland State Police.
Massachusetts	Motor Vehicle Traffic Accidents, Injuries, Deaths By Counties, years 1968-1972, Commonwealth of Massachusetts Registry of Motor Vehicles Statisticians Office, Boston, Massachusetts.
Minnesota	Cumulative Fatal Accidents and Deaths By Counties, 1968-1972, Minnesota Department of Public Safety.
New York	New York State Accident Facts. 1968-1972, New York State Department of Motor Vehicles. Albany, New York.
Virginia	Virginia Traffic Crash Facts, 1968-1972 Department of State Police, Richmond, Virginia.
Wisconsin	Computer Tape of Fatal Crashes. Division of Motor Vehicles, Department of Traffic, State of Wisconsin.

Let μ_{ijk} and ν_{ijk} denote the unknown mean values of the Poisson distributions associated with ASAP and comparison area fatalities in the j-th year of the k-th period for the i-th group, with $k = 1$ indicating the baseline and $k = 2$ the study periods ($i = 1, \ldots, I, j = 1, \ldots, J(k)$ where $I = 5$ for the first batch and $I = 8$ for the second batch of ASAPs, $J(1) = 3$ for both batches, $J(2) = 2$ for the first and $J(2) = 1$ for the second batch of ASAPs). The actual numbers of fatalities observed during the j-th year of the k-th period in the i-th group in each area will be written as X_{ijk} and Y_{ijk}. The conditional distribution of the proportion of fatalities occurring in ASAP areas

$$f_{ijk} = \frac{X_{ijk}}{X_{ijk} + Y_{ijk}} \tag{B1}$$

given the total number of fatalities in ASAP and comparison areas

$$N_{ijk} = X_{ijk} + Y_{ijk} \tag{B2}$$

is the same as the distribution of occurrences among N_{ijk} independent trials if in each of these

$$p_{ijk} = \frac{\mu_{ijk}}{\mu_{ijk} + \nu_{ijk}} \tag{B3}$$

is the probability of occurrence [Lehman and Scheffé, 1955].

Table A6. Groups of areas with 1972 ASAPs and matched groups of comparison areas

Group	Areas with ASAPS in study	Comparison Areas	Comments
1	Phoenix, Ariz.	Memphis, Tenn.; San Diego, Calif.; San Jose, Calif.	Matched on the basis of population size and growth rate of population from 1960 to 1970.
2	Baltimore, Md.; Boston, Mass.; Cincinnati, Oh.; New Orleans, La.	Cleveland, Oh.; Pittsburgh, Pa.; San Francisco, Calif.; Washington, D.C.; Oakland, Calif.	Same as for Group 1
3	Kansas City, Mo.; Oklahoma City, Okla. San Antonio, Tex.; Tampa, Fla.; Wichita Kansas	Atlanta, Ga.; Columbus, Oh.; Dallas, Tex.; Fort Worth, Tex.; Long Beach, Calif.	Same as for Group 1.
4	Little Rock, Ark.; Columbia, S.C.; Columbus, Ga.; Lincoln, Neb.	Jackson, Miss.; Knoxville, Tenn.; Topeka, Kans.; Winston-Salem, N.C.	Same as for Group 1 and considerations of geographic proximity.
5	Hennepin Co., Minn.; Marion Co., Ind.	Ramsey Co., Minn; Milwaukee Co., Wisc.	Matched on basis of geographic proximity and population size.
6	South Dakota	North Dakota; Wyoming	Matched on basis of geographic proximity
7	Fairfax Co., Va.	Montgomery Co., Md; Prince Georges Co., Md.	Fairfax, Montgomery, and Prince Georges are counties bordering on Washington, D.C., from the South, North West and North East, respectively.
8	New Hampshire, Cumberland and York Counties, Me.	Seven counties from New York state and two counties from Vermont for New Hampshire: Broome, Dutchess, Essex, Fulton, Herkimer, Montgomery, Oswego, N.Y. Addison, Bennington, Rutland, Vt.; two counties from New York state for Maine: Saratoga, Ulster.	Matched on basis of geographic proximity, population size, population density and population growth rate from 1960 to 1970

Table A7. Population and fatality data for groups of areas with 1972 ASAPs and matched comparison groups

Group		Number of fatalities(2)				Population size(1) (in 000's)				Ratio of 1970 to 1960 population size(1)		
		1969	1970	1971	1972	Total	Min.	Avg.	Max.	Min.	Avg.	Max.
1	ASAP	103	90	75	98	582	-(3)	582	-	-	1.33	-
	Comparison	244	261	244	257	1,767	446	589	697	1.22	1.38	2.19
2	ASAP	422	382	352	359	2,592	452	648	906	.90	.94	.96
	Comparison	502	446	376	377	3,106	362	621	757	.86	.93	.98
3	ASAP	398	341	351	372	2,085	277	417	654	1.09	1.09	1.14
	Comparison	465	469	432	446	2,633	359	526	844	1.02	1.13	1.15
4	ASAP	54	65	57	60	551	114	138	150	1.17	1.22	1.32
	Comparison	92	90	83	96	587	125	147	175	1.05	1.21	1.56
5	ASAP	259	260	225	226	1,754	794	877	960	1.14	1.14	1.14
	Comparison	175	175	167	184	1,530	476	765	1,054	1.02	1.05	1.13
6	ASAP	296	237	262	294	666	-	-	-	-	.98	-
	Comparison	403	371	393	405	950	-	-	-	.98	.99	1.01
7	ASAP	58	61	98	84	455	-	-	-	-	1.65	-
	Comparison	153	175	174	144	1,184	-	-	-	1.53	1.70	1.85
8	ASAP	264	296	285	252	1,043	-	-	-	-	1.17	-
	Comparison	327	321	318	335	1,125	-	-	-	-	1.14	-
Total	ASAP	1,854	1,732	1,705	1,745	9,728	-	-	-	-	1.11	-
	Comparison	2,361	2,308	2,187	2,244	12,882	-	-	-	-	1.11	-

(1) Webster's New Geographical Dictionary (1969, 1972)

(2) Fatality data sources are given in Table A5

(3) Not applicable

Another set of statistics with approximately unit normal distributions can be calculated from the observed numbers of ASAP and comparison area fatalities as follows.

The transformation

$$V^*_{ijk} = \arcsin\left[\sqrt{\left(\left(\frac{X_{ijk}}{N_{ijk}+1}\right)\right)}\right] + \arcsin\left[\sqrt{\left(\left(\frac{X_{ijk}+1}{N_{ijk}+1}\right)\right)}\right] \tag{B4}$$

suggested by Freeman and Tukey (1950) is employed first. The variances of these transformed variables are largely independent of variations in the magnitude of the proportions.

However, the variance of the variables given by the transformation in eqn (B4) is still inversely proportional to the sample size. This dependency is eliminated by multiplying the proportions in each group by suitably chosen weights. Let w_i

denote the weight for the i-th group. The i-th weight is defined as the square root of the average sample size in the i-th group:

$$w_i = \sqrt{\bar{N}_{i\cdot\cdot}}, \tag{B5}$$

where the customary "DOT" convention is employed to denote an average [Scheffé, 1959]. Then the second transformation

$$V_{ijk} = w_i V^*_{ijk} \tag{B6}$$

eliminates the dependence of the variance on the sample size. The mean of V_{ijk} is approximately equal to $2w_i$ arc sin (p_{ijk}) while their variances are approximately equal to unity.

In view of these observations, hypotheses of interest about the p's can be tested either by analyzing the variance of the V's or by analyzing the f's, the observed fatality proportions, directly.

