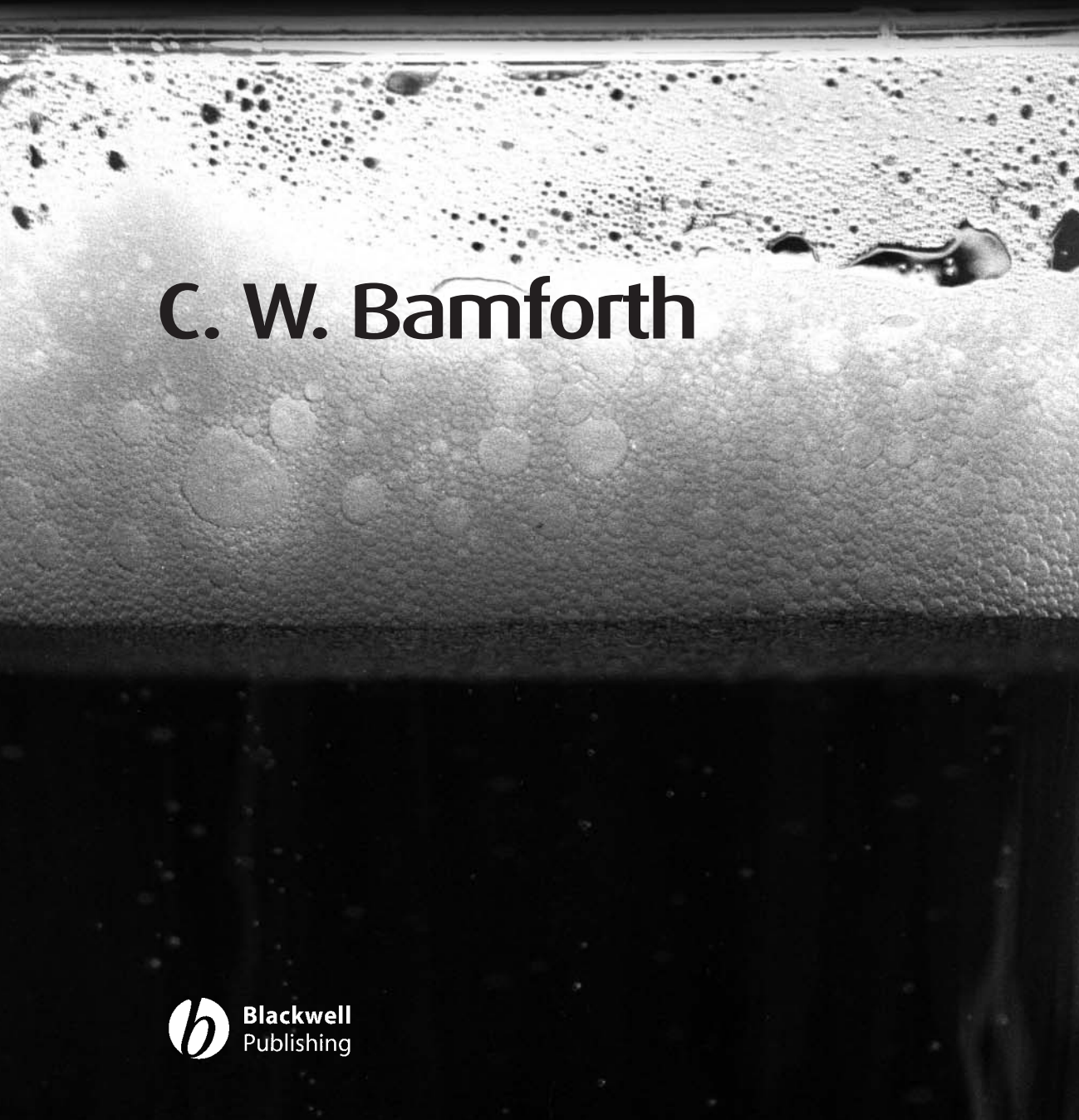
Beer

Health and Nutrition



C. W. Bamforth

Blackwell Publishing

Beer
Health and Nutrition

From man’s sweat and God’s love, beer came into the world

St Arnoldus

Beer
Health and Nutrition

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Dedicated to my forebears

Contents

Preface

John Hudson peered at me over his half-moons. A rm frown was on his forehead. His hands were folded rmly on his desk.

'Say that one more time, lad', he grunted in his familiar and frequently feared North Yorkshire accent.

I gulped and let it go one more time.

'I don't think the work I am doing here is worthwhile. I mean, I could be researching cancer – something bene cial for mankind. But I'm working on beer – what puts bubbles on a pint, why lager tastes of sweetcorn, how to choose the best barley. It's not exactly crucial, is it?'

I'd been worrying about my *raison d'être* for some while. Surely my expertise as an enzymologist could be put to better use?

Hudson, Deputy Director of the Brewing Research Foundation at Nut eld in leafy Surrey, was unexpectedly calm on that dull winter morning in 1980.

'Do people drink beer, Charlie?'

'Well, yes.'

'Who drinks beer?'

'Lots of people.'

'Such as the working class man and woman, for instance?

'Yeah.'

'Does it make them happy?'

'Well, sure, as long as they don't get drunk, and they can afford it, and nobody suffers as a result of them doing it.'

'True, but accepting all that, do they like their pint?'

'Well, yes.'

'So you don't think that helping brewers make grand beer, that people will enjoy, is worthwhile?'

I just looked at him. At that very moment I matured considerably. I realised that my humble place in society's tapestry was not insigni cant, that I did have a worthwhile role to play, and that there was no shame associated with the work that I was doing on a topic that, admittedly, I found to be fascinating.

Dr Hudson wasn't nished.

'Don't forget, lad, that beer has long since been important to the diet of some people. It gives them energy, vitamins, minerals. It soothes them. Don't knock it.'

Hudson was a wise man. Irascible for sure, but a man who loved beer in every respect and would have nobody badmouth it.

I, his young protégé, was certainly receptive to the fact that beer could actually be a worthy part of the diet. And, for a number of years prior to the conversation in question, it had formed a prominent part of my social activity, as it did for a great many young folk in late sixties and early seventies England.

I had my own clear appreciation of the merits and de-merits of alcohol consumption. As a young biochemistry student at the University of Hull, who worked ludicrously hard during the week, I looked forward eagerly to the weekend when my buddies and I would make for old town Hull and its plethora of outstanding pubs.

Sometimes I made a complete fool of myself. My conscience will not allow me to deny the fact that, from time to time, I imbibed to excess. It didn't take me long to learn the lesson, however, that this was disadvantageous, not least from the unpleasantness of the day after. Before long, though, I had come to understand the pleasure that is to be had from taking one's beer steadily and in moderation – a pint or two daily. It tasted good. It complemented the food I was taking, whether a sandwich, a curry or just a bag of crisps. It made me mellow and calmed. And, as I usually took the beer in a pub rather than at home, it was a valuable part of a holistic social experience.

For the majority of my beer-drinking life (33 years of cially – and still counting loud and strong), I have never contemplated beer in an overtly dietary manner. It has been taken for pleasure and not as part of a carefully considered diet. Few people would treat it as a foodstuff *per se*. And yet, as you will nd from reading this book, beer is very much a food. It is unreasonable for critics to refer to beer as 'empty calories' and, as we shall see in Chapter 5, it is entirely possible to tally the contribution of calories, bre, vitamins, minerals, and so on from beer alongside those of the other items on the dinner table. Proteins and carbohydrates, but (despite the myth) absolutely *not* fats, are very much a part of beer as they are of bread, meat, vegetables and cereal. Indeed, what is beer if it is not lique ed barley with added value?

As consumers become more and more health conscious and aware of the need for a well-balanced diet, it is not suf cient simply to bracket a product such as beer as 'something for pleasure', as if it was just water and contributing no nutritive quotient. It does kick plenty, in various ways, and people need to be aware of the extent of this and how it impacts the rest of their intake. It would never be my intention to advocate beer as an inherent substitute for any other component of the diet. It seems entirely logical, though, to include beer amongst the diverse other items on the menu in the ready reckoning exercise and even to fashion a sustaining and, of course, pleasurable meal

that incorporates a glass of beer. One less slice of bread perhaps? Skipping the stodgy or ludicrously sweet dessert?

The supermarket shelves are loaded with diverse choices and all manner of foodstuffs – forti ed with this or that, low calorie variants, 'organic', etc., etc. Beer is no different – except that there is no overt forti cation going on, rather the inherent components such as vitamins and minerals that can be in quite useful quantities.

What, I wonder though, would people say if I said that beer might as justi ably be located in the medicine cupboard as in the larder? The evidence is mounting that *moderate* consumption of beer (of the order of one to two pints per day) lowers the risk of mortality and morbidity and has a range of bene cial impacts on the body. When my wife was in the maternity ward with our rst born, the drinks trolley included stout alongside the other beverages on offer. It was accepted wisdom that beer is rich in valuable nutrients, as well as offering a soothing impact after an intense emotional and physical experience.

It would be stupid to argue against the fact that drinking alcoholic beverages to excess is dangerous (health-wise and accident-wise) and prone to lead to suffering, both for the imbiber and for those close to them. It is no surprise whatsoever, therefore, that organisations have sprung up with the aim of attacking the alcoholic beverages industry. It is equally unsurprising that those within the industry (and, as a professor whose specialisation is beer, I guess this includes me) should seek to counter such sieges. However, it is important that this is done in a responsible and conscientious manner, and with a rationality that seems to be too frequently lacking from those who decry alcohol.

The producers of alcoholic beverages must position their products for what they are: valuable and positive components of the human diet that should be enjoyed responsibly by adults. They should not be (but, too often, regrettably are) marketed with images of wild and irrational behaviour. And, when arguments for their positive contribution are made this should be done in as balanced and critical way as possible.

Would that those who oppose alcoholic beverages take the same approach in considering *all* the evidence. Perhaps then more of them might come to accept that, taken wisely and temperately, beer and other alcoholic beverages are a worthy component of society. The vast majority of people who take a beer are not drunken drivers, wife beaters, football hooligans, panhandlers or, above all, alcoholics. And neither will they go on to become these things. Certainly, excessive alcohol intake can reduce inhibitions that could increase the likelihood that a football yob will wreak havoc. However, it's not the alcohol, any more or less than the game of soccer itself, that has made the thug what he is.

Drinking of alcohol, including as beer, is so often an integral feature of social occasions for adults. As Gus eld (1987) says, a drink is a signal for an important change of pace or venue.

So, what is someone who has been employed either in the brewing industry or as a professor teaching its science and technology for a quarter of a century doing writing this book? Is it, as some will undoubtedly say, an exercise in self serving, an unashamed piece of biased lobbying to tout one's favourite beverage? I have very little doubt that the anti-alcohol lobby will come to that conclusion. With just as much vehemence, I would refute the inference. I must stress, too, that I have neither been commissioned to write this book nor am I directly paid by any brewing company. This volume seeks to discuss beer in a warts-and-all context. I have certainly not fought shy of discussing any of the adverse impacts that excessive consumption of alcoholic beverages can have.

I was driven to write the book by several forces:

(1) To consider dispassionately the role of beer in the human diet now and through history, as an exercise in scholarship.
(2) To consider the impact that beer (as part of the spectrum of alcoholic beverages) has on health, in an era when the average person has probably never been more conscious of, and concerned about, the state of their well-being.
(3) To redress the balance about the relative worth of beer and wine as bene cial parts of the diet.

It seems to me that those writing on the topic from within (or closely associated with) the alcoholic drinks industry tend to cover both the positive and negative aspects of alcohol. By contrast, those writing from the opposing stance seldom do other than consider the consumption of alcoholic beverages as entirely negative.

I believe that there is a key need for education, to present facts as we know them (and as they emerge consequent to state-of-the-art research) and not to shy away from any facet of the debate. In a class I teach to students of all ages on the Davis campus of the University of California we endeavour to do just this. I bring in guest speakers from breweries but also expose the students to medical experts able to articulate the perils of taking alcohol to excess. Some of the images can be quite gruesome. We want the students to understand, to nd themselves in this con icting arena.

For after all, is not a maxim from the Temple of Apollo at Delphi (Braun 1996), 'Know thyself and nothing to excess'?

Acknowledgements

Thanks to Lou Grivetti for hepful discussions. I am grateful to David Long for providing valuable statistical data and Jaime Jurado for making available analytical data on beers. As ever, I appreciate my wife Diane for her patience and support.

1 Beer as Part of the Diet

Beer has been drunk for more than 6000 years, from the time that it was first made by happenstance in the middle age of ancient times (Bamforth 2003). Ever since, it has become a staple part of the diet in many cultures. Furthermore, it has not only comprised a valuable addition to the table, but has served various medicinal roles, including mouthwash, enema, vaginal douche and applicant to wounds (Darby *et al.* 1977).

Beer (and other forms of alcohol) differs in its significance, acceptability and importance from culture to culture. At one extreme the prophet Mohammed forbade his followers to drink alcohol, thereby establishing a point of difference from Christianity. The *Koran* speaks of alcohol as being an 'abomination and the work of Satan' (5: 90). Conversely, the Kofyar of northern Nigeria believe that 'man's way to god is with beer in hand' (Netting 1964). In the Aztec nation, religious worshippers were obliged to get drunk for fear of displeasing the gods (Thompson 1940). In India, the various deities demand different approaches to the use of alcohol. Indeed, in some areas of India, alcohol is replaced by infusions of hashish (Carstairs 1957). What better illustration might one use to stress the need for tolerance of others' customs and beliefs and of what is or is not acceptable?

Mandelbaum, in discussing the Tiriki of Kenya, observes:

> Beer is a constant medium of social interchange for men; beer drinking is a preoccupying activity that few men reject. Drinking beer together induces physical and social mellowness in men. Very little aggressive behaviour is ever shown as a result of drinking, and that little is promptly squelched. Pathological addiction rarely, if ever, occurs.
>
> Mandelbaum (1979)

This thought-provoking view surely reminds us that we should view the consumption of beer (and other alcoholic beverages) from a holistic standpoint.

The historical importance within society of beer (and other alcoholic beverages, such as wine in climates where grapes could be grown) is illustrated by the argument that nomadic tribes gravitated to crop farming and organised communities in order to ensure a constant supply of beverages (Kendell 1987).

In many cultures, especially those of Northern Europe, beer was through generations the staple drink for the whole family, young and old. At least in part this was on account of beer being safer to drink than water in days when there were no water puri cation systems. The ale, after all, had been through a boiling stage, whereas the local supply of water had not. The ale tasted better too. Cesar de Saussure, a Swiss writing in 1720 (see de Saussure 1902), found in London that:

> Though water is to be had in abundance in London, and of fairly good quality, absolutely none is drunk. In this country beer is what everybody drinks when thirsty.

The early settlers in Virginia fell sick for want of ale, on account of the local infected water that they were obliged to drink. One of the rst settlers, Richard Ffrethorne, bemoaned the lack of any creature comforts, bitter that back in England folk were healthy on their strong ale whereas here there was only water to drink (Kingsbury 1906–1935).

It was only with the development of cleaner water and the advent of tea and coffee drinking in the seventeenth century that beer in countries such as Great Britain progressively shifted away from being the staple beverage at mealtimes for all members of the family unit, and became more of a luxury item.

Yet there remain cultures, notably the Czech Republic and Germany, where the consumption of beer to accompany a meal remains a key feature of the diet, which is re ected in the per capita consumption gures (Table 1.1).

Beer: a vice or a staple part of the diet?

Were we able to transport ourselves back to the Middle Ages and enquire in England, Flanders, Bavaria or Bohemia about the key features of the popular diet, ale or beer would unquestioningly and unhesitatingly be listed alongside meat, bread, milk and vegetables. The questioner would be regarded as being mightily peculiar if he or she were to question ale's legitimate place on the table. It was neither a comfort food nor an extravagance. It was an integral part of the food intake in all walks of society. In eighth-century England a monk might consume eight pints of ale a day.

Beer in Britain has long been considered to be a key part of the diet, as much so as wine in France. Henry Brougham MP (Brougham 1830) said that 'To the poor the beer is next to a necessity of life.'

Over 50 years ago the nutritive value of beer was emphasised. An admittedly weakish beer [3% alcohol by volume (ABV) in the austere early post-war years] was claimed to provide 200 calories and a fth of a working man's requirement for calcium, phosphorus,

Table 1.1 Worldwide consumption of beer, 2000.

Country	Consumption (litres per head)
Argentina	32.7
Australia	90.0
Austria	107.0
Belgium*	98.3
Brazil	48.2
Bulgaria	51.0
Canada	67.4
Chile	27.5
China	17.3
Colombia	32.7
Croatia	86.2
Cuba	20.3
Czech Republic	158.9
Denmark	98.6
Finland	80.2
France	35.9
Germany	123.1
Greece	39.0
Hungary	73.0
Ireland	125.0
Italy	28.9
Japan	55.9
Korea (Republic of)	35.5
Mexico	48.3
New Zealand	79.5
Netherlands	80.5
Nigeria	5.6
Norway	52.0
Peru	22.8
Philippines	15.9
Poland	62.8
Portugal	61.3
Romania	55.4
Russia	37.9
Slovak Republic	87.1
Slovenia	92.0
South Africa	53.8
Spain	72.0
Sweden	56.4
Switzerland	58.3
Ukraine	21.1
UK	95.4
USA	82.4
Venezuela	76.0

*Includes Luxembourg, because of inaccuracies introduced by cross-border trading.
Source: Tighe (2002).

nicotinic acid and riboflavin (Bunker 1947). The satisfaction of having at least part of one's dietary intake in a pleasurable form was not sneered at then.

Perhaps the first person to conduct a serious study of the impact of abstinence, moderation and excessive drinking on health was statistician Raymond Pearl. On the basis of interviews with over 2000 workers in Baltimore, he concluded almost 80 years ago that on average moderate drinkers lived longer than abstainers and much longer than those who were heavy drinkers (Pearl 1926).

Yet now, at the dawn of the twenty-first century, beer-drinking is regarded in many societies as a vice. It is surely astonishing that in the United States it is possible to buy cigarettes at the age of 18, but it is not legal to purchase alcohol until the age of 21. It would be a struggle to identify any merit associated with smoking, with the possible exception of its role as an anxiety relaxant. By contrast there is accumulating evidence that alcohol, including beer, *in moderation* can have a beneficial impact on health and wellbeing.

In passing, let us consider the legal age at which, in the US, it is possible to partake of other activities that surely might be considered a genuine risk to health and wellbeing, not only for the partaker but also for those around them. A child may legally drive a car, with relatively few restrictions, at the age of 16. More alarmingly, 35 states in the US have no licensing or registration requirements for guns (www.soros.org/crime/higlights.htm). Seven states lack a legal minimum age for buying a rifle or shotgun from an unlicensed dealer, while six states have no legal minimum age for a child to possess a handgun. In five states there is a minimum age – 16 in New York, Georgia, Vermont and Alaska, and just 14 in Montana. But the minimum legal age for drinking alcohol in all 50 states is 21!

Opinions about the relative merits and de-merits of smoking, driving, guns and alcohol will of course differ between individuals. Certainly if we consider the respective virtues of smoking, weapon use and alcohol (in restraint), then it seems to this author that there may be a warped set of priorities in one country at least. Nonetheless beer is the second most popular drink in the United States, with annual average per capita consumption at 357 8-ounce servings, after sodas and other soft drinks (861) (*Beverage Digest* 1998). Worldwide production of beer in 1999 ran at 0.13 billion litres.

It seems that we have lost sight of the real benefits of a foodstuff such as beer (and it *is* a foodstuff, as we will explore in Chapter 5) for the body and for overall wellbeing. P.G. Wodehouse, in *The Inimitable Jeeves*, wrote: 'It was my Uncle George who discovered that alcohol was a food well in advance of modern medical thought.'

In *Pearson's Weekly* (a rival to *Tit-Bits* and founded in 1890 by Sir Arthur Pearson, who went on to create the *Daily Express*), Bass Ale received the following testimonial:

> An old friend of mine, Colonel Worsley CB, when in India, had a very dangerous attack of dysentery and was given up by the doctors. When dying as it was thought,

> he begged the man in a faint whisper to give him some Bass and as it was thought his case was hopeless he was humoured. He then drank pint after pint and began to get better as soon as his yearning was satis ed much to the astonishment of the doctors and brother of cers.

Despite the fact that once upon a time I was research manager with Bass, I can't believe that there was anything magical about Bass Ale to make it superior in the context quoted as compared to any other beer. I remain open-minded about the veracity of the report, and about the likelihood of a causal link between Worsley's wellbeing and the consumption of beer.

The claims for Bass have been various. Doctors in its town of origin, Burton-on-Trent, are said to have recommended it as a laxative. Writing in *The Times*, Dr Mapother recommended Bass as a cure for gout. It is claimed that Bass cured Edward VII, when Prince of Wales, of typhoid. Perhaps this stimulated the music-hall song that ran

> I've tasted hock and claret too, Madeira and Moselle
> But not one of those boshy wines revives this languid swell
> Of all complaints from A to Z the fact is very clear
> There's no disease but what's been cured by Bass's Bitter Beer.

Remarkable testimony! But Bass isn't the only brand to have been championed in this way. 1928 saw Guinness launch the slogan *Guinness is Good for You*, and followed it with such as *My Goodness, My Guinness* and *Guinness for Strength* (Fig. 1.1).

The sweet stout, Mackeson, was marketed in the 1950s on a slogan of:

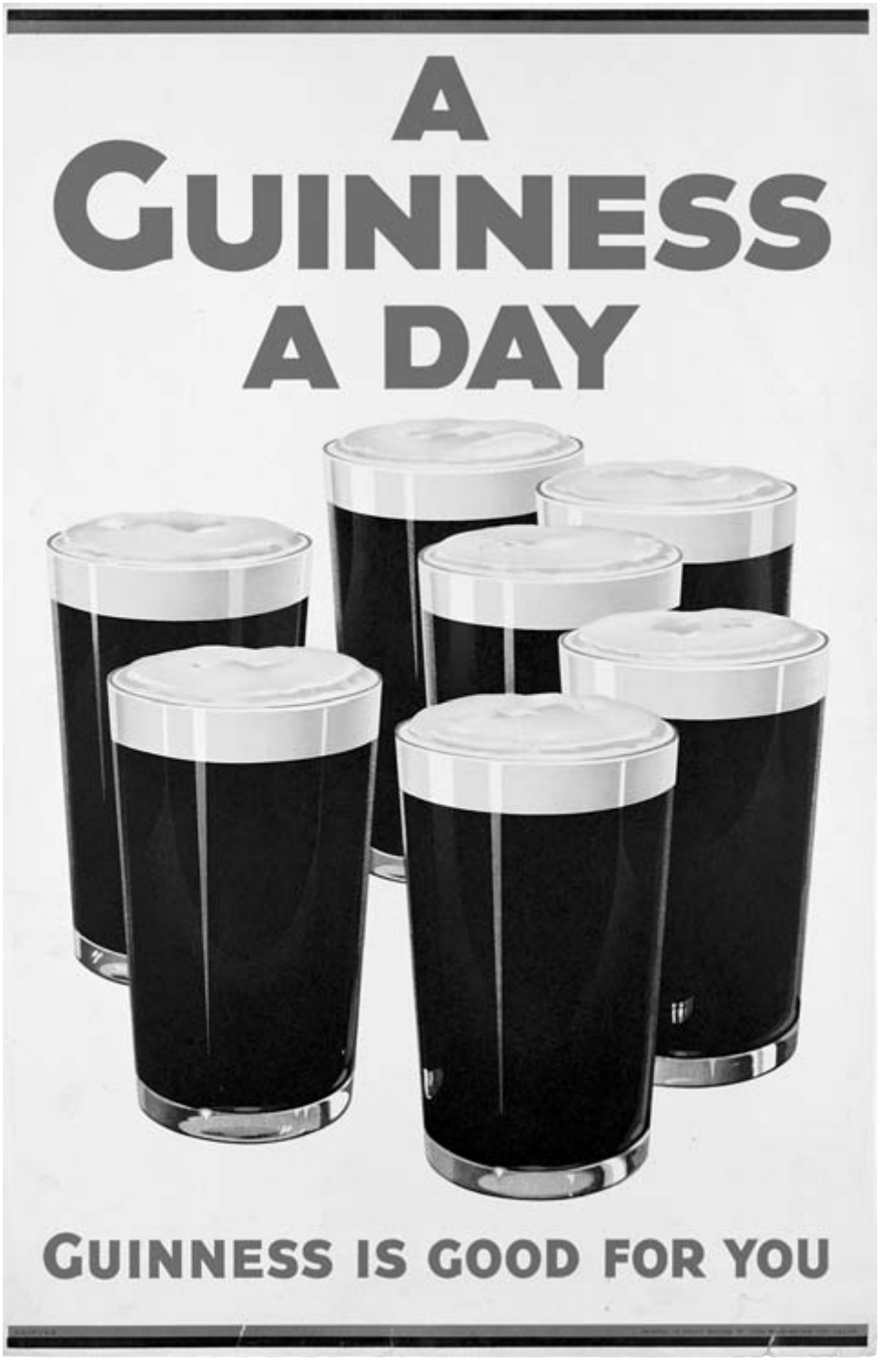
> It looks good, it tastes good,
> And, by golly, it does you good.

Nursing mothers were expected to enjoy a daily bottle of stout.

Those were the days when some governments were not hesitant to see the virtues that beer had as a social cement and catalyst of contentment. As Queen Victoria had said rather earlier: 'Give my people plenty of beer, good beer and cheap beer, and you will have no revolution among them.'

The British government in the middle of the last century was totally happy to see the trade association The Brewers Society champion their members' products with generic messages including *For Bodily Health – Beer is Best* and *To Set A Man up for Winter – Beer is Best* and *For an A1 People – Beer is Best* (Fig. 1.2).

Predictably, the temperance lobby countered with *Beer is Best Left Alone*.

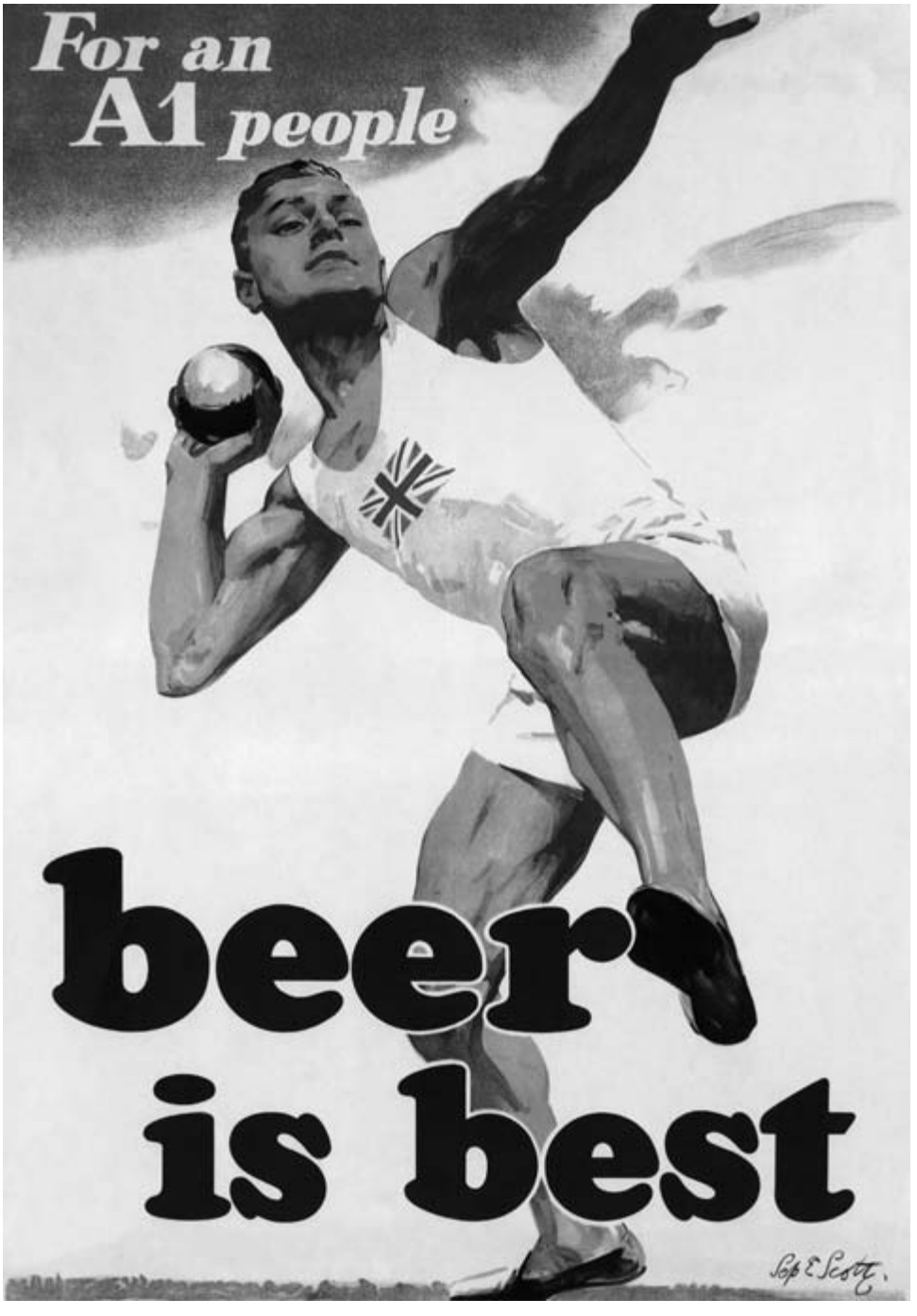


(a)

Fig. 1.1 Marketing slogans from Guinness. (a) Poster from 1932. The seven pints represented both the days of the week and the seven beneficial reasons for drinking Guinness: 'strength, nerves, digestion, exhaustion, sleeplessness, its tonic effects and for the blood'. (b) Poster from 1945. The Ministry of Information's 'Dig for Victory' slogan was adapted and integrated into the 'Guinness for Strength' campaign. The GUINNESS

(b)

word, HARP device and ARTHUR GUINNESS signature are trade marks and are reproduced together with the 'Poster from 1932' and 'Poster from 1945' advertisements with the kind permission of Guinness & Co. © Guinness & Co. All Rights Reserved. The 'GUINNESS IS GOOD FOR YOU' advertising campaign dates from the 1930s to 1960s and has not featured in subsequent campaigns to advertise GUINNESS™ beer.



(a)

Fig. 1.2 (a)–(e) Marketing slogans from the Brewers Society. Reproduced courtesy of the British Beer & Pub Association (formerly The Brewers Society).

(b)

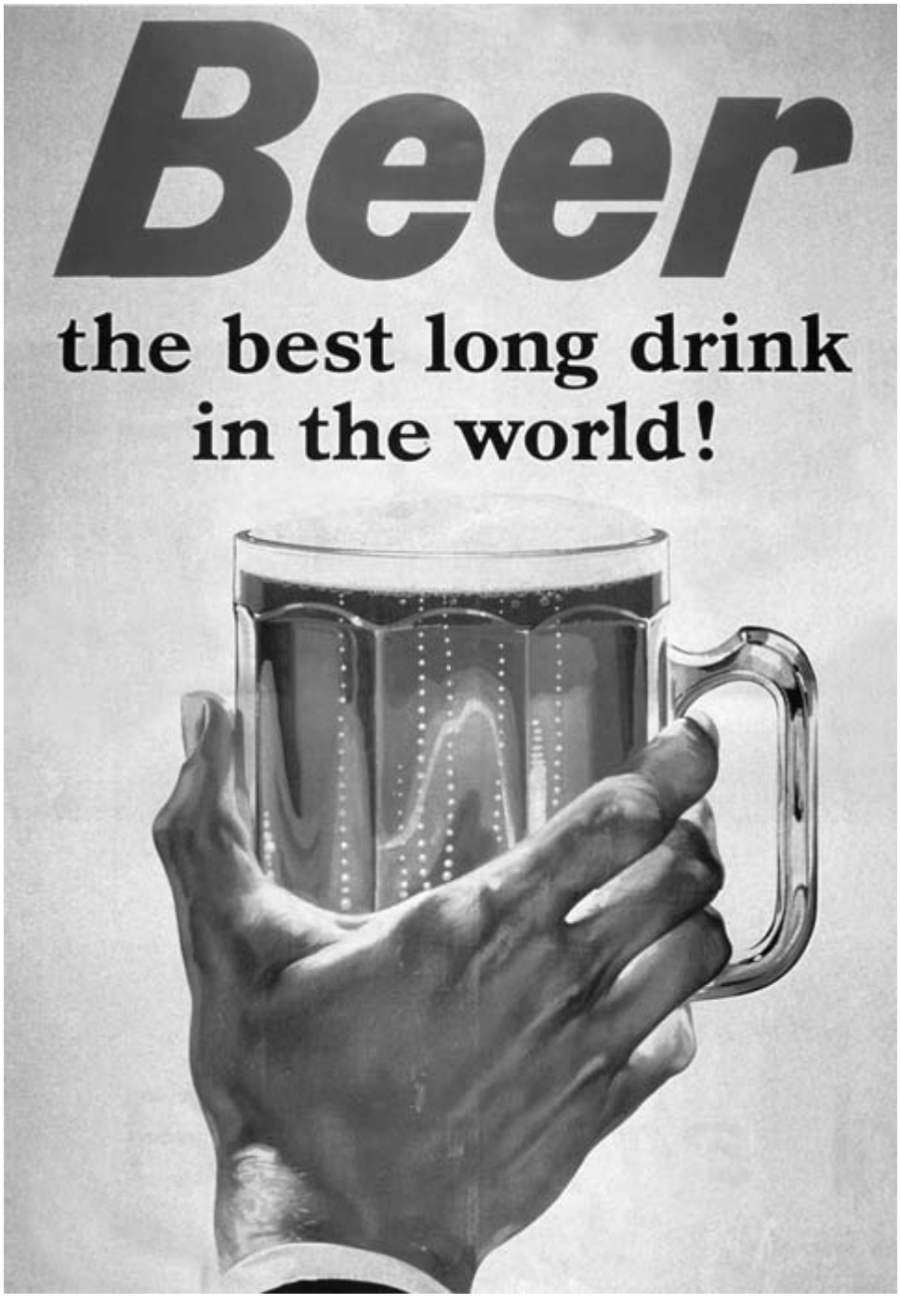


Fig. 1.2 (*Continued*.)

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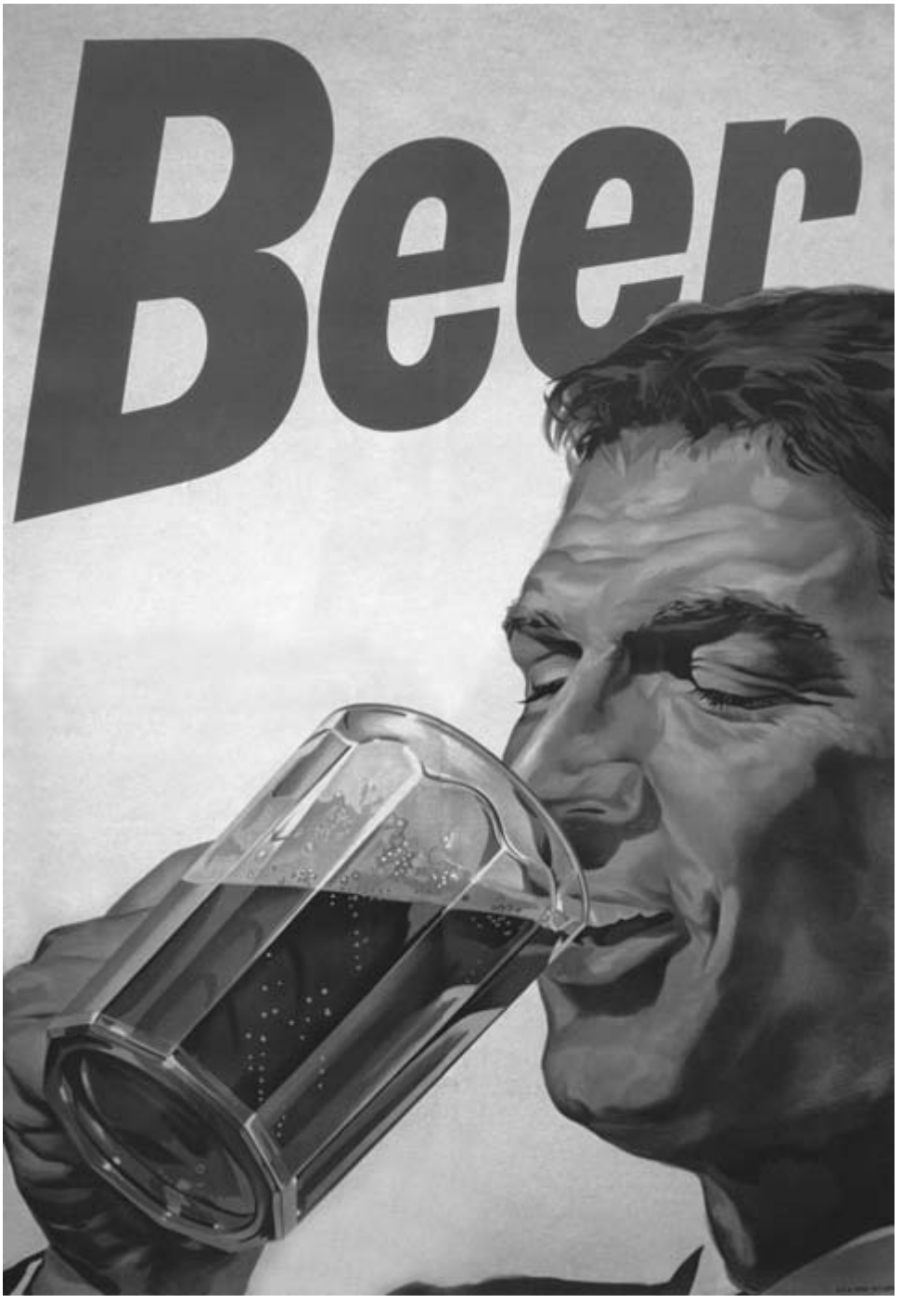

Fig. 1.2 (*Continued*.)

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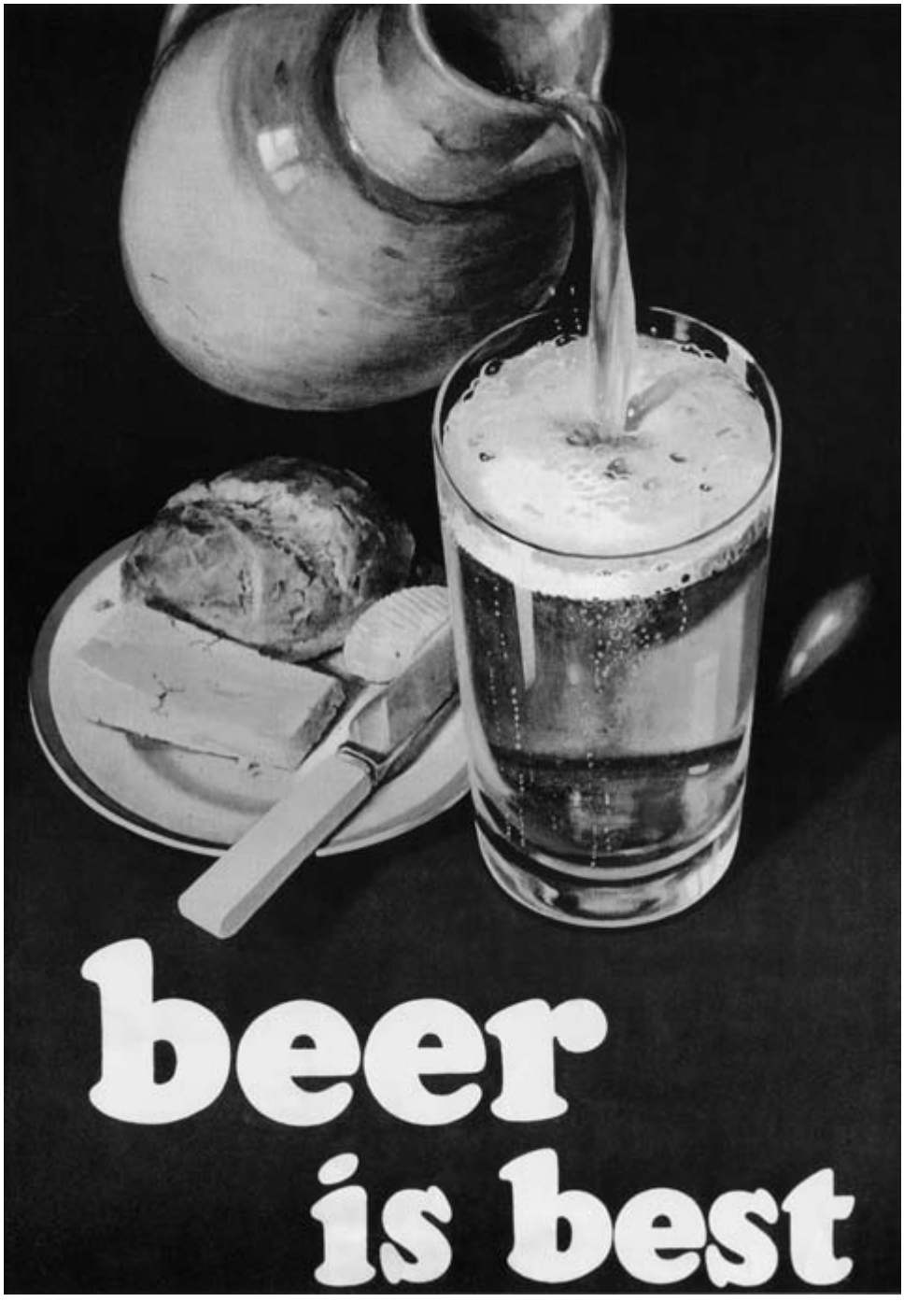

Fig. 1.2 (*Continued*.)