With the first method the total sum of squares (SS), $SS_{\text{"tot"}}$ of the V's is analyzed into the sum of 4 components [Scheffé, 1959].

$$SS_{\text{"tot"}} = SS_T + SS_C + SS_G + SS_e. \tag{B7}$$

where SS_T, SS_c, SS_G and SS_e denote the SS's due to ASAP effectiveness, deviations from the hypothesis that the fatality proportions are constant within each comparison group both before and after the start of ASAPs, between-group differences and the error term. Expressions for these components in terms of the V's are given in Table B1.

The hypothesis that fluctuations in the numbers of fatalities in the study and in the corresponding comparison areas were comparable both before and after the start of the ASAPs can be stated in terms of the p's as the hypothesis that the p's remained constant within each comparison group both before and after the start of the ASAPs. This hypothesis can be tested by means of the F-ratio.

$$F_{\nu_1\nu_2} = \frac{SS_C/\nu_1}{SS_e/\nu_2} \tag{B8}$$

where $\nu_1 = 3$, $\nu_2 = 16$ for the groups that include the 2-year ASAPs and $\nu_1 = 2$, $\nu_2 = 21$ for those that include the 1-year ASAPs (tests T1 and T2 in the Results section).

The hypothesis that the fatality proportions were not systematically changed after the start of the ASAPs in comparison with their baseline levels corresponds to the hypothesis that there was no overall change in the p's after the start of ASAPs in comparison with their baseline levels. This hypothesis can be tested by means of the F-ratio:

$$F_{\nu_1\nu_2} = \frac{SS_T/\nu_1}{(SS_C + SS_e)/\nu_2} \tag{B9}$$

where $\nu_1 = 1$, $\nu_2 = 19$ for the groups that include the 2-year ASAPs and $\nu = 1$, $\nu = 23$ for those that include the 1-year ASAPs (tests T3 and T4 in the Results section).

It is also possible to test the hypothesis that there is no comparison group x year interaction among the V's. Such interaction would be indicative of unsystematic changes in the fatality proportions, that is, changes occuring during certain years only in some of the comparison groups. Since the distribution of SS_e is approximately χ^2 with ν degrees of freedom ($\nu = 16, 21$, respectively, for the 2 batches of 1-year and 2-year ASAPs), the customary χ^2 test is applicable (tests T5 and T6 in the Results section).

Finally, the power of the test for the hypothesis that no systematic reduction in the fatality proportions followed the start of ASAPs against the alternative that such a systematic change in fact did take place can be calculated as follows.

The absence or presence of a systematic change can be written in terms of the p's as:

$$\text{Absence: } p_{ijk} = \bar{p}_{i\cdot\cdot}, i = 1,\ldots I, \quad j = 1,\ldots, J(k) \quad k = 1,2 \tag{B10}$$

$$\text{Presence: } \bar{p}_{i\cdot 2} = (1-\Delta)\,\bar{p}_{i\cdot 1}, i = 1,\ldots, I \tag{B11}$$

Table B1. Analysis of variance for the transformed fatality proportions of the 1971 and 1972 comparison groups

Source	SS	D.F. 1971	D.F. 1972
Pre-to post-ASAP change in fatality proportions	$SS_T = I\sum_{k=1}^{2} J(k)\,(\bar{V}_{\cdot\cdot k} - \bar{V}_{\cdot\cdot\cdot})^2$	1	1
Constancy of fatality proportions	$SS_C = I\sum_{k=1}^{2}\sum_{j=1}^{J(k)} (\bar{V}_{i\cdot\cdot} - \bar{V}_{\cdot\cdot k})^2$	3	2
Between group variation in fatality proportions	$SS_G = (J(1)+J(2))\sum_{i=1}^{I} (\bar{V}_{i\cdot\cdot} - \bar{V}_{\cdot\cdot\cdot})^2$	4	7
Error	$SS_e = \sum_{i=1}^{I}\sum_{k=1}^{2}\sum_{j=1}^{J(k)} (V_{ijk}+\bar{V}_{\cdot\cdot\cdot} - \bar{V}_{i\cdot\cdot} - \bar{V}_{ijk})^2$	16	21
"Total"	$SS_{\text{"tot"}} = \sum_{i=1}^{I}\sum_{k=1}^{2}\sum_{j=1}^{J(k)} (\bar{V}_{ijk} - \bar{V}_{\cdot\cdot\cdot})^2$	24	31

where 100Δ measures the percentage change from the baseline level following the introduction of ASAPs. After some simple algebra (B11) can be rewritten as

$$\bar{p}_{i.1} = \bar{p}_{i..} + r\Delta\bar{p}_{i.1},$$
$$\bar{p}_{i.2} = \bar{p}_{i.} - (1-r)\Delta\bar{p}_{i.1}) \tag{B12}$$

where $\bar{p}_{i..}$ is the fatality proportion in the i-th comparison group for the whole period:

$$\bar{p}_{i..} = \bar{p}_{i.1}(1 - r\Delta) \tag{B13}$$

and $r = J(1)/(J(1)+J(2))$. Retaining the terms involving the first power of Δ only in the power series expansion of $2\sqrt{(\bar{N}_{i..})}$ arc sin $\sqrt{(p_{ijk})}$ about $\bar{p}_{i..}$ results in:

$$2\sqrt{(\bar{N}_{i..})}\,\text{arc sin}\sqrt{p_{ijk}} = 2\sqrt{(\bar{N}_{i..})}\left(\text{arc sin}\sqrt{(\bar{p}_{i..})} + \tfrac{1}{2}\frac{\Delta\bar{p}_{i..1}u}{\sqrt{(\bar{p}_{i..1}(1-p_{i..1}))}}\right) \tag{B14}$$

where $u = r$ or $-(1-r)$ according as $k = 1$ or 2. The approximate increases ($A(\Delta)$ and $B(\Delta)$) in the expected values of SS_T and SS_e, the sums of squares that measure the systematic differences between the pre- and post-ASAP fatality proportions and the error component respectively, that are caused by a $100\Delta\%$ change in the fatality proportions from their baseline levels are calculated, using the approximation formula (B 14), as follows:

$$A(\Delta) = \Delta^2 I(\bar{H}.)^2(J(1)r^2 + J(2)(1-r)^2) \tag{B 15}$$

$$B(\Delta) = \Delta^2\sum_{i=1}^{I}(H_i - \bar{H}.)^2(J(1)r^2 + J(2)(1-r)^2) \tag{B 16}$$

where $H_i = (\bar{N}_{i.}\,\bar{p}_{i.}/(1-\bar{p}_{i.1}))^{1/2}$. It follows therefore, that under the alternative hypothesis that $100\Delta\%$ change in the fatality proportions followed the introduction of ASAPs the distribution of the F-ratio given in (B9) for testing the hypothesis of no change has a noncentral F distribution with the same degrees of freedom and the noncentrality parameter $\delta = (A(\Delta))^{1/2}$ provided that $B(\Delta)/\nu^2$ is sufficiently small. This observation allows to compute the approximate power of the test of the hypothesis of no change ($\Delta = 0$) in the fatality proportions following the introduction of ASAPs against the alternative of $100\Delta\%$ reduction for various values of Δ in accordance with the procedure described in Scheffé [1959]. It was thus possible to estimate the magnitude of the change that would result in a 90% detection probability.

With the second method the hypothesis that the fatality proportions are constant both before and after the start of ASAPs within each comparison group, i.e. the hypothesis that:

$$p_{ijk} = \bar{p}_{i.k} \tag{B17}$$

is tested directly. The applicable test statistic following Lancaster [1969]

$$Q_\nu^C = \sum_{i=1}^{I}\sum_{k=1}^{2}\sum_{j=1}^{J(k)}\left(X_{ijk} - N_{ijk}\frac{\bar{X}_{i.k}}{\bar{N}_{i.k}}\right)^2 / N_{ijk}\frac{\bar{X}_{i.k}\bar{Y}_{i.k}}{(\bar{N}_{i.k})^2} \tag{B 18}$$

has χ^2 distribution with ν degrees of freedom ($\nu = 15$ or 16 for the comparison groups that include two-year or one-year ASAPs, respectively). Tests T7 and T8 in the Results section employed this statistic.