(e)

Fig. 1.2 (*Continued*.)

This type of campaigning by the Brewers Society stressed the social element of beer as much as anything. There was scienti c understanding of the composition of beer and brewers realised that it could make a contribution to dietary intake of various key components, as you would expect from 'just another' foodstuff. *The Brewer's Journal* in 1939 reported (on the basis of a study by the Royal Society) that a barrel of beer was the equivalent in cumulative nutritive value of 10 pounds of beef ribs, 8 pounds of shoulder mutton, 4 pounds of cheese, 20 pounds of potatoes, 1 pound of rump steak, 3 pounds of rabbit, 3 pounds of plaice, 8 pounds of bread, 3 pounds of butter, 6 pounds of chicken and 19 eggs (Glover 2003). At that time the body of evidence was not available that now indicates that the moderate intake of beer has a clear impact in preventing certain diseases.

Getting beer into perspective

As my friend and colleague, Michael Lewis, is wont to say: 'There is nothing so disgusting as a drunken brewer.' I would go further, for the state of drunkenness is neither pretty nor conscionable in anybody. It is socially unacceptable, ugly and dangerous.

Stuttaford (1997) tells of how medical students memorise the various stages of drunkenness: 'dry and decent, delighted and devilish, delinquent and disgusting, dizzy and delirious, dazed and dejected, and dead drunk'.

Excessive consumption of alcohol can be fatal. At the very least it can lead to an unfortunate lack of inhibitions. Most extensively publicised of course are the incidences of drunken driving. There is no question that consumption of alcohol and driving do not mix. The legally permitted levels of alcohol consumption vary considerably between countries (Table 1.2). The safest option is to avoid alcohol completely when intending to drive. Interestingly, alcohol appears to play a part in 15% of fatal crashes in the UK where the legal drinking age is 18, but more than 30% in the US where the legal drinking age is 21 (Barr 1999).

There is ample evidence that drinking any alcoholic beverage to excess is harmful (Table 1.3). However, so too is the overconsumption of any dietary component or the pursuit of many activities to excess.

It is a fact that drunkenness has been around for millennia (Roueche 1960). The Chinese *Shu Ching* from about 650 BC said that:

> Men will not do without *kiu* (a beer made from millet or rice). To prohibit it and secure total abstinence from it is beyond the power even of sages. Here, therefore, we have warnings on the abuse of it.

The Mongolian chief, Genghis Khan, stated:

> A soldier must not get drunk oftener than once a week. It would, of course, be better if he did not get drunk at all, but one should not expect the impossible.

Table 1.2 Legal limits for blood alcohol content of drivers.

Country	Limit (mg/mL)	Country	Limit (mg/mL)
Albania	0.1	Lithuania	0.4
Argentina	0.5	Luxembourg	0.8
Armenia	0	Malta	0.8
Australia	0.5	Moldova	0.3
Austria	0.5	The Netherlands	0.5
Azerbaijan	0	New Zealand	0.8
Belarus	0.5	Norway	0.2
Belgium	0.5	Peru	0.5
Bosnia and Herzegovina	0.5	Poland	0.5
Bulgaria	0.5	Portugal	0.5
Canada	0.8	Romania	0
Croatia (Republic of)	0.5	Russia	'drunkenness'
Czech Republic	0	Singapore	0.8
Denmark	0.5	Slovak Republic	0
Estonia	0	Slovenia	0.5
Finland	0.5	South Africa	0.5
France	0.5	South Korea	0.5
Georgia	0.3	Spain	0.5
Germany	0.5	Sweden	0.2
Greece	0.5	Switzerland	0.8
Hungary	0	Thailand	0.5
Iceland	0.5	Turkey	0.5
Ireland	0.8	Turkmenistan	0.3
Israel	0.5	United Kingdom	0.8
Italy	0.5	United States	0.8
Kyrgyzstan	0	Zimbabwe	0.8
Latvia	0.5		

Source: International Center for Alcohol Policies (2002).

Table 1.3 Harmful effects of alcohol.

- Traf c accidents, falls, drowning
- Nervous system: cerebral, cerebellar, brain stem degeneration; optic atrophy; polyneuropathy; pellagra
- Digestive system: hepatitis; fatty degeneration of liver; cirrhosis; pancreatitis; peptic ulcer
- Cancers: mouth, pharynx, larynx, oesophagus, liver, colon (?), breast (?)
- Cardiomyopathy, hypertension
- Myopathy, porphyria, fetal alcohol syndrome

Source: Bamforth (2002).

Such pragmatic approaches sit uncomfortably with a good many people. However, in a mature and far-sighted society, it is only by confronting these issues that rational and realistic solutions and practices will emerge.

It is relevant at this point to consider statistics concerning drunkenness and instances of drink-related driving. Table 1.4 highlights that the current situation in the UK is far healthier in respect of all drunkenness offences than 25 years ago. Furthermore, the number of drivers involved in accidents that register above the legal limit for alcohol

Table 1.4 Drunkenness offenders in the United Kingdom.

Year	Rate per 10,000 people
1964	15.9
1965	14.9
1966	14.9
1967	15.2
1968	15.9
1969	15.9
1970	16.3
1971	17.0
1972	17.9
1973	19.8
1974	20.2
1975	20.5
1976	21.0
1977	20.7
1978	20.3
1979	21.4
1980	22.1
1981	19.4
1982	19.0
1983	18.9
1984	15.7
1985	14.3
1986	12.6
1987	15.2
1988	17.1
1989	16.7
1990	15.5
1991	13.5
1992	12.2
1993	10.6
1994	10.2
1995	7.5
1996	8.7
1997	9.4
1998	9.2
1999	8.3
2000 (estimate)	7.7

Source: Tighe (2002).

has remained around 2% of the total number involved in accidents since 1990 and is half the level of 25 years ago (Table 1.5). Incidentally, *Skynet Webmagazine* in May 2002 reported how the use of a mobile telephone (even a hands-free phone) presented a greater risk during driving than the consumption of up to two drinks. This should not be construed as an acceptance of even moderate alcohol consumption before driving – zero intake will always be the best option for those intent on such an activity – but

Table 1.5 Results of breath tests on car drivers involved in accidents in the United Kingdom.

Year	Percentage of drivers in accidents that were tested positive in a breath test
1969	1.7
1970	2.2
1971	3.0
1972	3.4
1973	4.0
1974	4.8
1975	4.8
1976	3.8
1977	3.6
1978	3.8
1979	4.3
1980	4.1
1981	3.8
1982	4.0
1983	3.9
1984	3.7
1985	3.7
1986	3.4
1987	3.2
1988	2.8
1989	2.6
1990	2.4
1991	2.4
1992	2.2
1993	2.0
1994	2.0
1995	2.1
1996	2.2
1997	2.1
1998	2.0
1999	2.0
2000	2.2

Source: Tighe (2002).

rather highlights that there are other even more potent dangers that do not attract the same focus or emotion.

Alcohol, though, raises passions to an extent wholly unlike most other components of the diet. It is anathema to some that a broad kirk within the world of medicine should be alerting society to the *bene ts* to be had from including alcohol in the diet *in moderation*. Moderation should surely be the byword for *all* parts of our menu.

Lord D'Abernon, editor of the early twentieth-century study championed by the British Medical Research Council, *Alcohol: Its Action on the Human Organism*, exclaimed:

> Alcohol is an ungrateful subject. Most people who are interested in the subject are already partisans on the one side or the other, and no body of impartial opinion exists which is ready to be guided by scienti c inquiry. The majority of those who would give any attention to original work on the subject would do so less to gain knowledge than to nd arms and argument to support their preconceived opinion.
>
> Roueche (1960)

In Chapter 6, I present the published facts about the negative impact of excessive alcohol consumption on health. However I do the same with the claimed bene ts of alcohol, especially beer. I hope I have been impartial.

In a speech to the National Press Club on 10 June 1991, Dr Arthur Klatsky, head of cardiology at the Kaiser Permanente Hospital in Oakland, California, said:

> Current evidence about lighter drinking and health suggests that:
> (1) The case is now quite strong that, for persons, at risk of coronary heart disease, there is an optimal amount, not just a safe amount of drinking.
> (2) This bene t of alcohol operates by reducing the risk of the commonest kind of heart disease – coronary heart disease.
> (3) We cannot yet de ne precisely the optimal amount of alcohol but that it is below 3 drinks per day.
> (4) It doesn't seem to matter what type of alcoholic beverage is taken.

Subsequent research from Klatsky's laboratory and various other researchers have re ned these statements, but their fundamental accuracy is unchanged.

Another major player in the eld has been Dr Norman Kaplan from the University of Texas Southwestern Medical Centre, who wrote in the *American Heart Journal*:

> I nd nothing wrong or unhealthy about my current practice – a beer or two after a heavy tennis game or a glass or two of wine after dinner…
>
> One last argument sometimes used against all alcohol consumption is that, even if moderate alcohol consumption is healthy, physicians cannot condone it because this condones heavier use and may even encourage those who now drink in moderation to become addicted abusers.
>
> To this I say 'baloney'.
>
> Kaplan (1991)

Dr Kaplan certainly hits the nail on the head, for there are so many who cannot seem to recognise that it is possible, indeed the norm, to consume alcohol in moderation. It is no more reasonable to advocate the elimination of alcohol consumption than it would be to lobby for the elimination of football because some people deliberately set out to

critically injure opponents, or the avoidance of prescription medicines because some people overdose.

Aspirin in regular small doses is a lifesaver. In excess it can be a killer. The same applies to alcohol. A dear friend of mine takes an aspirin a day to counter the risk of heart disease. He is roundly applauded for his conscientiousness and it is implicit that he would never take more than his prescribed ration. The evidence is increasing that a pint or two of beer per day may be just as ef cacious. Rather fewer people would approve if he swapped his aspirin for the beer, despite the fact that the beer has nutritional value absent in the aspirin. And, whereas everyone will naturally assume that he will know not to get heavy-handed with the aspirin, some will just as automatically assume that he won't know when to put down the bottle of booze.

Preventing reasonable-minded folk from drinking to their customary moderation is just as illogical as banning chocolate because some people pig out on it, or dispensing with kitchen knives because there is an occasional person predisposed to insert them into friends and neighbours.

What is moderation?

It is common for those writing on the topic of alcohol and health to refer to 'moderation'. What is it exactly?

In the *Second Special Report to Congress on Alcohol and Health* from the National Institute on Alcohol and Health in 1974, some de nitions of drinking habits were given:

- *Moderate occasional.* People who drink alcohol only in small amounts at any one time, never enough to become intoxicated and less frequently than daily
- *Moderate.* Same, except daily
- *Heavy occasional.* People who get drunk occasionally, with periods of abstinence or moderation
- *Heavy.* People who get drunk regularly and frequently

This is a general classi cation, but it still doesn't give any precise quanti cation. Most people would consider moderation to equate to one or two glasses of beer or wine per day. Even then, what a German consuming steins of lager or a Frenchman enjoying a bottle of wine daily would consider to be moderate might be considered to be excessive by those of other nationalities.

The World Health Organization suggests that 60 grams of alcohol per day should be a maximum. For a beer of 5% alcohol by volume, which equates to approximately 4% alcohol by weight, this means 1.5 litres, or a little over two and a half pints.

In his commendably balanced book, Stuttaford makes these perspicacious comments:

> Approximately 90 per cent of men and 80 per cent of women in this country [United Kingdom] enjoy drinking alcohol from time to time. Only a tiny fraction drink to excess; few would ever fall down any steps … and a couple of pints two or three nights a week will not turn most people into drunken hooligans. Opponents of drinking are selective in their reporting: they seize upon the disasters which overtake the minority who drink too much and draw conclusions from their behaviour and health which are then applied to the population as a whole. This way of generating statistics is unsound, and their misleading of the public is unjusti able. The medical advantages of alcohol have been hidden from the general public for thirty years, and the reason usually advanced for this obfuscation is the patronising one that alcohol, delightful as it is to take and good as it is for the heart, cannot be trusted to the masses lest they drink themselves to death.
>
> Stuttaford (1997)

If such is the case in the UK, then it is writ ten-fold larger in the US. Recently a colleague 'confessed' that he and his wife enjoyed a drink, but never in front of the children, for fear of giving them the wrong impression. The impact of this hypocritical behaviour is to persuade the younger element that drinking is some mysterious and hidden pleasure, a 'forbidden fruit'. Perhaps it is not to be wondered at that when a student here reaches the legal drinking age of 21 they too frequently succumb to the temptations of the drinking ritual, sometimes with such devastating consequences. Such ceremonies have generally involved the consumption of spirits, perhaps doubles, to match the number of years on the planet. Alas, too often they do not reach their next birthday. To consume that amount of alcohol in the form of beer would be virtually impossible on a volume basis, but that is not my point. Rather it seems to me that beer and other alcoholic beverages should be associated with messages of responsibility for the good that they can deliver when used in moderation – and not swept under the carpet.

Professor Pelc, a psychiatry lecturer at a leading Belgian university, was quoted in *Le Journal de Brasserie* (December 2002, page 17) as saying that banning the consumption of alcoholic beverages by young people actually increases the risk of harmfully excessive alcohol consumption and of criminal or other antisocial behaviour. He is said to have advocated what is surely the practice for many societies worldwide, namely an early introduction to the consumption of moderate quantities of alcohol in the family home as the best way to encourage safe and socially acceptable drinking habits. Y. Boes, writing in the same journal (page 7) suggests that traditional Belgian low-alcohol beer (*biere de table*) is healthier for children than cola or lemonade.

Between 1960 and 1980 there was an annual doubling of the amount of beer and spirits consumed in the US. Perhaps young people were rebelling against laws that restricted consumption of alcohol because of the association of alcohol with vice.

Fortunately there are legislatures that have far-sighted and common-sense attitudes. When the UK government freed up legislation to allow children to accompany their parents into public houses, the Home Secretary, Kenneth Clarke, suggested it would 'enable children to see people drinking sensibly and perhaps stop them becoming lager louts'. Incidentally, Clarke is by no means the only Member of Parliament to be favourably disposed to beer. In 1975 the then Prime Minister, Harold Wilson, having converted from whisky to beer, said: 'Contrary to all medical opinion, I've lost a lot of weight since I began drinking more beer. In fact, I've lost a stone in only a year.'

That prosaic Christian chronicler C.S. Lewis had written, rather earlier: 'The sun looks down on nothing half so good as a household laughing together over a meal, or two friends talking over a pint of beer.'

Wechsler and Isaac in 1989 produced evidence to show that the raising of the legal drinking age in the US from 18 to 21 had led to an increase in episodes of drunkenness from 25% up to 41% for men and from 14% to 37% for women (Wechsler & Isaac 1992). It seemed that the impact was a polarisation of drinking habits, with a disappearance of moderate consumers: students either drank not at all or to excess.

But what about addiction?

So many fear addiction. The former First Lady, Betty Ford, said in 1991 that alcohol was the number one addictive drug in the US. Yet the fact is that by far the majority of people who enjoy alcohol don't feel a compulsion to drink and don't suffer from withdrawal, which are the markers of an addictive drug. Roueche (1960) quotes a reformed alcoholic, the Reverend Ralph S. Pfau, in differentiating between a drunkard and an alcoholic: 'The drunkard drinks because he wants to. The alcoholic drinks because he has to.'

This is an important difference. As Harold Lovell, erstwhile clinical professor of neurology at the New York Medical College, said:

> Alcoholism is a condition characterised by uncontrolled, compulsive drinking. An alcoholic is *impelled* to drink against his will or judgement, even if will or judgement are functioning.

Stanton Peele (1985) says: 'Addiction may occur with any potent experience.' Orford (1985) reminds us that compulsive gambling, extremes of sexual behaviour and overeating are all addictions. Might we add to these watching television, shoplifting, sur ng the Internet, shopping (notably by credit card) and work?

Alcohol is less addictive than caffeine. It was shown by Strain *et al*. (1984) that caffeine in coffee, tea and cola induced all of the features of psychoactive dependence, including the continued use of the material despite side effects which include anxiety, sleeplessness and gastrointestinal dif culties, as well as the displaying of withdrawal symptoms. True, it is possible to get caffeine-free versions of this type of drink – but the 'fully charged' versions of each hardly attract the same attention as alcohol in the legislature. The words 'alcohol' and 'drug' are linked in the public consciousness, not so 'coffee' or 'cola' and 'drug'.

Those over-imbibing sodas or coffee would seldom be considered generally to have a disease. Yet the majority of the public would consider alcoholism to be a disease. The medical profession (Room 1983; Fingarette 1988) no longer holds this view.

The concept of alcoholism as a disease was rst propounded in the late 1930s (Mann 1950; Jellinek 1960). The argument is that certain people are vulnerable to alcohol and will develop the disease if they start to drink. Progressively they will consume ever-increasing amounts and suffer a range of symptoms, including amnesia and blackouts, and lose control over their ability to say yes or no to another drink. There is no alternative for such a person but to abstain.

It seems, however, that there is considerable scepticism about the disease concept (Kissin 1983). As Marlatt (1983) says: 'There is no adequate empirical substantiation for the basic tenets of the classic disease concept of alcoholism.'

There is a realisation that the tendency for some to abuse alcohol is little different to other forms of compulsive behaviour, such as addictions to drugs, cigarettes, gambling, shopping and caffeine. Peele (1985) embraces all these forms of 'excessive appetite' into a 'unitary theory'.

Jellinek (1960) largely de ned the concept of alcoholism as a disease. Fingarette (1988) detailed the various aws in Jellinek's approach, to the extent of pointing out that Jellinek himself questioned the adequacy of his techniques:

> In sum, Jellinek's highly in uential articles were based on questionnaires completed by 98 male members of AA (Alcoholics Anonymous). Of the 158 questionnaires returned, Jellinek had eliminated 60, excluding the data from some AA members who had pooled and averaged their answers on a single questionnaire because they shared their newsletter. Jellinek also excluded all questionnaires lled out by women because their answers differed greatly from the men's … Even in 1960, Jellinek acknowledges the lack of any demonstrated scienti c foundation for his proposals.
>
> Fingarette (1988)

There emerged diverse studies to contradict the disease concept, including the observation that those who have undertaken regular bouts of heavy drinking may very

well return to a style of moderate consumption (Clark & Cahalan 1976). The reader is referred to the autobiographical confessions of Jack London in *John Barleycorn* (1913) for a literary example of this. As Schuckit (1984) observes, in any given month half of all alcoholics will be abstinent, with an average of four months being 'dry' in a 1- to 2-year period. Keller (1972) points out that virtually all of the alcoholics that he had encountered said that they could frequently take just 1 to 3 drinks for a period of weeks without any episodes of being unable to stop. Keller observed that if there had been an unavoidable slide towards uncontrolled drinking as a result of simply taking one drink, then that would not explain why an alcoholic would lack the self-control simply to avoid taking that first drink. In other words, the lack of self-control exists *before* the drink is taken.

Several studies have presented powerful evidence that heavy drinkers do possess self-control. Mello and Mendelson (1972) (see also Heather & Robertson 1981) performed an experiment whereby heavy consumers of bourbon were allowed to earn ounces of bourbon in periods of between 5 and 15 minutes in response to their ability and preparedness to partake of simple tasks involving pushing a button according to instructions. Under conditions where they could certainly earn enough bourbon to become intoxicated, none of the subjects attempted to drink to gross excess. In fact they drank to maintain high but approximately constant blood alcohol levels, in spontaneously initiated and terminated sessions over a prolonged period as opposed to continuously. It was also concluded that the amount of alcohol consumed was related to the effort that needed to be exerted to get it – there was a benefit versus cost balance, which lies in the face of the lack of control supposition associated with alcoholism.

In another study it was shown that, when given the choice of more liquor or the ability to remain in a pleasant social environment, alcoholics mostly retrained themselves to moderate drinking (Cohen *et al.* 1971). Pattison *et al.* (1977), in a review of more than 50 clinical studies, drew the conclusion:

> Within a hospital or laboratory environment the drinking of chronic alcoholics is explicitly a function of environmental contingencies.

This must mean either that there is something about non-controlled environments that impacts on drinking behaviours or that properly controlled experiments and observations made out of a clinical or laboratory setting have not been made. If the former is the case, coupled with the observations made on individuals' drinking habits in relation to reward, then this argues for the importance of a range of other motivations for heavy drinking that are not chemical based.

Indeed, a compelling study by Marlatt *et al.* (1973) showed that alcoholics consume beverages in response to what they are directed to believe that those drinks comprise. Thus, if given tonic water alone but told that it contained vodka, the subject consumes

as much of that drink as they do of one that is genuinely a blend. However, if told that a product is pure tonic then, irrespective of whether the sample actually did contain vodka, the alcoholic would drink less of it and certainly no more of the sample that contained alcohol. This type of study ies directly in the face of arguments for a chemical-based rationale for alcoholism.

Fingarette (1988) opines that the retention of the disease concept by some in the medical profession and legislatures is one tactic for securing research funds and ensuring that those who do drink to excess seek help. As Vaillant puts it:

> Calling alcoholism a disease, rather than a behaviour disorder, is a useful device both to persuade the alcoholic to admit his alcoholism and to provide a ticket for admission into the health care system. I willingly concede, however, that alcohol dependence lies on a continuum and that in scienti c terms *behaviour disorder* will often be a happier semantic choice than *disease*.
>
> Vaillant (1983)

Jellinek (1960) himself said that 'A disease is what the medical profession recognises as such.'

The National Institute on Drug Abuse (Galizio & Maisto 1985) considered 43 different theories for what drives alcoholism. Fingarette (1988) says that some of them, at least, must be wrong, and that:

> there is no such single 'disease' and therefore there is no cause. The very proliferation of widely diverging unsupported hypotheses is not characteristic of solid scienti c research. It is characteristic of pseudo-science and faddism.

There are, however, rm adherents to the belief that there is a gene-based inheritance of alcoholism. Studies of relative tendency towards alcoholism in adoptive children and twins have now led to the view that the risk of alcohol dependence is due to the additive or interactive impact of multiple genes (Goodwin *et al.* 1973, 1974; Bohman *et al.* 1981; Hrubec & Omenn 1981; Heath *et al.* 1997; Kendler *et al.* 1997). The question is whether children born to an alcoholic parent and put up for adoption soon after birth show a greater tendency towards alcoholism that those adoptees who were born to non-alcoholic parents. In the work of Goodwin, there were 3.6 times more alcoholic adopted children from alcoholic fathers than from non-alcoholic fathers. It is important to stress, however, that 82% of the adoptees that came from an alcoholic biological father did *not* become alcoholic. This may be because they did not inherit the gene(s) or that there are other impacting factors, including environmental ones. Fingarette (1988) provides a calculation to illustrate that the majority of alcoholics are not born to alcoholic parents. Indeed, in a study analogous to that reported by Goodwin, it was found that daughters

of alcoholic parents were not predisposed to becoming alcoholics; indeed, there were more alcoholic women who did *not* have alcoholic parents (Cahalan *et al.* 1969).

Speci c genes for alcohol dependence have not yet been identi ed; there may be six or so linked to alcohol sensitivity, as well as others determining personality and general predilection towards addiction (Whit eld 2001).

It is believed by some that innate resistance to intoxication increases the risk of alcohol dependence, whereas sensitivity to the impact of alcohol decreases the risk (Whit eld 2001). Seemingly 5–10% of British and Germans and twice as many Swiss have forms of the enzyme, alcohol dehydrogenase, that allow up to 30% faster elimination of alcohol (Marshall & Murray 1989). The concern is that individuals who react less intensely to alcohol may lack the inherent feedback control to prevent the negative impact of higher alcohol intake (Finn *et al.* 1990). Another key factor that limits the extent to which people consume alcohol is its inhibition of the synthesis of glucose in the body (gluconeogenesis). This induces hypoglycaemia (shortage of sugar) and a healthy body should respond by limiting the intake of the inhibitor, i.e. ethanol.

Alcoholism, then, is held by many to run in families (Cotton 1979; Dietrich & Spuhler 1984; Goodwin 1985), with four- fths of male and female alcoholics in treatment possessing at least one close biological relative also displaying alcohol-related problems (Hesselbrock *et al.* 2001). Hesselbrock *et al.* say that the risk of alcoholism among sons of alcoholic fathers is 3–5 times greater than for the general population. It should be appreciated that, while there may be a genetic basis for this inheritance, there may equally be an environmental in uence. This may run in a counter-indicative way; for example (if I may be permitted a qualitative observation), I know several individuals who adopt an extremely abstemious lifestyle having been raised in households where the father has been troubled by abusing alcohol.

Fingarette (1988) amply illustrates how there are undoubtedly diverse causal impacts on individuals' likelihood to take alcohol to excess. There may be no uniformity between people in this respect. While there may be some genetic contribution to the effect, there are those who believe that there may equally be a signi cant contribution of 'learning theory': some people may simply learn to deal with life's dif culties in this way. Fingarette writes:

> There is no one cause of alcoholism; alcohol abuse is the outcome of a range of physical, personal and social characteristics that together predispose a person to drink to excess; and episodes of heavy drinking are triggered by immediate events in a person's life.

We are reminded, too, that there may be an economic impact. It is claimed that there is an inverse relationship between cirrhosis and the price of alcohol (Cook 1984). On this basis some rmly advocate higher taxation of alcohol to reduce alcoholism. For

this to be a legitimate tool inherently assumes that an individual does indeed have total control over their environment, psychiatry, physiology and genome, and will simply not purchase alcohol if it is highly priced. On the other hand, if it is accepted that there are individuals who, for whatever reason, are predisposed to abuse alcohol, then they will surely nd the wherewithal to acquire drink by whatever means it takes. Meanwhile the vast majority who enjoy and bene t from alcohol (see later) are penalised (Chaloupka *et al.* 2002).

It seems that diverse psychiatric conditions tend to be found in individuals displaying alcohol dependence. Thus it was shown in one study that only one- fth of people receiving treatment for alcohol dependence failed to report other psychiatric disorders. It seems, too, that those predisposed to 'abusing' alcohol are also increasingly likely to display anxiety, affective and antisocial disorders and other substance abuse problems (Burns 1994).

It is claimed that those people with an increased tendency towards alcohol abuse metabolise alcohol in distinctive ways. Acetaldehyde levels are seemingly higher in such people (Lindros 1978). However, Lindros does not believe that acetaldehyde is directly implicated in triggering a dependence on alcohol. Males have more alcohol-related problems than females (Dawson & Archer 1992), but females tend to accumulate higher levels of alcohol in the blood, metabolising it more slowly (Frezza *et al.* 1990).

Impacts on behaviour

I live in a city that was proud recently to vote in a new ordinance prohibiting the possession of open containers of alcoholic beverages in public places. It was argued that this would preserve some social ideal, denying rabble-rousers and itinerant panhandlers the opportunity to make a nuisance of themselves. Seemingly there was no thought given to the closing off of one avenue of contentment to the greater majority of people, i.e. those who enjoy a drink or two in accompaniment of a pleasurable all-round lifestyle. Those families and friends who enjoyed some conviviality over a bottle of wine or a couple of beers at the Farmers Market in Central Park were suddenly made to feel as if they were somehow socially inadequate. Legislators might ponder the work of Zarkin *et al.* (1998), which showed that men who consume alcohol enjoy approximately 7% higher wages than those who do not drink.

Alcohol, regrettably, is too often associated with antisocial behaviour. Starting with the observation that moderation can be associated with the use of any alcoholic beverage, what evidence is there for differences between drink type in their impact on social behaviour? And let us be careful when addressing matters of cause and effect. Thus football hooligans might be predisposed to beer consumption. That is quite different from saying that drinking beer causes all instances of football hooliganism. In just this

same way, a wife beater is predisposed to domestic violence from a aw in his character. The fact that he may enjoy a drink is by no means causally linked. Somebody who will in ict physical harm on a spouse is not made into such a person by consuming alcohol, although we might accept that the alcohol may remove inhibitions to increase the likelihood of it happening. Approximately half of adult males in the US who are heavy drinkers do not display drink-related personal or social problems, while nearly a half of those adult males that do have the very problems generally associated with drinking are not heavy drinkers (Cahalan & Room 1974).

Many laboratories have demonstrated the Mellanby effect (Mellanby 1919): the concentration of alcohol in the blood rises more rapidly and to higher levels after the consumption of spirits as opposed to beer (see e.g. Gardiner & Stewart 1968). Takala *et al.* (1957) showed that these differences were manifest even when the spirits were diluted to the alcoholic strength of beer. The differences were displayed in respect of performance – for example, driving tasks were more impaired for people who had taken brandy rather than beer (Bjerver & Goldberg 1950). Takala *et al.* (1957) found that brandy drinking led to more argumentative and aggressive behaviour than did beer drinking, even though blood alcohol levels were similar. Boyatzis (1974) made comparable observations. Pihl and colleagues (1981) feel that the different impact of beer and spirits is due to different *expectations* about their effects, and not the different type of beverage *per se*. However, we assume that Siamese ghting sh don't have expectations, and Raynes and Ryback (1970) found that aggression in such creatures was *decreased* by alcoholic beverages, with beer and wine having a greater impact than spirits.

Klein and Pittman (1993) claimed that emotional state impacts on the beverage of choice. Thus beer drinking increases in response to negative emotions, such as loneliness, whereas the intake of wine coolers was increased in association with positive emotional states. Seemingly, married people drink more wine when they are sad and bored.

Of course, we must not ignore the fact that there are substantial differences between the drinking public in where and when they will consumer beverages of different types. Also the perception of the different types of beverages varies. Klein and Pittman (1990) surveyed more than 2000 American adults to nd that underage drinking and antisocial behaviour were regarded as being associated more with beer and spirits than with wine. Conversely, Gaines (1985) found that the black population in three cities regarded beer as a soft drink and unlikely to be harmful. Lang *et al.* (1983) determined that undergraduates believed wine to be the most positively regarded of the alcoholic drinks, while Harford (1979) found that wine was more likely than beer or spirits to be consumed with a meal. It seems that bar customers taking beer will do so with greater rapidity and to a greater extent than will those taking other forms of alcohol (Storm & Cutler 1981; Stockwell *et al.* 1992).

Surveys seem to suggest that wine consumption is less associated with problems than is that of beer or spirits (e.g. Adlaf *et al.* 1993). However, Evenson (1986) found that

among more than 10,000 alcoholics in Missouri, those drinking beer alone had fewer alcohol-related symptoms and problems. Gronbaek *et al.* (2000) concluded that beer drinkers appeared to have more sensible drinking patterns than did wine drinkers.

Once again, I believe it to be important to distinguish cause and effect. The evidence seems to be that beer is *perceived* to be less healthful than wine even though the evidence (see also Chapter 6) does not support this contention. Beer is also more frequently associated with antisocial behaviour than is wine, though again good arguments can be made to say that either beverage is as good or bad as the other in this context. The simple truth is that 'high spirits' are more often associated with young men than with any other sector, and at the same time young men tend to be the fraction most likely to take in most beer (Single & Storm 1985). Without belabouring the point, it's rather like drawing a correlation between the absence of goatee growth and the predisposition to become a nurse. There are many more female than male nurses and (I assume) the majority of the former gender don't have goatees, and indeed, for reasons of hygiene, the male nurses won't either.

Thus the population drinking wine generally tend to be older (and wiser?) than those drinking beer. They are less likely to drink and drive for this very reason (Perrine 1970, 1975), and not on account of the beverage they drink. Berger and Snortum (1985) suggest that the problem is the beer drinker's culture, with the positioning of much beer advertising being one that appeals to gung-ho masculinity. Snortum *et al.* (1987) discovered that male students declaring a preference for beer regarded themselves as more 'drunk' than did those claiming to prefer wine. It was predicted that this self-concept would lead to an actual likelihood of increased drinking.

Booth (2003) points out that many effects of alcohol on mood and social behaviour are as much to do with the situation in which the drink is consumed as with the direct impact of ethanol on the neural system. He says:

> Merriment and perhaps sexual predation are what is expected at parties; personal aggressiveness and vandalism become a norm for soccer fans, and gloom is natural for the lone(ly) drinker. All these effects have been seen in experimental studies, but there tend to be large 'placebo' or expectancy effects, too. It seems that ethanol contributes some disinhibition or incapacitation but a participative spirit achieves the rest.

Diverse social pressures and norms can play an important role in conditioning individuals' approach to drinking. Religion is naturally high on the list (Single *et al*. 1997) and the reinforcement of standards by family and friends may be more effective than legal and regulatory controls (Heath 1990).

Grivetti (1985) reminds us that young people invariably start off by disliking the avour of alcoholic beverages, including beer. They pass through subsequent stages

of tolerance, acceptance and savouring. Impacting factors are peer impressions and adult mimicry. At first they stand in bars, saying 'boy, this stuff sure is great', when in fact they find the flavour challenging, to say the least. The same pressures lead to the impression that smoking is mature and socially sophisticated.

There are two possible reactions to such observations. Some would argue that the response should be to scare young people from the 'evils' and educate them so that this mimicry of adults is seen as futile and ill advised. The converse attitude, particularly when armed dispassionately with the facts in support of a very real positive impact of *moderate* alcohol consumption, is to educate with a more balanced approach. Sure, excessive consumption of alcohol is stupid, detrimental to health and antisocial. Restrained consumption, though, can be a boon. Schools in America teach 'Driver's Ed' to develop good road skills in young people. The person who advocated the banning of the automobile in response to the numerous instances of speeding, accidents (far from all traceable to drunkenness) and atmospheric pollution caused by such machinery would be viewed as eccentric at the very least.

Sutherland and Willner (1998) investigated problems of alcohol, cigarette and illicit drug use in English adolescents. They found that instances of drug use and smoking were lowest in those young people who drank beer or wine, was intermediate in those consuming 'alcopops' (nowadays the terms 'malternative' is in vogue for this type of product) and highest in those who drank spirits.

Schweitz (2001) made some very perceptive observations regarding beer drinking in Sweden. He says that many Swedes have been inculcated with a feeling that even very modest consumption of beverages of relatively low alcohol content (e.g. most beers) is morally wrong. He claims that the unjustified reaction of shame and guilt in turn leads to feelings of 'let's do something to feel guilty about', with attendant episodes of binge drinking. Such drinking patterns of over-indulgence separated by lengthy periods of abstinence are more prevalent in Sweden than in other countries. Schweitz also says that the proportionately higher taxation rate (on an alcohol basis) on beer as opposed to stronger products (wine, spirits) encourages people to consume the higher-alcohol products.

There is a strong appreciation that the most acute health and social consequences are most frequently associated with those who indulge in light drinking but then binge (Poikolainen 1995; Stockwell *et al.* 1996; Grant & Litvak 1998).

Understandably there is great concern from the medical profession in the face of the burgeoning evidence for the beneficial impact on the body of moderate alcohol consumption (which we will address in Chapter 6). To actually recommend that people drink is considered beyond the ethical pale. As W. Castelli, a principal in the famed Framingham Heart Study (see Chapter 6), wrote in 1979, 'With 17 million alcoholics in this country we perhaps have a message for which this country is not yet ready.' And

Criqui said in 1997: 'Alcohol is too dangerous to be employed as a pharmacological agent except in highly selected situations.'

How, then, to deal with observations like that of Sesso *et al.* (2000), who nd that, among men with low alcohol consumption (e.g. one drink per week or less), a subsequent moderate increase in alcohol consumption will lower their risk of cardiovascular disease?

Might I suggest that the sensible approach is to accept that a product such as beer can be a safe, pleasurable and even nutritious component of our diet, properly balanced against all other elements of our daily intake? It is not a medicine to be prescribed by doctors but rather a foodstuff that should be approached within social environs that are mature, considerate and reasonable. As was written in the *Wall Street Journal* on 13 January 1988:

> Drinking tends to be unproblematic when it is a normal, wholesome, enjoyable aspect of everyday life – not an unwholesome, dangerous and mysterious activity to be done in peculiar contexts that are set apart from friends, family and the normal routine of living. Drinking is much like eating, in the salutary view of Italians and many others, a view that contrasts markedly with the special quest for relaxation, relief of psychic stress, delusions of power or escape that prevail in much of Northern Europe and North America.

2 Beer Through History

It seems that the first domesticated grain dates from around 8000 BC in the regions of Tell Aswad, Jericho and Nahal Oren. A stamp seal from Tepe Gawra (one of the most important historic sites of ancient North Mesopotamia, now Northern Iraq) of some 6000 years ago is the first evidence of beer consumption: it depicts two people drinking beer from a single container using straws (Katz & Voigt 1986). Sumerian and Mesopotamian texts and artwork feature beer to a substantial extent, with the oldest known recipe being recorded as the *Hymn to Ninkasi* (Oriental Institute 2002). The lengthy verse (from which I quote extracts) refers to Ninkasi as

> the one who handles dough [and] …
> with a big shovel,
> Mixing, in a pit, the bappir with sweet aromatics.

This refers to the practice at the time of making a bread from sprouted barley, the bread subsequently being lightly baked:

> You are the one who bakes the bappir in the
> big oven,

We recognise that it was barley because of the retained hull (or husk, see Chapter 3):

> Puts in order the piles of hulled grain.

The 'malt' was then mixed with water, allowing the endogenous enzymes to digest the starch in the production of 'wort' and for adventitious yeasts to commence the fermentation process:

> You are the one who waters the malt set
> on the ground,
> You are the one who soaks the malt in a jar,
> You are the one who spreads the cooked mash on
> large reed mats,

Coolness overcomes …
You are the one who holds with both hands
the great sweetwort,
You place appropriately on [top of]
a large collector vat.
Ninkasi, the fermenting vat, which makes
a pleasant sound,

After fermentation there was a clari cation – and, by the sounds of it, there was rather a lot to lter:

When you pour out the ltered beer
of the collector vat,
It is [like] the onrush of the Tigris and the Euphrates.

And the poem goes on to indicate that the beer was prized and valued for its merits:

The gakkul vat, which makes the liver happy,
The lam-sá-re vat, which rejoices the heart,
The ugur-bal jar, a tting thing in the house.
The sa-gub jar, which is lled with beer,
The am-am jar, which carries the beer
of the lam-sá-re vat …
The beautiful vessels, are ready on [their] pot stands!
May the heart of your god be well
disposed towards you!
Let the eye of the gakkul vat be our heart!
What makes your heart feel wonderful,
Makes [also] our heart feel wonderful.
Our liver is happy, our heart is joyful.
While I circle around the abundance of beer,
While I feel wonderful, I feel wonderful,
Drinking beer, in a blissful mood,
Drinking liquor, feeling exhilarated,
With joy in the heart [and] a happy liver –
While my heart full of joy,

As we shall see in Chapter 3, the processes referred to are entirely recognisable in brewing practices to this very day.

In those far-off times, beer featured centrally as a foodstuff rather than as an accompaniment. Hesseltine (1979) indicates that a typical consumption must have been about a litre per day at 2% alcohol. The straw used for drinking was of clay or reed for the general population, but gold or silver for the rich and powerful. Some 40% of the grain in Sumeria was used for beer production. A workman in the temple got 1.75 pints per day, with senior dignitaries getting five times that level (Singer *et al.* 1954–58).

By the early Egyptian period the contemporary brewing practices were firmly in place (Tannahill 1973). Dough was made from sprouted and dried grains and partially baked. These loaves were then broken up and soaked in water and allowed to ferment for about a day. Then the liquid was strained off and the beer was ready for drinking. As Singer observes, Egyptian brewers were soon making variously spiced and flavoured beer breads, allowing for a diversity of beers. There was a superintendent of breweries to ensure that purveyors only made available the best and purest products (Fleming 1975).

Of course they had no control over the yeast because they had no notion that it existed, although they would have discovered that older cracked jars, with more hiding places for organisms 'naturally selected' for the purpose, would have given better results. It wasn't until later that Pliny the Elder (AD 23–79) reported that the Gauls and Iberians were skimming beer for the purpose of re-inoculating the next batch. The brewers were women, who sold their beer from home. The Code of Hammurabi (1750 BC) condemned alehouses for their under-strength and over-priced beers and also had a decree regarding those who diluted the beer (Saggs 1965). Those who overcharged for their beer were to be drowned.

In Egypt the most common beer was *haq* (*hek*) made from the red barley of the Nile (Tannahill 1973). Compared to some other products that we believe reached alcohol contents similar to modern wines (i.e. about 12%), *haq* seems to have been quite 'mild'. Bread, beer and onions seemed to form the basic diet of the dynastic Egyptian peasant. Beer was deemed to be essential for general wellbeing. The Ebers papyrus, a sort of pharmacists' standard text, listed the ingredients for diverse medicines, of which more than 100 of the 700 were made with beer (Fleming 1975).

Brewing travels west

The Egyptians passed on their brewing techniques to the Greeks, though wine was the preferred drink for that empire and also for the Romans. Greek tradition says that Dionysus fled from Mesopotamia in disgust owing to its people being addicted to beer (Tannahill 1973). Beer was the mainstay of more northern cultures and the Germanic and Celtic races. In the first century AD the Britons and Hiberni (Irish) were making *kourmi* from barley, a crop that had probably been cultivated in England since 3000 BC

(Dunn 1979). One member of St Patrick's (373–464) household was a brewer, a priest named Mecan (King 1947).

One of the earliest references to beer in England is perhaps not as complimentary as one might wish:

> Kourmi, made from barley and often drunk instead of wine, produces headaches, is a compound of bad juices and does harm to the muscles.

However, this was penned by a Greek (Dioscorides ca. 1st century AD), presumably biased in favour of wine!

The history of beer has always been entwined with the church. St Brigid brewed ale at Eastertide to supply to all churches in her neighbourhood (King 1947). Later, the monasteries spawned the first breweries in the British Isles. The word 'ale' comes from the Old English *ealu*, and we suppose that the malted grain was a cheaper option than the honey used in making mead.

The Danes and Anglo-Saxons drank ale because their homelands were too cold to cultivate grapes successfully. The Anglo-Saxons used ale for coughs, shortness of breath and curing hiccups (Fleming 1975). They rubbed it on to the knees to ease aches and pains. Beer was a drink for heroes and Norse seafarers were brave in battle believing that, should they perish, it would be to go to drink ale in Valhalla (Savage 1866). The Vikings sang about drinking well before putting out to sea, hence the phrase 'three sheets to the wind'. The Scandinavian word *bjor* became *beer* in the Anglo-Saxon. The foods enjoyed in Northern European countries were (and still are) heavy in carbohydrate and fat, needing to be washed down with large volumes of liquid (Tannahill 1973). Thus beer is highly suitable.

Restraining excess

King Edgar (959–975) was convinced by Archbishop Dunstan of Canterbury to close many alehouses because of drunkenness and it was decreed that there should be only one such establishment per hamlet. This early attempt at enforcing moderate consumption had the additional proviso that pins should be hammered inside drinking horns at stated points and 'whoever should drink beyond these marks at one draught should be obnoxious to a severe punishment' (King 1947). One might note, however, that medieval drinking vessels had a capacity of about four pints (a 'pottle') (Brown & Schwartz 1996). Drinking competitions sprang up to see who could uncover the most pins – in other words to 'take each other down a peg or two'.

In Norman times ale was used for casting out devils: the trick was to mix some herbs with 'clean ale', sing seven masses over the drink, add garlic and holy water and then drink it from an inverted church bell (King 1947). Ale was popular. William of Malmesbury wrote of the English in the early twelfth century (King 1947):

> Drinking was a universal practice, in which occupation they passed entire nights as well as days. They consumed their whole substance in mean and despicable houses; unlike the Normans and French who in noble and splendid mansions lived with frugality. They were accustomed to … drink till they were sick. These latter qualities they imparted to their conquerors.

Religious origins

All monasteries and abbeys featured breweries. The symbols X, XX and XXX were used as a guarantee of sound quality for beers of increasing strength (Savage 1866; King 1947).

The monasteries passed on their skills to those brewing in their own homes (notably the women: 'ale wyfes') and by the Middle Ages ale had become the drink at all meal-times. Out of the domestic brewing scene came the development of breweries, each selling their own beer in a room at the front – they would be known today as 'brew pubs'. They produced two main products: 'strong beer' fermented from the first runnings from the mash and 'small beer' from the weaker, later runnings.

In the early fourteenth century there was one 'brew pub' for every 12 people in England. In Faversham in 1327, 84 out of 252 traders were brewers. All ale was sold locally because of transport limitations and the difficulty of keeping beer for any length of time. Ale was sold in three types of premises: inns, where you also sought food and accommodation; taverns, which also sold wine; and ale-houses (Dunn 1979). And yet 90% of ale was still 'home-brew'.

One of the earlier attempts to regulate standards of quality was in Chester, where the penalty for a woman brewing bad ale was a drenching in the ducking chair (King 1947). The number of ordinances and regulations in the middle years of the second millennium that dealt with beer were nearly as many as dealt with another staple, bread (Drummond & Wilbraham 1958). In the *Liber Albus* of 1419 compiled by John Carpenter and Richard Whittington (of cat fame) there is mention of the 'aleconners of the Ward' whose job was to taste each brew and report on it to the Mayor.

In Medieval times ale was associated with festivals and family events – thus there were lamb-ales, bride-ales (*bridals*) and so on. A bride could sell ale on her wedding day and take the proceeds (King 1947).

Ale was sold to support Parish funds, hence at Sygatem Church in Norfolk we find the quotation:

> God speed the plough
> And give us good ale enow…
> Be merry and glade
> With good ale this church was made.

We look back to those times for the origins of terms like 'cheers' and 'good health' and diverse other 'toasts' (Fleming 1975). It was the custom to put a piece of toasted bread into the beer, which was passed around the guests in a 'loving cup'. Perhaps the toast improved the flavour. Finally the host received the cup, drank the remains and ate the bread.

Maintaining standards

Henry VI appointed surveyors and correctors of beer-brewers (King 1947):

> Both the malt and hops whereof beer is made must be perfect, sound and sweet, the malt of good sound corn – to wit, of pure barley and wheat – not too dry, nor rotten, nor full of worms, called wi es, and the hops neither rotten nor old. The beer may not leave the brewery for eight days after brewing, when of cials should test it to see that it is suf ciently boiled, contained enough hops and is not sweet.

Brewers of the time, though, were less than honest. In a popular play of the period, in which souls are able to escape from Hell, the Devil is allowed to keep the soul of one person as a souvenir. He chooses the brewer. In Oxford, where the University used to have its own brewery, brewers were ordered to assemble in the Church of the Blessed Virgin Mary and made individually to swear only to brew ale 'as was good and wholesome, so far as his ability and human frailty permitted him'.

The whole family drank. For instance, in 1512 the Earl of Northumberland's household – including the 8- and 10-year-old heirs – consumed 1 quart of ale or beer each mealtime (King 1947). In the poorest of homes, ale was still the drink of the whole family.

During the reign of Henry VIII [whose breakfast for three comprised a joint of roast beef, a loaf of bread and a gallon of ale (Katz 1979)] one owner of an ale brewery successfully fetched an action against his brewer for putting in 'a certain weed called a hop'. It was decreed that neither hops nor brimstone were to be put into ale (Savage 1866). We can be thankful that hops gained ascendancy, for they seem in nitely prefer-

able to materials such as wormwood, gentian, chicory or strychnia that were sometimes employed.

Savage (1866) has the date as 1524 when hops first came into the British Isles, from Flanders where they had been used for centuries. Prior to the arrival of hops, ale had sometimes been preserved with ground ivy.

Incidentally, Henry VIII was far from being the only monarch with a passion for ale. Seemingly, Queen Elizabeth I had the local ale sampled for suitability in advance of her travels around the nation. If it failed to pass muster, then her favourite London product was shipped ahead of her in time for her arrival (Katz 1979).

Concerning hops, by 1576 Henri Denham wrote:

> Whereas you cannot make above 8 or 9 gallons of indifferent ale out of one bushell of mault, you may draw 18 or 20 gallons of very good Beere, neither is the Hoppe more profitable to enlarge the quantity of your drinke than necessary to prolong the continuance thereof. For if your ale may endure a fortnight, your Beere through the benefit of the Hoppe shall continue a moneth, and what grace it yieldeth to the teaste, all men may judge that have sense in their mouths – here in our country ale giveth place unto Beere, and most part of our countrymen do abhore and abandon ale as a lothsome drink.

Gerard wrote in 1596 that:

> The manifold virtues in hops do manifestly argue the wholesomeness of beere above ale, for the hops rather make it a physical drink, to keep the body in health, than an ordinary drink for the quenching of our thirste.

This was one of the earliest attempts to position beer on a health-positive platform. In the sixteenth century, too, John Taylor penned:

> It is an Emblem of Justice, for it allowes and yeelds measure; It will put Courage into a Coward and make him swagger and fight; It is a seale to many a good Bargaine. The Physittian will commend it; the lawyer will defend it. It neither hurts, nor kils, any but those that abuse it unmeasurably and beyond bearing. It doth good to as many as take it rightly; It is as good as a paire of Spectacles to cleare the Eyesight of an old parish Clarke; And in Conclusion, it is much a nourisher of Mankinde, that if my mouth were as bigge as Bishopsgate, my Pen as long as a Maypole, and my Inke a fowing spring, or a standing fishpond, yet I could not with Mouth, Pen, or Inke, spak or write the true worth and worthiness of Ale.

Houses for the sale of beer had first become licensed in the reign of the boy king, Edward VI, in the mid-sixteenth century (Savage 1866).

By an Act of 1604, it was decreed that parish constables should inspect alehouses to ensure that they were operated properly (King 1947). It was emphasised that:

> the ancient, true and principal use of such places was for the relief of wayfaring men and women and also to fulfil the requirements of those people unable to store victuals in large quantities and not for the entertainment of lewd and idle people.

No workman was allowed to spend longer than one hour in an inn unless his occupation or residence obliged him so to do. Fines of 10 shillings were collected by churchwardens and given to the poor of the parish. At the time the cost of best ale was fixed at a penny a quart (one quart = two pints) and small beer at a halfpenny. Notwithstanding, the government in the middle of the seventeenth century was raising some 40% of its budget by taxing beer (Wilson 1991).

Beer: a nutritious dish for the whole family

By the late seventeenth century more than 12 million barrels of beer were drunk each year in Great Britain, when the population was only some 5 million. That's just about 2 pints per day per person. Even infants, who drank small beer, scarcely ever drank water. Although naturally there was no explanation for why it was the case, it was universally recognised that it was safer to drink beer. The boiling and the hopping were inadvertently water purification techniques.

In the era of Charles II, a family of seven in London would drink a barrel of small beer per week, this despite a tax of six pence a barrel (two shillings and sixpence for strong beer) (Savage 1866).

Tea seems first to have arrived in Holland and Portugal in about 1610 and in Germany in the 1630s, but the first public sale of tea in England was not until 1657 (Tannahill 1973). The first coffeehouse in England was to be found in Oxford in 1650. Soon there were choices available for a wholesome beverage at mealtimes and it no longer needed to be alcoholic. The progressive growth in tea drinking led to brewers brewing weaker beer (small beer was now 2–3% alcohol, compared to the previous 4–5%) and having to keep lower prices (Drummond & Wilbraham 1958). Beer, though, retained a key place in the diet, and at the end of the seventeenth century the beer allowance at Christ's Hospital school was 30 barrels per week for 407 people (Drummond & Wilbraham 1958). These authors stress the nutritive value of the beer (additional to its safety dimension when compared to water to drink). They estimate that small beer will have

had a calori c value of around 150–200 kilocalories per pint, so 3 pints per day for a small boy will have yielded some 20–25% of his energy needs. And furthermore it will have 'supplied a modest amount of calcium and appreciable quantities of ribo avin, nicotinic acid, pyridoxine, pantothenic acid and perhaps other vitamins' (Drummond & Wilbraham 1958).

This is not to ignore that the wholemeal bread still favoured in those days will also have supplied vitamins, including thiamine, which tends to be diminished in beer as it is readily consumed by yeast during fermentation.

> It is certain, however, that home-brewed beer was a good, sound, healthful drink and one which could not possibly do any harm to children when drunk in reasonable amounts.
>
> Drummond & Wilbraham (1958)

Moderation, however, was not universally displayed. And so the rst laws were already in place to reduce drunkenness, including xed hours when pubs must close at night, no opening on Sundays and a limit on any drinker of one hour at a time (King 1947).

In the early eighteenth century gin was developing popularity, and no licence was needed for its production, unlike beer. The duty on gin was merely tuppence per gallon (Drummond & Wilbraham 1958). By 1735 there were 5 million gin distilleries in England (King 1947). By 1750 it seems that every fourth or fth house in the slum areas of London sold gin, or something that passed for gin (Drummond & Wilbraham 1958). There were signs above doors claiming that 'here a man may get drunk for a penny, and dead drunk for tuppence' (Fleming 1975). Hogarth's paintings capture the sentiments: in Beer Street people seemed jolly and healthy, whereas in Gin Street they were debauched (Fig. 2.1). One London clergyman, James Townley, was driven to write:

> Gin, cursed end, with fury fraught
> Makes human race a prey;
> It enters by a deadly draught, And steals our life away.
> Virtue and Truth, driven to despair,
> Its rage compels to y;
> But cherishes, with hellish care,
> Theft, murder, perjury.
> Damned cup, that on the vitals preys,
> That liquid re contains;
> Which madness to the heart conveys,
> And rolls it through the veins.
>
> Roueche (1960)

(a)

Fig. 2.1 Depictions of drinking, by William Hogarth. (a) Beer Street. (b) Gin Lane. Reproduced courtesy of Haley & Steele (www.haleysteele.com).

(b)

Fig. 2.1 (*Continued*.).

There were no such verses about beer. In 1722, 33 million bushels of malt were used for brewing and annual consumption was running at a barrel of beer per head (King 1947).

There was, however, great concern regarding the wholesomeness of some of the brews that were being made, leading to a book in 1738, anonymously authored, entitled *The London and Country Brewer* (Drummond & Wilbraham 1958). The writer claimed that it was to inform a public who had long 'suffered great prejudices from unwholesome and unpleasant beers and ales, by the badness of malts, underboiling of worts, mixing of injurious ingredients, the unskilfulness of brewers'. Reference was made to the use of the seeds of a poisonous berry (*Cocculus indicus*) to afford bitterness and a 'heady' character. Coriander seeds and capsicum (red pepper) were used variously to give avour or 'bite' to thin beers or ones that had 'turned'. Tobacco and liquorice were not unheard of in the context of beer, despite an Act of Parliament in the reign of George III that prohibited many adulterants.

The brewer's concerns with the beer souring, however, were very real. *The London and Country Brewer* described the use of 'balls' to preserve beer in casks, such balls comprising alabaster or marble, oyster shells, chalk, horse-bean our, red saunders, grains of paradise, Florentine orrice-root, coriander seeds, cloves, hops, isinglass and treacle.

According to Drummond and Wilbraham (1958),

> …the marble, shells and chalk served to neutralise acidity as it developed, the bean- our and isinglass helped to ' ne' the beer, carrying down impurities to form a sludge at the bottom of the cask, while the coriander, orris-root, cloves etc imparted a avour which would help mask the earthy taste caused by the addition of so much lime.

The same authors observe, though, that the treatment tended to make beer go at, leading in turn to the addition of 'headings' to promote foaming. A popular treatment was iron sulphate, which produced a 'head like a colly ower'.

Twenty- rst-century beer drinkers should be relieved that none of these practices prevails, save for the use in some quarters of the entirely wholesome isinglass nings (see Chapter 3).

Towards the end of the eighteenth century, the impact of taxation and increasing imports of tea and coffee saw a change in domestic drinking habits – tea instead of ale for breakfast.

Temperance pressures

In the closing years of the eighteenth century less beer was brewed at home, with major brewing companies being spawned to supply beer to the millions employed in the newly developing industries. Only country folk retained their brewing traditions. The development of roads and railways provided distribution systems for the big brewers.

By 1810, there were 48,000 alehouses for some 8 million people in Britain (King 1947). Captains of industry were perturbed about wages being 'wasted' on excess drinking. This led to a tightening of licensing laws and many counties declared that public houses should be closed at 9 PM in winter and 10 PM in summer. Some were not satis ed even with that and the temperance movement developed. The rst pledge of 'teetotalism' was signed in Preston in 1832 (King 1947). [The word teetotal is said to have originated in an English temperance meeting, when a stammering man said 'We can't keep 'em sober unless we have the pledge total. Yes, Mr Chairman, tee-tee-total' (Fleming 1975).]

However, there were those who championed the merits of consuming beer. Savage (1866) wrote in the United States (where beer was very much the drink of moderation as compared to the much more prevalent distilled concoctions) that:

> The most useful temperance lecturer is he who advocates the temperate use of beverages which custom has sanctioned and which … man *will* have. A reform may, and we trust will be effected in favour of healthful and comparatively mild drinks; but it is more than doubtful if hard working, energetic and withal social people, such as form the bone and sinew of the Republic, will or can be induced to give up all drink which custom, and the large majority of clergymen and physicians, have sanctioned as refreshing.

Savage reminded the reader that in Bavaria at the time the average frugally drinking labourer consumed a gallon per day. With reference to England, Savage championed beer thus:

> With an impartial catholicity of palate the votary of the amber ale loves to see its 'beaded bubbles winking at the brim' and yet is never forgetful of the darker charms possessed by porter or stout. Boating men … cricketers, and the whole of the manly English sporting community, are sensible alike to the charms of the long, thin, narrow glass, the simple and unassuming tumbler, and the thorough going pewter pot. The prudent and industrious mechanic prefers the wholesome brew of native malt and hops to the ery foreign distillations that madden the brain and shatter the nerves. The statistics of beer drinking are simply stupendous. Mr. Gladstone … computed that every adult male in England consumed the astounding quantity

> of six hundred quarts per annum. Despite all the arguments and invectives of the agitators who advocate what is paradoxically described as a 'permissive bill', on account of its prohibitory character, we adhere to our faith that sound honest malt liquor does far more good than harm; nor should we dream of opposing any system of nancial legislation which would make it cheaper without in icting an extra burden upon the community.

And the beer strength in England at the time was formidable (Dunn 1979). In 1843 Burton Ale had original gravities between 1077 (19.25°P) and 1120 (30°P), while Common Ale was 1073 (18.25°P) and Porter 1050 (12.5°P) (see Chapter 3 for de nitions of beer strength).

Early nineteenth-century diets, though, retained beer as an integral feature, indeed the recommended 'family economy' for 'moderate persons in a frugal family' for 1826 comprised (per person, per week):

> 6 pounds meat (undressed)
> 4 pounds bread (quartern loaf)
> 0.5 pounds butter
> 2 ounces tea
> 0.5 pound sugar
> 1 pint per day of beer (Porter)
>
> Drummond & Wilbraham (1958)

The same authors cite a range of typical diets through the ages, reproduced in Table 2.1, and their estimated nutritive value is given in Table 2.2.

Table 2.1 Diets through history in England.

Diet 1	Diet 2	Diet 3	Diet 4	Diet 5
15th-century meat-eating classes (per day)	Sailor's diet, 1615 (per day)	St Bartholomew's Hospital, 1687 (per day)	Navy ration, 1745 (per week)	Navy ration, 1811 (per day)
cheese, 4 ounces meat, 1.5 pounds herring, 6 ounces fat, 1 ounce bread, 1 pound wine, 1 pint ale, 2 pints	cheese, 8 ounces bacon, 4 ounces butter, 4 ounces biscuit, 1 pound oatmeal, 3 ounces beer, 8 pints	cheese, 1.5 ounces milk pottage, 1 pint beef or mutton, 4 ounces broth, 1 pint butter, 1 ounce bread, 10 ounces beer, 3 pints	cheese, 12 ounces salt beef, 4 pounds salt pork, 2 pounds butter, 8 ounces biscuit, 7 pounds oatmeal, 2.5 pounds pease, 2 pints beer, 7 gallons	cheese, 1.75 ounces beef, 4.5 ounces pork, 2.25 ounces butter, 0.9 ounce suet, 0.25 ounce sugar, 0.9 ounce bread, 1 pound our, 3 ounces beer, 2 pints

Source: based on Drummond & Wilbraham (1958).

Table 2.2 Estimated nutritive value of the diets listed in Table 2.1 (and the 1826 diet referred to in the text).

	Diet 1	Diet 2	Diet 3	Diet 4	Diet 5	1826 diet	Requirement*
Energy (kcal)	4750	5800	2350	5500	2900	2050	2550
Protein (g)	200	150	70	160	80	70	63
Fat (g)	190	250	80	180	100	120	*a*
Calcium (mg)	1.3	2.6	0.9	1.9	0.7	0.1	800
Phosphorus (g)	4.2	3.7	1.7	3.7	1.9	1.2	0.8
Iron (mg)	39	24	14	36	18	18	10
Vitamin A (i.u.)	2800	6350	3200	1750	1450	1150	1 mg
Thiamine (mg)	1.5	1.9	1.1	2.6	1.6	0.8	1.5
Ribo avin (mg)	3.7	3.9	1.7	4.0	1.5	1.3	1.7
Nicotinic acid (mg)	68	84	40	100	46	28	19
Vitamin C (mg)†	?	?	?	?	?	0	60
Vitamin D (i.u.)	950	100	25	26	22	19	5 µg

*Requirement for adult male, aged 25–50, according to the Food and Nutrition Board, National Academy of Sciences and British Nutrition Foundation.
†Uncertain due to dif culty of estimating vegetable consumption and heat-dependent losses in cooking.
a For a diet containing alcohol, it is recommended that the total dietary energy should be 47% as carbohydrate, 33% as fat and 15% as protein.

Table 2.3 Expenditure on foodstuffs, 1881.

Item	Expenditure per head per day (pence)
Bread	0.59
Potatoes	0.27
Vegetables	0.14
Meat	0.79
Fish	0.11
Butter and cheese	0.28
Milk and eggs	0.33
Fruit etc	0.08
Sugar	0.21
Tea	0.12
Coffee etc	0.02
Beer	0.59
Spirits	0.32
Wines	0.07
Total	3.92

Source: British Association for the Advancement of Science (1981).
Values converted to decimal pence from the old shillings and pence.

In 1881 it was estimated that expenditure on beer in the average household was one of the three major outlays (Table 2.3) (Burnett 1966).

The development of teetotalism and the push for prohibition was moving apace in the late nineteenth century, featuring among others the Salvation Army. It was even suggested in 1903 that alcoholic drinks should only be taken with meals. Balance this

with the acceptance in the medical profession even then that alcohol had real merits. Before ether was discovered in 1846, alcohol was used to dull pain (Fleming 1975). In 1900, the distinguished physician Sir William Osler referred to alcohol as 'our most valuable medicinal agent'. In those days whisky, beer and brandy were stocked on the medicine shelves as 'stimulants'.

Meanwhile over a period of many years there was much debate and development in the area of licensing, primarily on account of concerns about the numbers of public houses. The Licensing Bill introduced in 1908 ruled that there could be one licence for every 400 persons for areas with populations averaging two individuals per acre; one for every 500 when the population was 2–25 per acre; and up to one per 1000 people when the population averaged 200 to the acre.

The Great War of 1914–18 led to fresh concerns about excessive drinking and its impact on the war effort. Lloyd George claimed: 'Drink is doing us more damage in the war than all the German submarines put together.' However, a bill proposing a doubling of the tax on alcohol was not passed (King 1947).

In World War II, also, formidable voices in the UK government urged a ban on alcohol, so as to divert raw materials to food production. Fortunately, rational minds applied logic to the situation (which seems seldom to be the case unfortunately when it comes to matters to do with alcohol): it was calculated that if the beer supply was halved and the barley thus saved diverted to chicken food, the net bene t would have been one egg per month in people's ration – and huge public discontent (King 1947).

Towards prohibition

The most famed instance of prohibition was of course the United States between 1920 and 1933. In the earliest days of that country everyone generally held that the human could not survive without alcohol (Fleming 1975 – from which reference I have sourced much of what follows in this section). As Fleming puts it:

> Men and women, old and young, rich and poor, regularly started the day with a morning dram. The drink might be anything from cherry brandy to wine mixed with sugar and water, as long as it contained alcohol. A daily glass of 'bitters' was considered essential for warding off disease, clearing the head, and keeping the heart in good working order.

Shopkeepers had barrels of rum on tap for customers (rather like a bank might have a pot of coffee on the go today). Labourers had a mid-morning break for 'bitters'. Jugs of rum were in the elds for agricultural workers. Note that the liquids provided were spirits, not the gentle (by comparison) beer.

It was Dr Benjamin Rush, a signatory to the Declaration of Independence, who in 1784 penned *An inquiry into the effects of spiritous liquors on the human mind and body,* and who argued that 'ardent spirits' caused *inter alia* obstruction of the liver, jaundice, hoarseness, diabetes, jaundice, gout, epilepsy, madness and 'frequent and disgusting belchings'. There were plenty of people prepared to buy into his argument. And so a group of Connecticut businessmen stopped making rum available to their employees, replacing it with cider and beer. And in New York State in 1808 the Union Temperance Society was founded. Beer was 'in', but the 44 members pledged to 'use no rum, gin, whisky, wine or any distilled spirits … except by the advice of a physician, or in case of actual disease, also excepting wine at public dinners'. A number of other such societies sprang up, arguing for moderation rather than abstinence. President Thomas Jefferson wrote to a friend in 1815 about beer: 'I wish to see this beverage become common instead of the whiskey which kills one third of our citizens and ruins their families'. As Divine *et al.* (1987) put it:

> The temperance movement was directed at a real social evil, more serious in many ways than the drug problems of today. Since the Revolution, whiskey had become the most popular American beverage. Made from corn by individual farmers or, by the 1820s, in commercial distilleries, it was cheaper than milk or beer and safer than water (which was often contaminated).
>
> Hard liquor was frequently consumed with food as a table beverage, even at breakfast and children sometimes imbibed along with adults. Per capita annual consumption of distilled spirits in the 1820s was almost triple what it is today, and alcoholism had reached epidemic proportions.

A Presbyterian minister, the father of Harriet Beecher Stowe (author of *Uncle Tom's Cabin*), became a particularly vocal opponent of alcohol in all its manifestations. The Reverend Lyman Beecher implored his congregation to join his crusade to rid the country of 'rum-selling, tippling folk, in dels and ruff-scruff'. His sermons were distributed nation-wide, with the impact that employers stopped giving drinks to their workforce and liquor rations ceased in the US Army. Beecher's American Temperance Union (ATU) sought to persuade every state to ban the production and sale of alcohol. At rst beer was accepted within the ATU, but that too fell foul of the zealots in 1836. The impact was a *decline* in membership. So many people realised the facts: it was hard spirits that were leading too many astray, not beer.

The ght against alcohol became easier in 1833 when the US Supreme Court ruled that state governments could regulate the liquor trade within their boundaries. Furthermore it permitted 'local option', in which individual counties and towns could introduce prohibition if they so wished. First off the blocks was Massachusetts in 1838, with the banning of sales of spirits in quantities less than 15 gallons. It didn't last long

– customers bought 15 gallons plus a gill, drank the latter and then returned the balance. Maine introduced total prohibition in 1851, causing Lyman Beecher to exclaim: 'The glorious Maine law is a square and grand blow right between the horns of the Devil.' Soon thirteen more states had joined Maine, but nine soon repealed the laws or declared them unconstitutional. Only Maine, Kansas and North Dakota held rm – and in each there were bootleggers and illicit taverns ('blind pigs').

By 1872 a political body, the Prohibition Party, had come into being and nominated James Black to run for President. He lost – and so have many other prohibition candidates since. Their best performance in the polls was 271,000 votes in 1892. The Party is still in existence (see http://www.prohibition.org/), and they observe that they are 'the oldest "third party" in the United States'. We might note their other stated 'values' include being anti-commercial gambling, against the homosexual agenda, preservation of US sovereignty and concerns about the United Nations and about international trade agreements.

Back to the late nineteenth century. Women soon led the charge against alcohol. One slogan was:

> We do not think we'll ever drink
> Whiskey or gin, brandy or rum
> Or anything that'll make drunk come.

Not classic verse – but at least no mention of beer.

The Women's Christian Temperance Union had prominent members, including the First Lady, Mrs Rutherford B. Hayes ('Lemonade Lucy'). And they warmly embraced the redoubtable Carry Nation, who declared 'hatchetation' in smashing up illicit taverns in her home state of Kansas and beyond, and set off on an enthusiastically received lecture tour in which hatchets could be bought as souvenirs. They do say that no publicity is bad publicity and soon liquor producers were marketing Carry Nation cocktails and bars were decorated with hatchets and signs that declared 'All Nations welcome but Carry.'

Carry Nation was probably emotionally disturbed for much of her life (Fleming 1975) and the most successful pro-prohibition lobby, the Anti-Saloon League originating in a Congregational Church in Ohio, ignored her. The tactics of this body were more subtle and low key, progressively persuading towns and counties to embrace prohibition. Soon they were successful at the state level: Georgia, Oklahoma and then half a dozen more fell into line. In 1913, after 20 years of existence, the Anti-Saloon League marched on Washington DC with a slogan 'A Saloonless Nation in 1920'. Several supporters were elected to Congress.

The 65th Congress, convening in March 1917, soon declared war on Germany following the sinking of the *Lusitania*. This demanded laws that would ensure that the

US was in t state to ght a war, including legislation concerning the production and distribution of food. A clause was inserted that outlawed the production and sale of alcoholic beverages, so that grain could be conserved. There was disagreement from the opponents of prohibition, and there was agreement to let the Senate vote on a separate resolution calling for a prohibition amendment to the Constitution. Astonishing to many, but the Eighteenth Amendment went speedily through Congress and it was rati ed by 36 State legislatures in little more than a year. Only Rhode Island and Connecticut held out on ratifying the amendment. The amendment was of cially adopted on 16 January 1919, with national prohibition being effected one year later.

It's perhaps not altogether strange that to deny people something that the majority enjoy and don't abuse will inevitably prove unsuccessful. In New York before prohibition there were 15,000 bars. After prohibition there were 32,000 speakeasies. Women and youngsters now decided that drinking was something they perhaps should entertain, having not bothered much before. Booze was coming in illicitly from Canada and Mexico and by ship from Cuba, the West Indies and Europe. And there was the illicitly brewed stuff in the States, much of it dangerous through a lack of regulation. There was plenty of corruption at high level and of course the making of some infamous criminal reputations among the gangsters. Bootleggers collected $2 billion annually, amounting to some 2% of the gross national product (Divine *et al.* 1987).

Bodies quickly sprang up, seeking to repeal the Volstead Act, including the Moderation League. In 1930 the American Bar Association adopted a resolution that called for a repeal of Volstead. They were supported by the Women's Organization for National Prohibition Reform. Those advocating 'dryness' were at risk of being perceived as defending the gangster culture.

By the early 1930s the nation was in the midst of the Great Depression. Many argued that it had been brought on by prohibition and that to repeal the Act would be to create jobs and put much needed taxation income into the exchequer.

The 1932 presidential campaign was in substantial part fought on the alcohol issue. Herbert Hoover said that prohibition had been an 'experiment noble in purpose' and he promised to do what he could to correct whatever shortcomings there were. Franklin Delano Roosevelt went a major step further: 'I promise you that from this date on the Eighteenth Amendment is doomed.'

Roosevelt was elected and nine days later he asked Congress to amend the Volstead Act so that the alcohol content of beer could be raised from 0.5% to 3.2% by weight. The law was passed. As he sat down to his evening meal on 12 March 1933, Roosevelt is quoted as saying: 'I think this would be a good time for a beer' (Barone 1990).

3 The Basics of Malting and Brewing: Product Safety and Wholesomeness

The fundamental shape of the processes by which beer is made has not changed for many generations [see Bamforth (2003) for a general introduction and overview, and a full glossary of brewing terms]. However, the control and predictability of those processes has improved. Beer nowadays is invariably a highly consistent consumable, closely controlled for the ef ciency of its production and its safety. There is little that is hit-and-miss about the making of beer. Despite its reliance on agricultural products (barley, sometimes other cereals, and hops) the understanding of the process means that seasonal and regional vagaries can be overcome such that the taste, appearance and composition of a beer are generally consistent from batch to batch. There is no such thing as a vintage in brewing.

Accordingly, the customer should realise as they explore their local supermarket shelves that one of the most consistent and reliable products to be had is the beer. It is also one of the safest, as we shall see.

Chemical beer?

The brewing of beer is complicated. The vast majority of beers comprise at least 90% water, with ethanol (it is customary to use 'alcohol' synonymously for this one alcohol – although there are other alcohols in beer) and carbon dioxide being quantitatively the next major individual components (Table 3.1). Beers also contain a wide range of chemical species in relatively small quantities that determine the properties of the beer in respect of appearance and avour.

Malting and brewing are processes designed to maximise the extraction and digestion of starch and protein from barley, yielding a highly fermentable extract that is known as wort. The processes are also designed to eliminate materials that can have an adverse effect on beer quality, such as the haze-forming polyphenol from barley and hops and the lipids and oxygen that, together, can cause beer to stale.

Malting and brewing within all companies, large and small, are very traditional processes. Relatively few chemicals are added to beer (or to the process) as opposed to being derived from its raw materials. In some markets (but by no means all) propylene glycol alginate is used as a foam stabiliser (Bennett 1993) and sulphur dioxide

Table 3.1 Composition of an all-malt Pilsen beer (ca. 12° Plato).

Component	Content (in mg/L unless otherwise indicated)†
Original extract	11.8 g/100g
Alcohol	3.93 g/100g
'Real extract'	4.15 g/100g
Water	919 g/L
Carbon dioxide	5 g/ L
Total carbohydrate	28 g/L
Glucose	150
Fructose	30
Sucrose	5
Maltose	1430
Maltotriose	1930
Maltotetraose	3360
Maltopentaose	1330
Maltohexaose	1150
Maltoheptaose	1090
Maltooctaose	1220
Maltononaose	1590
Maltodecaose	1750
Maltoundecaose	920
Maltododecaose	640
Maltotridecaose	760
Maltotetradecaose	1020
Maltopentadecaose	880
Maltohexadecaose	950
Maltohepatdecaose	800
Maltooctadecaose	1130
Higher dextrins	5490
Pentosans	60
β-Glucans	350
Proteins	5 g/L
Low molecular weight N componds	185
Medium molecular weight N componds	83
High molecular weight N componds	26
Histidine	36
Isoleucine	34
Leucine	55
Lysine	16
Methionine	2
Phenylalanine	77
Threonine	5
Tryptophan	20
Valine	73
Arginine	72
Proline	357
Aspartic acid	28
Serine	19
γ-aminobutyric acid	73
Glutamic acid	40
Glycine	31
Alanine	103
Tyrosine	76
Cysteine	12

(*Continued*.)

Table 3.1 (*Continued*.)

Component	Content (in mg/L unless otherwise indicated)†
Cystine	6
Potassium	493
Sodium	30
Calcium	34
Magnesium	107
Phosphorus	308
Copper	0.07
Iron	0.09
Manganese	0.17
Zinc	0.06
Silicon	107
Sulphate	176
Chloride	179
Nitrate	23
Thiamine	33 µg/L
Ribo avin	410 µg/L
Pyridoxin	650 µg/L
Pantothenic acid	1632 µg/L
Niacin	7875 µg/L
Biotin	13 µg/L
Vitamin B_{12}	0.1 µg/L
Folic acid	82 µg/L
Meso-inositol	10.1
Choline	18.1
Total polyphenols	172
Anthocyanogens	46
Catechin	5–55
Epicatechin	9–24
Rutin	1–6
Quercetin	5–125
Chlorogenic acid	2–20
Caffeic acid	2–20
Quinic acid	1–5
p-Coumaric acid	1–7
Ferulic acid	2–21
Sinapic acid	1–20
Kampferol	5–20
Myricetin	1
Gallic acid	5–29
p-Hydroxybenzoic acid	5–20
Isohumulones*	10–40
Sulphur dioxide	3.7
Putrescine	130 µg/L
Tyramine	1.69
Histamine	315 µg/L
Purines	134
Pyrimidines	144

Source: based on Moll (1994).
*Diverse closely related molecules are present, many of them being oxidation products.
†The balance is made up of organic acids (e.g. citric, acetic, malic, etc.) and various other fermentation secondary products (e.g. glycerol, propanol, ethyl acetate, iso-amyl acetate). These various components are much more signi cant for avour than wholesomeness.

or ascorbic acid (vitamin C) might be added to counter staling (Postel 1972). There is close regulation concerning the materials that are permitted. For example, in the US this is through the Food and Drug Administration (http://www.fda.gov/). However, the vast majority of the chemical constituents of beer are derived either directly from the malted barley, adjuncts, water and hops, or are produced through the metabolism of yeast during the alcoholic fermentation. In some markets, notably Germany within the German purity law of 1516 (the *Reinheitsgebot*), the raw materials for the production of beer are entirely restricted to malted barley, hops, yeast and water.

All raw materials of malting and brewing are subject to intense scrutiny by maltsters and brewers. The main raw materials of course are barley, hops and water.

Barley

Speci c malting varieties of barley (Fig. 3.1) are employed for beer production, characterised by their high yield of fermentable material that is readily obtainable from the stored starch (Table 3.2). Farmers are obliged to avoid excessive use of fertilisers, for fear of boosting the protein content of the barley – high protein means low starch, which in turn means low levels of fermentable sugar. Farmers are also obliged to be sparing with the use of pesticides and to use only those that are approved. However, in

Fig. 3.1 Barley in the field. The brewer produces beer from the grain.

Table 3.2 Composition of barley.

Component	% of total dry weight
Carbohydrates	78–83
Starch	63–65
Sucrose	1–2
Other sugars	1
Water-soluble polysaccharides	1–1.5
Alkali-soluble polysaccharides	8–10
Cellulose	4–5
Lipid	2–3
Protein	10–12
Albumins and globulins	3.5
Hordeins	3–4
Glutelins	3–4
Nucleic acids	0.2–0.3
Minerals	2
Other	5–6

Source: data derived from Harris (1962).

common with other crops, barley is susceptible to a range of infections and infestations (Briggs 1978).

Aflatoxins originate from some members of the genus Aspergillus, namely *Aspergillus avus, A. parasiticus, A. nomius* and *A. ochraceoreseus* (Moss 2003). (It will be noted that these don't include the strains such as *A. oryzae* that have a role in the production of alcoholic beverages such as sake or as a source of exogenous enzymes for brewers.) The most commonly a atoxin-contaminated foods are corn (maize) and peanuts, but all cereals may be affected. Infection is most commonly associated with post-harvest spoilage, when storage is under inapproporiate conditions of temperature and moisture.

Pesticides have real value in this context. Nonetheless there has been in-depth investigation of alternative ways of treating grain, particularly during storage, such that it does not develop infection. These studies have included the use of anaerobic storage (Baxter & Dawe 1990). Where pesticides are used much will be largely washed off the surface of the grain during steeping (Miyake *et al.* 2002).

It must be emphasised that authorities in most countries have regulations and systems for controlling the nature of pesticides that may be used, and those pesticides have been widely screened for their environmental and health impacts. Any perceived risks of using them are grossly outweighed by the very real problem that can accrue in any cereal from contamination with those micro ora capable of producing mycotoxins and ochratoxins (Petzinger & Weidenbach 2002). One such substance is deoxynivalenol (DON), which is produced by the fungus Fusarium (Wolf-Hall & Schwarz 2002). Brewers (and therefore maltsters) set rigorous standards for the level of DON in barley

and malt, and will not use grain that contains it. Fusarium infection is a bigger risk in wetter climates. Thus it was virtually unheard of in North America until the mid-1990s, when a substantial problem was encountered. The reason was a movement away from the burning of straw stubble after grain had been harvested. This burning, outlawed for supposed environmental damage, had served the valuable function of destroying Fusarium spores. Once burning was banned, it meant that the Fusarium was enriched in the soil and readily available to spoil crops the subsequent year.

Woller and Marjerus (1982) and Marjerus and Woller (1983) failed to detect any mycotoxins in a diversity of beers (detection limit 1–2 μg/L). It is not impossible to nd nite levels of mycotoxins – see for example Payen *et al.* (1983). However, provided all parties adhere to the strictest standards of hygiene from eld to glass, and the grain is maintained under the appropriately low levels of moisture and temperature, then this is not an issue.

Hops

The number of brewers employing whole hops (Fig. 3.2) is dwindling, with many using processed forms such as pellets or extracts made with liquid carbon dioxide. In any event, quantitatively the hop affords a minor fraction of the overall composition of beer, albeit a very important one in terms of quality. As we shall see, hops also offer intriguing possibilities from a health perspective.

Fig. 3.2 Hops. Reproduced courtesy of Yakima Chief Inc. (www.yakimachief.com).

Hops, perhaps even more so than barley, are prone to disease and infestation (Neve 1991). Accordingly they almost invariably demand some form of protection during their cultivation, with the same considerations as given above for barley.

The gross composition of hops is shown in Table 3.3. One advantage to the use of extracts of hops is that they have a somewhat lower nitrate content than the parent plant, nitrate presenting a potential cancer risk by comprising a precursor of nitrite. Even so, the contribution of nitrate to the daily human intake coming from any form of hops (or indeed beer) is extremely low in comparison to other sources.

Water

As the vast majority of beers are more than 90% water, its composition is of critical concern to the brewer. Any water that will end up in the beer or that will be in contact with tanks, pipes, etc. through which the process stream passes, must be of the highest chemical and microbiological quality. The water must ful l all legal requirements both chemically and microbiologically as well as satisfy the brewer's standards for clarity and lack of colour, taste and smell. Most, if not all, countries have their regulations concerning the quality of water. In the UK water quality is in the province of the Department for Environment, Food and Rural Affairs (http://www.defra.gov.uk/environment/water/index.htm). In the US potable water must satisfy the National Primary Drinking Water Regulations established by the Environmental Protection Agency (Table 3.4). Additionally there are National Secondary Drinking Water Regulations (Table 3.5). The latter are non-enforceable guidelines (though states may choose to adopt them as enforceable standards) regulating contaminants that may cause cosmetic effects (such as skin or tooth discolouration) or aesthetic effects (such as taste, odour or colour). The World Health Organization publishes *Guidelines for Drinking Water Quality* (http://www.who.int/water_sanitation_health/).