The hypothesis that the fatality proportions did not change in the comparison groups following the start of ASAPs can be tested by comparing the average pre- and post-ASAP fatality proportions for each comparison group separately with the average fatality proportion for that comparison group calculated over the whole study period. The test statistic

$$Q_I^T = \sum_{i=1}^{I}\sum_{k=1}^{2}\left(\bar{X}_{i.k} - \frac{\bar{X}_{i..}}{\bar{N}_{i..}}\bar{N}_{i.k}\right)^2 / N_{ijk}\frac{\bar{X}_{i..}\bar{Y}_{i..}}{(\bar{N}_{i.k})^2} \tag{B 19}$$

follows the χ^2 distribution with I degrees of freedom (tests T9 and T10 in the Results section).

The power of this last test against the alternative hypothesis that a systematic $100\Delta\%$ change did follow the start of ASAPs is calculated as follows. Re-writing the expression above for Q_I^T as

$$Q_I^T = \sum_{i=1}^{I}\frac{(\bar{f}_{i.1} - \bar{f}_{i.2})^2}{\bar{f}_{i..}(1-\bar{f}_{i..})}\frac{\bar{N}_{i.1}\bar{N}_{i.1}}{N_{1..}} \tag{B 20}$$

where $\bar{f}_{i.k} = \bar{X}_{i.k}/\bar{N}_{i.k}$, $k = 1,2$ and $\bar{f}_{i..} = \bar{X}_{i.}/\bar{N}_{i..}$ are the average fatality ratios in the i-th comparison group taken over the pre- and post-ASAP periods and over the whole study period, it follows from the normal approximation theory of frequencies that if a $100\Delta\%$ change in the fatality proportions followed the start of ASAPs, that is, if $\bar{p}_{i.2} = (1-\Delta)\,\bar{p}_{i.1}$, $i = 1,\ldots,I$, then, approximately,

$$Q_I^T = \Delta^2\sum_{i=1}^{I}\frac{\bar{f}_{i..}}{1-\bar{f}_{i..}}\frac{\bar{N}_{i.1}\bar{N}_{1.2}}{\bar{N}_{i..}} + \chi_I^2 \tag{B 21}$$

where χ_I^2 is a χ^2 variable with I degrees of freedom. The distribution of Q_I^T is thus a noncentral χ^2 distribution with noncentrality parameter

$$\lambda = \Delta^2\left(\sum_{i=1}^{I}\frac{\bar{f}_{i..}}{1-\bar{f}_{i..}}\frac{\bar{N}_{i.1}\bar{N}_{i.2}}{\bar{N}_{i..}}\right) \tag{B 22}$$

and I degrees of freedom.

Proceeding as in the case of the first method and using tables for the power of the non-central χ^2 test [Owen 1962], it was again possible to estimate the magnitude of the change that would result in a 90% detection probability.

APPENDIX C

NHTSA's *regression model*

In its analysis, NHTSA used the numbers of nighttime and daytime fatal crashes to estimate the parameters and to test their significance in the regression model below [Department of Transportation, 1974].

"$Y = B_0X_0 + B_1X_1 + B_2X_2 + B_3X_3 + B_4X_4$ where
X_0 is the usual correction for the mean,
X_1 is a sine function with a yearly period,
X_2 is a cosine function with a yearly period,
X_3 is a correction for linear trend and taken the form of the sequential order number for each data point (i.e., $1, 2, 3, \ldots n$).
X_4 is the project effect parameter with value:
0 for baseline
1 for operational periods"

As stated in the report "The test that $B_4 = 0$ tests project impact." The estimate for B_4 was not found to differ from zero significantly. NHTSA further asserts that "This model has been extended to account for other effects...", but the precise nature of the extension was not described. It was however subsequently asserted that the "period/daynight... interaction term is the key test for ASAP impact since it indicates that there was an interaction between the change from the baseline to operational period and the number of nighttime as compared to daytime crashes...." This interaction was found to differ from zero ($p \leq 0{\cdot}01$).

Annual variation in the proportion of nighttime fatal crashes

The transformed proportions of nighttime to all fatal crashes in areas with 1971 ASAPs and in comparison areas comprising 20 states were fitted with an additive model following the procedure described in Appendix B. Let v_{ij} denote the transformed proportions, where $i = 1$ for the ASAP areas, $i = 2$ for the comparison areas and $J = 1, \ldots, 5$ refers to years 1968-72. Then

$$v_{ij} = v + a_i + b_j + c_{ij} \tag{C1}$$

where

v_{ij} = transformed proportions,
v = grand mean of the v_{ij}'s,
a_i = area effect,
b_j = year effect,
c_{ij} = interaction between areas and years.

It was found that

$$\begin{aligned} v &= 129{\cdot}00 \\ a_1 &= -86{\cdot}15 \\ b_1 &= 0{\cdot}25 \\ b_2 &= 1{\cdot}57 \\ b_3 &= 0{\cdot}57 \\ b_4 &= -0{\cdot}22 \\ b_5 &= -2{\cdot}18 \end{aligned}$$

and, also, that the hypothesis of no year effect is rejected by the F test of the F-ratio $(SS_T + SS_C)/SS_e$(D.F. = 4,4, $p \leq 0{\cdot}01$). The fit of the model, when tested using the interaction terms $c_{11} = \times 0{\cdot}31$, $c_{12} = 0{\cdot}03$, $c_{13} = 0{\cdot}43$, $c_{14} = -0{\cdot}38$, $c_{15} = 0{\cdot}21$, seems to be quite good ($\chi_4^2 = 0{\cdot}95$, $p \geq 0{\cdot}95$). One is therefore justified in concluding that while the proportions of nighttime fatal crashes do change from year to year these changes are equally present for both ASAP and comparison areas.

Similar analyses yielded no significant year effects for either the 1971 ASAPs over the 1968–71 period or for the 1972 ASAPs over the 1969–72 period.

Comparison of nighttime fatal crashes and all fatal crashes in ASAP and comparison areas

The hypothesis that the proportions of nighttime fatal crashes in ASAP areas to nighttime fatal crashes in ASAP and comparison areas combined remained constant during the period 1968–72 was tested by means of the customary χ^2 test for comparing proportions [Lancaster, 1969]. The hypothesis was not rejected at the $p = 0{\cdot}05$ level.

The proportions of all fatal crashes were similarly tested. Again no significant differences were observed.

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A CRITIQUE OF THE PAPER "STATISTICAL EVALUATION OF THE EFFECTIVENESS OF ALCOHOL SAFETY ACTION"

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Abstract—This critique refutes Zador's claim [1976] that Alcohol Safety Action Projects, as large scale social programs have been ineffective. The discussion takes issue with Zador's comparison groups, statistical design, statistical methods, and use of 12-month fatality totals for data analysis. In addition to weaknesses and deficiencies in these areas, the authors find Zador's conclusion unfounded based on the analysis presented.

INTRODUCTION

Evaluation of social programs is in its infancy and few traffic safety projects have been scientifically assessed. The overly glowing, frequently self-serving "evaluations" sometimes conducted by project advocates have been justly criticized by research investigators. On the other hand, scientific evaluators can also be guilty of going beyond their data in rejecting official claims for project effectiveness. Traffic safety research is handicapped by inadequate data systems and the inability to maintain experimental control over the areas to be assessed. Moreover, because of funding limitations, most safety projects are too small or too short to permit collection of sufficient criterion data to provide a sensitive test of the project's effectiveness. Under these circumstances, it is incumbent upon the research investigator to be conservative in the interpretation of his results. Where he claims to have found no evidence of impact, he should indicate the sensitivity of his measures and the limitations they pose on the extent to which his findings can be accepted as an adequate overall assessment of the value of the project.