Table 3.3 Composition of hops.

Component	% of total dry weight
Resins	17
Essential oils	0.6
Tannins	4.5
Monosaccharides	2.5
Pectin	2.5
Amino acids	< 0.2
Proteins	17
Lipids and wax	3.5
Ash	1
Cellulose, lignin, etc.	45

Source: based on Hough *et al.* (1982).

Table 3.4 National Primary Drinking Water Regulations, United States (as from http://www.epa.gov/safewater/mcl.html#mcls).

Microorganisms				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Cryptosporidium	zero	TT[3]	Gastrointestinal illness (e.g. diarrhoea, vomiting, cramps)	Human and animal faecal waste
Giardia lamblia	zero	TT[3]	Gastrointestinal illness (e.g. diarrhoea, vomiting, cramps)	Human and animal faecal waste
Heterotrophic plate count	n/a	TT[3]	HPC has no health effects; it is an analytic method used to measure the variety of bacteria that are common in water. The lower the concentration of bacteria in drinking water, the better maintained the water system is	HPC measures a range of bacteria that are naturally present in the environment
Legionella	zero	TT[3]	Legionnaire's disease, a type of pneumonia	Found naturally in water; multiplies in heating systems
Total coliforms (including faecal coliforms and *E. coli*)	zero	5.0%[4]	Not a health threat in itself, it is used to indicate whether other potentially harmful bacteria may be present[5]	Coliforms are naturally present in the environment; as well as faeces; faecal coliforms and *E. coli* only come from human and animal faecal waste.
Turbidity	n/a	TT[3]	Turbidity is a measure of the cloudiness of water. It is used to indicate water quality and ltration effectiveness (e.g. whether disease-causing organisms are present). Higher turbidity levels are often associated with higher levels of disease-causing microorganisms such as viruses, parasites and some bacteria. These organisms can cause symptoms such as nausea, cramps, diarrhoea, and associated headaches	Soil runoff
Viruses (enteric)	zero	TT[3]	Gastrointestinal illness (e.g. diarrhoea, vomiting, cramps)	Human and animal faecal waste

Disinfection byproducts				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Bromate	zero	0.010	Increased risk of cancer	Byproduct of drinking water disinfection
Chlorite	0.8	1.0	Anemia; infants and young children: nervous system effects	Byproduct of drinking water disinfection
Haloacetic acids (HAAs)	n/a[6]	0.060	Increased risk of cancer	Byproduct of drinking water disinfection
Total trihalomethanes (TTHMs)	none[7]; n/a[6]	0.10; 0.080	Liver, kidney or central nervous system problems; increased risk of cancer	Byproduct of drinking water disinfection

Disinfectants				
Contaminant	MRDL[1] (mg/L)[2]	MRDL[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Chloramines (as Cl_2)	MRDLG=4[1]	MRDL=4.0[1]	Eye/nose irritation; stomach discomfort, anaemia	Water additive used to control microbes
Chlorine (as Cl_2)	MRDLG=4[1]	MRDL=4.0[1]	Eye/nose irritation; stomach discomfort	Water additive used to control microbes
Chlorine dioxide (as ClO_2)	MRDLG=0.8[1]	MRDL=0.8[1]	Anaemia; infants and young children: nervous system effects	Water additive used to control microbes

Inorganic chemicals				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Antimony	0.006	0.006	Increase in blood cholesterol; decrease in blood sugar	Discharge from petroleum re neries; re retardants; ceramics; electronics; solder
Arsenic	0[7]	0.010 as of 23 Jan 06	Skin damage or problems with circulatory systems, and may have increased risk of getting cancer	Erosion of natural deposits; runoff from orchards, runoff from glass and electronics production wastes
Asbestos (bre >10 µm)	7 million bres per litre	7 MFL	Increased risk of developing benign intestinal polyps	Decay of asbestos cement in water mains; erosion of natural deposits
Barium	2	2	Increase in blood pressure	Discharge of drilling wastes; discharge from metal re neries; erosion of natural deposits
Beryllium	0.004	0.004	Intestinal lesions	Discharge from metal re neries and coal-burning factories; discharge from electrical, aerospace, and defence industries
Cadmium	0.005	0.005	Kidney damage	Corrosion of galvanised pipes; erosion of natural deposits; discharge from metal re neries; runoff from waste batteries and paints

(*Continued*.)

Table 3.4 (*Continued.*)

Inorganic chemicals (continued)				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Chromium (total)	0.1	0.1	Allergic dermatitis	Discharge from steel and pulp mills; erosion of natural deposits
Copper	1.3	TT[8]; action level=1.3	Short-term exposure: gastrointestinal distress. Long-term exposure: liver or kidney damage. People with Wilson's disease should consult their personal doctor if the amount of copper in their water exceeds the action level	Corrosion of household plumbing systems; erosion of natural deposits
Cyanide (as free cyanide)	0.2	0.2	Nerve damage or thyroid problems	Discharge from steel/metal factories; discharge from plastic and fertiliser factories
Fluoride	4.0	4.0	Bone disease (pain and tenderness of the bones); children may get mottled teeth	Water additive which promotes strong teeth; erosion of natural deposits; discharge from fertiliser and aluminium factories
Lead	zero	TT[8]; action level=0.015	Infants and children: delays in physical or mental development; children could show slight de cits in attention span and learning abilities. Adults: kidney problems; high blood pressure	Corrosion of household plumbing systems; erosion of natural deposits
Mercury (inorganic)	0.002	0.002	Kidney damage	Erosion of natural deposits; discharge from re neries and factories; runoff from land lls and croplands
Nitrate (measured as nitrogen)	10	10	Infants below the age of 6 months who drink water containing nitrate in excess of the MCL could become seriously ill and, if untreated, may die. Symptoms include shortness of breath and blue-baby syndrome	Runoff from fertiliser use; leaching from septic tanks, sewage; erosion of natural deposits
Nitrite (measured as nitrogen)	1	1	Infants below the age of 6 months who drink water containing nitrite in excess of the MCL could become seriously ill and, if untreated, may die. Symptoms include shortness of breath and blue-baby syndrome	Runoff from fertiliser use; leaching from septic tanks, sewage; erosion of natural deposits
Selenium	0.05	0.05	Hair or ngernail loss; numbness in ngers or toes; circulatory problems	Discharge from petroleum re neries; erosion of natural deposits; discharge from mines
Thallium	0.0005	0.002	Hair loss; changes in blood; kidney, intestine, or liver problems	Leaching from ore-processing sites; discharge from electronics, glass, and drug factories

Organic chemicals				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Acrylamide	zero	TT[9]	Nervous system or blood problems; increased risk of cancer	Added to water during sewage/wastewater treatment
Alachlor	zero	0.002	Eye, liver, kidney or spleen problems; anaemia; increased risk of cancer	Runoff from herbicide used on row crops
Atrazine	0.003	0.003	Cardiovascular system or reproductive problems	Runoff from herbicide used on row crops
Benzene	zero	0.005	Anaemia; decrease in blood platelets; increased risk of cancer	Discharge from factories; leaching from gas storage tanks and land lls
Benzo(a)pyrene (PAHs)	zero	0.0002	Reproductive dif culties; increased risk of cancer	Leaching from linings of water storage tanks and distribution lines
Carbofuran	0.04	0.04	Problems with blood, nervous system, or reproductive system	Leaching of soil fumigant used on rice and alfalfa
Carbon tetrachloride	zero	0.005	Liver problems; increased risk of cancer	Discharge from chemical plants and other industrial activities
Chlordane	zero	0.002	Liver or nervous system problems; increased risk of cancer	Residue of banned termiticide
Chlorobenzene	0.1	0.1	Liver or kidney problems	Discharge from chemical and agricultural chemical factories
2,4-D	0.07	0.07	Kidney, liver, or adrenal gland problems	Runoff from herbicide used on row crops
Dalapon	0.2	0.2	Minor kidney changes	Runoff from herbicide used on rights of way
1,2-Dibromo-3-chloropropane (DBCP)	zero	0.0002	Reproductive dif culties; increased risk of cancer	Runoff/leaching from soil fumigant used on soybeans, cotton, pineapples, and orchards
o-Dichlorobenzene	0.6	0.6	Liver, kidney, or circulatory system problems	Discharge from industrial chemical factories
p-Dichlorobenzene	0.075	0.075	Anaemia; liver, kidney or spleen damage; changes in blood	Discharge from industrial chemical factories
1,2-Dichloroethane	zero	0.005	Increased risk of cancer	Discharge from industrial chemical factories
1,1-Dichloroethylene	0.007	0.007	Liver problems	Discharge from industrial chemical factories
cis-1,2-Dichloroethylene	0.07	0.07	Liver problems	Discharge from industrial chemical factories
trans-1,2-Dichloroethylene	0.1	0.1	Liver problems	Discharge from industrial chemical factories
Dichloromethane	zero	0.005	Liver problems; increased risk of cancer	Discharge from drug and chemical factories
1,2-Dichloropropane	zero	0.005	Increased risk of cancer	Discharge from industrial chemical factories
Di(2-ethylhexyl) adipate	0.4	0.4	Weight loss, liver problems, or possible reproductive dif culties	Discharge from chemical factories

(*Continued.*)

Table 3.4 (*Continued.*)

Organic chemicals (continued)				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Di(2-ethylhexyl) phthlate	zero	0.006	Reproductive dif culties; liver problems; increased risk of cancer	Discharge from rubber and chemical factories
Dinoseb	0.007	0.007	Reproductive dif culties	Runoff from herbicide used on soybeans and vegetables
Dioxin (2,3,7,8-TCDD)	zero	0.00000003	Reproductive dif culties; increased risk of cancer	Emissions from waste incineration and other combustion; discharge from chemical factories
Diquat	0.02	0.02	Cataracts	Runoff from herbicide use
Endothall	0.1	0.1	Stomach and intestinal problems	Runoff from herbicide use
Endrin	0.002	0.002	Liver problems	Residue of banned insecticide
Epichlorohydrin	zero	TT[9]	Increased cancer risk, and over a long period of time, stomach problems	Discharge from industrial chemical factories; an impurity of some water treatment chemicals
Ethylbenzene	0.7	0.7	Liver or kidneys problems	Discharge from petroleum re neries
Ethylene dibromide	zero	0.00005	Problems with liver, stomach, reproductive system, or kidneys; increased risk of cancer	Discharge from petroleum re neries
Glyphosate	0.7	0.7	Kidney problems; reproductive dif culties	Runoff from herbicide use
Heptachlor	zero	0.0004	Liver damage; increased risk of cancer	Residue of banned termiticide
Heptachlor epoxide	zero	0.0002	Liver damage; increased risk of cancer	Breakdown of heptachlor
Hexachlorobenzene	zero	0.001	Liver or kidney problems; reproductive dif culties; increased risk of cancer	Discharge from metal re neries and agricultural chemical factories
Hexachlorocyclope-ntadiene	0.05	0.05	Kidney or stomach problems	Discharge from chemical factories
Lindane	0.0002	0.0002	Liver or kidney problems	Runoff/leaching from insecticide used on cattle, lumber, gardens
Methoxychlor	0.04	0.04	Reproductive dif culties	Runoff/leaching from insecticide used on fruits, vegetables, alfalfa, livestock
Oxamyl (Vydate)	0.2	0.2	Slight nervous system effects	Runoff/leaching from insecticide used on apples, potatoes and tomatoes
Polychlorinated biphenyls (PCBs)	zero	0.0005	Skin changes; thymus gland problems; immune de ciencies; reproductive or nervous system dif culties; increased risk of cancer	Runoff from land lls; discharge of waste chemicals
Pentachlorophenol	zero	0.001	Liver or kidney problems; increased cancer risk	Discharge from wood preserving factories
Picloram	0.5	0.5	Liver problems	Herbicide runoff
Simazine	0.004	0.004	Problems with blood	Herbicide runoff
Styrene	0.1	0.1	Liver, kidney, or circulatory system problems	Discharge from rubber and plastic factories; leaching from land lls

Tetrachloroethylene	zero	0.005	Liver problems; increased risk of cancer	Discharge from factories and dry cleaners
Toluene	1	1	Nervous system, kidney, or liver problems	Discharge from petroleum factories
Toxaphene	zero	0.003	Kidney, liver, or thyroid problems; increased risk of cancer	Runoff/leaching from insecticide used on cotton and cattle
2,4,5-TP (Silvex)	0.05	0.05	Liver problems	Residue of banned herbicide
1,2,4-Trichlorobenzene	0.07	0.07	Changes in adrenal glands	Discharge from textile nishing factories
1,1,1-Trichloroethane	0.20	0.2	Liver, nervous system, or circulatory problems	Discharge from metal degreasing sites and other factories
1,1,2-Trichloroethane	0.003	0.005	Liver, kidney, or immune system problems	Discharge from industrial chemical factories
Trichloroethylene	zero	0.005	Liver problems; increased risk of cancer	Discharge from metal degreasing sites and other factories
Vinyl chloride	zero	0.002	Increased risk of cancer	Leaching from PVC pipes; discharge from plastic factories
Xylenes (total)	10	10	Nervous system damage	Discharge from petroleum factories; discharge from chemical factories

Radionuclides				
Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential health effects from ingestion of water	Sources of contaminant in drinking water
Alpha particles	none[7]; zero	15 picocuries per litre (pCi/L)	Increased risk of cancer	Erosion of natural deposits of certain minerals that are radioactive and may emit a form of radiation known as alpha radiation
Beta particles and photon emitters	none[7]; zero	4 millirems per year	Increased risk of cancer	Decay of natural and man-made deposits of certain minerals that are radioactive and may emit forms of radiation known as photons and beta radiation
Radium 226 and Radium 228 (combined)	none[7]; zero	5 pCi/L	Increased risk of cancer	Erosion of natural deposits
Uranium	zero	30 µg/L as of 8 Dec 03	Increased risk of cancer, kidney toxicity	Erosion of natural deposits

(*Continued*.)

Table 3.4 (*Continued.*)

Notes

1 De nitions

Maximum contaminant level (MCL) The highest level of a contaminant that is allowed in drinking water. MCLs are set as close to MCLGs as feasible using the best available treatment technology and taking cost into consideration. MCLs are enforceable standards.

Maximum contaminant level goal (MCLG) The level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs allow for a margin of safety and are non-enforceable public health goals.

Maximum residual disinfectant level (MRDL) The highest level of a disinfectant allowed in drinking water. There is convincing evidence that addition of a disinfectant is necessary for control of microbial contaminants.

Maximum residual disinfectant level goal (MRDLG) The level of a drinking water disinfectant below which there is no known or expected risk to health. MRDLGs do not re ect the bene ts of the use of disinfectants to control microbial contaminants.

Treatment technique (TT) A required process intended to reduce the level of a contaminant in drinking water.

2 Units are in milligrams per litre (mg/L; equivalent to parts per million) unless otherwise noted.

3 EPA's surface water treatment rules require systems using surface water or ground water under the direct in uence of surface water to (1) disinfect their water, and (2) lter their water or meet criteria for avoiding ltration so that the following contaminants are controlled at the following levels:
- *Cryptosporidium* (as of 1 January 2002 for systems serving > 10,000 and 14 January 2005 for systems serving < 10,000): 99% removal.
- *Giardia lamblia:* 99.9% removal/inactivation.
- Viruses: 99.99% removal/inactivation.
- *Legionella:* No limit, but EPA believes that if *Giardia* and viruses are removed/inactivated, *Legionella* will also be controlled.
- Turbidity: At no time can turbidity (cloudiness of water) go above 5 nephelometric turbidity units (NTU); systems that lter must ensure that the turbidity go no higher than 1 NTU (0.5 NTU for conventional or direct ltration) in at least 95% of the daily samples in any month. As of 1 January 2002, turbidity may never exceed 1 NTU, and must not exceed 0.3 NTU in 95% of daily samples in any month.
- HPC: No more than 500 bacterial colonies per millilitre.
- Long Term 1 Enhanced Surface Water Treatment (Effective Date: 14 January 2005): Surface water systems or (GWUDI) systems serving fewer than 10,000 people must comply with the applicable Long Term 1 Enhanced Surface Water Treatment Rule provisions (e.g. turbidity standards, individual lter monitoring, *Cryptosporidium* removal requirements, updated watershed control requirements for un ltered systems).
- Filter Backwash Recycling: The Filter Backwash Recycling Rule requires systems that recycle to return speci c recycle ows through all processes of the system's existing conventional or direct ltration system or at an alternate location approved by the state.

4 More than 5.0% samples total coliform-positive in a month. (For water systems that collect fewer than 40 routine samples per month, no more than one sample can be total coliform-positive per month.) Every sample that has total coliform must be analysed for either faecal coliforms or *E. coli* if two consecutive TC-positive samples, and one is also positive for *E. coli* faecal coliforms, system has an acute MCL violation.

5 Faecal coliform and *E. coli* are bacteria whose presence indicates that the water may be contaminated with human or animal wastes. Disease-causing microbes (pathogens) in these wastes can cause diarrhoea, cramps, nausea, headaches, or other symptoms. These pathogens may pose a special health risk for infants, young children and people with severely compromised immune systems.

6 Although there is no collective MCLG for this contaminant group, there are individual MCLGs for some of the individual contaminants:
- Trihalomethanes: bromodichloromethane (zero); bromoform (zero); dibromochloromethane (0.06 mg/L). Chloroform is regulated with this group but has no MCLG.
- Haloacetic acids: dichloroacetic acid (zero); trichloroacetic acid (0.3 mg/L). Monochloroacetic acid, bromoacetic acid, and dibromoacetic acid are regulated with this group but have no MCLGs.

7 MCLGs were not established before the 1986 Amendments to the Safe Drinking Water Act. Therefore, there is no MCLG for this contaminant.

8 Lead and copper are regulated by a treatment technique that requires systems to control the corrosiveness of their water. If more than 10% of tap water samples exceed the action level, water systems must take additional steps. The action level is 1.3 mg/L for copper and 0.015 mg/L for lead.

9 Each water system must certify, in writing, to the state (using third-party or manufacturer's certi cation) that when acrylamide and epichlorohydrin are used in drinking water systems, the combination (or product) of dose and monomer level does not exceed the levels speci ed, as follows:
- Acrylamide = 0.05% dosed at 1 mg/L (or equivalent);
- Epichlorohydrin = 0.01% dosed at 20 mg/L (or equivalent).

Table 3.5 National Secondary Drinking Water Regulations, United States (as from http://www.epa.gov/safewater/mcl.html#mcls).

Factor	Permissible level
Aluminium	0.05–0.2 mg/L
Chloride	250 mg/L
Colour	15 (colour units)
Copper	1.0 mg/L
Corrosivity	noncorrosive
Fluoride	2.0 mg/L
Foaming agents	0.5 mg/L
Iron	0.3 mg/L
Manganese	0.05 mg/L
Odour	3 threshold odour number
pH	6.5–8.5
Silver	0.10 mg/L
Sulphate	250 mg/L
Total dissolved solids	500 mg/L
Zinc	5 mg/L

Basic outlines of malting and brewing

Beer is the product of the alcoholic fermentation by yeast of extracts of malted barley (see Figs 3.3 and 3.4).

The sugars that are converted to alcohol for the most part arise from the starch of barley. It was pure happenstance that the first beers were brewed from barley 6000–8000 years ago (Bamforth 2003), but barley has been retained ever since because, unlike other cereals, it retains its husk on threshing. This husk has traditionally formed the filter bed through which the liquid extract of sugars is separated in the brewery.

The starch in barley is enclosed in cell walls and proteins (Fig. 3.5) and these wrappings are first stripped away in the malting process (which is essentially a controlled and limited germination of the barley grains), to leave 85–90% of the starch behind, but in a form accessible for hydrolysis to sugars in brewing. The controlled germination softens the grain, rendering it more readily milled. Unpleasant grainy and astringent characters are also removed. Malt has diverse food uses additional to the production of beer (Table 3.6), and it is not only tastier than barley, but the malting process makes its components more nutritionally available.

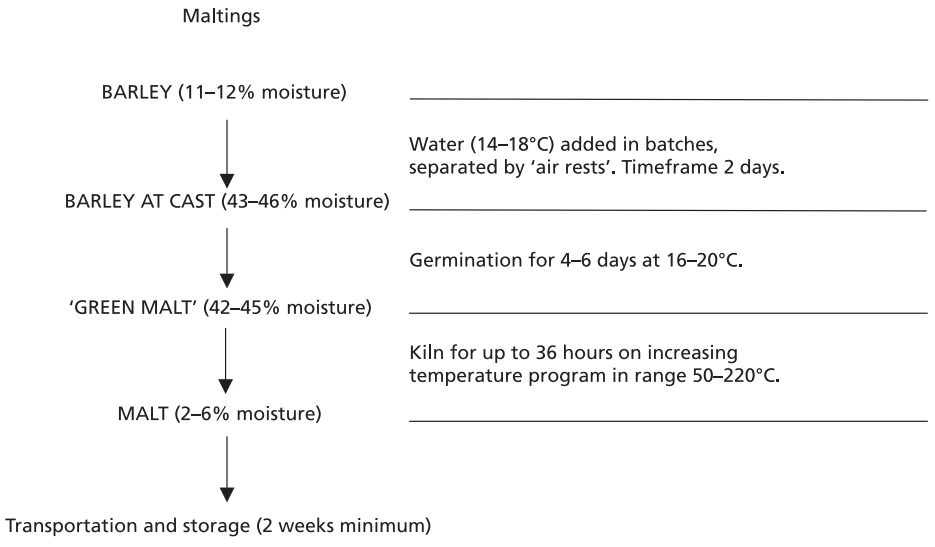

Fig. 3.3 Outline of malting.

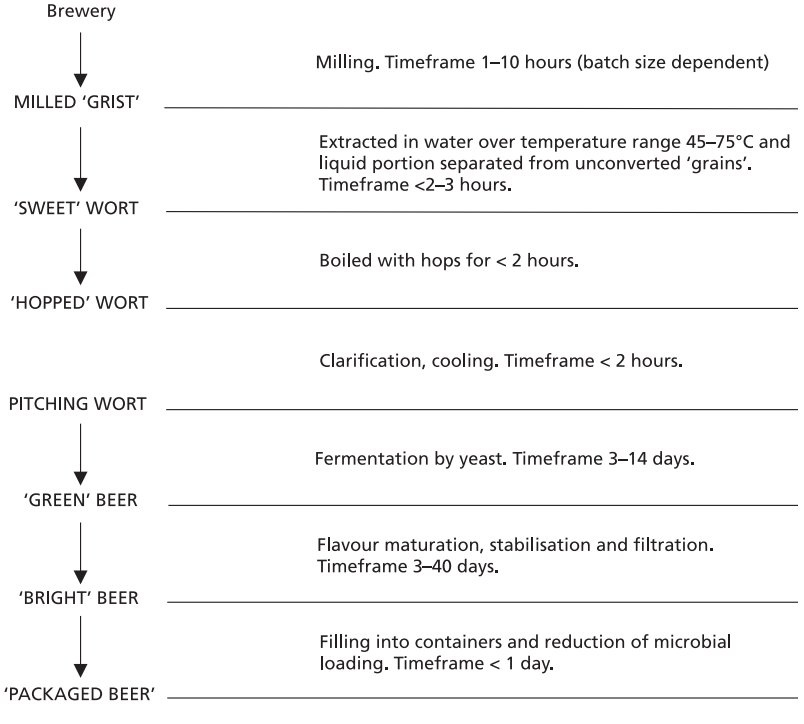

Fig. 3.4 Outline of brewing.

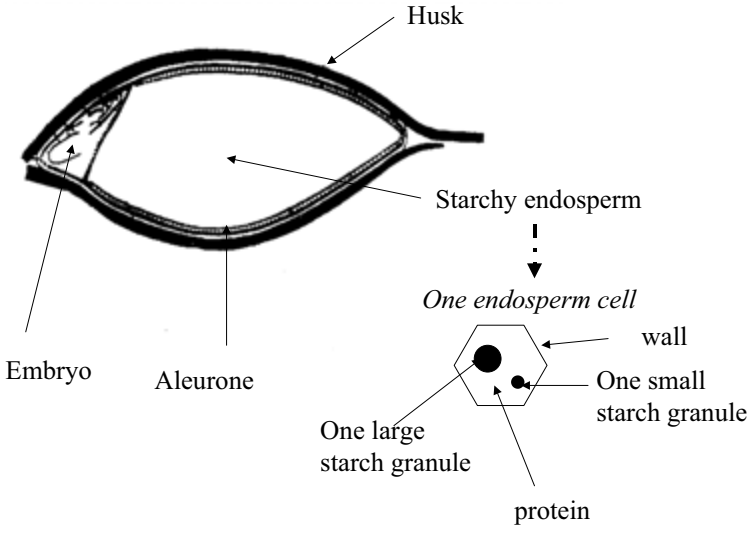

Fig. 3.5 The structure of the barley grain. Only two of the thousands of starch granules are depicted.

Table 3.6 Uses for malt.

Used in foodstuff	Used for colour	Enzymes	Flavour	Sweetness	Nutrition
Beer	✓	✓	✓	✓	✓
Biscuits and crackers	✓	✓	✓	✓	✓
Bread	✓	✓	✓	✓	✓
Breakfast cereal			✓	✓	✓
Cakes	✓		✓	✓	
Coffee alternative	✓		✓		
Confectionery	✓		✓	✓	✓
Desserts	✓		✓		
Gravy	✓				
Ice cream	✓		✓		
Infant food		✓	✓	✓	✓
Malted food drinks		✓	✓	✓	✓
Meat products	✓				
Mincemeat	✓				
Pickles	✓				
Preserves	✓				
Sauces	✓		✓	✓	
Soft drinks	✓		✓	✓	✓
Soups	✓				
Stock cubes	✓				
Whisky		✓	✓		

Source: based on Bamforth & Barclay (1993).

Malting

The first stage of malting comprises the steeping of barley in water at 14–18°C for up to 48 h, until it reaches a moisture content of 42–46%. Raising the moisture content allows the grain to start to germinate, a process that usually takes less than a week at 16–20°C. In germination, the enzymes break down the cell walls and some of the protein in the starchy endosperm (the grain's food reserve), rendering the grain friable. The amylases that break down the starch are produced (or released) in germination and these are important for the subsequent mashing process in the brewery, which is where they convert starch to fermentable sugars. Over the years a number of agents have been employed to assist the maltster to efficiently produce malts that will satisfy the brewer in terms of quality and cost. In a great many markets these materials are banned, even though there is little or no evidence that they are harmful. Thus the natural gibberellin hormones of the barley, which have a key role in stimulating enzyme production, can be supplemented with gibberellic acid (GA), which is produced using industrial fermentation processes (Tudzynski 1999). GA is very closely similar to the native molecules in barley, but nonetheless is outlawed in the Scotch whisky industry and the North American brewing industry. Where it is used, its undesirable impact in excessively stimulating the production of rootlets (which is a waste of potentially fermentable material) has been countered by the use of potassium bromate. A detailed study showed that this latter molecule does not survive in significant quantities into beer (Brewing Research International, unpublished). Very few malting operations nowadays use bromate, but it is widely used in the baking industry where it is used to help bread rise.

There was a time, long ago, when maltsters experimented with the use of formaldehyde, as an agent to remove tannins from the surface of the grain and render the malt less prone to giving the beer a tendency to cloud (haze) formation (Macey 1970). I know of no maltster (or brewer) that has used this material for many years.

One recent development has been the proposal to seed barley with lactic acid bacteria during the malting process (Laitila *et al.* 2002). These bacteria are widely employed in the production of wholesome foodstuffs, e.g. sauerkraut and cheeses, and indeed natural infection of worts in German breweries has a very long history as an exercise in 'naturally' lowering the pH to a more favourable level. The rationale for using lactic acid bacteria in the maltings is that they will consume surface nutrients from the grain, thereby preventing undesirable organisms such as *Fusarium* from prospering.

Germination is arrested by kilning, in which there is a lowering of the moisture content. Regimes with progressively increasing temperatures over the range 50 to perhaps 110°C are used to allow drying to < 5% moisture, while preserving those enzymes that are particularly sensitive to heat. The more intense the kilning process, the darker the malt that is produced and the more roasted, coffee-like and smoky are the flavour characteristics developed. Essentially, malts used for making very pale lager-style beers are

kilned quite gently, whereas those going into the somewhat darker ales are subjected to more heating. The very dark colours in stouts come from the incorporation into the grist of a proportion of malt that is roasted intensely.

One of the biggest concerns with the intense heating of grain raised over 20 years ago was the risk of developing nitrosamines (Havery *et al.* 1981). These molecules have been demonstrated to be carcinogenic in model animal systems, but not so far for man. They are primarily produced when precursors in grain, notably hordenine, react under heat with oxides of nitrogen, which tend to be present in the atmosphere, especially in regions with heavy industry. The malting and brewing industries responded with tremendous alacrity to the 'scare' and within a very short period of time nitrosamine levels had been reduced to very low levels (Sen *et al.* 1996, and see Chapter 5). The key change in practice was the use of indirect kilning such that the nitrogen oxides no longer contacted the malt.

Brewing

Brewing (and malting) is nowadays conducted in well-designed and highly hygienic facilities, for the most part fabricated from stainless steel. The equipment is repeatedly cleaned using regimes of acid or caustic, followed by thorough rinsing with clean water and perhaps a sterilant of the type that would nd use in the domestic kitchen.

In the brewery, the malted grain must rst be milled to generate relatively ne particles, which are then intimately mixed with hot water in a process called mashing. Mashes typically have a thickness of around three parts water to one part malt and contain a stand in the vicinity of 65°C. At this temperature the granules of starch are converted in a transition called gelatinisation into a 'melted' form that is much more susceptible to digestion by amylases. These enzymes are developed during malting, but only start to act once the gelatinisation of the starch has occurred in the mash tun. Some brewers will add starch from other sources, such as unmalted barley, maize or rice, to supplement that from malt. These other sources are called adjuncts. It may be necessary for the brewer to add extra enzymes at this stage, to help deal with some of these adjuncts. Many brewers, though, outlaw the adoption of such 'exogenous' enzymes, even though they are fully recognised as safe and are derived from harmless organisms, e.g. *Aspergillus* and *Pencillium*, which naturally thrive throughout nature, including on the surface of grain (Flannigan 2003).

After a period typically of one hour, the liquid portion of the mash, known as wort, is recovered in a 'lautering' or ltration operation and run to the kettle where it is boiled, again typically for an hour. Boiling serves various functions, including sterilisation of wort, precipitation as 'trub' of proteins and tannins (which would otherwise come out of solution in the nished beer and cause cloudiness), and the driving away of unpleasant

grainy characters that originate in the cereal. Many brewers add some adjunct sugars at this stage, and most brewers also introduce at least a proportion of their hops.

The hops have two principal components: resins and essential oils. The resins (so-called α-acids) are changed ('isomerised') during boiling to yield iso-α-acids, which provide the bitterness to beer. This process is rather inef cient. Nowadays, hops are often extracted with lique ed carbon dioxide and the extract is either added to the kettle or is isomerised outside the brewery for addition to the nished beer (thereby avoiding losses due to the tendency of bitter substances to stick on to yeast).

The oils in hops are responsible for the 'hoppy nose' on beer.

After the precipitate produced during boiling has been removed, the hopped wort is cooled and pitched with yeast. There are many strains of brewing yeast (*Saccharomyces cerevisiae*), and brewers carefully select and maintain their own strains because of their importance in determining brand identity. Yeast needs a little oxygen to trigger off its metabolism, but otherwise the alcoholic fermentation is anaerobic. Ale fermentations are usually complete within a few days at temperatures as high as 20°C, whereas lager fermentations at as low as 6°C can take several weeks. Fermentation is complete when the desired alcohol content has been reached and when an unpleasant butterscotch a-vour, which develops during all fermentations, has been mopped up by yeast. The yeast is harvested for use in the next fermentation. It may be washed with acid to eliminate contaminating microbes that can produce non-volatile nitrosamines (Simpson *et al*. 1988).

In traditional ale brewing the beer is now mixed with a small quantity of hops (to supplement hoppy avour), some priming sugars and isinglass nings, which settle out the solids in the cask. Isinglass is basically hydrolysed collagen, a protein found in many animal tissues. The collagen used for brewing comes from the swim bladders of certain species of sh that breed in the South China Seas. The swim bladders are dried, and then partially hydrolysed using sulphurous acid to generate a solution that has good capability for reacting with beer proteins to form large aggregates, which precipitate and settle. Under Draft Directive 2000/13/EC of the European Union it will in future be required that process aids or ingredients that are included in one of the major allergen groups be labelled. As sh and sh products are in the list that forms an annex to the Directive, this means that isinglass would need to be declared. Phillips (2003) has argued convincingly why this seems preposterous, for the collagen is vastly modi ed during processing and the levels that survive into beer are minimal.

In traditional lager brewing the 'green beer' is matured by several weeks of cold storage, prior to ltering. Filtration generally involves the use of lter aids that keep the lter bed loose and prevent it from clogging up. The two main types of lter aid are kieselguhr and perlite. They leave no residue in the beer.

Nowadays many beers, both ales and lagers, receive a relatively short conditioning period after fermentation and before ltration. This conditioning is ideally performed

at –1°C for a minimum of three days, under which conditions more proteins drop out of solution, making the beer less likely to go cloudy in the package or glass. The long-term stability of beer may also be aided by the use of materials downstream that remove haze-forming protein or polyphenol. For the latter, the one choice is polyvinyl-polypyrrolidone. Protein may be removed in three ways: by adsorption on silica gels that are made from sand, by precipitation with tannic acid derived from gallnuts, or by hydrolysis with the enzyme papain from the pawpaw. This is the same enzyme that comprises meat tenderiser.

The filtered beer is adjusted to the required carbonation before packaging into cans, kegs or glass or plastic bottles. The packaging operations are rigorously designed to ensure that the product is delivered in secure (tamper-proof or at the very least tamper-evident) packages that minimise the opportunity for air ingress (oxygen promotes staling). Modern packaging lines incorporate highly efficient systems to ensure that packages will not contain foreign bodies and furthermore that such items cannot be introduced during the packaging process itself.

Countries such as the UK have regulations which stipulate that packaging materials may not react with or alter the organoleptic properties of the food which they contact (Partington 2003). Aluminium or stainless steel cans, casks or kegs, therefore, are lined with epoxy lacquer coatings to prevent metal from leaching into the relatively low pH beer.

Styles of beer

One fundamental approach to classifying beers is based on whether they are generated by 'top fermentation' or 'bottom fermentation', i.e. whether the yeast congregates at the top of the vessel or sinks to the base. In modern fermenters with their high hydrostatic pressures the distinction is blurred. Top fermentation tends to be at relatively warm temperatures (15–25°C) with the yeast producing higher levels of flavour volatiles such as esters, affording fruity characteristics. Bottom fermentation beers are produced at much lower temperatures (e.g. 6–15°C) and frequently possess significant sulphury notes.

The main top fermentation beers are the ales. Alcohol content will generally be in the range 3 to 7.5% by volume (ABV), and more frequently in the bottom half of the range. The major grist material will be well-modified malt, kilned to relatively high temperatures to impart a copper colour. 'Mild' is a sweeter, darker product, the colour being either due to caramel or in part to a low proportion of heavily kilned malt, though not so much as to impart burnt flavours. It tends to have a lower alcohol content (less than 3.5% ABV) and when bottled may be referred to as 'Brown Ale'. 'Barley wines'

are fermented at very high gravities and so develop much higher alcohol contents (up to 10% by volume). They are usually sold in smaller volumes, in bottles called 'nips'.

Porters (named after the main customers in eighteenth-century London) are traditionally very dark, due to the use of a proportion of roasted barley in the grist, and not overwhelmingly strong (about 5% ABV). Stouts are close relatives of porter, originating in Ireland, with intense colour and burnt, smoky avours due to the use of roasted barley adjuncts, and high bitterness. These robust avour characters are frequently mellowed by the use of nitrogen gas, which 'smoothes' the palate as well as affording the rich, white and creamy foam. Alcohol content may be between 4 and 7%, with up to 10% in Imperial stouts. Sweet stouts are a British variant, of lower alcohol content (up to 4% ABV), with less roast character (often due to the use of caramel and less roast barley as colourant). Trappist beers, from Belgium, are relatively dark, intensely bitter, acidic products of up to 12.5% alcohol by volume. Lambic and gueuze have very complex avours, owing to the use of a more complex micro ora than brewing yeast alone. They are sour (low pH) and usually hazy. Various avourants may be added, including cherries (Kriek) or raspberries (Framboise). The German wheat beers comprise a further class of top fermentation beers. Weizenbier is made from a grist of at least 50% wheat malt. The products are relatively highly carbonated, affording a refreshing nature alongside the fruity and phenolic (clove-like) characters. Often they are cloudy due to yeast, which is employed traditionally to carbonate the bottled product through 'natural conditioning'. The products are relatively lightly coloured (straw-like) and have alcohol contents of 5–6% by volume. Weissbier ('white beer') is much weaker (e.g. 2.8% alcohol by volume), made from a grist of less than 50% wheat malt, with the addition of lactic acid bacteria to generate a low pH of 3.2–3.4. Therefore such beers are quite sour, and may be taken with raspberry or sweet woodruff syrups.

The classic style of bottom fermentation beers originated in Pilsen and is known as Pilsner. It is quite malty with typically 4.8–5.1% ABV and a pale gold colour. Particularly important is the 'late hop character', which is introduced by retaining a proportion of the hops for addition late in the kettle boil. The term 'lager' is used by many, inaccurately, as a synonym for Pilsner. Lager as a term is really an umbrella description for relatively pale beers, fermented and dispensed at low temperatures.

Malt liquor is a term used to describe alcoholic products (6–7.5% ABV) which are very pale, very lightly hopped and quite malty and sweet.

Light beers comprise the most rapidly growing segment of the beer market. 'Standard' beers retain a proportion of carbohydrate that is not fermentable by yeast, whereas a light beer has most or all of this sugar converted into alcohol. These beers therefore have fewer calories, provided that the extra alcohol is diluted to the level found in 'normal' beers.

There are many definitions worldwide about what constitutes low-alcohol products. Perhaps the most stringent is in UK, where non- and low-alcohol beers (NAB/LABs) contain less than 0.05% or 1.2% ABV, respectively. They are produced either by removing the alcohol from a full-strength brew (by techniques such as vacuum distillation or reverse osmosis), or by restricting the ability of yeast to ferment wort (either by making a wort containing very low levels of fermentable sugars or by ensuring that the contact between yeast and wort is at a very low temperature and for a relatively brief time).

The chemistry of beer

Ethanol

As we shall see in Chapter 6, there is increasingly good evidence for the beneficial impact of moderate levels of ethanol on the body. There are several other effects of alcohol on the quality of beer. It contributes directly to flavour, by impacting characters variously described as warming and sweet as well, of course, as *alcoholic*. It also moderates the contribution of other components to flavour by influencing their partitioning between the body of the beer and its headspace ('the nose'). Ethanol also influences the foaming properties of beer (Brierley *et al.* 1996). It lowers surface tension, and so aids bubble formation, but it also competes with other surface-active molecules (notably proteins) for places in the bubble wall, thus detracting from stability of the head.

Beer strength is usually defined in terms of alcohol by volume (ABV), i.e. the number of cm^3 of ethanol per 100 cm^3 of beer. Sometimes alcoholic strength is described in terms of weight per volume. As the specific gravity of ethanol is 0.79, this means that a beer that contains 5% alcohol by volume has approximately 4% alcohol by weight. One of the most relevant examples to use by way of illustration is the so-called '3–2 beer' in Utah. Most of the beer in that US state is in this category, which refers to the fact that it contains no more than 3.2% *by weight*. This is of course 4% when quoted on the basis of volume.

Another way of describing the strength of a beer is on the basis of its 'original gravity' (known as 'original extract' in the US). This is variously quoted on the basis of specific gravity or, increasingly commonly, degrees Plato. It is basically a measure of the strength (approximating to the sugar content) of the wort prior to fermentation. During fermentation, the fermentable sugars are converted into alcohol, leaving behind that proportion of the solubilised starch that is not fermentable. Sugar solutions have a high specific gravity (weight per unit volume), as compared to water (1 mL of which weighs 1 g – i.e. the specific gravity is 1.00) and to ethanol (specific gravity 0.79). Thus there is a fall in specific gravity during fermentation and the final specific gravity of a beer reflects the balance between ethanol and the residual unfermentable 'dextrins' (see

later). By measuring the speci c gravity and ethanol content and putting the values into an equation, the brewer can calculate the original extract, that is, the original strength of the wort.

One degree Plato basically represents a 1% by weight solution of sugars. Thus a wort that is 10° Plato is the equivalent of a 10% sugar solution. A 12°P wort is a 12% sugar solution. If they contain the same proportion of fermentable sugars, then the latter would go on to give a more alcoholic beer. For most beers the sugars originate from malted barley, but some brewers use adjuncts. Thus, for instance, if the grist comprised 70% malt : 30% corn syrup, then, when compared to one of the same strength in degrees Plato derived from an all-malt grist, the former would contain less of the other components that are derived from malt (protein, vitamins, polyphenols, bre, etc.). Thus, although a knowledge of the original gravity of a beer is useful for 'normalising' analytical data on beers, it is important to bear in mind that the exact nature of the grist has a key role to play.

Compared to other alcoholic beverages, beer contains relatively low levels of ethanol. In the UK the mean alcohol content of all beers is 4.1% whereas in the US the average alcoholic strength is 4.6% ABV. Table 3.7 illustrates the typical alcohol content of a diversity of other beverages. Naturally, those of higher alcohol content are consumed in smaller servings. However, there is an obviously greater risk with the drinks of higher alcohol content. Thus, if a whisky is poured without the use of an optic, then a 'heavy hand' delivering 30 mL rather than the standard unit of 25 mL has a profound effect on the amount of alcohol being given. In the vast majority of instances the amount of alcohol being served in the form of beer is inherently self-regulated. If on draft it is de ned as the volume of the glass (e.g. pint or half-pint) whereas if in small pack it is determined by the size of the container (viz. bottle or can). Of course beers do vary substantially from brand to brand in their alcoholic strength (see Tables 3.8–3.11); however, the vast majority are in the range 3–6%. The average alcohol content of beers on a national basis is given in Table 3.12.

Table 3.7 Alcohol content of a range of beverages.

Beverage	Typical alcohol content (% ABV)
Premium beer	4.5
High-strength beer	9.0
Wine	12.0
Whisky	40.0
Gin	40.0
Vodka	45.0
Vermouth	15.0

Source: Bamforth (2003).

Table 3.8 Alcohol content of a range of ales.

Brand	Alcohol (% w/w)
BridgePort India Pale Ale	4.45
India Ale	3.80
Greene King IPA	2.74
Deuchars IPA	3.32
Indian Pale Ale	2.25
James Squire IPA	4.04
Imperial Pale Ale	6.19
Indica IPA	5.76
Full Sail IPA	4.99
Woodstock IPA	4.99
India Pale Ale	4.34
Quail Springs IPA	4.75
Hop Ottin' IPA	5.06
Pyramid Indian Pale Ale	5.15
Wolaver's India Pale Ale	5.09
Rogue XS Imperial Ale	7.26
India Pale Ale	4.74
Old Nick	5.45
Old Horizontal/Barleywine Style Ale	7.90
Hobgoblin Extra Strong Ale	4.02
Rogue XS Imperial Ale	7.26
Maredsous Abbey Ale Dobbel 8.1%	6.24
Ballantine Burton Ale	6.74
Dominion Millennium	8.00
Druid Fluid Barley Wine	6.68
Blue Heron Ale	3.09
Old Brewery Pale Ale	3.88
Organically Produced Ale	4.08
Bass Pale Ale	3.84
Augustinian Ale	4.11
Golden Thread	3.84
Old Speckled Hen	3.74
Sparkling Ale	4.33
St. Andrews Ale	3.60
Young's Ram Rod	3.89
Fuller's ESB	4.63
Fuller's 1845	4.90
Belhaven Scottish Ale	2.93
Old Peculiar	4.46
Speights Pale Ale	3.23
Maudite	5.89
Little Creatures Pale Ale	3.99
Point Pale Ale	4.29
Sierra Nevada Pale Ale	4.32
Three Floyds X-Tra Pale Ale	3.85
Dead Guy Ale	5.63
Black Oak Pale Ale	3.89
Liberty Ale	4.87
Arrogant Bastard Ale	5.67
Full Sail Pale	3.94
Paci c Ridge Pale Ale	4.23
Sunnyside Pale Ale	3.18
Pale Ale	4.22

(*Continued*.)

Table 3.8 (*Continued.*)

Brand	Alcohol (% w/w)
Dog Town Pale Ale	3.79
Single Track Copper Ale	3.56
Old Slugger Pale Ale	3.64
Mirror Pound Pale Ale	4.04
Saranac Pale Ale	4.15
Ruedrich's Red Seal Ale	4.56
Porch Swing Single Ale	4.30
Ruth All American Ale	5.51
Pale Ale	3.90
Syracuse Pale Ale	3.78
Hop Jack Pale Ale	4.15
Union Pale Ale	5.36
Mobjack Pale Ale	3.73
Wild Salmon Pale Ale	3.82
Telemark Ale	3.52
Shelter Pale Ale	3.83
Seneca Trail Ale	4.69
Sam Adams Pale Ale	4.32
Holyoke Dam Ale	3.92
Beast Bitter	4.11
Long Trail Pollenator Ale	3.00
Yuengling Black & Tan	3.53
Ballantine Ale	4.34
Summit Extra Pale Ale	4.11
Jackman's American Pale Ale	4.06
Casta Pale Ale	4.13
Casta Dark Ale	4.37
Pete's Wicked Red Rush	3.78
Ebenezer Ale	4.75
Clancy's Harvest Ale	3.8
Kilkenny Irish Cream Ale	3.31
Smithwick's Irish Ale	3.74
Leinenkugel Red Lager	3.85
Original County Ale	3.92
Irish Red Ale	4.24
Blue Ridge ESB Red Ale	3.41
Irish Red Ale	3.85
Augustiner Lager	3.39
Grolsch Amber Ale	4.22
Hammer & Nail Vienna Style Ale	3.43
McSorley's Ale	4.49
Oktoberfest Marzen Amber	4.12
Avalanche	4.31
Ommegang	6.28
Michael Shea's Irish Amber	3.64
Iron Range Amber Lager	3.81
Bob's 1st Ale	3.17
Killarney's Red Lager	3.83
George Killian's Irish Red	3.88
MacTarnahan's Scottish Style Amber Ale	3.79
Beamish Irish Cream Stout	3.73
Guinness Extra Stout	4.27

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

Table 3.9 Alcohol content of a range of lagers.

Brand	Alcohol (% w/w)
Budweiser	4.82
Becks	5.13
Pilsner Urquell	3.4
Michelob	4.9
Bud Light	3.56
Molson Canadian	5.19
Pete's Signature Pilsner	3.79
CD Pils	3.96
Premium Pils	3.98
Stone Hammer Pilsner	3.37
Stoudt's Pils	3.05
Summer Pils	4.21
Harpoon Pilsner	4.12
Saratoga Pilsner	3.81
Paper City Pilsner	3.91
Prima Pils	4.45
Pils	4.48
Pilsner	4.2
Pilsner	4.66
Zephyrus Pilsner	4.28
Blue Paddle Pilsner	3.75
Golden Pilsner	3.83
Pete's Wicked Helles	3.85
Andechser Spezial Hell	4.81
Lagerbier Hell	4.17
Kaltenberg Hell	4.03
Original Bayrisch Mild	4.16
Urtyp Hell	4.02
Wurziges Helles	3.69
Edelstoff	4.63
Meistersud Spezialbier	4.43
Lowenbrau Original	4.00
Lowenbrau	3.90
Schloss Gold	4.17
Urtyp Hell	3.84
Spezial	4.42
Münchner Hell	3.84
Export Hell	4.26
Original München	4.09
Helles Export	4.35
Lager 2000	4.25
Original Münchner	3.82
Edel-Helles	4.04
Brau Hell	4.16
Münchner Hell	3.86
Appenzeller Bier	3.78
Premium Pils	3.90
Shiner Blonde	3.44
Black Oak Lager	3.84
Golden Ale	3.26
Blonde Ale	4.27
Vienna Style Lager	3.44
Ichiban Special Reserve	3.86
Amstel Lager	3.85
Black Label	4.13

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

Table 3.10 Alcohol content of a range of wheat beers.

Brand	Alcohol (% w/w)
Shiner Winter Ale	4.09
Shiner Hefeweizen	4.12
Pete's Honey Wheat	3.84
Half Ton Hefeweizen	4.20
Hefeweizen	4.28
Eramosa Honey Wheat	3.40
Celis White	3.03
Penn Weizen	4.38
Weizen Bock	6.51
Ramstien Kristall Wheat Beer	3.33
Classic Wheat Beer	4.53
Hefeweizen	4.88
Hefe-Weizen	4.21
Hefeweizen	3.78
Hefe Weizen	3.57
Wheat Beer	3.47
Whistlepin Wheat Ale	4.11
Kristall Weizen	4.05
Wheat Beer	3.65
Bert Grant's Hefeweizen	3.64
Ramstein Blonde Wheat Beer	4.88
Hefeweizen	3.90
Jack Whacker Wheat Ale	3.79
Honey Weiss Bier	3.67
Sunshine Wheat Bear	3.51
Franziskaner Hefe-Weisse	3.95
Paulaner Hefe-Weizen	4.43

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

Carbon dioxide

Carbon dioxide is produced molecule for molecule alongside ethanol during the fermentation of glucose by *Saccharomyces cerevisiae*:

$$\underset{\text{glucose}}{C_6H_{12}O_6} \xrightarrow{\text{yeast}} \underset{\text{ethanol}}{2C_2H_5OH} + \underset{\text{carbon dioxide}}{2CO_2}$$

CO_2 provides the 'sparkle' in beer, affording a pleasurable pain sensation through interaction with the trigeminal nerve. Like ethanol, it plays a substantial role in establishing the quality of beer. Apart from its influence on mouthfeel, CO_2 determines the extent of foamability (foam formation) and naturally influences the delivery of volatiles into the headspace of beers.

Table 3.11 Alcohol content of a range of seasonal beers.

Brand	Alcohol (% w/w)
Pete's Wicked Winter Brew	4.00
Pintail Ale	3.87
Pete's Wicked Summer Brew	3.70
Shiner Summer Stock Koelsch-Style	3.85
Summer Ale	3.04
Young's Summer Beer	3.47
St. Peter's Summer Ale	5.16
Hopback Summer Lightning	4.20
Curve Ball Kolsch Style Ale	3.65
Sommerbrau Kolsch Beer	3.96
Zommerfest Kosch Style Summer Ale	3.97
Spring Brew Speciality Lager	4.78
Sam Adams Spring Ale	4.13
Summerfest	3.59
Sam Adams Summer Ale	4.14
Juju Ginger Ale	2.05
Pete's Wicked Oktoberfest	4.50
Oktoberfest Marzen Amber	4.27
Original Oktoberfest Hacker-Pschorr	4.39
Ayinger Oktober Fest-Marzen	4.21
Sam Adams Oktoberfest	4.57
Frambozen	4.60
Framboise Lambic	1.46
Blue Moon Abbey Ale	4.10
Thomas Kemper Roggen Rye	3.75
Rogue Honey Cream Ale	3.63
Apricot Ale	3.79
Young's Waggledance Honey Ale	3.85
Pete's Wicked Strawberry Blonde	3.99
Samuel Smith's Winter Welcome Ale	4.56
Winterbraun Holiday Ale	5.63
Christmas Brew	4.47
Royal X-Mas Brew	4.51
Jubel	3.98
Victory Dark Lager	4.71

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

Most cans or bottles of beer contains between 2.2 and 2.8 volumes of carbon dioxide (that is, between 2.2 and 2.8 cm^3 of CO_2 is dissolved in every cm^3 of beer). At atmospheric pressure and 0°C, a beer will dissolve no more than its own volume of CO_2 and so achievement of these high levels of CO_2 demands the pressurising of beer. The carbon dioxide that is used to pressurise beer and to bring up the gas content is subject to the same stringent quality control procedures as other raw materials used in the production of beer. The use of gases in this way is not without its risks, and some years ago there was a crisis

Table 3.12 Average alcohol levels of beers in different countries.

Country	Average beer strength (% ABV)
Argentina	4.8
Australia	4.3
Austria	5.1
Belgium*	5.2
Bulgaria	4.8
Canada	5.0
Chile	4.5
Colombia	4.2
Croatia	5.0
Cuba	5.0
Czech Republic	4.5
Denmark	4.6
Finland	4.6
France	5.0
Greece	4.9
Hungary	4.7
Ireland	4.1
Italy	5.1
Japan	5.0
Korea (Republic of)	4.0
Mexico	4.0
New Zealand	4.0
Netherlands	5.0
Nigeria	4.5
Norway	4.5
Philippines	4.7
Poland	5.2
Portugal	5.2
Romania	4.5
Slovak Republic	4.5
Slovenia	4.9
South Africa	5.0
Spain	5.2
Sweden	4.0
Switzerland	4.9
UK	4.1
USA	4.6

*Includes Luxembourg, because of inaccuracies introduced by cross-border trading.
Source: Tighe (2002).

in the soft drinks industry with the detection of benzene in the CO_2 used for carbonating those drinks (see http://www.scotland.gov.uk/food/hazards/haz980701.htm).

Other gases

Two more gases from air can be found in beer. *Oxygen*, which can enter into beer when it is transferred between tanks and during the packaging process unless precautions are

taken, is severely detrimental to quality because it oxidises components of beer, leading to staling and the formation of haze (Bamforth *et al.* 1993).

The most oxidisable molecules in beer are the polyphenols (Owades & Jakovac 1966). On the one hand this serves to protect beer against staling, as these substances act as oxygen scavengers (Walters 1997). However, following their oxidation, they polymerise and crosslink with proteins (the tanning reaction) to form insoluble complexes, which afford an unsightly turbidity (McMurrough & Delcour 1994). Generally speaking, the brewer will err on the side of caution and seek to remove polyphenols as much as possible, by adsorbing them on to polyvinylpolypyrrolidone (PVPP) after the filter process. This will take the total polyphenol content down to less than 100 mg/L, which means that this class of compounds is somewhat less in beer than in products such as red wine and cider, where they contribute to astringency. The PVPP does not enter the beer.

Nitrogen has been added to beer for many years, mostly in Ireland and the UK, to promote foam stability (Lindsay *et al.* 1996). As little as 20 mg of N_2 per litre is sufficient to enhance beer foam quality, levels which are vastly lower than those of CO_2. In small pack beer the nitrogen is usually accompanied by the use of widgets, which promote nucleation. These plastic or metal inserts are perfectly safe, provided they do not display any disintegration in the container. As the atmosphere is some 79% nitrogen it hardly seems that we need worry about the quantities deliberately introduced into beer.

Water

As already stated, most beers comprise 90–95% water and so its composition is critical as a determinant of beer quality. Brewing demands much more water (5–20 times) than the amount which ends up in the beer (UNEP 1996). A lot is needed for cleaning and for raising the steam needed for heating vessels.

The water must contain no taints or hazardous components and a brewer may treat all water coming into the brewery by procedures such as charcoal filtration and ultrafiltration (Katayama *et al.* 1987). The water must also have the correct balance of ions (Taylor 1990). Traditionally ale brewing was established in towns such as Burton-on-Trent in England. The level of calcium in the water of the region is relatively high (about 350 mg/L), and it is claimed that this is good for ales, whereas low levels of calcium, such as the less than 10 mg/L in Pilsen, is best for bottom-fermented lagers. In many places in the world the salt composition of the water is adjusted to match that first used by the monks in Burton in the year 1295, a process known as 'Burtonisation'. Often the brewer will simply add the appropriate blend of salts to achieve this specification. To match a Pilsen-type water it is usually necessary to remove existing dissolved ions by deionisation, perhaps by a filtration technique.

Carbohydrates

While most of the sugar found in wort is fermented to ethanol by yeast, some carbohydrates remain in the beer. Furthermore, extra sugars ('primings') may be added to sweeten the final product.

The carbohydrates surviving into beer from wort are the non-fermentable dextrins and some polysaccharide material. The dextrins are remnants of starch degradation, whereas the polysaccharides derive from cell walls in barley.

Most of the starch in the endosperm of barley survives malting, because it is relatively resistant to enzymatic hydrolysis over anything other than prolonged contact times. However, if starch is gelatinised (which can be likened to melting) by heat treatment, then its constituent molecules, amylose and amylopectin, become much more accessible to enzymes. Thus the start of brewing involves gelatinisation, typically at 65°C, a stage known as 'conversion'. Other cereals, which may be used as adjuncts, have starches that need higher gelatinisation temperatures, e.g. rice and corn, in which starch gelatinises over the range 70–80°C. As stated above, amylase enzymes in the malt degrade the gelatinised starch to fermentable sugars; however, a proportion (usually around 20–25%) remains in the form of unfermentable dextrins. A range of beers is available, which are termed 'super-attenuated' but generally marketed as 'light', in which all of the available starch is converted into ethanol. To effect this, an exogenous heat-stable glucoamylase or pullulanase of microbial origin is often added to the mash or to the fermenter (Bamforth 1985a). It is not obligatory to approach the problem in this way. By judicious use of the mashing regime, and also perhaps the addition of an extract of lightly kilned or unkilned malt to the fermenter, the enzymes native to malt are sufficient to deal with all the dextrins.

The world's first approved, genetically modified (GM) brewing yeast was transformed to express a glucoamylase; however, as yet this strain has not been used in any commercial operation (Hammond & Bamforth 1994). Indeed, no GM material is knowingly or deliberately introduced into beer by any brewer. The only commodities that are based overtly on products of gene technology are some of the commercial enzymes. However, most brewers do not use these and, where they are used, they are added to the mash, and are denatured and precipitated in the kettle boil. Even so, it needs to be stressed that GM commercial enzymes are themselves rigorously screened before approval for commercial use.

Another major carbohydrate component in brewing systems is in the cell walls of barley, a β-glucan comprising β 1–4 links (as in cellulose) but disrupted by occasional β 1–3 links. This molecule is very similar to the β-glucan that is found in oats and which is well known as the 'soluble fibre' championed as part of oat-based breakfast cereals (Lasztity 1998). While one of the main purposes of malting is to degrade the cell walls through the action of β-glucanase enzymes during germination, in practice some glucan

always survives into malt (Bamforth & Barclay 1993). Unless it is properly degraded it renders the wort extremely viscous, with attendant problems in the operations of separating the wort from the spent grains and with downstream beer ltration (Bamforth 1994). Thus some brewers mash-in at low temperatures (say 50°C) to allow the β-glucanase (which is sensitive to heat) to act. Additionally a heat-stable glucanase from bacteria (such as *Bacillus subtilis*) or fungi (such as *Trichoderma reesei* or *Penicillium funiculosum*) may be employed (Bamforth 1985a). Barley has been transformed to express a heat-resistant β-glucanase, but it is not yet cleared for commercial use (Mannonen *et al.* 1993). All of these efforts to eliminate β-glucan are important if production problems are to be avoided, as well as quality problems, for the glucan can cause hazes and precipitates in beer. The beers that will contain the most residual glucan are those that are produced with a high charge of barley adjunct, for instance some well-known stouts. The products of β-glucan breakdown in malting and mashing are not fermentable by yeast, so they survive into beer. Even those beers in which most of the glucan has been converted to low molecular-weight oligosaccharides may be of some value as sources of bre, as it is now understood that any β-linked sugar, no matter how small, may retain some bene cial properties when they reach the lower gut (Schneeman 1999).

β-Glucan is not the only polysaccharide found in the cell walls of barley, the other being arabinoxylan. For reasons that are not entirely understood, this seems to survive malting and brewing more readily than does β-glucan, such that beers tend to contain more arabinoxylan than glucan (Schwarz & Han 1995). It also ranks as soluble bre. In the cell wall the arabinoxylan is covalently linked to ferulic acid (Ahluwalia & Fry 1986). This phenolic acid is released during mashing (McMurrough *et al.* 1996) and survives into beer (unless the beer is made with yeasts, such as those used in the fermentation of wheat-based beers, which contain an enzyme that can decarboxylate the ferulic acid to 4-vinylguiacol, a substance that gives the classic clove-like character to such products). There is huge interest in ferulic acid as an antioxidant (Kroon & Williamson 1999).

Proteins, polypeptides and amino acids

The presence of polypeptide material in beer is important for the contribution it makes to foam (Bamforth 1985b). In the processes of malting and brewing, the native proteins of barley undergo considerable degradation and denaturation, such that those present in the nished beer bear little resemblance to those found in the barley kernel. While polypeptides can be bene cial for foaming, they are detrimental in another respect: they can crosslink with polyphenols to form hazes (McMurrough & Delcour 1994).

The amino acids in beer provide no real bene t to the beer. If present in excess, they potentiate infection of a product by acting as nitrogen sources for spoilage microorganisms. This is why brewers seek to optimise the level of amino acids in wort, so that the yeast uses up all that is readily assimilable.

Lipids

Barley contains about 3% w/w lipid, most of it congregated in the living tissues (embryo and aleurone) (Anness & Reed 1985). Very little lipid, however, survives into beer, making this beverage essentially a fat-free food. This is just as well, from an aesthetic point of view, because lipids are very bad news for beer foam (Bamforth 1985b).

The other adverse in uence of lipids is through their ability to act as precursors of stale avours in beer (Drost *et al.* 1971). The unsaturated fatty acids, such as linoleic acid, may get a good press for their health-giving properties; however, they can be oxidised, ultimately to yield carbonyl compounds that afford aged character to beer. For this reason many brewers try to ensure that as little lipid as possible survives the brewing process and therefore they are meticulous about eliminating solid material at all stages, because the insoluble lipid associates with solids.

Flavours from hops

Hops play several roles in the production of beer, but in particular they are crucial as a source of bitterness (from the hop resins) and aroma (from the essential oils) (Neve 1991).

The chemistry of hop resins is somewhat complex, but of most importance are the α-acids, which can account for between 2% and 15% of the dry weight of the hop, depending on variety and environment. The higher the α-acid content, the greater the bitterness potential. When wort is boiled, the α-acids are isomerised to form iso-α-acids. The latter are much more soluble and bitter than the α-acids. Isomerisation in a boil is not very ef cient, with perhaps no more than 50% of the α-acids being converted to iso-α-acids and less than 25% of the original bittering potential surviving into the beer.

Apart from imparting bitterness to beer, the iso-α-acids also promote foaming by crosslinking the hydrophobic residues on polypeptides with their own hydrophobic side-chains, rendering the foam almost solid-like and able to cling to ('lace') the walls of the drinking glass (Hughes & Simpson 1994). Furthermore they have strong antimicrobial properties and are able to suppress the growth of many Gram-positive bacteria (Fernandez & Simpson 1995). Beer is not entirely resistant to spoilage but certainly the bitter acids have a strong antimicrobial in uence. Other key factors that render beer extremely inhospitable to microbes are its very low pH (typically in the range 3.8–4.6), lack of oxygen, minimal levels of residual nutrients such as sugar and amino acids, its content of ethanol and perhaps the presence of some other antimicrobial constituents such as polyphenols. No pathogens will grow in beer, even alcohol-free beer. All too familiar food scares such as those due to Listeria, *Escherichia coli* O-157 and *Clostridium botulinum* cannot be caused by beer.

Increasingly used nowadays are isomerised resin extracts in which one or more of the side-chains of the iso-α-acids has been reduced, using hydrogen gas in the presence of a

palladium catalyst (Hughes & Simpson 1993). This is because one of the side-chains is susceptible to cleavage by light, yielding a radical breakdown product that reacts with traces of sulphidic materials in beer to produce 3-methyl-2-butene-1-thiol (MBT), a compound that affords a reprehensible skunky aroma. If the side-chain is reduced, it no longer produces MBT. For this reason, beers that are likely to be exposed to light in package (e.g. by being sold in green or clear glass bottles) often contain these modi ed bitterness preparations, which have the added advantage of possessing increased foam-stabilising properties. Once again, these products are fully cleared for safe use.

Hops contain between 0.03% and 3% w/w of oil, which comprises a complex mixture of at least 300 compounds contributing to beer aroma (Gardner 1997).

Phenolic materials

In just the same way that the chemistry of the essential oil fraction of hops is enormously complex, so too is that of the phenolic materials contributed to beer by both barley and hops (Verzele 1986).

We encountered ferulic acid above. Other monomeric phenolic species present in beer include catechin and quercetin. Catechin is rmly accepted as an antioxidant, through its ability both to scavenge oxygen radicals and to inhibit the enzyme lipoxygenase, which promotes the initial breakdown of unsaturated fatty acids to staling carbonyls.

Low molecular-weight contributors to beer aroma

Many people misguidedly believe that most of the avour of beer is derived from its taste. In fact they are detecting the avoursome materials by the nose, there being only four true characters detected on the tongue: bitterness, sweetness, sourness and saltiness (Bamforth & Hughes 1998).

The confusion about what is detected by tongue and what by nose arises because there is a continuum between the back of the throat and the nasal passages. A beer's smell is the net effect of a complex contribution of many individual molecules. No beer is that simple as to have its aroma determined by one or even a very few substances. The perceived 'nose' is a balance between positive and negative avour notes, each of which may be due to more than a single compound from different chemical classes. Some of these volatile substances come from the malt and hops. A great many, though, are side products of the metabolism of yeast.

Tables 3.13–3.18 indicate examples of the classes of compounds that contribute to the aroma of beer and which come from yeast metabolism. They can be classi ed as esters (Table 3.13), alcohols (Table 3.14), organic acids (Table 3.15), vicinal diketones (Table 3.16), sulphur-containing substances (Table 3.17), aldehydes (Table 3.18) and

fatty acids. Additionally we can consider the aroma-contributing compounds arising from the malt and hops (see earlier).

Table 3.13 Some esters in beer.

Ester	Flavour descriptor	Range detectable (mg/L)
Ethyl acetate	Solvent, fruity	8–42
Butyl acetate	Banana, sweet	0.04–0.4
Isoamyl acetate	Banana, apple	0.6–4
Ethyl butyrate	Papaya	0.04–0.2
Isoamyl propionate	Pineapple, aniseed	0.015
Phenylethyl acetate	Roses, honey	0.05–0.2
Ethyl caprate	Goaty	0.01–1
Ethyl caprylate	Apple	0.1–1.5
Ethyl myristate	Vegetable oil	0.4

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

Table 3.14 Some alcohols in beer.

Alcohol	Flavour descriptor	Typical range detectable (mg/L)
Ethanol	Alcoholic, strong	< 5,000–100,000
Propan-1-ol	Alcoholic	3–16
Glycerol	Sweetish, viscous	1,300–2,000
Isoamyl alcohol	Vinous, banana, sweet	30–70
Cis-3-hexen-1-ol	Fresh cut grass	0.025
2-phenylethanol	Roses, bitter, perfumed	8–35
Phenol	Phenol	0.01–0.05
Tyrosol	Bitter	3–40
4-vinylguiaicol	Clove-like	0.05–0.55

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

Table 3.15 Some acids in beer.

Acid	Flavour descriptor	Typical range detectable (mg/L)
Acetic	Vinegar	30–200
Propionic	Milky	1–5
Butyric	Buttery, cheesy	0.5–1.5
Valeric	Sweaty	0.03–0.1
Hexanoic	Vegetable oil	1–5
Hexenoic	Dry leaves	0.01
Oxalic		2–20
Succinic		16–140

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

Table 3.16 Some vicinal diketones and their reduced derivatives in beer.

Material	Flavour descriptor	Typical range detectable (mg/L)
Diacetyl	Butterscotch	0.01–0.4
2,3-pentanedione	Honey	0.1–0.15
2,3-hexanedione	Strawberry	< 0.01
Acetoin	Fruity, mouldy, woody	1–10
3-hydroxy-2-pentanone		0.05–0.07

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

Table 3.17 Some sulphur-containing compounds in beer.

Sulphur compound	Descriptor	Typical range detectable (mg/L)
Hydrogen sulphide	Rotten egg	0.001–0.02
Ethyl mercaptan	Rotting leek, onion, garlic, egg	0.001–0.02
Dimethyl sulphide	Cooked vegetable, corn, blackcurrant	0.01–0.2
Diethyl disulphide	Garlic, burnt rubber	0.001–0.01
Methionyl acetate	Mushrooms	0.013–0.03
Methional	Mashed potato	< 0.05
3-methyl-2-butene-1-thiol	Skunk	0.00001–0.03

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

Table 3.18 Some aldehydes in beer.

Aldehyde	Flavour descriptor	Typical range detectable (mg/L)
Acetaldehyde	Green apples	2–20
Butyraldehyde	Melon, varnish	0.03–0.2
3-Methylbutanal	Unripe banana	0.01–0.3
Hexanal	Bitter, vinous	0.003–0.07
trans-2-nonenal	Papery, cardboard	0.00001–0.002

Source: most numbers given in Tables 3.13 to 3.18 are derived from Moll (1991).

4 The Basics of Human Nutrition

If we are to make reasoned judgements on the interrelationship of beer and human health, then it is important that we first consider the key elements of nutrition.

Essentially our bodies require, in the correct balance, the key nutrients for healthy functioning and development. Additionally the diet should be devoid of materials that are damaging. In this context there may be components of our daily intake that, while not of themselves essential nutrients, may serve to counter negative impacts of adverse food constituents or materials present in the environment. For more detailed considerations of human nutrition the reader is referred to Boyle and Zyla (1996).

Our bodies need food to provide energy (calories) and the building blocks of our tissues (notably amino acids), for the most part taken into the body in the form of protein, carbohydrates, lipids, vitamins, minerals and water. Our wellbeing is therefore incontrovertibly related to what we eat and drink, in terms of the content of the essentials, the form in which they are present in the food (e.g. carbohydrate in the form of fibre acts beneficially in a way quite distinct from that carbohydrate that will overtly provide energy through digestion) and the presence or absence of molecules in the food that may be beneficial or damaging to the body.

If any individual component of the diet is present in excess or is insufficient in quantity, then the diet is out of balance.

Energy

The main sources of energy for the human body are carbohydrates, fats and proteins. However, especially in the context of this book, we must stress that alcohol is a source of energy.

Energy in food is quantified on the basis of calories, one calorie being defined as the amount of heat required to raise the temperature of one gram of water by one degree Celsius. It is customary to talk in terms of kilocalories (or Calories with a capital C) which equate to 1000 calories. These days it is more scientifically correct to talk in terms of kilojoules, for the joule has replaced the calorie as the primary unit of energy under the international system of units (SI). (Incidentally, James Prescott Joule, 1818–89, after whom the unit of energy was named, was a member of a famous Staffordshire brew-

ing family.) One joule is de ned as the amount of energy exerted when a force of one newton is applied over a displacement of one metre. It is the equivalent to one watt of power radiated or dissipated for one second. However, calorie is so widely known and used as a term that I employ it here: the term calorie is proving impossible to shake from popular parlance. The reader should be warned that often calorie (without the capital C) is employed in the literature rather than kilocalorie.

The number of calories in a foodstuff can be determined in the laboratory by combustion. However the 'true' calori c content of a food as it pertains to the diet depends on the extent to which those calories are available to the body.

This applies to all components of the diet. Just because something is present in high quantity in a foodstuff it does not necessarily follow that it will get into the body to exert any effect. Many factors may impact, including the form in which the nutrient is present in a food. A metal such as iron may not be assimilated if it is attached to some other component of the diet that passes straight through the gut. Much of the modern work on antioxidants is awed in this way. For example, only if the speci c antioxidants get into the body will they get to the key site where they are able to act.

Returning to carbohydrates, those such as starch and sugar are almost completely digested and oxidised by the body and they are ascribed a calori c value of 3.75 kcal/g. Fats, which are digested up to 95%, afford a higher energy level (9 kcal/g) because they are less oxidised than the carbohydrates. The calori c value of protein is generally held to be similar to that of carbohydrate, at 4 kcal/g. Ethanol is ascribed a calori c value of 7 kcal/g, indicating that, molecule for molecule, it is an extremely rich source of energy, second only to fat.

If calories in excess of those needed to maintain the body in equilibrium are taken in, then the surplus will be built up in the form of fat, for the simple reason that, pound for pound, fat is a richer energy store than is starch or protein. The converse applies: enhanced energy demand through exercise will 'burn up' fat provided that the extra calorie requirement is not met from fresh food intake.

We will address the calorie composition (and other key analytical measures) of a range of foodstuffs, including beer, in the next chapter. In North America groups including the National Academy of Sciences and the Institute of Medicine collaborated on the establishment of dietary reference intakes (DRIs). The precise requirement that a human will have for the various components of the diet will differ, depending on issues such as age, sex, climate, activity and weight. Individuals, too, will differ to varying degrees in their metabolic activity. Pregnant and breast-feeding women will need more of each type of nutrient. The DRIs re ect some of these differences. As this is a book dealing with beer, I will restrict consideration to adults over the age of 18 (see Table 4.1). (The reader must bear in mind that the legal drinking age in some countries, including the US, is higher than 18, at 21.)

Table 4.1 Nutritional recommendations.

	Men			Women		
	19–24	25–50	51+	19–24	25–50	51+
Weight (kg)	72	79	77	58	63	65
Height (cm)	177	176	173	164	163	160
Resting energy expenditure (kcal/day)*	1780	1800	1530	1350	1380	1280
Average energy allowance (kcal/kg)†	40	37	30	38	36	30
Average energy allowance (kcal/day)†	2900	2900	2300	2200	2200	1900
Protein (g)	58	63	63	46	50	50
Vitamin A (retinol equivalents)	1000	1000	1000	800	800	800
Vitamin D (µg)	10	5	5	10	5	5
Vitamin E (mg)	10	10	10	8	8	8
Vitamin K (µg)	70	80	80	60	65	65
Vitamin C (mg)	60	60	60	60	60	60
Thiamine (mg)	1.5	1.2	1.2	1.1	1.1	1.0
Ribo avin (mg)	1.7	1.7	1.4	1.3	1.3	1.2
Niacin (mg equiv)	19	19	15	15	15	13
Vitamin B_6 (mg)	2	2	2	1.6	1.6	1.6
Folate (µg)	200	200	200	180	180	180
Vitamin B_{12} (µg)	2	2	2	2	2	2
Calcium (mg)	1200	800	800	1200	800	800
Phosphorus (mg)	1200	800	800	1200	800	800
Magnesium (mg)	350	350	350	280	280	280
Iron (mg)	10	10	10	15	15	10
Zinc (mg)	15	15	15	12	12	12
Iodine (µg)	150	150	150	150	150	150
Sodium (mg)	>500	>500	>500	>500	>500	>500
Chloride (mg)	>750	>750	>750	>750	>750	>750
Potassium (mg)	>2000	>2000	>2000	>2000	>2000	>2000
Chromium (µg)	50–200	50–200	50–200	50–200	50–200	50–200
Molybdenum (µg)	75–250	75–250	75–250	75–250	75–250	75–250
Copper (mg)	1.5–3	1.5–3	1.5–3	1.5–3	1.5–3	1.5–3
Manganese (mg)	2–5	2–5	2–5	2–5	2–5	2–5
Fluoride (mg)	1.5–4	1.5–4	1.5–4	1.5–4	1.5–4	1.5–4
Selenium (µg)	70	70	70	55	55	55

*REE represents the energy expended by a person at rest.
†Rounded values that take into consideration an estimated degree of daily physical activity.
Note: For a diet containing alcohol the recommendation is that the population average should have 15% of total dietary energy in the form of protein, 47% as carbohydrate and 33% as fat.
Source: Boyle & Zayla (1996) and, in turn, based on the recommendations of the National Academy of Sciences.

The values in Table 4.1 presuppose 'normal' conditions of health and activity. The number of calories required will vary depending upon the amount of physical exertion. For a male the range might be 2500 through to 5000 kcal per day for the most physically demanding lifestyles. Clearly a foodstuff rich in lipid, and to an only slightly lesser extent alcohol, allows the consumer to take in the energy in a more concentrated form. This must be balanced with satisfying the other nutritional needs as delineated in Table 4.1. Balance is the key word. There are real concerns, for instance, about the tendency of people to shift to sugar-rich drinks as an alternative to, for example, milk. A consequence might be a de ciency in the intake of calcium.

In the US, dietary recommendations are also encapsulated within a food pyramid (Fig. 4.1), which was developed by the US Department of Agriculture. The higher up in the pyramid, the more sparing the intake should be. Its emphasis is a plant-based diet high in bre, rich in vitamins and minerals, and low in fat. Beer as a grain-based foodstuff clearly would feature in the lower part of the pyramid, accepting that considerable processing has taken the added-value product away from the whole grain.

Other pyramids exist. Two of relevance are the Mediterranean pyramid (Fig. 4.2) and the California pyramid (Fig. 4.3). The former recognises the so-called French Paradox (see Chapter 6), which describes the lower than expected incidence of heart disease and some cancers in Mediterranean countries. This has been ascribed by some to antioxidants but by others to alcohol. In particular much has been written about the merits of red wine

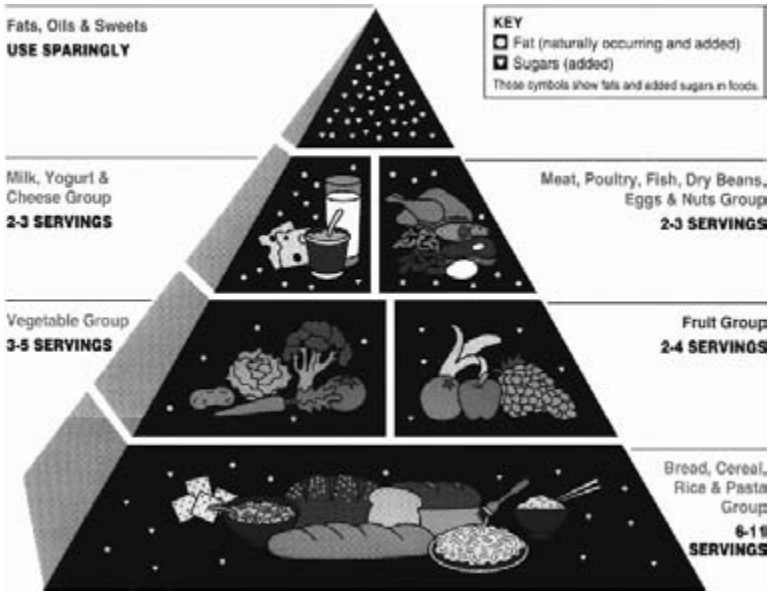

Fig. 4.1 The food pyramid. Reproduced courtesy of US Department of Agriculture and US Department of Health and Nutrition Services.

Fig. 4.2 The Mediterranean food pyramid. Reproduced courtesy of Chef Depot (www.chefdepot.com).

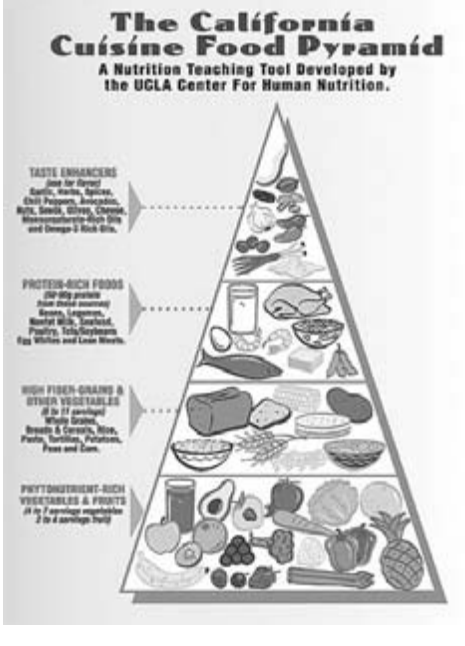

Fig. 4.3 The California food pyramid. Reproduced courtesy of David Heber, MD, PhD, *The Resolution Diet,* Avery Publishing Group, 1999.

in this context. Notwithstanding, the Mediterranean pyramid refers to 'wine in moderation'. It also reinforces messages about exercise underpinning the correct diet.

Phytonutrients

The importance of antioxidants is highlighted in the California pyramid, with the baseline here occupied by foodstuffs, notably fruits and vegetables, which are rich in these and other 'phytonutrients' (i.e. plant-derived nutrients). People living on plant-rich diets generally appear to have lower incidence of disease. This has prompted a search for the active ingredients, of which some are undoubtedly antioxidants. Others may regulate enzyme action and in uence the production or elimination of relevant components. Thus there has developed a large market for herbal supplements. It is in this context that attention has been paid to the hop (see Chapter 6).

Phytochemicals are de ned by the US Food Administration as substances of plant origin that may be ingested by humans daily in gram quantities and which exhibit the potential for modulating metabolism such as to be favourable for cancer prevention and cardiovascular protection (Rincon-Leon 2003). The word 'nutraceutical' has crept into common parlance.

For those preferring their phytonutrients in food – as opposed to supplement – form, Gollman and Pierce (1998) offer one useful recipe book. The authors endeavour to present their recipes from an underpinning scienti c perspective. Alas, beer is not featured. Wine is – yet we will discover in Chapter 6 that beer is likely at least the equal of wine from a health perspective.

Carbohydrate, fat and protein

Although carbohydrate, fat and protein are interchangeable through pathways of intermediary metabolism in the body, the relative amounts of each are not irrelevant. Carbohydrates, then, can 'spare' protein if they are present in adequate quantities. If they are not, then the body will use protein, which is a key component of muscles and other body tissues. Health experts suggest that about 60% of calorie intake should be as carbohydrate. Even within a category, there can be signi cant differences. More complex forms of carbohydrate, e.g. starch, will linger in the body longer than will simpler sugars, allowing the growth of microbes to take place and the attendant enrichment of vitamins in the uxing food. The converse can apply. Some individuals are lactose-intolerant, with this sugar being poorly absorbed and leading to attendant diarrhoea.

For proteins, a key feature of their value in the diet is their relative content of the various amino acids. The best proteins are those containing all of the essential amino

acids (which the human body cannot synthesise) presupposing that those proteins are indeed taken up by the body. Meat, fish, milk and egg proteins are generally good. Barley protein is relatively deficient in two amino acids, lysine and (to a lesser extent) threonine, though high lysine variants have been developed (Kasha *et al.* 1993).

Of course most diets don't usually contain just a solitary source of protein, and generally there is an appropriate mix of animal and vegetable proteins.

The fats provide the essential fatty acid, linoleic acid, which the human body cannot synthesise. Unsaturated fatty acids of this type are associated with a lower incidence of coronary heart disease: they lower cholesterol levels. Beer is essentially fat free.