Paul Zador's conclusions go far beyond the data he presents and the analysis he was able to conduct of the effectiveness of the National Highway Traffic Safety Administration's (NHTSA) Alcohol Safety Action Projects (ASAPs). His paper ends with the statement that "... it is only possible to conclude scientifically that the ASAPs, as large scale social programs, have been ineffective" [Zador, 1976]. This conclusion is not permissible "scientifically" or logically from the data presented in the analysis conducted by this investigator because: (a) His study is very incomplete. He considers only one of several objectives of the ASAPs. He analyzes data from less than half the operational period of the ASAPs. He reviews only one of many of the ASAP reports. (b) His study is insensitive. He uses a very gross, indirect criterion. His statistical technique lacks sufficient sensitivity to have detected the changes the NHTSA claimed to have observed. He readjusts the borders of the ASAPs to suit the convenience of his analysis technique. (c) His selection of control or comparison areas is invalid. (d) His interpretations of his results go beyond the data presented, particularly the assumption that because he could not detect an impact with his analytical technique, no impact in fact, occurred. These limitations in the Zador study are considered at greater length below.

A. COMPLETENESS OF STUDY

1. After listing the 3 major goals of the ASAPs [Zador, 1976], Zador chooses to study a portion of one objective and ignores the others. Even if his results and his interpretations of those results are accepted, his conclusions can apply to only a portion of the purposes of the ASAPs.

2. The data from which Zador is deriving his conclusions are based on less than half of the ASAP operations. His study covers only 2 of 3 years of operation of the first 8 ASAPs; 1 of 3 years of operation of the second group of 20; while the results of 7 of the 35 projects were excluded entirely. The NHTSA report covering these same data clearly labeled the report as "interim" [DOT, Vol II, p. 8, para. 3]. Zador was less modest—he sat through the first half of the game and called a winner without waiting for the final results.

3. The author did not review the evaluation reports of the individual projects (in part because these reports were not readily available) nor did he review or comment on other types of criterion data, such as the roadside surveys of breath alcohol levels which were presented in the original NHTSA report. Therefore, his conclusions are necessarily based on only a portion of the available data on ASAP effectiveness. A final verdict on the "effectiveness of the ASAPs as large scale social programs," cannot be reached solely on the basis of reductions in fatalities but must consider the societal benefits resulting from the totality of countermeasure effects on the drinking driving problem.

B. SENSITIVITY OF STUDY

1. Zador's conclusions are limited by the criterion he chose which was total fatalities. The ASAPs were directed at crashes resulting from the excessive use of alcohol. Therefore, their goal was to reduce alcohol related (A/R) crashes, which comprise about half of all fatal crashes. Reductions in A/R crashes should ultimately be reflected in a reduction in total crashes, which in turn should result in a reduction of total fatalities. However, this gross measure is an indirect and less sensitive indicator of the effectiveness of alcohol safety programs. A disappointing aspect of this paper is that, while the original NHTSA document [DOT, 1974] had an extensive discussion of the criterion problem, Zador chooses to ignore the issue and set up his own straw man criterion of "total fatalities."

2. Zador concludes that the ASAPs were "ineffective" when, by his own estimate [Zador, 1976], which we dispute, (see Appendix 1), his analytical procedure would not have detected a real reduction of less than 10% in total crashes (or 20% in A/R crashes). Thus, by "ineffective" the investigator means that during the first half of the operational period the ASAPs failed to produce at least a 20% change in one of its criterion measures. Stated in this way, the implications to be drawn from this study's results are quite different than those suggested by the final paragraph of his paper.

3. It should be noted that the NHTSA report claimed a significant reduction in night to day fatal crashes and in total fatal crashes only for the first 8 projects for which 2 years of operational data were available. The study specifically concludes [DOT, 1974], that there was "no statistically significant evidence for impact... for the 21 projects for which only 1 year of operational data are available." Zador comes to essentially the same conclusion regarding these projects. In this, he simply confirms the NHTSA results. The only issue between the 2 studies, then, is whether there is evidence for impact in the first 2 years of the first 8 projects. Because the numbers of fatalities at these sites are relatively few, the significance of the power of the statistical procedures used by Zador becomes particularly important. Using his methodology, it does not appear that he could have detected a difference as small as that reported by NHTSA (see Appendix 1).

4. There is a considerable discrepancy in the data reported for the ASAP areas by NHTSA and the data used by Zador (see Appendix 2). It is not entirely clear why so many discrepancies arise, however State and local data records quite frequently contain significant inaccuracies. In the evaluation of the ASAPs, considerable expense and time was devoted to purifying the data base. In the present instance, Zador used different sources for his data and slightly different geographical boundaries for the ASAP areas. Whether this contributed to the different results of his study is unclear.

C. SELECTION OF COMPARISON AREAS

1. Zador claims [1976] that the use of comparison areas is a minimal requirement for a "scientific" demonstration of a change in fatalities. By implication he finds the NHTSA study inadequate because comparison groups were not used. This is clearly not in keeping with current practice in this field.

Campbell and Stanley [1963] presented an extensive discussion of the use of "quasi-experimental designs" and of the sources of invalidity in these designs. These "time series" designs are not dependent on the use of comparison groups for their validity. Rather, they are dependent upon testing and elimating "alternate hypotheses." One of the most persuasive and well accepted impact analyses of a traffic safety program is that of Ross [1973] who used time series analysis to evaluate the British Road Safety Act of 1967. His study did not employ a

comparison community but rather depended upon the contrast between the crash reductions at "drinking hours" and the smaller reductions seen at others hours of the day.

The NHTSA study, far from ignoring this experimental design issue, (1) specifically discussed this methodological problem [DOT, 1974]; (2) listed Campbells and Stanley's "6 threats to validity [DOT, 1974] and discussed their potential impact on the ASAP evaluation; (3) and specifically qualified the results of the analysis of crash trends [DOT, 1974] based on the need to eliminate the "competing hypothesis" that the difference between night and day crashes at the ASAP sites was reflecting a national trend. The NHTSA demonstration project impact for the first 8 projects is based on comparing nighttime (drinking) with daytime (nondrinking) hours which is essentially similar (through developed in less detail) to the method used by Ross [1973].

2. Zador selected his comparison sites based on population and population growth. This is probably an inadequate basis for selecting comparison groups, since a number of other factors may be equally or more important in determining the number of alcohol related crashes (see Appendix 3). Zador [1976] attempts to meet this issue by testing for significant variations in the year to year fatality rates during the baseline period at the ASAP communities and the comparison communities. But since his statistical technique is relatively insensitive (see Appendix 1), this demonstration is unconvincing.

3. Zador [1976] states but ignores, the most essential requirement in the use of comparison communities: they must not have a similar countermeasure effort during the period of the comparison. Zador apparently made no effort to determine what countermeasure activities were being undertaken in his comparison communities. When a check was made by the present authors, it was found that a number of these communities had special enforcement efforts and one had a State funded ASAP! (See Appendix 3.) This result was not surprising. A number of the ASAPs chose control communities at the beginning of their projects, only to have these communities decide to initiate special alcohol safety projects of their own, midway in the study period.

D. INTREPRETATION OF RESULTS

1. Zador makes the basic error of concluding that since the specific statistical technique he used on a limited portion of the data failed to find a significant effect, the ASAPs were ineffective "as large scale social programs." Zador should have limited his claims to the hypothesis he tested which only related to reduction of total fatalities in ASAP sites to certain comparison areas. He makes no test of the broader social, catalytic organization impacts of the ASAPS. Even his claims with regard to fatalities are strictly limited by his insensitive analysis. A more appropriate statement, assuming a full acceptance of Zador's methodology, is that the effectiveness of the ASAPs in reducing total fatalities is *unproven.*

2. In addition to the analysis of his own data on fatalities, Zador [1976] reanalyzed the NHTSA data on nighttime vs daytime crashes, using his own statistical method (shown in his Appendix C). In reanalyzing the NHTSA data, Zador notes a decrease in the proportion of nighttime crashes in both the ASAPs and a sample of crash data from 20 States provided in the NHTSA report. He states that this is positive evidence that the ASAPs could not have produced the change in nighttime crashes [Zador, 1976]. This national trend he "discovers" is extensively discussed in the NHTSA report [DOT, 1974].

The DOT report states that until the possibility that a national trend to fewer nighttime crashes accounts for the ASAP results can be eliminated, it is not possible to conclude that the 8 ASAPs had a significant impact [DOT, 1974]. The evidence for a national trend is skimpy, however, since it is based on only 1 year (1972) and on only 20 States. NHTSA has contracted for a 10-year (1965–1974) study of a carefully selected sample of States to determine whether a national trend to fewer nighttime crashes can be demonstrated. NHTSA has also recently completed a study of a national sample of U.S. communities to determine the extent to which ASAP counter-measures have been adopted by the States. This study indicates that during the period of ASAP operations ('71–74), there has been a significant increase in the implementation of these activities throughout the nation (see Appendix 4). Thus, the NHTSA effort to get these measures adopted nationally may be the reason for this national trend, and far from disproving the effectiveness of the ASAPs, it may be the ultimate demonstration of their effectiveness.

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APPENDIX 1

The Zador analysis is based upon the use of highly aggregated annual data, i.e. 3 data points for baseline compared with 1 or 2 data points for the operational period depending upon whether the project was started in 1971 or 1972. F and Chi-Square tests were performed to determine if significant changes occurred over time in the ASAP groups as well as the comparison groups. In addition, a second set of tests using the F ratio and Chi-Square to determine significant differences between pre and post ASAP fatality proportions was conducted. The calculations show no significant differences in annual fluctuations of the fatality proportions within ASAP and comparison groups as well as no significant differences in annual fluctuations between baseline and operational periods.

Because of the limited number of data points used (25 data points for 1971 ASAP and comparison groups, 32 data points for 1972 ASAP and comparison groups), it is very important to examine the power of the Zador experimental design for its sensitivity relative to committing a Type II error.

The power of the test is defined as (1-B) where B is the Type II error. The power of the test is a measure of the design's capability to accept the alternative hypothesis when it is true. Therefore, a power of 0·9 indicates that the experimental design has the capability to accept the alternative hypothesis when it is true nine out of 10 times and to accept it once where it is false.

In general, the power of a test varies with the number of observations to be studied. The greater the number of observations, the higher the power within a given design concept. In addition, estimates of residual or experimental errors and specifications of significant differences between treatment groups are generally required. Given these specifications, the researcher can design an experiment to determine the number of observations required for given Type I and II errors, experimental errors, and magnitude of treatment differences.

In the analysis of the ASAP impact, Chap. 2, Vol. II, ASAP Program Evaluation Methodology and Overall Program Impact, page 10, Fig. 7 contains the Analysis of Covariance sum of squares of the combined first 8 ASAPs for 3 years of baseline versus two years of operations. In all, 456 data points were included. The ASAP experiment was not designed to maximize the power of the test; however, its power can be calculated based upon the design used, the number of observations, and the experimental error noted.

Using B. J. Winer's [1972] methodology for determining sample sizes, a value of ϕ' is computed as follows:

$$\phi' = \frac{\sqrt{(\chi_i - \mu_i)/K}}{\sigma^2}$$

where: μ_i, average of each treatment group;
K, number of treatments;
σ^2, experimental error.

In the ASAP analysis, H_1, H_2 are the null and alternative hypotheses being tested; H_1, there is no significant difference in monthly fatal crashes among the first 8 ASAPs from baseline period through the 2-year operational period; H_2 there is a significant difference in the level of monthly fatal crashes between baseline and operations for the first 8 ASAPs.

For discernability or detection of an approximately 15% reduction in average monthly fatal crashes between baseline and operational periods, ϕ' is calculated to be approximately 0·15. Using Tables 1 and 2 [Winer, 1962], curves of constant power for tests on main effects, it is estimated that the power of the test used in the ASAP evaluation for a Type 1 error of 0·05 lies between 0·8 and 0·9, assuming an average of 278 observations per treatment. In nonstatistical terms, the design used by NHTSA has the capability of accepting the null hypothesis when true 95 out of 100 times and accepting the alternative hypothesis when it is true 90 times out of 100 for detection of a 15% reduction in monthly fatal crashes.

Using the same specifications for the Zador model, where the analysis of variance was computed using annual fatalities for the 5 years separately, one concludes that the design has little or no power to discern whether the alternative hypothesis is true. The power of the test was estimated to be 0·1 to 0·2 for the same specifications. Thus, the utilization of annual data for the 5 year period makes the Zador model almost totally insensitive in accepting alternative hypotheses; that is that the ASAPs *were* effective.

The use of inferential techniques in analysis of dependent data must be done with caution, since methods normally used with independent data may not be directly applicable. The confidence intervals chosen for the Chi-Square and F tests shown in [Zador, 1976, p. 56] may be subject to question on this basis. Glass [1972], Gastwirth *et al.* [1971] showed through analysis of dependent time series that what would represent a 90% confidence interval for an independent series can actually produce confidence intervals significantly different in either direction when applied to a dependent time series. Since the series of yearly fatality totals could conceivably be a dependent time series, additional attention to confidence intervals for the tests used must be reevaluated in the light of the above discussion. There is no indication that this was done.

Another consideration which has been overlooked in the Zador design is that accident and fatality data are time series having characteristics which could preclude the use of classical statisticial techniques such as Analysis of Variance, F ratio and Chi-Square tests. Apparently no