Vitamins

Vitamins are organic substances that the human body cannot synthesise itself and which must be provided in the diet (Finglas 2003). They have various functions in the body and are customarily divided into the water-soluble vitamins and the fat-soluble vitamins; they are summarised in Table 4.2. For the most part they are not required in very large quantities, but it must be borne in mind that the composition of the food matrix in which they are present can impact on their availability. One example is the higher requirement

Table 4.2 Vitamins and their significance.

Vitamin	Notes
Fat soluble	
A (retinol)	Not present in plants, but precursor β-carotene is, and this can be converted by human to retinol. Shortage leads to blindness, bone/teeth failures of development; diseases of cells in throat, nose and eyes leading to infection risk. Excess toxic
D	Actually can be formed in skin by contact with light. Needed for efficient use of calcium and phosphate. Deficiency causes rickets. Can be formed by irradiation of ergosterol from yeast
E	α-Tocopherol. Antioxidant, protecting e.g. unsaturated fatty acids and vitamin A
K	Needed for normal blood clotting
Water soluble	
C	Ascorbic acid. Deficiency causes scurvy. An antioxidant, e.g. for beer
B_1	Thiamine. Deficiency causes beri-beri. Sensitive to sulphur dioxide that is sometimes used for preserving beer. Like all the B vitamins a very good source is yeast and cereal germ and bran, e.g. from barley and wheat
B_2	Riboflavin. Very sensitive to light
Niacin	Deficiency causes pellagra
B_6	Pyridoxine. Reduces risk of cardiovascular disease and osteoporosis. No recognised deficiency disease
Pantothenic acid	Symptoms in case of shortage include depression
B_{12}	Shortage causes pernicious anaemia
Folic acid	Prevents certain anaemias
Biotin	Important for healthy nails

for thiamine if alcohol is present at high levels. It is equally important to stress that excessive intake of vitamins may have adverse effects. For the most part this pertains to two of the fat-soluble vitamins, A and D, though B_6 at levels above 50 mg per day or nicotinic acid in excess of 2–6 g per day are of concern for neurological damage and liver damage respectively (Finglas 2003).

Minerals

Table 4.3 lists the requirements of the human for minerals and their various impacts. Minerals comprise only 4–6% of the body (Freeland-Graves & Trotter 2003) and some of them are needed only in vanishing quantities. Calcium, chloride, magnesium, phosphorus, potassium and sodium are the major minerals. Chromium, copper, uoride, iodide, iron, manganese, silicon and zinc are needed in trace quantities. Arsenic, boron, molybdenum, nickel, selenium and vanadium are 'ultra trace' minerals.

Table 4.3 Minerals and their significance.

Mineral	Notes
Calcium	Needed for teeth and bones (e.g. lack causing osteoporosis in older women) and blood clotting. By interaction with phosphate these two minerals mutually antagonise one another's uptake. Also binding with other components will limit uptake, e.g. oxalate
Phosphorus	Needed for teeth and bones and energy metabolism
Magnesium	Needed for nerve and muscle function
Iron	Component of haemoglobin and myoglobin. Uptake may be limited by complex formation, e.g. with phytate and phosphate
Copper	Component of key oxidative enzymes
Cobalt	Part of vitamin B_{12}
Zinc	Needed by several enzymes; implicated in reproduction, growth, skin integrity and wound healing
Sodium	Maintenance of osmotic equilibrium and body uid volume
Chloride	Maintenance of osmotic equilibrium and body uid volume. Also needed in manufacture of stomach hydrochloric acid
Potassium	Helps sodium in regulating ionic balance across membranes
Iodine	Part of thyroid hormone, de ciency causing goitre
Fluoride	Needed for sound teeth and bones
Chromium	Glucose metabolism, insulin sensitivity
Manganese	Cartilage and bone equity, lipid and carbohydrate metabolism
Silicon	Bone calci cation and cartilage formation
Arsenic	Taurine and polyamine metabolism
Selenium	Antioxidant, thyroid hormone metabolism
Boron	Energy utilisation, bone development
Molybdenum	Sulphur and nucleic acid metabolism
Nickel	Production of hormones
Vanadium	Iodine metabolism

Fibre

The term is unfortunate, for not all of the components generally considered under this heading are actually brous. Perhaps 'roughage' after all is no worse a term (Kritchevsky & Bon eld 1995).

The majority of materials considered to be dietary bre are plant cell wall components including celluloses, hemicelluloses (such as are found in the cell walls of barley) and pectins. There can be a further division into soluble and insoluble fractions, though it must be remembered that this refers to what is solubilised in standard laboratory analytical procedures and not necessarily what happens in the gastrointestinal tract.

Insoluble components may serve to delay the digestion of other components via physical blocking. The soluble components, on the other hand, will afford increased viscosity if they are of high molecular weight, thereby lengthening transit time in the gut and also the rate at which digestion products (e.g. glucose) are taken through the gut wall. This may also explain the impact of dietary bre in reducing the absorption of cholesterol.

These materials hold water, lead to a softening of stools and accelerate the passage of the stool through the large intestine. Research in recent years has demonstrated the merits of bre in lowering plasma cholesterol levels, reducing cancer incidence, lessening the need for diabetics to take insulin, and so on. The understanding of the precise structural features in bre which lead to best effect is less than clear (see Johnson 2003). The beer carbohydrates comprising soluble bre (which will include the degradation products of barley cell wall polysaccharides and also the dextrins produced during starch degradation; see Chapter 3) escape absorption in the small intestine, thus becoming nutrients for bacteria located in the large bowel. The importance of these organisms to gut function and health has become well recognised in recent years and has led to the concept of probiotics and prebiotics. Probiotics are organisms, notably lactobacilli and bi dobacteria, which are added to diet to boost the ora in the large intestine. For example they are added to yoghurt (Young 1998). Prebiotics are nutrients that boost the growth of these organisms. These may include oligosaccharides that may promote the growth of the appropriate organisms (Gibson 1999; Roberfroid 2001). Microbes in the large intestine produce methane and other gases as a result of their metabolism, and the atulence experienced after drinking beer may relate to this activity (but see Chapter 6).

It also needs to be borne in mind that materials capable of binding to the bre passing straight through the digestive system will also be less available to the body. This might include certain minerals and vitamins (Prosky 2003).

Water

The human body is almost two-thirds water. Loss of 5–10% of the body weight as water leads to symptoms of dehydration. Evidently the greater the risk of water loss, the greater the need for rehydration. Clearly if the water is also carrying away with it other nutrients, e.g. minerals, then these will need to be replaced in quantities that restore the *status quo*.

Balance

To reiterate: the diet needs to be in balance. And this includes 'trendy' food ingredients – the so-called functional food ingredients. Excessive bre can lead to problems with intestinal gas, perhaps intestinal obstruction, and a reduced absorption of essential minerals such as zinc, iron and calcium. Uptake of minerals can also be restricted by chelating agents such as phytate and oxalate. Polyphenolics can bind metals such as iron and so reduce uptake. Phosphates reduce the uptake of zinc while calcium interferes with assimilation of manganese. Another example is that high levels of antioxidants such as vitamin C can switch over and become pro-oxidants.

As is said more than once in this book, beer should be taken in moderation as part of a balanced diet. The same goes for all other foodstuffs.

5 The Composition of Beer in Relation to Nutrition and Health

In Chapter 2 we encountered the changing opinions on the importance of beer as part of the diet. Seemingly on Captain Cook's ships beer contributed as many calories to the sailors' diets as biscuits (bread) and meat combined (Feeney 1997). Of course this *a priori* signi cance of beer is tilted rather differently nowadays; however, beer can still offer signi cant contributions to the diet, quite apart from its role as a thirst quencher and substantial contribution to the holistic dining experience.

Norris (1946) and Stringer (1946) contributed some of the earliest and most authoritative assessments of the worth of beer to the adult diet. These papers were based on presentations to a joint meeting of the Institute of Brewing and the Nutrition Panel of the Society of Chemical Industry in December 1945. World War II had just concluded and Norris observed that:

> … there has been great activity on the nutrition front, largely as a result of the stress of war, and it is not unpro table to examine the position in regard to beer in the light of recently acquired knowledge of dietary requirements …
>
> Norris (1946)

In the discussion recorded after that meeting, which was held at the historic Horse Shoe Hotel on Tottenham Court Road, Dr S.K. Kon was moved to offer his opinions, recorded as follows:

> The two papers had underlined the nutritional importance of fermented beverages for a civilian community in war. He believed it was an open secret that when Dr Sydenstricker came here from the United States, in 1941, when nutritional problems were very dif cult, he found much less de ciency disease than was expected, and there seemed little doubt that the explanation, or part explanation, was the ribo- avin and nicotinic acid intake from beer, and possibly from tea. In that way this country seemed to have solved one or two nutritional problems more satisfactorily than the otherwise more fortunate USA. But the importance of beer becomes even greater when the nutrition is considered of the more primitive natives such as those of Africa. From the studies carried out there recently it would really seem that the local fermented native beer may be at times almost the sheet anchor of nutrition.

Energy

Too often in nutritional texts all beers are lumped together with one generalised compositional listing. It must be borne in mind, though, that beers can differ enormously in their composition, depending on their strength and how they were made, including the grist materials employed (see Chapter 3). Thus the alcohol content may range from in excess of 10% (v/v) in beers produced in Trappist monasteries to < 0.05% in the alcohol-free products. Most beers worldwide have an alcohol content in the range 3–6% (v/v). Note that ethanol has an energy contribution of 7 kcal per g (c.f. protein 4 kcal/g and carbohydrate 3.75 kcal/g). Additionally, conventionally fermented beers may retain some 25% of the starch in a partially degraded, non-fermentable form which will also contribute to the calorie count. By contrast, so-called light beers generally contain minimal levels of carbohydrate. Some two-thirds of the energy value in a regular beer originates in the alcohol.

Brewers use the following formula (ASBC 1992) to calculate the calori c value of a beer:

kcal in 100 g beer = 6.9(A) + 4(B – C)
where A = alcohol (% by weight), B = real extract (% by weight) and C = ash (% by weight)

The 'real extract' is a measure of the total dissolved solids in the beer. The major components of this are residual unfermented carbohydrates, some protein and ash (inorganics). The myriad of avour components contributes relatively little and can be ignored in this context. The ash has no calori c value and is therefore subtracted from the residual extract number.

Martin (1982) suggested a more exact formula, which takes into consideration more precisely the individual contributions of the major beer components:

Calori c value (kcal/100 mL) = [ethanol (g/100 mL) × 7] + [total carbohydrates (as glucose g/100 mL) × 3.75] + [proteins (g/100 mL) × 4]

Tables 5.1 to 5.4 compare the calori c values of a diversity of beer brands, respectively ales, lagers, wheat beers and seasonal beers.

Ethanol is just as assimilable as other sources of energy (Hawkins & Kalant 1972; Wei *et al.* 1972). Forsander (1998) has amply shown why he claims that, as a source of energy, 'ethanol should be an excellent nutrient'. It is used by the body as ef ciently as other energy sources, it requires no digestion by the body before it enters the bloodstream by diffusion, and it is transferred to all cells without the need for an energy-demanding transport system.

Table 5.1 Calorific value of a range of ales (per 355 mL).

Brand	kcal
BridgePort India Pale Ale	180.6
India Ale	161.7
Greene King IPA	124.2
Deuchars IPA	139.3
Indian Pale Ale	101.6
James Squire IPA	177.5
Imperial Pale Ale	229.9
Indica IPA	211.8
Full Sail IPA	199.7
Woodstock IPA	199.7
India Pale Ale	184.6
Quail Springs IPA	191.9
Hop Ottin' IPA	207.6
Pyramid Indian Pale Ale	220.8
Wolaver's India Pale Ale	202.2
Rogue XS Imperial Ale	278.5
India Pale Ale	174.0
Old Nick	259.8
Old Horizontal/Barleywine Style Ale	346.6
Hobgoblin Extra Strong Ale	165.0
Rogue XS Imperial Ale	278.5
Maredsous Abbey Ale Dobbel 8.1%	222.0
Ballantine Burton Ale	232.0
Dominion Millennium	320.2
Druid Fluid Barley Wine	279.0
Blue Heron Ale	145.3
Old Brewery Pale Ale	152.7
Organically Produced Ale	160.0
Bass Co's Pale Ale	150.1
Augustinian Ale	155.4
Golden Thread	152.6
Old Speckled Hen	163.2
Sparkling Ale	151.3
St. Andrews Ale	139.6
Young's Ram Rod	159.4
Fuller's ESB	183.4
Fuller's 1845	202.6
Belhaven Scottish Ale	128.2
Old Peculiar	177.6
Speights Pale Ale	127.8
Maudite	222.7
Little Creatures Pale Ale	163.5
Point Pale Ale	177.0
Sierra Nevada Pale Ale	170.2
Three Floyds X-Tra Pale Ale	145.8
Dead Guy Ale	207.0
Black Oak Pale Ale	151.0
Liberty Ale	189.7
Arrogant Bastard Ale	238.5
Full Sail Pale	179.8
Paci c Ridge Pale Ale	198.0

(*Continued*.)

Brand	kcal
Sunnyside Pale Ale	150.9
Pale Ale	160.3
Dog Town Pale Ale	165.6
Single Track Copper Ale	154.5
Old Slugger Pale Ale	164.3
Mirror Pound Pale Ale	169.2
Saranac Pale Ale	177.2
Ruedrich's Red Seal Ale	173.6
Porch Swing Single Ale	172.0
Ruth All American Ale	183.5
Pale Ale	169.3
Syracuse Pale Ale	159.2
Hop Jack Pale Ale	169.8
Union Pale Ale	186.3
Mobjack Pale Ale	159.0
Wild Salmon Pale Ale	156.2
Telemark Ale	151.1
Shelter Pale Ale	143.6
Seneca Trail Ale	166.3
Sam Adams Pale Ale	163.7
Holyoke Dam Ale	156.1
Beast Bitter	160.0
Long Trail Pollenator Ale	151.2
Yuengling Black & Tan	151.0
Ballantine Ale	175.2
Summit Extra Pale Ale	156.0
Jackman's American Pale Ale	172.4
Casta Pale Ale	179.0
Casta Dark Ale	190.0
Beamish Irish Cream Stout	131.4
Guinness Extra Stout	152.7

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

Although for many people the focus on alcoholic beverages is the potential negative impacts when consumed in excess, the question of their contribution to obesity is perhaps the major concern, as obesity is associated with many other health problems, including hypertension, cancer, cardiovascular disease and type II diabetes. In the US 39% of men and 36% of women are overweight (National Research Council 1989). The research of Peeters *et al.* (2003) suggests that obesity is at least as dangerous as smoking as a causal agent of death. The US Surgeon-General recently expressed the concern that obesity will soon overtake cigarette smoking as the leading cause of preventable disease and death (see http://www.surgeongeneral.gov/topics/obesity/default.htm).

Most drinkers add alcohol to their normal diet (Prentice 1995) rather than substitute it. Thus total calorie intake is increased and, if not metabolically utilised by exercise,

Table 5.2 Calorific value of a range of lagers (per 355 mL).

Brand	kcal
Budweiser	142
Bud Light	107
Michelob	156.2
Pilsner Urquell	157.5
Pete's Signature Pilsner	161.5
CD Pils	151.9
Premium Pils	149.4
Stone Hammer Pilsner	143.6
Stoudt's Pils	145.5
Summer Pils	168
Harpoon Pilsner	160.1
Saratoga Pilsner	159.4
Paper City Pilsner	145.5
Prima Pils	166.4
Pils	170.6
Pilsner	158.1
Pilsner	172
Zephyrus Pilsner	159.6
Blue Paddle Pilsner	155.7
Golden Pilsner	161.6
Pete's Wicked Helles	163.5
Andechser Spezial Hell	178.2
Lagerbier Hell	148.6
Kaltenberg Hell	148.9
Original Bayrisch Mild	154.3
Urtyp Hell	156.3
Wurziges Helles	152.2
Edelstoff	165.6
Meistersud Spezialbier	173.7
Lowenbrau Original	152.7
Lowenbrau	148.4
Schloss Gold	159.8
Urtyp Hell	149.2
Spezial	170.3
Münchner Hell	146.3
Export Hell	162.1
Original München	145.5
Helles Export	159.0
Lager 2000	150.6
Original Münchner	149.3
Edel-Helles	164.6
Brau Hell	156.4
Münchner Hell	151.6
Appenzeller Bier	141.5
Premium Pils	146.3
Shiner Blonde	141.8
Black Oak Lager	144.0
Golden Ale	128.9
Blonde Ale	166.1
Vienna Style Lager	154.2
Ichiban Special Reserve	145.5
Amstel Lager	146.6
Black Label	137.7
Michelob Ultra	96

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Table 5.3 Calorific value of a range of wheat beers (per 355 mL).

Brand	kcal
Shiner Winter Ale	189.2
Shiner Hefeweizen	168.0
Pete's Honey Wheat	154.6
Half Ton Hefeweizen	172.9
Hefeweizen	173.7
Eramosa Honey Wheat	130.2
Celis White	182.9
Penn Weizen	170.5
Weizen Bock	264.8
Ramstien Kristall Wheat Beer	154.1
Classic Wheat Beer	189.0
Hefeweizen	179.7
Hefe-Weizen	157.6
Hefeweizen	147.5
Hefe Weizen	148.8
Wheat Beer	148.9
Whistlepin Wheat Ale	156.4
Kristall Weizen	157.8
Wheat Beer	145.8
Bert Grant's Hefeweizen	153.4
Ramstein Blonde Wheat Beer	180.5
Hefeweizen	155.5
Jack Whacker Wheat Ale	133.3
Honey Weiss Bier	143.5
Sunshine Wheat Bear	139.8
Franziskaner Hefe-Weisse	151.9
Paulaner Hefe-Weizen	169.0

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

weight gain will result. If a person consumes 1.6 MJ (roughly the level of calories in a couple of pints of beer) more than is needed as an energy supply to maintain bodily functions then this may result in approximately 1 kg per month gain in weight. However 250 calories is 'knocked off' by cycling briskly for 25 minutes, jogging for a similar period, swimming for 30 minutes, gardening for 50 minutes or walking for 60 minutes. (In the days when malting and brewing processes demanded hefty manual labour, such as turning the malt by fork or shovelling out spent grains, the operatives had a generous daily beer allowance. They didn't get fat: the beer rehydrated them and replenished the calories they were burning off.)

Of course it would be totally incorrect to label beer as being a prime factor in causing obesity in moderate drinkers. Any foodstuff loaded with calories will impact and, certainly in a consumer society such as the US with its fast food and generously sized portions, it is likely that for most people alcohol is not the prime source of their excess

Table 5.4 Calorific value of a range of seasonal beers (per 355 mL).

Brand	kcal
Pete's Wicked Winter Brew	170
Pintail Ale	160
Pete's Wicked Summer Brew	163
Shiner Summer Stock Koelsch-Style	150
Summer Ale	123.9
Young's Summer Beer	136.0
St. Peter's Summer Ale	206.7
Hopback Summer Lightning	140.9
Curve Ball Kolsch Style Ale	143
Sommerbrau Kolsch Beer	145
Zommerfest Kosch Style Summer Ale	151
Spring Brew Speciality Lager	186
Sam Adams Spring Ale	172.9
Summerfest	150
Sam Adams Summer Ale	163.2
Juju Ginger Ale	106
Pete's Wicked Oktoberfest	189
Oktoberfest Marzen Amber	178.0
Original Oktoberfest Hacker-Pschorr	178.8
Ayinger Oktober Fest-Marzen	171.1
Sam Adams Oktoberfest	192
Frambozen	192
Framboise Lambic	173
Blue Moon Abbey Ale	183
Thomas Kemper Roggen Rye	167
Rogue Honey Cream Ale	148
Apricot Ale	162
Young's Waggledance Honey Ale	147
Pete's Wicked Strawberry Blonde	160
Samuel Smith's Winter Welcome Ale	183.9
Winterbraun Holiday Ale	230.5
Christmas Brew	165.0
Royal X-Mas Brew	166.3
Jubel	170
Victory Dark Lager	169.2

Sources: Most of the data in this table is reproduced courtesy of Carlos Alvarez & Jaime Jurado (Gambrinus). The data was originally published by Jurado in a series of articles in *The Brewer International*. Most of the remaining information is from http://brewery.org/brewery/library/AlClbinger.html.

calories. Table 5.5 compares the calorie count in a pint of regular beer with that of other components of the diet.

Nonetheless there is considerable interest in so-called Light beers, with their reduced calorie content (Table 5.5). Such brands represent the big growth segment of the brewing sector in the US.

According to MacDonald *et al.* (1993), some 4–6% of the energy intake of the western diet is in the form of alcohol. They highlight that separate studies have led to different

Table 5.5 A comparison of beer with other foodstuffs – energy, protein, fat, carbohydrate and fibre.

Food	Size of serving (weight or volume)	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Fibre (g)
Beer*	UK pint (568 mL)	250	2.8	0	16	ca. 1
Light beer	UK pint (568 mL)	158		0	9	
Cola	12 uid ounces (355 mL)	152	0	0	38	0
Milk	1 cup	150	8	8	11	0
Tea (black)	6 uid ounces (178 mL)	2	0	0	1	0
Coffee (black)	6 uid ounces (178 mL)	4	0	0	1	0
Wine, white	5 uid ounces (148 mL)	100		0	1	0
Wine, red	5 uid ounces (148 mL)	106		0	2	0
Whisky (80 Proof)	1.5 uid ounces (44 mL)	97		0	0	0
Apple	1 medium	81	0	0	21	4
Banana	1 medium	109	1	1	28	3
Cabbage, cooked	0.5 cup	17	1	0	3	2
Carrot, cooked	0.5 cup	35	1	0	8	3
Lettuce, Iceberg	1 cup	7	1	0	1	1
Tomato	1 medium	26	1	0	6	1
Potato, baked	1	220	5	0	51	5
Bread, white	1 slice	67	2	1	12	1
Corn akes	1 cup	102	2	0	24	1
Spaghetti, cooked	0.5 cup	99	3	0	20	1
Sirloin steak, broiled	3 ounces (85 g)	229	23	14	0	
Pork sausage, cooked	3 ounces	314	17	27	1	
Chicken breast, roasted	3 ounces	141	27	3	0	
Egg, raw	1 large	75	6	5	1	
Cod, cooked (dry)	3 ounces	89	19	1	0	
Cheese. Cheddar	1.5 ounces	171	11	14	1	0
Chocolate, milk	1 bar (1.5 ounces)	226	3	14	26	

* For a beer of 12° Plato produced from an all-malt grist. 1° Plato is approximately equal to a 1% solution of carbohydrate in the wort prior to fermentation. The higher this value, the higher will be the concentration of alcohol produced during fermentation (see also Tables 5.1 to 5.4). The amount of carbohydrate left in the beer will be much lower than in the wort; however, that which is not fermented will remain, together with any that is added to the nished beer as a sweetener to balance bitterness.
Source: *Encyclopedia of Foods: A Guide to Healthy Nutrition* (2002).

conclusions concerning the impact of alcohol on body mass index (BMI): those which say there is a positive correlation, those saying that there is a negative correlation, and those claiming no correlation whatsoever. It seems that in many of the studies confounding factors have not been taken into consideration, including physical activity, tobacco use and other lifestyle attributes. Jacobsen and Thelle (1987) refute the notion of the 'beer belly' with their demonstration of a negative correlation between BMI and beer intake. Incidentally BMI is de ned as weight in kg/(height in metres)^2 or [weight in pounds/(height in inches)^2] × 703. Overweight is de ned as a BMI of 25–29.9, and obesity is a BMI of > 30. For those with a BMI > 30 the all-causes risk of mortality is 50–100% higher than for those with a BMI between 20 and 25.

Alcohol consumption bears an inverse relationship to sugar use (Kubler 1990). In fact most studies suggest that at moderate levels alcohol is itself ef ciently used as a

fuel by the liver (Mitchell & Herlong 1986). Gibney *et al.* (1989) suggest that there is an inverse relationship between energy derived from alcohol and that from dietary fat. On the other hand, Le Marchand *et al.* (1989) claim that abstainers consume more vitamins, calcium, fruit and raw vegetables, while drinkers took in more fat (especially polyunsaturates), meat, pickled vegetables and dried fish. It appears as if alcohol is causing a higher metabolic rate (Klesges *et al.* 1994; Orozco & de Castro 1994) perhaps with an increased burning up of fats (Suter *et al.* 1992). Rumpler (1994) from the USDA Human Nutrition Research Center claimed that consuming moderate amounts of alcohol did not cause weight gain or an excess of body fat. He suggested that alcohol may help the body to regulate appetite.

People who consume alcohol but who are not alcoholics appear to add the energy from alcohol to their normal energy intake rather than replace food with alcohol (Jones *et al.* 1982). That this increased energy intake does not necessarily translate into body mass may be because alcohol stimulates the basic metabolic rate (MacDonald *et al.* 1993). So it does not appear that additional calories from alcohol are compensated for by a reduction in calorie intake from other foods (Westerterp-Plantenga & Verwegen 1999). In contrast, pre-dinner drinks in which carbohydrate, protein or fat was the prime energy source led to a reduction in the amount of food eaten during the meal. Richter (1926) and Eriksson (1969), however, presented evidence to suggest that voluntary alcohol intake depresses food consumption in proportion to its energy content. Forsander (1988) showed that ethanol suppresses the consumption of carbohydrate but not fat or protein. There have been various reports that a high carbohydrate/low protein food depresses voluntary alcohol intake, while a low carbohydrate/high protein diet increase it (Hauser & Iber 1989). Candy is recommended to those with a predilection to consume alcohol to excess (Biery *et al.* 1991). Istvan *et al*. (1995) says that those who drink regularly, but each time in relatively small amounts, have lower body weights than those who drink a lot at once or don't drink at all.

Direct studies in which alcohol was 'control fed' to humans showed that, under normal living conditions, moderate alcohol consumption (e.g. 60–75 g alcohol per day, which is equivalent to approximately 2 litres of average strength beer daily) had no measurable impact on energy balance and body weight over a period of approximately one month (MacDonald *et al.* 1993).

Another index of body mass (perhaps of most interest to women) is waist : hip ratio (WHR). Just as for BMI, it has variously been concluded that alcohol lowers (Kaye *et al.* 1990), raises (Lapidus *et al.* 1989) or has no effect (Haffner *et al.* 1986) on WHR.

In a recent investigation, Buemann *et al.* (2002) measured the amount of food consumed by subjects given beer, wine or a carbonated soft drink with the meal. When people were given a designated quantity of each drink there was no significant difference between any of the beverages in respect of impact on the amount of food consumed. However, when they were given less of the drink and allowed to consume the whole

serving or a lesser amount at their will, then the total energy intake (food plus drink) was rather higher for those taking wine as opposed to beer or the soft drink.

Carbohydrate, fat and protein

Beer is essentially fat free. Fats are highly water-insoluble molecules which, when present in foodstuffs, are either in the form of emulsions or within a solid matrix. Beer, of course, is largely water, and most beers contain very few insoluble solids.

A range of carbohydrates can be found in beers. For most beers, the majority of these are the partial degradation products of starch, which generally amount to 20–25% of the original starch. These dextrins (see Chapter 3) will afford calories if the body uses them, but will contribute to the soluble fibre component if they survive to the large gut where they may form part of the feedstock for the microflora. The polysaccharides that originate in the barley cell walls, and their breakdown products, also contribute to the soluble fibre complement. Some sugars may survive fermentation, but if there are sugars in beer it is usually because brewers have added them in small quantities to balance sourness and bitterness.

Although beer does contain some protein, indeed rather more than in other alcoholic beverages, the levels are somewhat lower than in many other foodstuffs. Beer contains the essential amino acids, at levels of the order of 5–10 mg per 100 g (Table 5.6).

Table 5.6 Amino acid composition of beers.

			after Hough *et al.* (1982)		
Amino acid (mg/L)	after Darby (1979)	after Hardwick (1995)	India Pale Ale (10.75 P)	Draught Bitter (10.2 P)	Stout (11.25 P)
Histidine	5.9–20.4	9–50			
Isoleucine	2.1–6.6	5–40		0.06	2.5
Leucine	2.0–10.9	3–60		0.19	2.5
Lysine	0.2–4.4	5–60	2.6	3.1	1.25
Methionine	1.4–2.7	0–10		1.5	
Phenylalanine	3.1–32.4	5–99		3.1	
Threonine	3.7–4.6	0–10		4.1	1.25
Tryptophan	8.6	1–12	11.1	12.9	3.1
Valine	2.9–17.8	5–80		0.19	
Arginine	2.0–9.4	9–110		1.1	
Proline	151–169	± 400	177	178	238
Aspartic	4.5–20.6	6–45		1	1.25
Serine	5.3	2–12		0.9	1.25
Glutamic acid	1.2–6.6	9–50		0.9	
Glycine	8.1–11.5	9–45	1.3	0.5	
Alanine	14.5–21.6	10–130		2.7	
Tyrosine	14.7–28.4	9–80		2.2	
Cysteine	Trace	0–11			
Cystine		0–6			

Water

The recommended daily intake of water for an adult male in temperate climates is 2.5 litres, to be increased in relation to local temperature and/or physical exertion. Nutritionists recommend the consumption of at least eight 8-ounce glasses of water daily. Beer, being at least 90% water, can clearly be a signi cant contributor to water intake. In regions of heavy industry, beer has long been championed. We cannot ignore the fact that alcohol exerts a diuretic effect (see chapter 6). Clearly, though, as beer is a drink customarily of lower alcohol content than other alcoholic beverages it is the more useful as a source of water. The lower alcohol beers have been promoted as sports drinks, as an opportunity for replenishing water, minerals and energy to the body (Piendl 1990).

Vitamins

The observation that alcohol suppresses the desire to take up calories from other foodstuffs (see above) raises concerns about unbalanced diets, in particular that those who depend on alcohol as a source of calories run the risk of vitamin shortage. In this context beer, with its nite vitamin content, would be a wiser beverage than other alcoholic drinks (though, of course, it is wisest to use it in moderation as part of a properly balanced diet).

Table 5.7 shows the vitamin content of beers and Table 5.8 that of beer in relation to a range of other foods. Beer can be a valuable source of many of the water-soluble vitamins, notably folate, ribo avin, pantothenic acid, pyridoxine and niacin. As much as 10% of the daily intake of folate might come from beer in some countries. The fat-soluble vitamins do not survive into beer and are lost with insoluble components in processing (grains, trub and yeast). Some beers will contain vitamin C, because this material is added to protect the beer from oxidation.

Table 5.7 Vitamin content of beers.

	Derived from Hough *et al.* (1982)		
Vitamin (µg/L)	Lagers	Ales	Derived from Moll (1994)
Biotin	7–18	11–12	2–15
Nicotinic acid	4494–8607	7500–7753	3000–8000
Pantothenic acid	1093–1535	1375–1808	40–2000
Pyridoxine	329–709	341–546	70–1700
Ribo avin	219–420	331–575	20–800
Thiamine	15–58	59–181	3–80
Folic acid			40–600
B_{12}			3–30

Table 5.8 Vitamin content of beers in comparison with other foodstuffs.

Food	Size of serving (weight or volume)	Thiamine (mg)	Ribo avin (mg)	Niacin (mg)	B_6 (mg)	Folate (µg)	B_{12} (µg)
Beer*	UK pint (568 mL)	0.003–0.08	0.02–0.8	3–8	0.07–1.7	40–600	3–30
Cola	12 uid ounces (355 mL)	0	0	0	0	0	0
Milk	1 cup	0.1	0.4	0	0.1	12	0.9
Tea (black)	6 uid ounces (178 mL)	0	0	0	0	9	0
Coffee (black)	6 uid ounces (178 mL)	0	0	0	0	0	0
Wine, white	5 uid ounces (148 mL)	0	0	0	0	0	0
Wine, red	5 uid ounces (148 mL)	0	0	0	0	3	0
Whisky (80 proof)	1.5 uid ounces (44 mL)	0	0	0	0	0	0
Apple	1 medium	0	0	0	0.1	4	0
Banana	1 medium	0.1	0.1	1	0.7	23	0
Cabbage, cooked	0.5 cup	0	0	0	0.1	15	0
Carrot, cooked	0.5 cup	0	0	0	0.2	11	0
Lettuce, Iceberg	1 cup	0	0	0	0	31	0
Tomato	1 medium	0.1	0.1	1	0.1	18	0
Potato, baked	1	0.2	0.1	3	0.7	22	0
Bread, white	1 slice	0.1	0.1	1	0	24	0
Corn akes	1 cup	0.4	0.4	5	0.5	99	0
Spaghetti, cooked	0.5 cup	0.1	0.1	1	0	49	0
Sirloin steak, broiled	3 ounces	0.1	0.2	3	0.3	8	2.3
Pork sausage, cooked	3 ounces	0.6	0.2	4	0.3	2	1.5
Chicken breast, roasted	3 ounces	0	0.1	12	0.5	3	0.3
Egg, raw	1 large	0	0.3	0	0.1	24	0.5
Cod, cooked (dry)	3 ounces	0.1	0.1	2	0.2	7	0.9
Cheese. Cheddar	1.5 ounces	0	0.2	0	0	8	0.4
Chocolate, milk	1 bar (1.5 ounces)	0	0.1	0	0	4	0.2

*Range reported across beers.
Source: *Encyclopedia of Foods: A Guide to Healthy Nutrition* (San Diego: Academic Press).

Stringer (1946) noted that the levels of vitamins in beer are proportional to original gravity (see Chapter 3). Of course, this will depend on the nature of the grist materials employed. If the beer is all malt, or is produced with the employment of cereal-based adjuncts, then the vitamin level would be higher than one produced from a grist including a high proportion of sugar.

Earlier I mentioned the meeting at the Horse Shoe Hotel. It is intriguing to quote another contributor to the discussion, Colonel C.J. Newbold:

> [I have] a strong belief that, speaking quite generally, the human body knows what it wants. In that connection [I want] to say something about gravity. [I believe that] England and New Zealand are the only two countries in the world that tax beer on its strength. [I am] not arguing that this system is not a good one from a revenue and perhaps other points of view, but there is another system adopted by all other beer-drinking countries and that is to tax it on volume irrespective of strength. In the latter system the average gravity in that country will probably settle down at the gravity that the people want.

His point was that beer gravity (and presumably selection of grist materials) are heavily impacted by tax considerations in countries where the levy is on the basis of strength as opposed to volume, and that this will have implications for the content of 'useful' materials in the beer. In the UK duty is no longer levied on the wort upstream, but since the late 1980s has been on the basis of alcohol content of the end product. There remains, therefore, a prevalence of products that are comparatively low in alcohol as compared to those in other countries (e.g. the US) where, for the most part, all beers attract the same rate of taxation, irrespective of strength.

Beers tend to contain very low levels of thiamine, owing to the fact that it is taken up by yeast (Stringer 1946). Agranoff (2000) hypothesises that it wasn't ever thus. The high levels of residual yeast present in eighteenth-century beer will have provided vitamins to the diet and might have been part of the reason why beer was portrayed by William Hogarth as leading to a healthier lifestyle (e.g. less beri beri and other neurological diseases) than gin. There is no modern evidence for the relative vitamin 'charge' in ltered beers and their counterparts that still contain yeast (i.e. naturally conditioned beers), though the latter would be expected to make a greater contribution providing the yeast is consumed. (A former colleague of mine was devoted to his Worthington White Shield, with its goodly charge of yeast in the bottom of the bottle. He would pour out the beer with extreme caution, such that the glass only contained bright beer. Then, with gusto, he would drain the sediment directly from bottle to throat, declaring with satisfaction that he was 'getting his vitamins'.) However, in attempts to fortify beer with thiamine, it was found that when the vitamin was added to beer it was soon eliminated by unknown

reactions with other components of the product (Thompson *et al.* 1990). Furthermore, ethanol inhibits the absorption of thiamine by the body (Hoyumpa 1980).

Thiamine de ciency stimulates alcohol consumption (Pekkanen 1979): thiamine shortages interfere with glucose metabolism, so perhaps the same causal inverse link referred to earlier between intake of alcohol and carbohydrate is at play. The body does not need thiamine to deal with ethanol and there are better substrates than fats, so thiamine de ciency may be expected to promote the tendency toward alcohol consumption (Segovia-Riquelme *et al.* 1971; Yki-Jarvinen 1988).

Levels of ribo avin increase through malting and brewing, whereas nicotinic acid levels increase in malting and decline during brewing.

Mayer *et al.* (2001) have demonstrated the worth of beer as a source of folic acid, leading to a decreased homocysteine content in blood (hyperhomocysteinemia is a signi cant risk factor for vascular diseases – see Chapter 6). Chronic alcoholism leads to the obverse effect, although beer drinkers had signi cantly lower serum concentrations of homocysteine than did those consuming wine or spirits (Cravo *et al.* 1996). Cereals are rich in folate and so it is no surprise that beer is a richer source of this material than are other alcoholic beverages (Savage *et al.* 1995).

Walker *et al.* (2001b) report folate levels of between 47 and 125 μg/L in a range of lagers, ales and a weissbier, which displayed the highest concentration. The extractable level of folate increased during germination of barley, which the authors ascribe to its synthesis in the embryo, though it may be a result of increased availability for extraction. Typically about 4 mg/kg folate is present in ale and lager malts, with less in barley and other adjuncts, so beers produced from a malt-rich grist might give more folate.

Substantial loss of folate occurs during mashing and this is thought to be due to oxidation and heat inactivation. Losses, though, are low in wort boiling and wort clari cation. There is no net effect of fermentation on folate – yeast makes folate to balance that which is lost presumably through adsorption effects. There is some loss of folate (up to 50%) in the nal package due to ill-de ned changes occurring during packaging and storage.

Vitamin C is found in barley and green malt, but is destroyed on kilning (Harden & Zilva 1918). Some brewers add it to beer as an antioxidant.

Minerals

The mineral content of beer is illustrated in Table 5.9, while that of beer in relation to other foods is shown in Table 5.10. Beer is rich in magnesium and potassium, but

Table 5.9 Mineral content of beers.

Inorganic component (mg/L)	British beers*	German beers*	Lager-style beers*	Unspeci ed†
Potassium	330–1100	396–562 (476)	253–680 (362)	200–500
Sodium	40–230	9–120 (35)	15–170 (58)	20–110
Magnesium	60–200	75–250 (114)	34–162 (82)	60–140
Calcium	40–140	3.8–102 (32.7)	10–135 (46)	20–160
Iron	0.1–0.5	0.02–0.84 (0.02)	0.04–0.44 (0.12)	0.01–0.3
Copper	0.3–0.8	0.04–0.8 (0.19)	0.01–0.41 (0.11)	0.02–0.4
Zinc		0.1–1.48 (0.1)	0.01–0.46 (0.06)	0.02–4.5
Manganese		0.04–0.51 (0.2)		0.03–0.2
Lead			0.06	<0.01–0.1
Arsenic			0.02	<0.02–0.05
Chloride	150–984	143–365 (210)		150–400
Sulphate	150–400	107–398 (182)		60–300
Phosphate	260–400	624–995 (860)		
Phosphorus	90–400			
Nitrate		1.4–101.3 (34)		0–30
Nitrite				0–2
Fluoride		0.08–0.64 (0.15)		0.09–0.2
Cobalt				0.01–0.11
Silica				50–120
Aluminium				0.1–2

*Hough *et al.* (1982).
†Moll (1991).
Values in parentheses represent mean values.

relatively de cient in iron, zinc and calcium. The presence of iron in beer is avoided deliberately by brewers, on account of its acting as a pro-oxidant. Nevertheless it has been used as a foam stabiliser in Belgium. Just as effective as a foam enhancer is zinc, and this mineral is used by brewers at a concentration typically of 0.2 mg/L to stimulate fermentation. However at this addition rate zinc does not survive into beer. To the author's knowledge no brewer is presently employing zinc as a foam stabiliser, though it would be a low-risk possibility. Walker & Baxter (2000) claim beer as a good source of silicon. It should be borne in mind that alcohol is a diuretic and so can stimulate a loss of minerals (Buday & Denis 1974).

Beer is frequently cited as being a signi cant dietary source of selenium.

The relatively high potassium:sodium ratio (typically 4 : 1) is consistent with a low-sodium diet. The recommendation is that the intake of sodium should be less than 6 g per day.

Brewing yeast is a rich source of the so-called glucose tolerance factor, a chromium-containing complex which may aid insulin in the regulation of body glucose levels (Zetic *et al.* 2001).

Table 5.10 Mineral content of beer in relation to other foodstuffs.