Table 1. Curves of constant power for tests of main effects ($K = 2, 3$)*

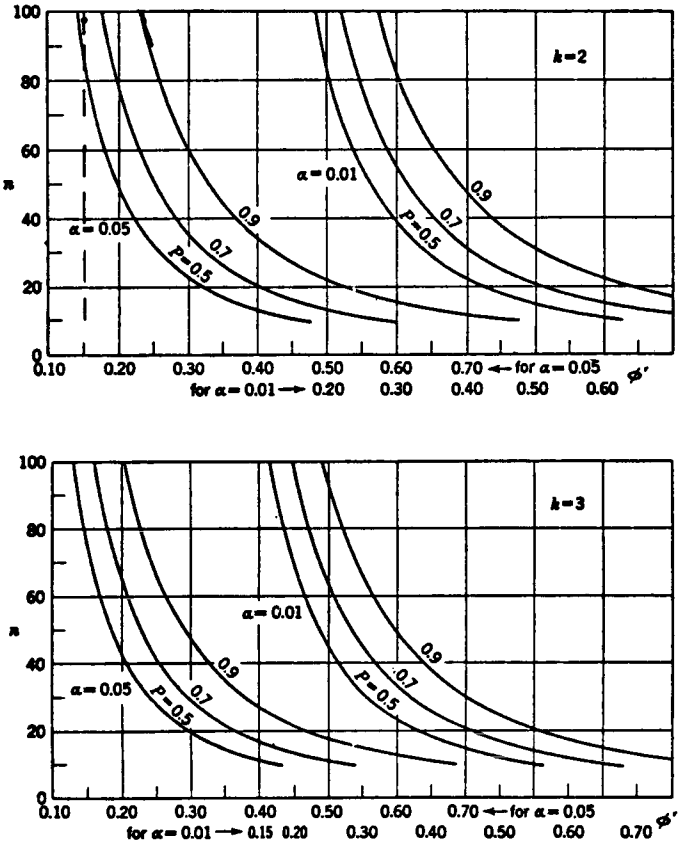
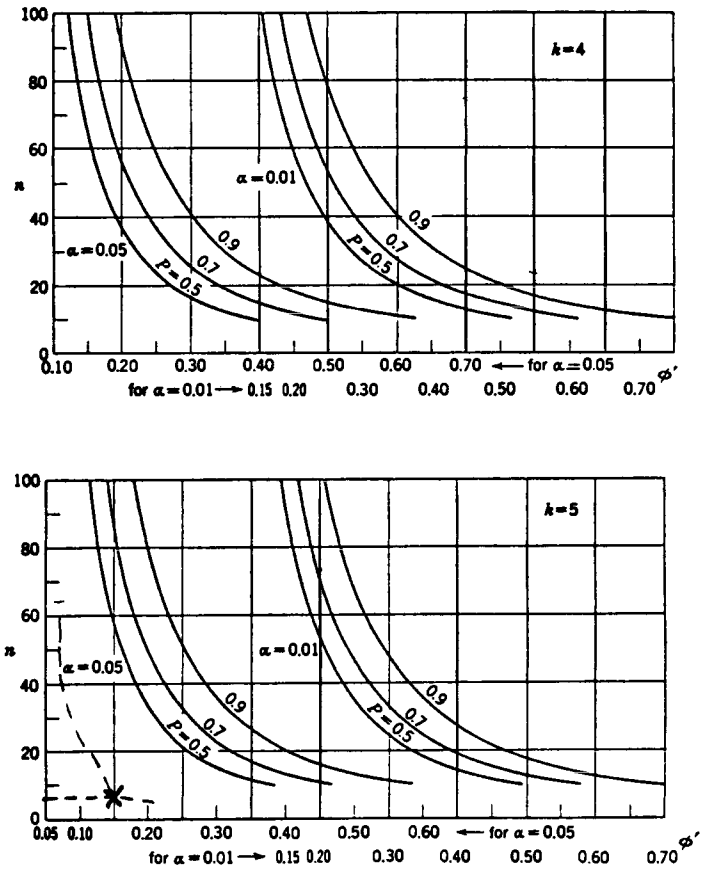


Table 2. Curves of constant power for tests of main effects ($K = 4, 5$)*



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tests were conducted which validated the presence or absence of autocorrelation in the data. Although tests for constancy were conducted, they were based on at most 5 data points per group using an Analysis of Variance design of very low power.

The nighttime fatal crash analysis referred to in the Zador report was conducted on an aggregated basis using the same Analysis of Variance approach as the fatality analysis. Weaknesses noted previously concerning power of the test, assumption of randomness and aggregation of data apply to the nighttime fatal crash analysis as well. Acceptance of alternative hypotheses regarding ineffective ASAP programs are completely invalid. Using a design of low power, it is incorrect to state that because a significant effect was not found, the program is therefore ineffective.

APPENDIX 2

Comparison of the Zador fatality data with the ASAP reported fatalities for the 4 and 5 year periods shows agreement with only 16 out of the 28 ASAPs. Significant differences were noted, especially in sites where partial populations were used or where geographic areas were not alligned, such as Baltimore. Table 3 indicates the differences in the 2 sources of data. The 12 sites where significant discrepancies were noted affect the group totals in which they were included. These comparisons can be found in Tables 4 and 5. These discrepancies are imbedded in the 4 totals which form the basis for the analysis of variance calculations. The degree of error propagated in the transformed ratios is difficult to determine due to the transformation of the ratio defined in the paper.

Table 3. Site comparisons of fatalities

Z=ZADOR A=ASAP ASAP PROJECT		% POP. INCL.	1968	1969	1970	1971	1972	TOTAL
DENVER	Z	47	98	99	80	61	72	410
	A		125	122	116	106	114	583
PORTLAND, ORE.	Z	83	62	59	74	57	80	332
	A		64	60	73	57	84	338
SEATTLE	Z	46	101	68	56	69	53	347
	A		222	216	197	172	159	966
ALBUQUERQUE	Z	77	30	47	48	48	56	229
	A		67	67	79	83	100	396
CHARLOTTE	Z	68	41	41	55	46	40	223
	A		79	82	87	95	82	425
NASSAU	Z	100	145	196	173	174	161	849
	A		144	180	176	173	157	830
WASHTENAW	Z	100	64	57	78	67	69	335
	A		63	57	78	67	66	331
MARATHON AND SHEBOYGAN, WISC.	Z	100	57	53	47	46	57	260
	A		57	53	47	46	57	260
PHOENIX	Z	100		103	90	75	98	366
	A			111	101	89	108	409
BALTIMORE	Z	100		127	109	90	104	430
	A			169	148	147	150	614
BOSTON	Z	100		112	76	75	93	356
	A			110	76	74	87	347
CINCINNATI	Z	100		75	84	89	82	330
	A			73	84	88	81	326
NEW ORLEANS	Z	100		108	113	98	80	399
	A			109	112	100	80	401
KANSAS CITY	Z	100		102	89	89	95	375
	A			101	91	89	97	378
OKLAHOMA CITY	Z	100		102	76	82	86	346
	A			102	75	82	86	345
SAN ANTONIO	Z	100		106	97	92	93	388
	A			100	99	92	93	384
TAMPA	Z	57		63	49	56	60	228
	A			171	129	166	167	633
WITCHITA	Z	79		25	30	32	38	125
	A			57	52	63	57	229

Table 4. Site comparisons of fatalities

Z=ZADOR (Sept., A=ASAP 1974) ASAP PROJECT		% POP. INCL.	1968	1969	1970	1971	1972	TOTAL
PULASKI CO.	Z	46		22	24	15	17	78
	A			54	64	51	61	230
RICHLAND CO.	Z	49		6	10	14	14	44
	A			63	63	67	47	240
COLUMBUS	Z	93		11	16	16	18	61
	A			19	26	16	19	80
LINCOLN	Z	100		15	15	12	11	53
	A			15	15	12	11	53
HENNEPIN CO	Z	100		118	143	112	94	467
	A			118	143	113	94	468
INDIANAPOLIS	Z	100		141	117	113	132	503
	A			76	66	53	58	253
(Zador used Marion Co Ind.)								
SOUTH DAKOTA	Z	100		296	237	262	294	1,089
	A			296	237	262	292	1,087
FAIRFAX CO.	Z	100		58	61	98	84	301
	A			67	66	104	82	319
NEW HAMPSHIRE	Z	100		189	* 196	214	179	778
	A			189	196	214	180	779
PORTLAND	Z	100		75	100	71	73	319
	A			74	108	70	73	325

GROUP I FY 1971 ASAPs		1968	1969	1970	1971	1972	TOTAL
Zador (Sept., 1974) ASAP							
1	Z	261	226	210	187	205	1,089
	A	411	398	386	335	357	1,887
2	Z	71	88	103	94	96	452
	A	146	149	166	178	182	821
3	Z	145	196	173	174	161	849
	A	144	180	176	173	157	830
4	Z	64	57	78	67	69	335
	A	63	57	78	67	66	331
5	Z	57	53	47	46	57	260
	A	57	53	47	46	57	260

Table 5. Group 2 comparison of fatalities

GROUP II FY 1972 ASAPs		1969	1970	1971	1972	TOTAL
Zador (Sept. 1974) ASAP						
1	Z	103	90	75	98	366
	A	111	101	89	108	409
2	Z	422	382	352	359	1,515
	A	461	420	409	398	1,688
3	Z	398	341	351	372	1,462
	A	531	446	492	500	1,969
4	Z	54	65	57	60	236
	A	151	168	146	138	603
5	Z	259	260	225	226	970
	A	194	209	166	152	721
6	Z	296	237	262	294	1,089
	A	296	237	262	292	1,087
7	Z	58	61	98	84	301
	A	67	66	104	82	319
8	Z	264	296	285	252	1,097
	A	263	304	284	253	1,104

APPENDIX 3

The comparison areas selected by Zador were assembled on the basis of population size, population growth, area type and geographic location. These criteria are not sufficient for constructing valid comparison areas against which to measure experimental change. If comparison groups are desired for a statistical model, it is necessary to develop them so that they can be considered predictors of the behavior that would have occurred in the ASAP group. If similarity in driving-related factors is not established between the ASAP and its comparison group, the behavior in the comparison group during the test period could be totally irrelevant to the behavior that would have occurred without the ASAP effect. Factors which need to be examined include:

Population	Driving Environment
Percentage of licensed drivers	Miles of roadway in the areas
Distribution by age and sex of drivers and total population	Distribution of mileage by roadway type
Fatality distribution by age, sex and fatality type	Distribution of fatalities by roadway type
Number and type of vehicles operated	
Distribution of night and day fatal crashes	

Certainly, there are more variables which could be added to this list to extend comparisons of the 2 groups. The depth of any comparison, of course, is a function of the time and data available. The point is that nothing in Zador's population based comparison groups indicates characteristics similar enough in *traffic related* experience to be compared for an ASAP study.

The composition of the comparison groups indicates that some groups contain significantly less population and/or fatalities than the ASAP sites while other groups contain significantly more population and fatalities than the ASAP sites, with no particular rationale given for their construction. As a result, the fatality ratios for given levels of population ratios vary significantly between comparison groups, and the transformed ratios are heavily affected by the fatality levels within the the comparison group. This can be illustrated by a comparative analysis of Groups 1 and 3 of the 1971 ASAPs in Table A4 of Zador's paper. Group 1 ASAPs have a total population of 1,427,000 while its comparison group has a population of 616,000, a ratio of over 2 to 1. Group 3 ASAPs have an almost identical population 1,429,000 and a comparison group population of 894,000. The fatality level of Group 1 comparison areas is approximately 500 per year while the fatality level for Group 3 comparison area is approximately 130 fatalities per year.

The calculation of the transformed ratio in Group 1 is of a lesser order of magnitude, primarily due to the high fatality level in its comparison area. The opposite is true in Group 3, namely its transformed ratio is higher, primarily due to the lower level of fatalities in the comparison area. The Group 1 trend of the fatality ratio shows an almost constant level while Group 3 trend moves upward during the baseline period followed by a decline in the operational period. If we were to exchange the Group 1 and Group 3 comparison groups or even substitute other cities, then their corresponding trends in fatality ratios would be reversed, i.e. Group 3 would show a constant level trend and Group 1 would then show an increase in the baseline period followed by a decrease during the operational period. Thus, the arbitrary selection of the comparison areas based on gross and insensitive criteria can induce artificial and spurious results leading to the wrong conclusions.

Further investigation by Zador in selection of his comparison groups would have revealed that many areas in his groups were not free from countermeasure activity, as was assumed.

Table 6 summarizes traffic safety and alcohol related programs which were in effect in the comparison groups during the ASAP program. Programs were found in all of the 1971 comparison groups and in all but one of the 1972 comparison groups. Further programs designed to reduce *alcohol related acidents and fatalities* were found going on during the period when the comparison groups were supposed to be free of the countermeasure. For example, Ohio implemented a breathalyzer law, passed an implied consent law and administered specialized training for police in handling alcohol cases, all in 1971 and 1972. Therefore, Toledo and Akron should not have been included in comparison groups 1 and 2 for 1971 ASAPs; Cleveland and

Table 6. Groups of areas with 1971 ASAPs in study and matched groups of comparison areas

Group	Areas with ASAPs in Study	Comparison Areas	Comments
1	Denver, Colorado; Portland, Oregon; Seattle, Washington;	Akron, Ohio; Atlanta, Georgia; Long Beach, California; Milwaukee, Wisconsin; Newark, New Jersey; Norfolk, Virginia; Oakland, California; San Francisco, California.	Akron-Selective law enforcement program, expanded EMS program--Milwaukee-Special Pedestrian program in high incident area. Ohio Statewide: 1971-1972: (1) Implied Consent passed; (2) Breathalyzer Law went into effect; (3) Specialized training (i.e., alcohol); (4) Police traffic service training for Selective Law Enforcement. San Francisco-FARE operational July 1972-June 1973. Denver-Only 47% of ASAP population included in study. In six municipalities within three counties excluded from study, special ASAP patrols were funded in Denver.
2	Albuquerque, New Mexico; Charlotte, North Carolina	Corpus Christi, Texas; El Paso, Texas; St. Petersburg, Florida; Toledo, Ohio.	Ohio Statewide: 1971-1972: See group 1 El Paso-STEP operational January 1972-June 30, 1973
3	Nassau County, New York	Westchester County, New York	Westchester 1971: New Rochelle-Driver education simulator lab--Peekskill-Speed enforcement. Briarcliff Manor-Use of breath testing device--Port Chester-radar equipment and use of breath testing device. Greenburgh-Police traffic services--Westchester-Breath testing program, declaration of emergencies, determination of no passing zones and speed limits, and identification and surveillance of accident locations. Hastings-on-Hudson-Use of breath testing device--Irvington-Alcohol testing Mount Pleasant-Breathalyzers purchased, communications and radar equipment--White Plains-Traffic record computerization Yonkers-Intoxicated driver enforcement and traffic law enforcement--Mount Vernon-Police department safety vehicle-North Castle-Alcohol testing Eastchester-Accident investigation and breath testing--North Pelham-use of Breath Testing Device Pelham Manor-Speed enforcement traffic--Rye-Police services--Scarsdale-Alcohol testing Tuckahoe-Driving While Intoxicated, alcohol testing and recording--Westchester-Accident investigation and rescue , Emergency Medical Communications--White Plains-Speed enforcement.
4	Washtenaw County	Ingham, Kalamazoo, Saginaw Counties, Michigan	Ingham-Special selective enforcement in Lansing, special pedestrian programs have had special police training, EMS program expanded plus of M.V. Inspection program--Kalamazoo-Selective enforcement, expansion of EMS program Saginaw-Expansion of EMS program
5	Marathon and Sheboygan Counties, Wisconsin	Adams, Clark, Lincoln, Portage, Price, Shawano, Taylor, Waupaca, Wood Counties, Wisconsin for Marathon and Manitowac and Racine Counties, Wisconsin for Sheboygan.	Manitowac-FARE program, special alcohol enforcement and special PIE program on alcohol Racine-FARE program, expansion of EMS program Wisconsin Statewide: 1971-1972: (1) Implied Consent, (2) Statewide breathalyzer training, special traffic law enforcement except Adams Co., (3) Traffic court conferences in all counties, (4) Counseling for licensing revokees in all counties, (5) Traffic safety coordinator, commission appointed in each county. Clark Co.-Police traffic service equipment, EMS program expanded--Lincoln-EMS Program expanded Portage Co.-Special summer emphasis enforcement program initiated EMS expansion--Price Co.-EMS expansion bridge inspection program, special PIE program Shawano Co.-Police equipment purchased, expanded EMS Taylor Co. Expanded EMS police equipment purchased Waupaco Co.-Special summer emphasis enforcement program, special alcohol enforcement Wood Co.-EMS expansion, police traffic service training and equipment, special summer emphasis enforcement