Food	Size of serving (weight or volume)	Calcium (mg)	Iron (mg)	Magnesium (mg)	Phosphorus (mg)	Potassium (mg)	Sodium (mg)	Zinc (mg)	Copper (mg)	Manganese (mg)	Selenium (μg)
Beer*	UK pint (568 mL)	40–140	0.1–0.5	60–200	90–400	330–1100	40–230	0.01–1.48	0.1–0.5	0.04–0.51	<0.4–7.2
Light beer*	UK pint (568 mL)	40–140	0.1–0.5	60–200	90–400	330–1100	40–230	0.01–1.48	0.1–0.5	0.04–0.51	<0.4–7.2
Cola	12 uid ounces (355 mL)	11	0	4	44	4	15	0	0	0	0
Milk	1 cup	291	0	33	228	370	120	1	0	0	5
Tea (black)	6 uid ounces	0	0	5	2	66	5	0	0	0	0
Coffee (black)	6 uid ounces	4	0	9	2	96	4	0	0	0	0
Wine, white	5 uid ounces	13	0	15	21	118	7	0	0	1	0
Wine, red	5 uidounces	12	1	19	21	165	7	0	0	1	0
Whisky (80 proof)	1.5 uid ounces	0	0	0	2	1	0	0	0	0	0
Apple	1 medium	10	0	6	13	207	1	0	0	0	0
Banana	1 medium	7	0	34	24	467	1	0	0	0	1
Cabbage, cooked	0.5 cup	23	0	6	11	73	6	0	0	0	0
Carrot, cooked	0.5 cup	24	0	10	23	177	51	0	0	1	1
Lettuce, Iceberg	1 cup	10	0	5	11	87	5	0	0	0	0
Tomato	1 medium	6	1	14	30	273	11	0	0	0	0
Potato, baked	1	20	3	54	115	844	16	1	1	0	2
Bread, white	1 slice	27	1	6	24	30	135	0	0	0	7
Corn akes	1 cup	1	9	3	11	25	298	0	0	0	1
Spaghetti, cooked	0.5 cup	5	1	13	38	22	1	0	0	0	15
Sirloin steak, broiled	3 ounces	9	3	24	187	309	53	5	0	0	
Pork sausage, cooked	3 ounces	27	1	14	156	307	1,101	2	0	0	15
Chicken breast, roasted	3 ounces	13	1	25	196	220	64	1	0	0	24
Egg, raw	1 large	25	1	5	89	61	63	1	0	0	15
Cod, cooked (dry)	3 ounces	12	0	36	117	207	66	0	0	0	32
Cheese, cheddar	1.5 ounces	307	0	12	218	42	264	1	0	0	6
Chocolate, milk	1 bar (1.5 ounces)	84	1	26	95	169	36	1	0	0	2

* Range reported across beers.
Note: the mineral composition of the diet will be greatly impacted by the ionic composition of the water supply.
Source: *Encyclopedia of Foods: A Guide to Healthy Nutrition* (San Diego: Academic Press).

Fibre

Various polymeric carbohydrates survive into beer. These may include the dextrin degradation products of starch with a degree of polymerisation of four or greater, and which are unfermentable by yeast. There is some evidence that the straight-chain dextrins may be digested by salivary amylase to sugars assimilable by the body, and that the branched-chain dextrins may be similarly hydrolysed by an enzyme (oligo-1,6-glucosidase) in the intestinal mucosa, but any which emerge into the lower gut will contribute to soluble bre. Soluble bre particularly comprises the degradation products of cell wall polysaccharides (β-glucans and arabinoxylans). Gromes *et al.* (2000) quote bre levels in beer as high as 6 g/L, with average levels in the vicinity of 2 g/L. Schwarz and Han (1995) report 4 g/L. These levels might be compared with the recommended daily intake of 18 g (UK). A couple of pints a day would provide roughly a quarter of the recommended intake of bre. A comparison of beer with other foodstuffs for bre content is given in Table 5.5.

Comparison of beer with other foodstuffs for nutrient value

From data provided in the UK National Food Survey (http://www.defra.gov.uk/esg/Work_htm/publications/cf/nfs/nfs.htm) it is possible to compare the nutrient value of beer with other components of the diet on a normalised basis in respect of calori c value (Tables 5.11–5.13). This style of presentation refutes the notion of 'empty calories' in the context of beer.

Table 5.11 Vitamin density of foods (per 1000 kcal).

	Beer	Cereals	Meat	Fruits and vegetables
Consumption (kcal)	380 (l litre)	670	260	260
Thiamine (mg)	0.005	0.1	0.05	0.08
Pyridoxine (mg)	0.07	0.04	0.1	0.19
Niacin (mg)	0.82	0.56	2.2	0.69
Ribo avin (mg)	0.033	0.033	0.1	0.03
Folate (μg)	7.4	6.9	3.1	22.2
B_{12} (μg)		0.02	0.33	0
Biotin (mg)	0.002			
Pantothenate (mg)	0.19			
Ascorbic acid (mg)	Variable – depends on addition	0.17		12.4
E (mg)	0	0.1	0.09	0.57
A (retinol equiv)	0	0.005	0.04	0.057
D (μg)	0	0.05	0.14	0.005

Source: based on Righelato (2001).

Table 5.12 Mineral density of foods (mg/1000 kcal).

	Beer	Cereals	Meat	Fruits and vegetables
Potassium	72	32.5	74.1	203
Magnesium	14	6.9	5.3	12.7
Iron	0.07	0.5	0.26	0.43
Calcium	5.5	19.1	6.2	14.8
Zinc	0.07	0.2	0.5	0.24

Source: based on Righelato (2001).

Table 5.13 Contribution of foods to fibre consumption.

Food	Fibre (g/1000 kcal)
Beer	0.72
Cereals	0.5
Meat	Trace
Fruits and vegetables	1.36

Based on Righelato (2001)

Antioxidants

Few (if any) texts give much credit to beer as a source of antioxidants. And yet beers are generally a valuable source of polyphenols (Table 5.14). Paganga *et al.* (1999) analysed the major avone, avonol, anthocyanidin and hydroxycinnamic acid constituents of various foodstuffs and assessed their antioxidant activities. Using a standard index for oxidation they showed that the antioxidant activities of one glass (150 mL) red wine = 12 glasses white wine = 2 cups of tea = 4 apples = 5 portions of onion = 5.5 portions of eggplant (aubergine) = 3.5 glasses of blackcurrant juice = 3.5 (500 mL) glasses of beer = 7 glasses of orange juice = 20 glasses of apple juice (long life). Some degree of caution must always be displayed when analysing data of this type. Such comparisons are generally made using a standard test-tube based assay in which the foodstuffs are compared for their relative ability to prevent oxidation in a model system. This does not necessarily mean that the antioxidants from the various foodstuffs are taken into the body with equal ef ciency, or that their ability to protect sensitive molecules in the body is the same as their ability to prevent oxidation of a 'marker' molecule *in vitro*.

According to some workers, levels of antioxidants in beer are of the same order of magnitude as those found in fruit juices, teas and wines (Vinson *et al.* 1999; Gorinstein *et al.* 2000). Flavonoids in foodstuffs have attracted the most attention for their potential value as chemoprotective agents (Horvathova *et al.* 2001). The polyphenols derived from beer are much more ef cacious as inhibitors of the oxidation of LDL and CLDL

Table 5.14 Phenolic compounds in beer.

Fraction	Examples	Levels (mg/L)
Phenolic alcohols	Tyrosol	3–40
Phenolic acids	Ferulic acid, p-coumaric acid, vanillic acid, caffeic acid, gallic acid	10–30
Phenolic amines and amino acids	Hordenine, tyramine, tyrosine	10–20
Flavan-3-ols	Catechin	0.5–13
	Epicatechin	1–10
Flavan-3,4-diols	Leucocyanidin	4–80
Flavonols	Quercetin, myrecetin, rutin	< 10
Condensed polyphenols	Dimeric catechins	5–8
	Polymeric catechins	< 1
	Proanthocyanidins	20–60
	Prodelphinidins	3–10

lipoproteins (see Chapter 6) than are ascorbic acid, alpha-tocopherol and beta-carotene (Vinson *et al.* 1995). However, in studies looking at lipoprotein-bound activity, they were not as good as polyphenols from tea and wine (Vinson *et al.* 1999).

Walker *et al.* (2001a) emphasise that the quality of antioxidants is more relevant than their absolute quantity. In other words, measuring the total polyphenol level in a foodstuff is probably of less value than measuring the type of polyphenol. In their work they have measured (using a luminescence-based system) the antioxidant activity of individual fractions emerging from high-performance liquid chromatography. They divided the antioxidants into ascorbic acid and related compounds, polyphenolic avonoids, catechin and related compounds and epicatechin and related compounds. Such a test revealed cider to perform best from among the beverages tested (Fig. 5.1). The correlation between total polyphenol content and antioxidant activity is weak. It seems that there is a greater diversity of phenolic compounds in beer than in red wine or cider. Walker *et al.* (2001a) compared a range of foodstuffs for their relative antioxidant value as determined in a standard laboratory assay (Table 5.15). Clearly beer measures up well; however, I stress again that this is very much an *in vitro* assay – it is one thing having antioxidants at high levels, but are they taken up into the body and do they reach the parts where they need to exert an effect? This will be in uenced by the food matrix itself, the type of antioxidants and the presence of other factors, including alcohol. Smaller molecules tend to be more readily assimilated, and so are those which are more water-soluble.

Gorinstein *et al.* (2000), using laboratory animals and clinical investigations, showed that the content of total polyphenols is higher in white wine than in beer. However, beer possessed a higher antioxidant activity. The authors ascribed this to the fact that levels of procyanidins, epicatechin and ferulic acid were signi cantly higher, statistically, in beer than in white wine.

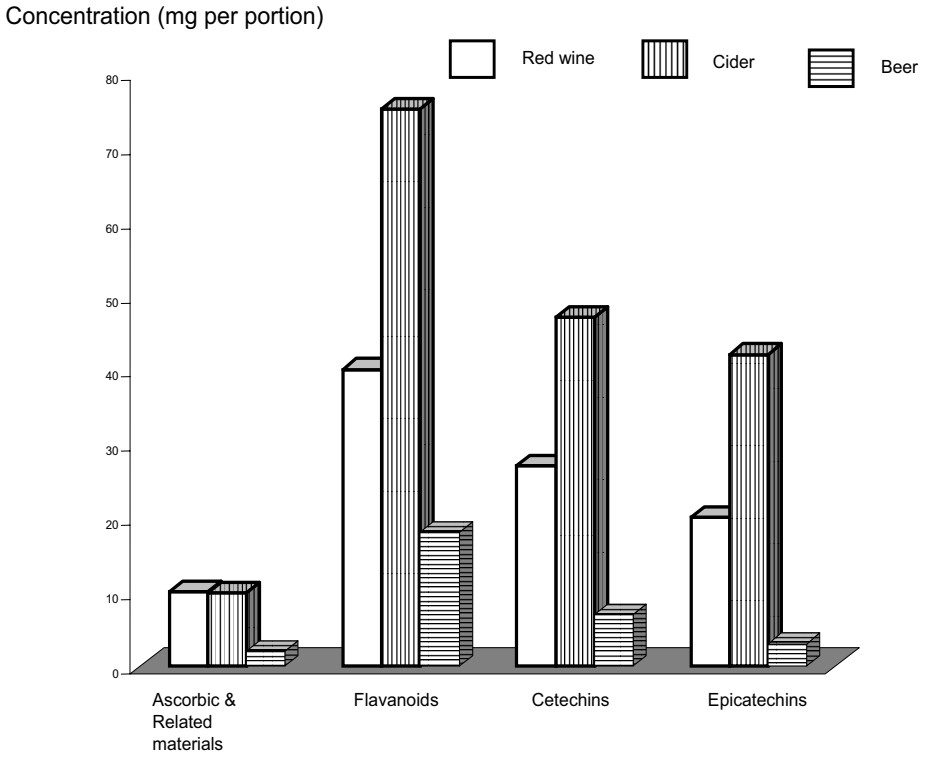

Fig. 5.1 A comparison of antioxidant potential in various alcoholic beverages. (Redrawn from Walker *et al.* 2001a).

Table 5.15 Antioxidant activity of various foodstuffs (based on Walker *et al.* 2001a).

Food	Amount	Total antioxidant activity (μmol Trolox equivalents)
Apple (peeled)	100 g	640
Tomato	100 g	160
White wine	150 mL	220
Black tea	150 mL	1400
Apple juice	150 mL	140
Orange juice	150 mL	400
Beer	500 mL	910–1340
Cider	500 mL	200–5190
Red wine	150 mL	1340–3400

An antioxidant-rich material unique to the production of beer is hops (De Keukeleire *et al.* 2001). These authors report:

> The diversity of natural hop constituents undoubtedly accounts for the varied and rich panoply of bioactivities hitherto reported: sedative, antistress, sopori c activities, estrogenicity, treatment of complaints related to the menopause, anticancer properties (in particular inhibition of hormone-dependent cancers of breast, uterus and prostate), bacteristatic activity, anti-in ammatory action, stimulation of the digestive tract, diureticum and agent against bladder complaints, diaphoretic and perspiration-stimulating effects, and anaphrodiasiacum.

The active ingredients of the hop are laid down in lupulin near the end of the growing period with a primary purpose of protecting the plant against pests. The prenylated avonoids are a family of compounds restricted to a relatively small number of plant families. Prenylated avonoids are present in lupulin glands at levels from 0.2–0.6%, while up to 4 ppm of prenylated avonoids can be found in beer.

Stevens *et al.* (1999b) found that the prenylated avonoids, including xanthohumulone, are largely isomerised during wort boiling and lost as a result of incomplete extraction from hop and adsorption onto wort proteins and yeast cells. Consequently only 22–30% ends up in beer. Some 10% of the hop desmethylxanthohumol and the 3′-geranylchalconaringenin survive into beer in the form of prenylnaringenin. De Keukeleire *et al*. (1997b) reports up to 21 ppb 8-prenylnaringenin in beers, although Stevens *et al.* (1999a) report up to 240 ppb. The levels will depend on the mode of hopping.

Forster *et al.* (2002) demonstrated that the xanthohumol levels in beer can be enhanced using xanthohumol-rich hop preparations. This substance is ordinarily lost to a sizeable extent during the brewing process, but by introducing more into the stream, especially later on in the process, levels might be raised. The scope for tailoring raw materials to enhance the levels of potentially healthful components of beers is considerable – 'functional beers'? Certainly the prenylated chalcones from hops and beer have been shown to be effective in combating lipid oxidation in model systems based on rat liver microsomes (Rodriguez *et al.* 2001).

Potentially deleterious components of beer

Baxter *et al.* (2001) employed arti cially high levels of ochratoxin A to show that most is probably lost as a result of proteolysis in mashing, through adsorption on spent grains and during fermentation. However, in these studies some 13–32% survived into beer. It is clearly necessary to ensure that the levels of these materials in raw materials are as low as possible. Blank (2002) observed that the main sources of mycotoxin in the diet were

bakery products and breakfast cereals, but that for these, as for products such as beer, the risks were acceptably low provided proper precautions are taken, such safety measures hinging around proper storage conditions in respect of moisture content and temperature. Pesticides are effective (providing the intention is not to deliver a strictly 'organic' crop), such materials being thoroughly screened for health and environmental impacts. Surveys show that the pesticides used in the production of malting barley do not nd their way into malt in levels exceeding the recommended maximum (Baxter 2003).

Nitrate is present in barley (and therefore malt), hops and water (Baxter 1988). Less is found in hop extracts. The concern with nitrate re ects its role in the production of the potentially carcinogenic nitrosamines. However, levels of such compounds in beer have dropped enormously from the time that they were rst reported in beer at concentrations of up to 2–10 μg/L (Spiegelhalder *et al.* 1979). The precautions taken in malting and brewing (see Chapter 3) meant that within short order the levels had been lowered to an average of 0.2 μg/L (Klein *et al.* 1982).

Two of the most recent food safety 'scares' have revolved around 3-monochloropropanol (MCPD – see www.ifst.org/hottop37.com) and acrylamide (go to http://www.europa.eu.int/comm/food/fs/sc/scf/out131_en.pdf). MCPD is formed in many foods by the reaction of chloride with lipids at high temperatures and has been shown to be a carcinogen in laboratory animal studies. Soy sauce and hydrolysed vegetable proteins were agged up as especial concerns; however, there is a risk in any foodstuff that contains the key precursors and which involve baking, boiling, drying, grilling and toasting in their production. Particular focus in the brewing industry was on certain of the speciality coloured malts that are produced with intense heating (see Baxter 2003); however, it does not appear that the MCPD is detectable in beers produced with a proportion of such malts.

Acrylamide, another potential carcinogen, is also produced under conditions of high heat, from starch-rich foods. Clearly this must mean that any cereal, pulse, tuber, etc. employed in food production presents a risk. However the World Health Organization tabulation of values cited at http://www.europa.eu.int/comm/food/fs/sc/scf/out131_en.pdf reveals the acrylamide level in beer to be at the limit of detection using current methodology (Table 5.16).

Beer as a 'treat'

For the vast number of years that have elapsed since beer was rst brewed this drink was regarded as a staple part of the diet. Today, rightly or wrongly, it is much more regularly regarded as a provider of pleasure and not as an integral component of the menu. As such – and despite evidence to the contrary that we have previously explored – it is

Table 5.16 Levels of acrylamide (μg/kg) in a range of foodstuffs.

	Mean
Crisps	1312
French fries	537
Batter-based products	36
Bakery products	112
Biscuits, crackers, toast, bread crisps	423
Breakfast cereals	298
Corn crisps	218
Bread, soft	50
Fish and seafood products, crumbed, battered	35
Poultry or game, crumbed, battered	52
Instant malt drinks	50
Chocolate powder	75
Coffee powder	200
Beer	< 30

Data from the European Commission Opinion of the Scientific Committee on Food on new findings regarding the presence of acrylamide in food (European Commission, 2002, see http://www.europa.eu.int/comm/food/fs/sc/scf/out131_en.pdf)

often regarded as something that will 'add to the calories' and provide little more than hedonic benefit. In concluding this chapter, then, I compare the relative contribution to the nutritive intake of beer and a selection of other 'luxury' items (Table 5.17). It will be seen that beer affords substantially fewer calories and vastly less fat than the other 'temptations', while at the same time stacking up favourably with regard to components such as B vitamins and potassium : sodium balance.

Table 5.17 Comparison of beer with 'temptation foods'.

	Beer (regular, 12 uid ounces)	Ice cream, vanilla (0.5 cup)	Snickers bar	Doughnut, yeast-leavened, glazed (3.25 inches dia.)	Lemon meringue pie (1 piece)	Milk shake, vanilla (10 uid ounces)
Energy (kcal)	146	179	278	242	375	314
Protein (g)	1	2.5	6	4	2	10
Carbohydrate (g)	13	16.5	37	27	66	51
Fibre (g)	3	< 0.5	2	1	2	< 1
Fat (g)	0	12	14	14	12	8
Calcium (mg)	18	86	70	26	78	345
Iron (mg)	0.11	0.035	0.48	1.23	0.85	0.25
Magnesium (mg)	0.21	8	37	13	21	34
Phosphorus (mg)	43	71	129	56	147	289
Potassium (mg)	89	128	200	65	125	492
Sodium (mg)	18	42	164	205	204	232
Zinc (mg)	0.07	0.3	0.7	0.46	0.69	1.02
Vitamin A (RE)	0	136	19	6	73	91
Thiamine (mg)	0.04	0.03	0.03	0.22	0.09	0.13
Ribo avin (mg)	0.11	0.12	0.11	0.13	0.29	0.52
Niacin (mg)	1.6	0.06	1.83	1.71	0.91	0.52
Vitamin B_6 (mg)	0.18	0.03	0.11	0.03	0.04	0.15
Folate (μg)	21	3.5	24	13	11	9
Vitamin C (mg)	0	0.5	< 1	0	4	2

Source: extracted from Boyle & Zyla (1996).

6 The Impact of Alcohol on Health

In this chapter we consider the effect that alcohol, including in the form of beer, might have on the overall state of healthfulness of the body. What harm might it do – and might it actually do some good? And let us start from a baseline statement that alcohol is relatively non-toxic, with an oral LD_{50} for the rat of 13.7 g/kg (i.e. the amount of ethanol which will kill half of the animals in an experimental population) (Bakalinsky and Penner 2003)

Increasingly the evidence is that there appear to be bene ts in drinking beer (and other types of alcoholic beverage). Guallar-Castillon *et al.* (2001) concluded that the consumption of total alcohol (wine and beer) was associated with a lower prevalence of sub-optimal health. Hospitalisation is less acute for daily moderate drinkers (Longnecker & McMahon 1988), especially for women who had consumed between 29 and 42 alcoholic beverages in the fortnight prior to lling in the questionnaire. Artalejo *et al.* (2000) found that moderate drinkers in Spain were less likely than abstainers to use healthcare services. Meanwhile Wiley and Camacho (1980) showed that moderate alcohol consumption (17–45 drinks per month) was associated with the most favourable adjusted health scores.

Beer drinkers were shown by Richman and Warren (1985) to have signi cantly lower rates of morbidity (sickness) than expected – one drink per day giving 15% less disability than was the case for the general population.

There will be those reading this who will not be able to countenance such ndings. If these people nd it hard to swallow that drinkers, imbibing in *moderation*, could be *less* ill, then they might note that they have certainly not been shown to be *more* sick. However, we must stress always that many of these studies are dealing with correlation, not necessarily causality. Some will argue that there may be other confounding factors not explored in the studies, and that those who tend to drink in moderation may have other lifestyle attributes that are the true reason for their enhanced healthiness. However, the sheer frequency of studies that have demonstrated the bene ts of restricted alcohol intake, which we will explore in this chapter, weigh heavily in support of the merits of sensible drinking.

In the mid-1990s, the Department of Health within the British government addressed the matter of recommended safe limits for drinking. After (we presume) careful consideration of the scienti c and medical evidence available up to that stage, they increased

the recommended limit for men from 21 units to 28 units per week, with the advice to women being to drink no more than 21 units per week (previously it had been 14). They stressed that the daily maximum should be 4 units and that binge drinking (the equivalent of taking all of the weekly allocation at one sitting) is absolutely undesirable.

Table 6.1 describes a unit of alcohol in terms of volume of beer and other alcoholic drinks in the UK. It should be noted that the de nition of a unit differs between countries (Table 6.2). This table also indicates the recommendations concerning alcohol consumption in those countries. It must be stressed that beers can differ substantially in their alcohol content (see Chapter 3). Thus a mainstream ale or lager in most parts of the world is likely to contain between 3.5 and 5% alcohol by volume (ABV) and one unit is basically a half-pint (284 mL) of such a product. There are some beers

Table 6.1 What constitutes a 'unit of alcohol' in the United Kingdom.

Drink	Typical alcohol content (% ABV)	Volume of drink constituting a 'unit'
Premium beer	4.5	approx. half a pint
High-strength beer	9.0	approx. quarter pint
Wine	12.0	approx. one-tenth of a 75-cL bottle
Whisky	40.0	20 mL
Gin	40.0	20 mL
Vodka	45.0	15–20 mL
Vermouth	15.0	approx. 1/15 of a bottle

Source: Bamforth (2003).

Table 6.2 Definitions of a unit of alcohol.

Country	Grams of alcohol contained in one unit (u) or one drink (d)	Of cial recommendation (drinks or units)
Australia	8–10 (d)	Men < 4 per day Women < 2 per day
Austria	6.3 (u)	
Canada	13.6 (d)	Men 2 per day Women 0.7 per day
Denmark	12 (d)	Men < 21 per week Women < 14 per week
Japan	19.75 (u)	
New Zealand		Men 3–4 drinks per day Women 2–3 drinks per day
Sweden		Men and women < 50 g alcohol per week
UK	8 (u)	Men < 4 per day Women < 3 per day
USA	12 (d)	Men, 2 per day Women, 1 per day

Source: after Hughes & Baxter (2001).

containing rather more than this, for instance the Trappist beers, the barley wines and the so-called 'super lagers'. This latter genre might contain 9% ABV, so here a half-pint would constitute 2 units. Butterworth (1993) offers 8–12 g of alcohol as being a 'standard serving' (remembering, as we discovered in Chapter 3, that a beer that is 4% alcohol *by volume* is 3.2% alcohol *by weight*).

Individuals differ substantially in their bodily response to alcohol. Various factors will play a role, including body weight, general state of health, amount of activity, and whether the alcohol is being consumed on its own or alongside food. The UK guidelines are precisely that: blueprints to give some guidance to people to judge sensibly what is and what is not an advisable amount of alcohol to consume. They are not recommendations to drink: they are certainly not instructions. Rather they are a common-sense judgement on what is likely to be healthful for a sensible and healthy adult. And the fact that the levels were *increased* is testimony to the burgeoning evidence that there is real merit in moderate consumption of alcohol.

The author of a newspaper article in California once highlighted the number of times I had invoked the word 'moderation' when she interviewed me. I make no apology for using the word again here (particularly as a glance at the thesaurus in my computer offers the word temperance as a suggested alternative!). As the reader should surmise from what follows, there is more than ample evidence for the harmful effects of sustained, heavy intake of alcohol in all its forms. However, it will be noted that the serious ailments are primarily associated with extreme *alcoholism*, and a consequence of vastly more alcohol ingestion than is the norm for the great majority of adults.

The metabolism of ethanol

Unlike drugs, alcohol is completely metabolisable by the body, at a rate of 10–15 g/h. The enzyme alcohol dehydrogenase in the stomach commences the metabolism of ethanol. This is referred to by Halsted (2003) as ' rst pass' metabolism. The enzyme has a rather poor af nity for ethanol, and it seems that this enzyme deals with about 30% of ethanol metabolism in men, but only about 10% in women, partly explaining the lower tolerance of women to alcohol.

The rest of alcohol metabolism is in the liver. Here the alcohol dehydrogenase has a much greater af nity for alcohol, leading to the production of acetaldehyde and much reducing power. The latter may also spill over to lipid synthesis, lowered production of carbohydrate (gludoneogenesis), enhanced lactate production and lessened excretion of uric acid. Consequences may therefore include transient fatty liver, hypoglycemia, acidosis and gout.

This enzyme seems to deal with all the alcohol (not dealt with by the stomach) in moderate drinkers. At higher alcohol levels a microsomal enzyme called CYP2E1 kicks

in. This enzyme produces more acetaldehyde but no assimilable energy. The enhanced acetaldehyde blocks respiration in the mitochondria, thus exaggerating the accumulation of fat in the liver. Ketoacidosis is promoted and lipid peroxidation and collagen synthesis promoted, which contributes to alcoholic hepatitis and cirrhosis. There is also free radical generation, which can promote liver damage.

Direct and indirect impacts

There are at least two ways in which an alcoholic beverage such as beer might impact bene cially on the body: rst, through a direct physiological impact on bodily tissues and functions (which will be focused upon here); second, through indirect impact, but founded equally on a physiological interaction. The mellowing in uence that moderate consumption of alcohol has, with its calming and relaxing impact, will of itself have a sparing effect on stress-related illnesses (Morrell 2000). Cleophas (1999) concludes that there is a signi cant psychological component in the bene cial relationship between moderate alcohol consumption and mortality.

In either instance it will be recognised that excessive alcohol consumption will shift the *status quo* in a negative direction. We will address the incontrovertible direct damage to body organs that can be caused by overconsumption, and there is no denying the antisocial impact of excessive alcohol consumption in terms of behavioural changes and drink driving. One problem emphasised by many writers is the impact of under-reporting alcohol consumption.

Dr Thomas Stuttaford (who for years has written a most engaging column in *The Times*) presents a fascinating experiential account of the likely reasons why his patients in rural Norfolk enjoyed a lesser incidence of cardiovascular problems and tended to live longer than did their counterparts in London (Stuttaford 1997). First, they had enjoyed less sedentary lives, with less dependence on the automobile. Second, they took aspirin daily to counter the osteoarthritis brought on by working in soggy agricultural conditions. Third, they weren't teetotallers. And their chosen drink was beer, with the occasional celebratory whisky.

It is of course not possible to con rm with any certainty that there was a causal link between any of those three factors and Stuttaford's observations on mortality. Indeed, the reader will recognise the dif culty of pursuing robust research in this entire area, for the simple reason that studies relating health to any type of food intake must inherently try to remove as many interfering factors as possible and this is not easy:

> Additional methodological problems are presented by a number of 'confounding factors' such as age, sex, body mass index, diet, physical activity, smoking, coffee consumption, educational attainment, type A/B behaviour, socio-economic status, and medical history, that may be factors in particular health problems in persons

> who have been the subjects of the reported studies. For example, a generally poor nutritional condition could possibly play a signi cant role in various health problems associated with heavy drinkers.
>
> Butterworth (1993)

Studies based on individuals' reporting of their dietary intake are not as controlled as those in which feeding trials are performed with laboratory rats with de ned diets. Yet, of course, what is observed with a rat does not necessarily extrapolate to the human. We must critically evaluate the breadth of evidence that is presented. Most assume that if suf cient evidence of diverse origin is offered then 'there must be something in it'.

Much of the attention that has been paid to the impact of alcohol on the body has been for its negative effect on those who abuse it. These effects are amply described in the *Oxford Textbook of Medicine* (Weatherall *et al.* 1996) and, in more prosaic form, by Stuttaford (1997). In the discussions that follow I refer to these impacts and the reader is referred to those texts for more information.

The heart and the circulatory system

Lichtenstein (2003) states that 15 million deaths in the late 1990s could be attributed to cardiovascular disease. The American Heart Association has pointed out that coronary heart disease and the related cardiovascular disease is the number-one killer in the US, accounting for almost one in two deaths among Americans and more deaths than are caused by all the forms of cancer combined. The impact on disability and the attendant economic loss are enormous.

Atherosclerosis ('hardening of the arteries') is the term used to describe a number of pathological events occurring in arteries and which are responsible for coronary heart disease, stroke and diseases of the peripheral circulatory system (Fisher 1991).

Atheroma (from the Greek *ather* = porridge) comprises deposits of fatty material on the walls of arteries – a material comprising cholesterol, triglycerides, brous tissue and red blood cells. As it builds it restricts blood ow and if this is in the coronary artery then heart attack and death may follow, as the heart muscle does not receive suf cient oxygen. Atheroma has also been associated with the development of cataracts, macular degeneration in the retina and the development of cancers (Emerit *et al.* 1991; Tunick *et al.* 1994). If the atheroma accumulation (plaque) is ruptured a blood clot may form which not only can accelerate the blockage of the artery concerned but also may break loose and plug another artery, increasing the risk of heart attack or, if the newly blocked artery is in the brain, a stroke.

Plainly, the intake of saturated fats and cholesterol increases the risk, although it must be realised that four- fths of the cholesterol is made in our bodies and does not

come through the diet. The quantity of cholesterol produced is increased in proportion to the level of saturated fatty acids in the diet (polyunsaturated fatty acids reduce blood cholesterol), and also the *trans* saturated fatty acids, i.e. those that are produced industrially by catalytic hydrogenation (Krisetherton 1995). High sugar intake can lead to high formation of saturated fats in the body. Indeed, any imbalance in metabolism such that there is an excess of calories over those needed to sustain the body will lead to an accumulation of fat. Obesity, hypertension, diabetes, sedentary living and the use of cigarettes all increase the risk of atherosclerosis.

As cholesterol and other lipids such as the triglycerides are insoluble in aqueous systems, they are transported through the body by combination with proteins, as lipoproteins. The principal carrier of cholesterol is low-density lipoprotein (LDL) and there is a strong positive correlation between its level and the risk of atherosclerosis. Hence LDL is frequently referred to as 'bad cholesterol'.

A lower percentage (20–30%) of the blood cholesterol is in the form of high-density lipoprotein (HDL), which is responsible for transporting cholesterol away from the arteries to the liver where it is metabolised. This role has caused HDL to be named 'good cholesterol', such that high levels of HDL appear to afford protection against heart attack. Thus there is an inverse correlation between levels of HDL and atherosclerosis.

There is now a plethora of papers arguing that moderate consumption of alcohol counters coronary heart disease [see, for example, Dyer *et al.* 1977; Hennekens *et al.* 1978; Ramsey 1979; Marmot *et al.* 1981; Gordon & Kannel 1983 (the Framingham study); Kozarevic *et al.* 1983; Yano *et al.* 1984; Moore & Pearson 1986; Klatsky *et al.* 1992; Maclure 1993; Verschuren 1993]. Alcohol causes a lowering of LDL cholesterol in the plasma and an increased level of HDL cholesterol (HDL_2 and HDL_3) and apolipoproteins A-I and A-II (Clevidence *et al.* 1995; Goldberg *et al.* 1995; Jansen *et al.* 1995; Parker *et al.* 1996).

Alcohol also appears to lower the risk of blood clotting by reducing the level of brinogen in blood plasma (Stefanick *et al.* 1995) and lessening the tendency of blood platelets to aggregate (Renaud *et al.* 1992). The bene ts apply to both men and women (Nanchahal *et al.* 2000).

Doyens of the eld have included Arthur Klatsky in Oakland, California, Norman Kaplan of the University of Texas Southwestern Medical Center, and Sir Richard Doll in Oxford, England.

The phenomenon has taken the name the 'French paradox', on account of the unexpectedly low risk of cardiovascular disease in a country noted for its intake of very fatty foods. We can look back nearly two centuries to the rst noting of this effect, when an Irish doctor, Samuel Black, remarked on the much greater incidence of angina in France as opposed to Ireland, which he believed was ascribable to 'the French habits and modes of living, coinciding with the benignity of their climate and the peculiarity of their moral affections' (Black 1819). The occurrence is now sometimes called

the European Paradox because it re ects dietary characteristics beyond France alone (Bellizzi *et al.* 1994).

Various laboratories have reported U-shaped curves (e.g. Doll *et al.* 1994) or J-shaped curves (e.g. Tsugane *et al.* 1999) (Fig. 6.1) to illustrate the impact of various intakes of alcohol on coronary heart disease and on all causes of mortality. For the most part it seems that the J shape relates to the relationship between alcohol intake and total mortality, with the U shape better describing that between alcohol consumption and coronary heart disease. The clear evidence is that the intake of some alcohol has a bene cial impact. In many instances consumption of between 1 and 3 units daily perhaps offers the best advantage, with higher intake progressively shifting the risk upwards again.

The low point (nadir) in these curves has been reported at various levels, for example, 69 g alcohol per week for men in the US (26 g per week for women), but 116 g per week for men in the UK (White 1999). It seems that bene ts for women are especially notable after the menopause (Fuchs *et al.* 1995; Nanchahal *et al.* 2000).

Even the American Cancer Society reported this type of effect (Boffetta & Gar nkel 1990). The study began in 1959 with 276,802 men between the ages of 40 and 59. Assigning 1.0 as a standard value for risk of death in non-drinkers, it was shown that the risk of death dropped to 0.84 (i.e. by 16%) for those taking one alcoholic drink per day. The risk of death for those claiming to consume six drinks per day was still lower than for abstainers, at 0.92.

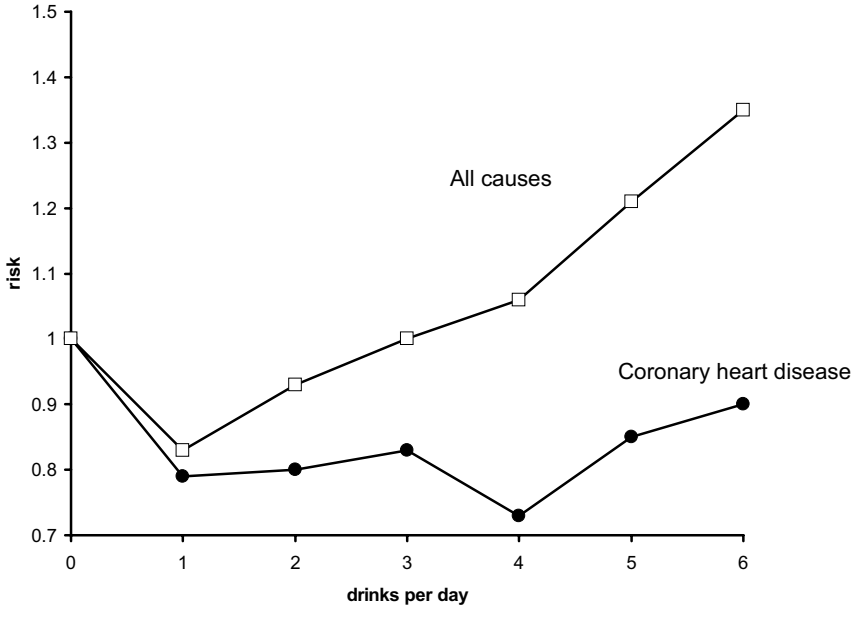

Fig. 6.1 The relationship between alcohol consumption and all risks of mortality. (Derived from Renaud *et al.* 1993.)

It seems that not only does limited alcohol intake reduce the risk of heart attack, but even after myocardial infarction, the moderate consumption of alcohol reduced the risk of a subsequent episode (Muntwyler *et al.* 1998).

Predictably, critics of these various claims have asserted that the phenomenon is an artefact arising from the fact that some of the non drinkers were either not consuming alcohol because of ill health or were previous heavy consumers who had stopped drinking for health reasons ('sick quitters') (Shaper 1990). However studies that have painstakingly eliminated such purported problems have continued to demonstrate the validity of the U- or J-shaped curves (Criqui 1990, 1996; Kannel & Ellison 1996).

Dawson (2000) stresses that alcohol dependence nulli es any bene t from moderate drinking. Excessive consumption of alcohol has unquestioned detrimental effects (Poikolainen 1996). For example, Kauhanen *et al.* (1997) demonstrated the adverse impact on all causes of mortality, including myocardial infarction, of binge drinking of beer. Britton and McKee (2000) highlighted how the apparently contradictory nding that alcohol intake in certain populations (e.g. Russia) was positively correlated with cardiovascular disease could in fact be linked to binge drinking. This emphasises the importance of moderation in terms of not only the amount of intake but also the frequency.

Remarkably, however, Mukamal *et al.* (2003) showed from a study of 38,077 male health professionals over a 12-year period that men who consumed alcohol 3–4 or 5–7 days per week had a decreased risk of myocardial infarction when compared to those who drank less than once per week. They found that the risk was similar for men taking 10 g alcohol per day or 30 g or more per day, and furthermore it didn't matter whether the beverage was beer, red wine, white wine or spirits. In other words, these authors would claim that the frequency of drinking has as much, if not more, effect than the absolute level of drinking.

At least two components of alcoholic beverages have been suggested as being the key factors in the reduction of atheroma: antioxidants and the alcohol itself.

It is understood that alcohol increases the concentration in blood serum of HDL cholesterol – i.e. it lessens accumulation of cholesterol in blood vessels (Hulley & Gordon 1981; Thornton *et al.* 1983). It has also been suggested that alcohol reduces the risk of atherosclerosis by lessening the tendency of blood platelets to aggregate in blood clotting (Renaud *et al.* 1992; Hendriks & van der Gang1998) and bene cially impacting clotting/ brinolysis mechanisms (Kluft *et al.* 1990; Ridker *et al.* 1994). Alcohol may decrease platelet stickiness (Mikhailidis *et al.* 1983), lower levels of brinogen (Ridker *et al.* 1994) and promote the release of plasminogen activator (Laug 1983).

Components of alcoholic beverages other than alcohol may also combat coronary heart disease (Klatsky 1999), and these include the polyphenols (Halpern *et al.* 1998). Beer, wine and spirits seem to have similar effects on plasma HDL (Parker *et al.* 1996; Rimm *et al.* 1999), which may argue against agents such as polyphenols, which are

somewhat lower in most beers and white wine as opposed to red wine, and not a major component of most spirits (see Chapter 5).

Moderate consumption of alcohol might also reduce the risk of heart problems through its role in decreasing stress (Baum-Baicker 1985b; Pohorecky 1990; Marmot *et al.* 1993; Vasse *et al.* 1998). Perhaps beer is particularly valuable in this regard because the hop-derived bittering agents are said to have sedative and hypnotic in uences (Cooper 1994). Williams (1997) showed that exercise and alcohol independently bene ted the levels of HDL in men and women.

Imhof *et al.* (2001) demonstrated that the blood of non-drinkers and heavy drinkers had higher concentrations of a protein called CRP than did that of moderate drinkers. CRP is a marker of in ammation, and the authors suggest that an anti-in ammatory action of alcohol might be a factor in the bene cial impact of moderate consumption of alcohol.

Cleophas (1999) and Rimm and colleagues (1996) have summarised the literature comparing the relative impact of beer, wine and spirits on coronary heart disease. Rimm highlights the problems with so-called 'ecological studies' that are based on using existing data previously collected as part of census and surveillance programmes. This type of study tends to report that wine has bene cial impacts, but that beer and spirits don't. However, by way of illustration, Renaud *et al.* (1992) observed that wine drinking tends to be associated with other habits and that these are actually responsible for the additional bene ts (i.e. a secondary correlation is occurring). Wine drinkers often belong to socio-economic categories enjoying a healthier lifestyle and superior health care (Klatsky *et al.* 1997; Galobardes *et al.* 2001). The overall diet of those drinking wine may be better than that of people for whom beer is the drink of choice (Tjonneland *et al.* 1999) and they may exercise more (Woodward & Tunstall-Pedoe 1995). Burke *et al.* (1995) elaborate on this point of lifestyle by stressing how Australian men who preferred beer to wine also drank larger quantities, smoked more and had a generally less healthy diet. Analyses of dietary patterns have seen beer bracketed with 'convenience food' (Pryer *et al.* 2001). Watten (1999) reported a relationship between daily smoking and beer consumption, clearly highlighting the confounding factor scenario. It has even been observed (Osler 1998) that a partner's smoking promotes consumption of certain foodstuffs, including beer.