Groups of areas with 1972 ASAPs and matched groups of comparison areas

Group	Areas with ASAPS in Study	Comparison Areas	Comments
1	Phoenix, Arizona	Memphis, Tenn., San Diego, Calif.; San Jose, Calif.	Memphis, Tenn.-Comprehensive alcohol safety program (see attached)
2	Baltimore, Md.; Boston, Mass.; Cincinnati, Ohio; New Orleans, La.	Cleveland, Ohio; Pittsburgh, Pa.; San Francisco, Calif.; Washington, D.C.	Ohio Statewide: 1971-1972: (1) Implied Consent passed; (2) Breathalyzer law went into effect; (3) Specialized training (i.e., alcohol as result statewide); (4) Police traffic service training for Selective Law Enforcement.
3	Kansas City, Mo.; Oklahoma City, Okla.; San Antonio, Texas; Tampa, Florida; Wichita, Kansas.	Atlanta, Ga.; Columbus, Ohio; Dallas, Tex.; Fort Worth, Tex.; Long Beach, Calif.	Columbus, Ohio-Selective Law Enforcement Program expanded EMS Program Ohio Statewide-See group 2 Dallas, Texas-State funded ASAP operational Atlanta, Ga.-Alcohol safety school for persons convicted of DWI
4	Little Rock, Ark.; Columbia, S.C.; Columbus, Ga.; Lincoln, Neb.	Jackson, Miss.; Knoxville, Tenn.; Topeka, Kans.; Winston-Salem, N.C.	Topeka, Kansas-Fatal accident and fatality statistics by time of day for the years of 1969 through 1973 Jackson, Miss.-Alcohol safety school, mass media program on drinking and driving, increased emphasis on drinking and driving by police department Knoxville, Tenn.-FARE Program (increased effort regarding traffic violations including alcohol) Winston-Salem, N.C.-Purchase of breath testing devices and training of operators, special pedestrian safety programs
5	Hennepin, Co., Minn.; Marion Co., Ind.	Ramsey Co., Minn; Milwaukee Co., Wisconsin	Milwaukee Co., Wisc.-Big emphasis in county on alcohol, special training for police, viedo tape purchase, large expansion of EMS program
6	South Dakota	North Dakota; Wyoming	North Dakota-STEP from August 1972 through June 30, 1975 and FARE from July 1, 1973 through December 31, 1973 in Bismarck, N. D. Wyoming-FARE from May 25 through December 31, 1973.
7	Fairfax Co., Va.	Montgomery Co., Md.; Prince Georges Co., Md.	
8	New Hampshire, Cumberland and York Counties, Me.	Seven counties from New York state and two counties from Vermont for New Hampshire: Broome, Dutchess, Essex, Fulton, Herkimer, Montgomery, Oswego, N.Y. Addison, Bennington, Rutland, Vt.; two counties from New York state for Maine: Saratoga, Ulster.	Addison Co., Vermont-This county was a part of the ASAP Project in Vermont in that Judicial Rehabilitation and PIE countermeasures were operated, in addition, the county was directly adjacent to the most active enforcement countermeasure area in the Vermont ASAP. Bennington Co., Vermont-Initiated a locally funded crash school in late 1972. The county also had an active PIE Countermeasures program operational (under CRASH). Dutchess, 1972-Monthly County Accident Analysis Poughkeepsie-Traffic Law Enforcement Amsterdam-Traffic control enforcement Montgomery-Traffic control enforcement and traffic safety & enforcement Oswego, Fulton-Location and Preventive Surveillance and enforcement of DWI laws Oswego-County Traffic Division & traffic control device inventory and and evaluation program Essex-Radios for Ambulances in Essex County and purchase of miscellaneous rescue equipment for ambulances in county Herkimer-Purchase of Speed Measuring device Mohawk-Purchase of speed measuring device Binghamtom-School traffic safety program and speed computer for highway safety and vehicle registration reader Broome-Breathalyzer a/o Alco-Tector, driver simulators, "911" emergency telephone system, sonic light bar and power call siren, speed computer and recorder Chenango Forks-Exemplary classroom driver Telemate Tutor #16 Chenango Valley-Multi-unit driver education program Endicott-Breathalyzer, purchase of recording equipment, 600 aluminum sign blanks Val 1 Scotchlite Brand Vacuum Application, sonic light bar and power call siren and VASCAR Johnson City-Breathalyzer, driver simulators and speed computers for highway safety Vestal-Ambulance for emergency Medical Services (use of) Breathalyzer, multi-unit program for driver education, speed computers for highway safety, vehicle registration reader Whitney Point-Ambulance for Emergency Medical Service and exemplary driver education classroom program

Table 7. Growth of drinking-driver countermeasures

Countermeasures:	Number of Jurisdictions 1969	1972	1973	1974
Alcohol Enforcement Patrols	240	370	510	590
Background Investigations Performed	4060	4580	4720	4960
Court Referrals to Rehabilitation	1550	2690	3500	4060
Post-Rehabilitation Follow-up	170	580	1130	1520
Public Information & Education	1260	1910	2080	2340
Alcohol Program Staffs	630	1270	1720	2070
Alcohol Program Coordinators	490	1360	1730	2090

Columbus should have been excluded from comparison groups 1 and 2 for 1972 ASAPs. Further study of the list shows alcohol safety schools implemented in Atlanta, Georgia [1972]; State funded ASAP in Dallas, Texas implemented [1972]; Alcohol Safety School and coordinated media campaign in Jackson, Miss. [1972] and a comprehensive alcohol safety program implemented in Memphis, Tenn. [1972]. These are only a few of the areas where there were active programs which may have affected fatalities and accidents, and such areas should have been excluded from any comparison group. In effect then, the comparison groups used were inappropriate for this study and have no value as "controls" for the ASAP groups. The NHTSA did not make use of specific comparison areas because valid comparison areas could not be found.

APPENDIX 4

No adequate data are available at this time to determine whether there is a national trend toward fewer alcohol related crashes in the United States. A reason why there might be is indicated in the table below taken from the NHTSA Statewide Highway Safety Program Assessment. This study was conducted to determine the extent to which the NHTSA highway safety programs are being implemented by the 50 States.

The data in Table 7 are based on a sample of 105 jurisdictions. This sample has been expanded to provide an estimate for the 6435 U.S. jurisdictions with 2500 or more residents. As can be seen, countermeasure activities similar to those implemented in the ASAPs, have been growing throughout the country. For example, in 1969 1500 of the nearly 6500 jurisdictions in the country referred drinking drivers to a rehabilitation program; in 1974 this number had more than doubled to 4000. Thus, the increasing safety activity of the states and the NHTSA national program are "contaminating," and thereby reducing the validity of the non ASAP areas as comparison areas for evaluating the effectiveness of these projects. If the ASAP counter-measures are indeed effective, then the fact that the 20 State comparison samples also demonstrated a shift to fewer nighttime crashes might be further evidence for the effectiveness of the ASAPs rather than proof, as Zador claims, that the ASAPs could not have produced the reduction in nighttime crashes.

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FORTHCOMING EVENT

The 7th International Conference on Alcohol, Drugs and Traffic Safety is scheduled to take place at Melbourne University, Australia, 23–28 January 1977. The Australian Forensic Science Society will be holding its annual meeting in Melbourne at about the same time, while a summer school on Alcohol and Alcohol Studies is usually held in New Zealand a week or so later. Titles and abstracts of papers proposed for the Conference should be sent to: Royal Australian College of Surgeons, Spring Street, Melbourne 3000, Australia. The tentative program is as follows: 23rd January, *Registration and Reception*: 24th January, *The Dimensions of the Problem*; 25th January, *Pharmacology and Analytical Methods*; 26th January, *Community Attitudes and Behaviour*; 27th January, *Control and Enforcement*; 28th January, *Public Information and Education.*