Rogers and Green eld (1999) claim that hazardous drinking (de ned as those occasions when ve or more drinks are consumed daily) is associated more with beer than with other types of alcoholic beverage and, predictably enough, this correlates with younger, unmarried males. Mortensen *et al.* (2001) found that those selecting wine over beer tended to have higher IQ, higher parental educational attainment and higher socio-economic status. Furthermore beer drinkers fared less well than wine drinkers on scales of psychiatric and health-related behaviour. Mortensen concluded that the apparent superior health bene ts of wine over beer were related to better social and

psychological performance. On the other hand, and perhaps unsurprisingly, those engaging in team sports (presumably leading to increased tness) consume more beer and spirits (Watten 1995).

An additional criticism of studies that are based on surveys is the reliability of individuals' reporting of alcohol intake (Dawson 1998; McCann *et al.* 1999). As Klatsky (2001) says: 'In data based upon surveys, systematic 'underestimation' (lying) probably tends to lower the apparent threshold for harmful alcohol effects.'

Rimm *et al.* (1996) highlight the greater reliability of case-control studies, in which controlled observations are made relating consumption of a speci c alcoholic beverage to ailments such as coronary heart disease. From such investigations (see also Cleophas 1999) it is clear that wine, beer and spirits all confer a reduction in coronary heart disease. As Rimm and co-workers (1996) say:

> We conclude that if any type of drink does provide extra cardiovascular bene t apart from its alcohol content, the bene t is likely to be modest at best or possibly restricted to certain sub-populations.

Barefoot *et al.* (2002) concur entirely, highlighting the signi cance of confounding factors as establishing the apparent difference in health impact of wine and beer. Wine drinkers had healthier diets than did those drinking beer (or spirits) and they were less likely to smoke. They reported that they ate more servings of fruit and vegetables and fewer servings of red or fried meats. The diets of wine drinkers contained less cholesterol and saturated fat, but more bre. Non-drinkers consumed fewer vegetables and more bre, and they were less likely to exercise regularly. They had a higher mean body mass index.

Nonetheless, there have been several reports suggesting that one type of alcoholic beverage is superior to another on a health basis. Rimm *et al.* (1996) insist that this relates to aspects of lifestyle associated with consuming drinks of a certain type. Klatsky *et al*. (1997) observed that wine consumers were less likely, and beer drinkers more likely, to develop coronary heart disease than spirit drinkers were, but that when the data was corrected for parameters such as sex, race, cigarette smoking and consumption of coffee, the correlations were eliminated. To emphasise the point: *differences in bene t or risk associated with beer, wine or spirits are probably associated with other lifestyle parameters and not due to the different drinks themselves.*

It seems that, if there is a preference for one type of drink within a population, then studies within that populace relating moderate alcohol consumption to health bene ts tend to highlight the advantages of that particular beverage. For instance, a study in Honolulu where a minority of the population consumes wine showed a signi cant inverse population between coronary heart disease and beer drinking (Yano *et al.* 1977). Similar results were obtained in countries noted for the popularity of beer, namely Germany (Keil *et al.* 1997) and the Czech Republic (Bobak *et al.* 2000). Hoffmeister

et al. (1999) went so far as to suggest that if European beer drinkers stopped imbibing there would be a decrease in life expectancy of two years – and a lot of unhappiness. In a study in Caerphilly, Wales, a clear bene t of alcohol consumption as a cardioprotective was demonstrated, even though the vast majority of the alcohol was consumed as beer, with little wine being consumed in that society (Fehily *et al.* 1993). In contrast, in wine-drinking rural Italy, the bene ts of wine were seen (Farchi *et al.* 2000).

Hendricks *et al.* (1994) demonstrated that alcohol (40 grams) taken at dinner in the form of wine, beer or spirits impacted bene cially on the plasminogen system, consistent with the notion that moderate consumption of alcohol in any of these forms reduced the risk of coronary heart disease. Similar results were reported by Rimm *et al.* (1999), who monitored changes in several parameters related to coronary heart disease and concluded that 30 g of alcohol per day offers the drinker almost 25% reduced risk of this disease.

Van der Gaag *et al.* (2000) report that alcohol taken as red wine and spirits causes an increase in the level of homocysteine in blood serum, an event that is associated with heart disease. They claimed that when the alcohol was taken as beer it did not lead to such an increase, and it was hypothesised that this was because of the presence of vitamin B_6 in beer.

Klatsky *et al.* (1997) decided that there might be minor additional bene ts linked to drinking both beer *and* wine, and not especially red wine. However, their study of 3931 people hospitalised for coronary disease showed an inverse relationship to CHD for each type of beverage, the weakest correlation being for spirits drinkers. For men the inverse relationship was signi cant for beer; for women it was signi cant for wine. But when analysis was controlled for total intake of alcohol, a signi cant relationship only remained for beer use by men. No signi cant differences in risk could be found when red or white wine was the drink of choice. Gaziano *et al.* (1999) were clear in their conclusions that beer, wine and spirits were equally advantageous (in moderation) (see also Renaud *et al.* 1993; Klatsky 1994; Klatsky *et al.* 1997; Hein *et al.* 1996; Rimm *et al.* 1996). As Klatsky (2001) says:

> …it seems likely that ethyl alcohol is the major factor with respect to CHD risk. There seem to be no compelling health-related data that preclude personal preference as the best guide to choice of beverage.

One particularly intriguing study was that of Hlavacek *et al.* (1999). They rst showed that rats preferred beer to water or to an aqueous solution of alcohol, and furthermore that the rats' apparent preference for different beers coincided with that of their human counterparts. The authors concluded that these rats were therefore a good model to compare drinkability of different beers. More pertinent to the present discourse, however, were Hlavacek's observations that the propensity of hamsters to develop atherosclerosis was minimised by the beers used in the rat study to an extent equal to that of red wine.

Such direct studies are important to underpin the much more prevalent investigations that are based on surveys and questionnaires.

It will be appreciated that components of the diet, such as antioxidants, will only be of bene t if they are demonstrated to enter the body and indeed reach the tissues that they are to protect. The number of studies demonstrating a direct uptake of antioxidants into the body is limited.

Bourne *et al.* (2000) showed that ferulic acid in alcohol-free beer (selected to maintain the sobriety of test subjects) could be detected in the urine (Fig. 6.2). Comparable results were observed for a series of polyphenolics (Walker & Baxter 2000). To reach the kidney a material must be absorbed into the digestive system. While this is not an *a priori* guarantee that the antioxidant reaches all of the tissues where it might have a protective role, it does appear that all of the ferulic acid is absorbed by the body, suggesting the ready availability of the substance in beer. By contrast, the absorption of ferulic acid from tomatoes was only 11–25%. This is an important illustration of how the food matrix can make a direct impact on the availability of a component. Just because it is present in high quantities in a food, it does not necessarily mean that the food is a better source of a material than another food in which it is present in lower quantities. If questioned, most people would say that tomatoes are a healthier food than beer. In at least one respect, though, this appears not necessarily to be the case.

It seems that the presence of alcohol stimulates the uptake of antioxidants into the body (Ghiselli *et al.* 2000). After only one hour, a 17% increase in the measurable antioxidant level (TAA) was observed in the volunteers' blood.

The main category of substances discussed in the context of antioxidant potential of red wines is the polyphenols (Frankel *et al.* 1993). Hertog *et al.* (1993) indicated that a

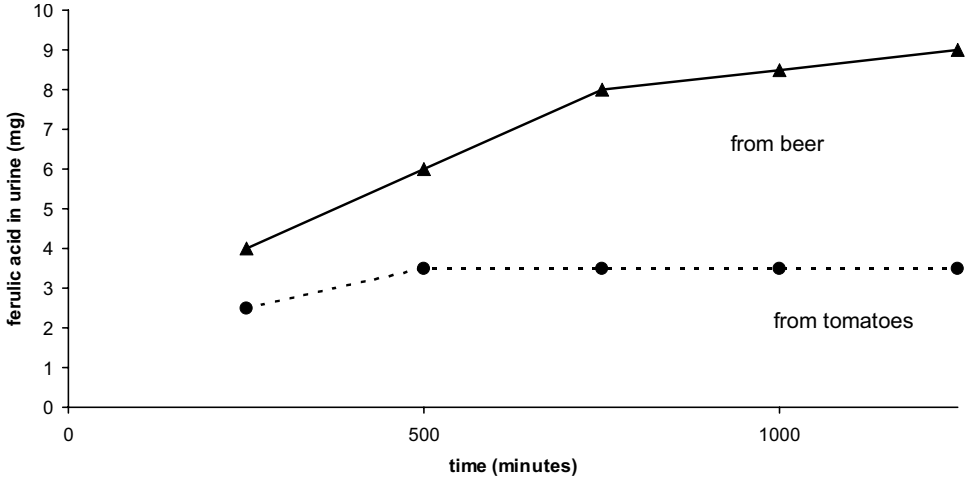

Fig. 6.2 Uptake of ferulic acid from beer and tomatoes. (Redrawn from Bourne *et al.* 2000.)

prime class among the polyphenols, the avonoids, could reduce risk of diseases of the cardiovascular system in humans. We ascertained in Chapter 5 that beer also contains this class of molecules.

An inverse link between cardiovascular disease and folate levels in the diet has been strongly implicated (Riddell *et al.* 2000). High levels of homocysteine arising from the metabolism of methionine are associated with increased cardiovascular disease, perhaps because homocysteine is toxic to endothelial cells (Bellamy *et al.* 1999; Langman & Cole 1999). Accordingly homocysteine levels are used as a diagnostic tool for cardiovascular disease. Increased folate leads to decreased homocysteine (Eikelboom *et al.* 1999; McDowell & Lang 2000). Folate might also protect against Alzheimer's disease (Clarke *et al.* 1998) and cancer of the colon and cervix (Mason & Levesque 1996).

Ubbink *et al.* (1998) assessed the homocysteine concentration in the serum of 2290 men to predict ischaemic heart disease. The mean serum total homocysteine concentration in the men who experienced an ischaemic heart disease event was signi cantly higher than for the 2136 men who experienced no such event, after standardising for the effects of differences in age, social class, smoking, body mass index, diabetes, HDL-cholesterol and prevalent ischaemic heart disease. The vitamin nutritional status and extent of alcohol intake were signi cant for their reduction of total homocysteine concentration in the blood serum. The authors explain the effect of alcohol by the folic acid content of beer, which is the preferred alcoholic beverage in Caerphilly, where the study was conducted. Halsted *et al.* (2002) have stressed, however, that binge-drinking alcoholics display an impediment to folate absorption by an inhibition of the carrier system needed to transport the vitamin. It is claimed that the folate de ciency accelerated changes in the methionine metabolism of the liver, with attendant oxidative damage and alcoholic liver disease.

The hop constituent, xanthohumol, has a strong inhibitory effect on the enzymes in liver microsomes (rat) that convert diglycerides to triglycerides and so it may reduce the extent of atherosclerosis (Tabata *et al.* 1997). Xanthohumol is also active in the oxidation of low-density lipoprotein (Miranda *et al.* 2000a).

A nal observation in the area of atherosclerosis is one that perhaps best illustrates the complexity of the human body and its many interrelated facets, as well as perhaps the risk of drawing correlations that may or may not have simple causal bases. Thus Lotufo *et al.* (2000) conclude that vertex pattern baldness (i.e. balding from the top of the scalp as opposed to from the front) is a marker for an increased risk of coronary heart disease. The authors offer an explanation, in that men prone to hair loss in this way seem to have more androgen receptors and higher levels of testosterone. It is claimed that the sex hormones directly impact events in the vascular system, leading to atherosclerosis, thrombosis and hypertension. Baldness, these authors would contend, is a symptom associated with cardiovascular disease in men, not a cause of it. The logic

would extend to an expectation that moderate consumption of alcohol in vertex-pattern bald men would have a more favourable effect than in hirsute or front-balding men, though I have not come across such a study.

Excessive consumption of alcohol is detrimental to the heart muscle, leading to a disease called cardiomyopathy (McDonald *et al.* 1971; Schoppet & Maisch 2001). It tends to be associated with excessive alcohol consumption over a prolonged period: one estimate is 120 g alcohol (roughly 7 pints of beer) every day for 20 years (Urbano-Marquez *et al.* 1989). Other muscles in the body are probably affected also.

In some circumstances it may not have been the alcohol alone that has led to the disease. For example, arsenic contamination led to major heart problems in Manchester in 1900 (Reynolds 1901), while cardiomyopathy was one consequence of the ill-reasoned use of cobalt as a foam stabiliser in Quebec and a few other places in the mid-1960s (Morin & Daniel 1967).

Excessive drinking has been proposed (Alvarez *et al.* 1999) as one of the several causes of atrial brillation – an irregular heartbeat – with attendant breathlessness and perhaps palpitations and angina. Koskinen (1991), by contrast, did not nd a causal link between misuse of alcohol and the occurrence of this disease. This type of con ict in the literature is not uncommon, because many studies involved drawing correlations between disease (or lack of disease) and records (often self-reported by patients) of dietary information, including the intake of beer. Frequently other correlations are at play that may confound the observations. A good example might be that people who smoke are often heavy drinkers. A direct correlation of a disease with the smoking that might be genuine would also correlate with alcohol intake, but this would not necessarily be a causal link. Of course the converse might apply equally. With a weather eye for these dif culties, let us continue.

Studies have indicated that hypertension (increased blood pressure) is twice as common in heavy drinkers as opposed to light drinkers, seemingly unconnected with any weight increase (Kannel & Ellison 1996). Beer use is said to be associated with higher blood pressure (Nevill *et al.* 1997). Potter and Beevers (1984) found that 4 pints daily over a period of 3–4 days had a demonstrable effect on this measurement. Keil *et al.* (1993) describe a causal link between 'chronic' intake (> 30–60 g alcohol per day, perhaps less for women) and the elevation of blood pressure in men and women. As a rule of thumb, the authors claim that at levels greater than 30 g alcohol per day, each increase of 10 g per day alcohol increases systolic blood pressure by an average of 1–2 mmHg and diastolic blood pressure by 1 mmHg. Only obesity is a greater risk factor. The precise way in which alcohol exerts its effect is unknown, but there appear to be neural, humoral and direct vascular elements.

Williams (1997) has shown that despite the bene cial effect of alcohol (and exercise) on levels of HDL-cholesterol in the body, the intake of alcohol continued to increase

the blood pressure in men, regardless of the level of exercise. Nanchahal *et al.* (2000) recommend that women should restrict their alcohol intake to less than 14 units per week to avoid an increase in hypertension.

There is a contrary view. Moline *et al.* (2000) suggest that there is an inverse relationship between avonoid levels circulating in the blood and the risk of hypertension. Indeed 40% of studies show that the blood pressure of non-drinkers is higher than is the case in those consuming 10–20 g alcohol per day (Keil *et al.* 1993). Thadhani *et al.* (2002) con rm that low to moderate alcohol intake leads to a lesser incidence of hypertension in women, and this is not related to any particular type of beverage. Truelsen *et al.* (1998), however, suggest that wine drinkers fare better than beer drinkers in the blood pressure stakes.

Strokes represent the third leading cause of death worldwide, after coronary artery disease and cancer (Suter 1999). There are essentially two types of stroke: blockage strokes and rupture strokes. The former is akin to a heart attack in the brain and is due to a blockage in an artery in the brain. A rupture stroke is caused by breakage of a cerebral blood vessel and resultant increase in pressure in the brain. Alcohol protects against the former type of stroke exactly as it protects against a heart attack. However, higher levels of alcohol intake, particularly habitually, increase the risk of a rupture type of stroke.

Potassium, magnesium and bre have been identi ed as signi cant modulators of the risk for stroke in men, perhaps through both direct and indirect effects on blood pressure and regulatory functions, such as endothelial function. These nutrients are readily obtained from a diet rich in fruits and vegetables and, as we saw in Chapter 5, beer is also a signi cant source of these components.

The biggest risk factor for strokes is hypertension. One report has it that moderate drinkers (< 60 g alcohol per day) have an equal or slightly increased risk of stroke compared to non-drinkers (Van Gijn *et al.* 1993). Other reports claim quite the opposite, with a reduced risk of stroke for light to moderate drinkers (Gill *et al.* 1988; Stampfer *et al.* 1988a; Gill *et al.* 1991; Rodgers *et al.* 1993; Palomaeki & Kaste 1993; Berger *et al.* 1999; Sacco *et al.* 1999). Moderate alcohol consumption was shown to protect against blockages of the chief arteries going to the brain (Bogousslavsky *et al.* 1990).

Wannamethee and Shaper (1996) stress that heavy drinkers (> 6 drinks per day) run an increased risk of stroke. Binge drinking is associated with an elevated risk of stroke (Juvela *et al.* 1995, Hillbom *et al.* 1999).

Instances of sudden cardiac death are increased by heavy alcohol consumption (Wannamathee & Shaper 1992) and reduced with light to moderate drinking (Albert *et al.* 1999).

The liver and the digestive system

Heavy drinkers develop a poor and idiosyncratic appetite, which will focus on the ingestion of fats and proteins rather than carbohydrates (Roe 1979). Alcohol in moderation, however, stimulates the appetite (Hetherington *et al.* 2001).

Alcohol suppresses the ow of saliva (Enberg *et al.* 2001), making the meal tend to be somewhat drier. Clearly beer would be expected to be better than wine in respect of hydrating the bolus. Swelling of the salivary glands (parotids) is a symptom of a heavy drinker (Santolaria *et al.* 1997).

Very heavy drinkers may also occasionally be prone to oesophagitis (Seifert 1995), the re uxing of stomach hydrochloric acid into the oesophagus leading to 'heart burn' and 'acid brash'.

Beer and to a lesser extent wine encourage the production of the hormone gastrin that switches on the ow of the gastric juices in the stomach (Chari *et al.* 1993). The stomach absorbs alcohol more ef ciently when it is full and it is metabolised more quickly (Sedman *et al.* 1976). Less alcohol then passes to the duodenum, from which alcohol is absorbed into the bloodstream very rapidly. Here is one explanation for the reduced feelings of lightheadedness when a drink accompanies the eating of a full meal, as opposed to nibbling.

Fasting appears to reduce the level of alcohol dehydrogenase (ADH; Riveros-Rosas *et al.* 1997), a key enzyme involved in metabolising alcohol (Baraona *et al.* 1994), 90% of which is metabolised by the liver. ADH produces acetaldehyde, which is toxic if not adequately dealt with by the next enzymes in the cascade, aldehyde dehydrogenase. An accumulation of acetaldehyde causes hangovers and liver damage in the long term (Agarwal & Seitz 2001).

In men, 80% of ADH is in the liver and the remainder in the stomach (Myerson 1973). Women produce less ADH, meaning that alcohol has more signi cant effects on them (Seitz *et al.* 1993), although ADH levels in women increase after the menopause, so that presumably older women are better able to deal with alcohol.

A secondary alcohol-metabolising system in the body, known as the microsomal ethanol oxidising system (Lieber 1999), is probably stimulated to an increased extent by regular consumption of alcohol. When oxidised through this system, alcohol provides only two-thirds of calories that are generated when ADH deals it with.

The ADH system is polymorphic – and different in Asians (Li & Bosron 1986; Yoshida *et al.* 1991). It is claimed that the genetic make-up of ADH impacts the response of individuals to alcohol in respect of the levels of acetaldehyde produced (Yin & Agarwal 2001). Acetaldehyde may be the causative agent in several problems ascribed to excessive alcohol consumption. The Chinese and Japanese ush very readily, due to a mutation in one of the aldehyde dehydrogenases (Crabb *et al.* 1989).

Through adaptation, heavy drinkers probably metabolise alcohol more rapidly than do non-drinkers, provided that their digestive system has not been damaged. Forsander and Sinclair (1992) produced evidence based on studies with rats that rates of alcohol elimination and alcohol consumption are partially determined by genetics. Rats displaying higher rates of alcohol elimination or levels of ADH had higher voluntary intakes of alcohol than rats with lower elimination rates. Although alcohol elimination itself probably does not exert direct control over drinking, some factor related to the rate of alcohol elimination appears to be among the mechanisms in uencing the level of alcohol consumption.

Heavy drinkers commonly suffer from chronic gastritis, an in ammation of the stomach lining (Figlie *et al.* 2002). It is a particular problem for those who also smoke. Alcohol to excess also affects the blood supply to and motility of the small intestine (Chiba & Phillips 2000). There is good evidence for a link between the organism *Helicobacter pylori* and ulceration of the stomach and duodenum, as well as stomach cancer. It has been reported that alcohol protects against infection by *H. pylori* (Brenner *et al.* 1997, 1999, 2001; Ogihara *et al.* 2000), in fact countering the effect of coffee. However, it is also claimed that alcohol lessens the incidence of this organism in older people, but appears to promote the growth of the organism in younger folk. Ohsugi *et al*. (1997) showed that the hop β-acid lupulone could inhibit growth of *H. pylori*, so conceivably it is not alcohol alone that is responsible for the effect.

There appears to be an increased risk of pancreatitis in heavy drinkers (Dreiling *et al.* 1952; Haber *et al.* 1995; Sakorafas & Tsiotou 2000), with about 1 in 20 people suffering. As this tissue is responsible for making digestive enzymes and also insulin, there are attendant problems with digestion and diabetes. The reduced digestive ef ciency leads to an increased level of triglycerides and therefore atheroma and increased risk of cardiovascular disease (compare this statement with the observed upturn in the U-shaped curve for high alcohol intake; see earlier). Schmidt (1991) suggests that the consumption of distilled spirits, but not wine or beer, is a risk factor for pancreatitis.

Gall bladder activities are improved by alcohol. Its consumption speeds up the emptying of the gall bladder after a meal and increases the rate of lling, too – so people with reasonable alcohol intake develop fewer gallstones. Leitzmann *et al.* (1999) showed that, after adjusting for other risk factors, an increased amount of alcohol consumed correlated with a decreased risk of symptomatic gallstone disease. It seemed that frequency of intake was an important factor, with intake 5–7 days per week leading to a decreased risk, as compared with non-drinkers. In contrast, infrequent alcohol intake (1–2 days per week) led to no change of risk. The nature of the alcoholic beverage did not appear to be signi cant.

The *Oxford Textbook of Medicine* (Weatherall *et al.* 1996) suggests that consumption rates of 80 g alcohol daily by a man and 50 g by a woman gives a 15% chance of liver damage. These levels equate to more than 5 pints and 3 pints of average-strength beer,

respectively. However, consumption has to be regular and spread over many years (e.g. 15 years or more). It seems that the number of heavy drinkers who will actually develop cirrhosis is one in ten (Stuttaford 1997). Furthermore it needs to be recognised that there are various other causes of cirrhosis, such as in countries where hepatitis B is endemic.

Gruenewald and Ponicki (1995) reported a link between cirrhosis and excessive consumption of spirits, but not beer or wine. Similar results were reported by Kerr *et al.* (2000), based on data garnered from Australia, Canada, New Zealand, the UK and the US. Tverdal and Skurtveit (2003) observed an inverse relationship between the consumption of coffee and the instance of cirrhosis, including alcoholic cirrhosis.

Alcoholics, of course, tend to be malnourished (Lieber 2001). This has led to the concept in many critics' minds of alcoholic beverages representing merely 'empty calories' – i.e. they provide just energy (calories) without other nutrients. However, reference to Chapter 5 will illustrate why this can be refuted, at least for beer. Of course beer is de nitely not a meal in itself and it would be ridiculous to suggest it. There *are* people who abuse alcohol, and they will tend to be malnourished, at least in part because they do not have (or devote) money for the selection of a well-balanced diet. Somebody addicted to, say, chocolate would similarly be malnourished if they primarily consumed that and not the other key diet constituents described in Chapter 4. In abusers, though, alcohol will have a direct impact on metabolism (Bitsch 2003). High levels of ethanol impair the intestinal absorption and transport of some of the amino acids, e.g. isoleucine, arginine and methionine. There are also adverse impacts on the uptake of folate, and the oxidation product of ethanol (acetaldehyde) triggers the breakdown of that vitamin. This, together with the impact of high ethanol levels per se, causes damage to the intestinal mucosa, with attendant impairment of general nutrient uptake.

It is claimed that there is an increased risk of liver cancer from excessive consumption of alcohol; however, this is confounded by and perhaps related to the incidence of cirrhosis (Ohnishi 1992). Those imbibing alcohol to excess can also display fatty in ltration, a swelling of the liver and attendant lowered appetite and nausea (Kishi *et al.* 1996). Binge and prolonged drinking can cause alcoholic hepatitis (Maher 2002).

However, it would be wrong to conclude that there are no positive impacts of a food such as beer on the digestive system. Faist *et al.* (2002) showed that water-soluble melanoidins from roasted malt promoted the activity of detoxifying enzymes (NADPH–cytochrome *c* reductase and glutathione-S-transferase) in intestinal cells. Even at the very front end of the gut there may be bene ts. Tagashira *et al*. (1997) showed that hop polyphenols could inhibit growth of streptococci and delay the development of caries. Nakajima *et al.* (1998) found that dark beers (more so than paler beers) inhibited the synthesis of the polysaccharide that anchors streptococci to the teeth. They did not identify the inhibitory material(s), but suggested that all three stages of roasting, mashing and fermentation are signi cant in its development. On the other hand, beers that

contain signi cant levels of residual sugar and unusually low pH (< 4.0) have potentially harmful effects on teeth (Nogueira *et al.* 2000).

Too much alcohol can affect absorption of all foods, but especially vitamins and other micronutrients through an effect on gastrointestinal motility and intestinal permeability (Knight *et al.* 1992). Once again we encounter the importance of balance in the diet. For example, some of the useful avonoids will not be available if essential vitamins are absent. In turn there is a requirement to have enough fat in the diet if these vitamins are to be utilised. An excess of one trace element can restrict the intake of another.

Alcohol may improve glucose tolerance (Baum-Baicker 1985a). It seems that alcohol attenuates the increase in blood glucose concentration in subjects given a glucose load, with an accompanying increase in the concentration of insulin in plasma (Facchini *et al.* 1994). The implication is that alcohol increases the sensitivity of susceptible cells to insulin. This in turn reduces demand on the pancreas. In a study of 735 'middle-aged' British men, moderate drinkers (16–24 units per week) displayed a reduced risk of developing non-insulin dependent diabetes (Perry *et al.* 1995).

Recently there have been some intriguing studies on the relationship between alcohol consumption and the development of type II diabetes mellitus. This is the type of diabetes that arises because the body does not make suf cient insulin and the system does not work properly to control glucose levels, leading to hyperglycaemia. It was formerly called 'adult-onset diabetes' and it accounts for 85–90% of diabetes in people over the age of 30. The biggest risk is obesity.

Wannamethee *et al.* (2002) found that heavy drinkers run a greater risk of type II diabetes. However, light and moderate drinkers did not run this risk. Stampfer *et al.* (1988b) found a lower incidence of non-insulin dependent diabetes in moderate drinkers (female nurses). Rimm *et al.* (1995) observed that moderate alcohol consumption among healthy people might be associated with increased insulin sensitivity and a reduced risk of diabetes. Moderate alcohol consumption may have a bene cial effect on the risk of death due to coronary heart disease in those people displaying type II diabetes (Valmadrid *et al.* 1999; Solomon *et al.* 2000). Tsumura *et al.* (1999) discovered that among men with a body mass index of 22.1 or more, moderate alcohol consumption was associated with a reduced risk of type II diabetes. However, among lean men (BMI below 22.1), heavy alcohol consumption was associated with an increased risk of type II diabetes.

If glucose accumulates through diabetic conditions then it is converted into sorbitol by aldose reductase, the accumulating sorbitol leading to damage of tissues such as eyes and kidneys. It has been shown that components of beer, including quercetin and the iso-α-acids, inhibit aldose reductase (Shindo *et al.* 2002).

Alcohol enhances the absorption of glucose and galactose (Carreras *et al.* 1992). There is little effect on fat absorption, provided there is an adequate intake of proteins.

Heavy drinking interferes with uptake of several nutrients (Chiba & Phillips 2000) including essential amino acids.

Thiamine de ciency is often claimed to be prevalent in heavy drinkers, and is frequently cited as a hallmark of malnutrition. Poupon *et al.* (1990), however, presented evidence to suggest that thiamine de ciency is either slight or absent in chronic drinkers. It is important to re-emphasise that a key reason why an alcohol abuser may have a substandard diet is because they do not have available funds to secure that diet, rather than a direct effect of alcohol on the ability to use the various components of the food intake.

Zinc is recommended as a dietary supplement for heavy drinkers (Zhou *et al.* 2002). There is evidence that excessive levels of alcohol deplete the body's reserves of this element, which is important for the reproductive systems of both sexes (Bedwal *et al.* 1991).

The bre content of beer is likely to be a contributor to atus in beer drinkers. Bolin and Stanton (1998) demonstrated a clear link between bre intake and frequency of daily emissions, which averaged 12.7 (range 2–53) for men and 7.1 (range 1–32) for women. There was also a correlation between men's beer drinking and the aroma of the resultant atus – indeed, men generally felt compelled to report more aromatic wind.

Beer, being produced from a cereal base, may present a dietary risk to those suffering from coeliac disease (Ellis *et al.* 1990; see later).

The reproductive system

As William Shakespeare observes in *Macbeth*, alcohol 'provokes the desire but takes away the performance'. Many men would identify with that, though it seems that there is little evidence that there is any downturn in their ability to procreate after the overindulgence has passed. E.M. Jellinek (cited by Roueche) is quoted as saying:

> Germ tissue is the toughest of all human tissues. Germ tissue could be damaged by very high concentrations of alcohol, but it is so wonderfully protected that before such concentrations of alcohol would occur, the alcoholic father or mother would be dead.
>
> Roueche (1960)

Roueche also quotes Miles Weatherall of the London Hospital Medical College:

> The available facts suggest that a man must drink rather a lot before alcohol is seriously harmful to him and that it is not impossible that the consumption of a little alcohol daily may even be a bene cial practice.
>
> Roueche (1960)

Juhl *et al.* (2001) produce evidence to refute the claim that there is any link between alcohol consumption and waiting time to pregnancy (i.e. fecundity).

Lapcik *et al.* (1998) and Gavaler (1998) demonstrated that beer contains a range of health-promoting iso avonoids (phytoestrogens) (see also Walker 2000). There have been concerns that such materials may adversely modify the hormonal status of men, but Promberger *et al.* (2001) have concluded that the risk is negligible owing to the extremely low levels of these substances found in beer. Equally, of course, this does bring into question whether such low levels have any bene cial effects either. The level of the principal iso avonoid found in beer (isoxanthohumol, 1.5 mg/L or less) is about 20-fold lower than the effective human dose for anti-cancer treatments (Forster & Koberlein 1998). More on this later.

The xanthohumol found in hops is devoid of oestrogenic activity but 8-prenylnaringenin is not (De Keukeleire *et al*. 1997a). It seems that this is the most potent oestrogen yet identi ed. Hops are more effective than the widely used plant preparations to relieve postmenopausal symptoms (Dixon-Shanies & Shaikh 1999). Hop extracts suppress menopausal hot ushes (Goetz 1990).

Hops are now being included in some herbal preparations for women for breast enhancement. Milligan *et al.* (2000) investigated the relative oestrogenic, androgenic and progestogenic activities of 8-prenylnaringenin in comparison to 6-prenylnaringenin, 6,8-diprenylnaringenin and 8-geranylnaringenin. While the latter three exhibited some oestrogenicity, their potency was less than 1% of that of 8-prenylnaringenin, which alone competed strongly with 17 β-estradiol for binding to both the α- and β-oestrogen receptors. None of the compounds tested, which also included xanthohumol, isoxanthohumol, 3′-geranylchalconaringenin, 6-geranylnaringenin and 4′-O-methyl-3′-prenylchalconaringenin, as well as polyphenolics from hops, showed progestogenic or androgenic bioactivity. This indicates that the endocrine properties of hops and hop products are due to the very high oestrogenic activity of 8-prenylnaringenin.

It has been estimated (Milligan *et al.* 1999) that beer might account for some 10% of the daily intake of phytoestrogens (vegetables, cereal grains and especially soya beans are rich sources too). The phytoestrogens are said to counter breast and prostate cancer, as well as cardiovascular disease (Knight & Eden 1996).

Emanuele and Emanuele (1998) present an alarming review of the impact of excessive alcohol consumption on male potency. There is interference with the function of each of the three main components of the male reproductive system, namely the hypothalamus, the anterior pituitary gland and the testes. The impact is impotence, infertility and reduced secondary sexual characteristics. Alcohol can adversely affect the Leydig cells of the testis that produce and secrete testosterone. Alcohol also impairs the function of the Sertoli cells that play an important role in sperm maturation in the testis. Alcohol can decrease the production, release and activity of luteinising hormone

and follicle-stimulating hormone in the pituitary gland. These have importance for reproductive functions. Alcohol can interfere with hormone production in the hypothalamus. In moderate drinkers any effects are short-lived, but the problem for alcoholics is signi cant (O'Farrell *et al.* 1998).

This report should be contrasted with that of Heaton and Varrin (1991), who intriguingly made a study of the impact of alcohol ingestion on two physiological events: yawning and penile erections. They found that 0.25 mg/kg ethanol had no impact on either response, whereas twice this amount suppressed erections but not yawning. Relatively high doses (1–3 mg/kg) had to be injected intraperitoneally for there to be an effect on both yawning and the erectile response. The authors suggest that alcohol interferes with neural receptor systems.

One of the most widely reported effects of alcohol is fetal alcohol syndrome (FAS), in which there are malformations in the child (Streissguth 2001). The child will tend to be underweight, shorter and possessed of a decreased skull circumference. There may be certain facial abnormalities, internal de ciencies in joints, and congenital heart disease. According to Weatherall *et al*. (1996) this occurs only in women who drink at least 4 units of alcohol daily throughout early pregnancy, and is greatly increased if the rest of the diet is not good. It appears that the risks are rather greater when women binge drink (four or ve drinks at one sitting) rather than consume alcohol moderately, e.g. one drink daily (Streissguth 2001). A 1987 American study of 32,870 women, of whom nearly half had taken alcohol during pregnancy, found no cases of FAS. There was a correlation with prosperity.

Lesser but related problems in children of heavy drinkers are known as fetal alcohol effect (Clarren & Smith 1978). Here the children are smaller and more excitable. However, it is unclear how much of the effect is due to alcohol and how much to maternal deprivation. There appears to be little risk if women drink less than 10–12 units a week, unless they also smoke.

Stuttaford (1997) says that women need not be teetotal when pregnant, but that they should only drink at formal or special occasions and with a meal, no more than 2 units per session and less than 7 units per week.

Alcohol is excreted in breast milk – but at levels unlikely to make baby more than drowsy (Stuttaford 1997). Koletzko and Lehner (2000) remind us of the received wisdom that moderate beer consumption may help in the initiation and success of breast-feeding. It seems that a component of beer, perhaps a barley polysaccharide, promotes prolactin secretion. The authors further suggest that the relaxing effects of alcohol and hop components might also have a bene cial impact on lactogenesis. Mennella (2001) offers a contrary view, saying that maternal alcohol consumption may slightly reduce milk production and that alcohol transferred to the infant may adversely affect the infant's sleep and motor development.

Brain and cognitive function

One of the most alarming trends in recent years in the US has been the habit of binge drinking among young people. Students, newly freed from constraints, partake of ludicrous drinking rituals, frequently with tragic consequences. These episodes generally involve high-alcohol, low-volume beverages rather than beer, whose sheer volume is not commensurate with excessive consumption in short periods of time. Having said that, there is no question that any signi cant intake of alcohol has at least some impact on the brain. Modest intakes may be bene cial, excessive intakes are certainly not.

Those who have imbibed excessive amounts of alcohol and who sleep or lose consciousness run the risk of swallowing their tongue due to a relaxation of the airways through inhibition of medullary centres of the brain. They may inhale vomit because the preventive re ex centres in the brain are anaesthetised.

The Royal College of Physicians guidelines on the relationship between the alcohol content of blood and mental and motor response are:

- 30 mg/100 mL in blood (2 British units) makes the drinker relaxed.
- At 50 mg/100 mL (3 units) the consumer is cheerful, with some loss of inhibitions.
- At 80 mg/100 mL there is an impairment of driving ability.
- 150 mg/100 mL leads to a loss of self-control.
- By the time 400 mg/100 mL is reached, the drinker is oblivious to events and surroundings.
- A further 8 drinks will result in coma.
- Those who take 30 drinks in one evening are likely to die.

The amount of alcohol that will be found in the blood depends on various factors, of which sex and body weight are two signi cant ones. Table 6.3 allows an approximate computation of the blood alcohol that will arise from consumption of different numbers of drinks.

Alcohol is being metabolised as it is being drunk. Clearly there are far greater risks when the alcohol does not take a lot of drinking. Thus the sheer volume of beer relative to alcohol content makes the short-term risks from its consumption less than for the drinking of spirits.

An average person's body clears alcohol at the rate of 15 mg per 100 mL blood per hour. That is, one unit of alcohol is removed hourly. The rate is not in uenced by the amount of alcohol – i.e. higher concentrations of alcohol are not metabolised at a faster rate. Therefore excessive drinking can have carryover effects the next day. The ability to metabolise alcohol decreases with age.

Tremoliere *et al.* (1975) compared the rate of alcohol utilisation in the body after challenging with beer, wine or spirits. Fourteen subjects with empty stomachs were given 0.5 g alcohol per kg body weight as beer, wine or whisky over periods of 15 days

Table 6.3 Estimated blood alcohol content (mg/mL) for people of different weight consuming different numbers of drinks (1 drink = 355 mL of beer of around 5% ABV).

Weight (pounds)	1 drink	2 drinks	3 drinks	4 drinks	5 drinks
Men					
100	0.43	0.87	1.3	1.74	2.17
125	0.34	0.69	1.03	1.39	1.73
150	0.29	0.58	0.87	1.16	1.45
175	0.25	0.5	0.75	1.0	1.25
200	0.22	0.43	0.65	0.87	1.08
225	0.19	0.39	0.58	0.78	0.97
250	0.17	0.35	0.52	0.7	0.87
Women					
100	0.5	1.01	1.52	2.03	2.53
125	0.4	0.8	1.2	1.62	2.02
150	0.34	0.68	1.01	1.35	1.69
175	0.29	0.58	0.87	1.17	1.46
200	0.26	0.5	0.76	1.01	1.26
225	0.22	0.45	0.68	0.91	1.13
250	0.2	0.41	0.61	0.82	1.01

Source: derived from Fox (1997).

to one month. They found that the ethanol in beer was oxidised twice as fast as that in wine and seven times faster than that in whisky. Absorbed alcohol passes into the blood in 15–30 minutes if the stomach is empty, but in 1–3 hours if the stomach is full. There is a constant rate of metabolism of approximately 100 mg per hour per kg body weight. Thus an adult male weighing 70 kg drinking of 750 mL of beer on an empty stomach would not reach the French drinking limit of 0.8 g/L of blood.

Alcohol should not be consumed with aspirin, which disables alcohol dehydrogenase (Roine *et al.* 1990).

The predisposition to hangovers differs considerably between people with regard to the intensity of the condition in relation to how much alcohol has been taken. Furthermore it also depends on the type of drink. Thus, among the spirits, brandy has a more severe impact than whisky, with vodka having proportionately less tendency to cause hangovers. There has been much discussion but few rm conclusions concerning the prime causative agents of hangovers (Pradalier & Ollat 1991). They are believed partly to be due to a build-up of acetaldehyde produced via the oxidation of alcohol (Wall *et al.* 2000). The aldehyde interacts with components of brain cells to exert its effect. Congeners such as traces of methanol are also oxidised by alcohol and the resultant formaldehyde is even more unpleasant in its effects than acetaldehyde.

Other compounds that may contribute to hangovers include some of the biogenic amines that are found in relatively small quantities in beer (see Table 6.4). These include 15–200 μg/L histamine, 0.7–35.5 mg/L tyramine, 0.5–07 mg/L cadaverine, 3.1–5.6 mg/L putrescine and < 0.1–0.8 mg/L β-phenylethylamine (Cerutti *et al.* 1989).

Table 6.4 Amine content of beer, wine and cheese.

	Spermine	Spermidine	Putrescine	Cadaverine	Histamine	Tyramine	Tryptamine
Beer							
Lager	n.d.–1.41	n.d.–6.0	0.85–9.8	0.15–2.6	n.d.–0.9	0.3–3.1	n.d.–0.8
Stout	n.d.–2.05	0.31–1.38	1.99–5.84	0.3–1.37	n.d.–0.85	0.48–36.8	n.d.–10.1
Ice	n.d.–0.3	0.6–0.8	3.9–4.5	0.1–0.2	n.d.	0.7–1.4	n.d.
Bock	n.d.–1.73	0.25–2.1	1.55–6.3	0.15–1.72	n.d.–1.46	0.81–5.05	n.d.–3.5
Non-alcoholic	n.d.–1.2	1.35–2.3	2.3–4.95	n.d.–0.5	n.d.–0.62	0.6–3.3	n.d.–1.41
Wine							
Pinot Noir	n.d.–2.38	n.d.–2.35	2.43–203	n.d.–2.07	n.d.–23.98	n.d.–8.31	n.d.–5.51
Cabernet	n.d.–1.17	n.d.–4.03	3.15–23.6	n.d.–1.51	n.d.–10.1	n.d.–7.53	n.d.
Cheese							
Blue	—	—	9.6–23.7	42.3–227	n.d.–409	2.2–166	n.d.–110
Cheddar	—	—	n.d.–99.6	n.d.–40.8	n.d.–154	n.d.–153	n.d.–30
Gorgonzola	—	—	1.2–124	5.8–428	1.7–191	8.9–255	2.4–43
Gouda	n.d.– 1.13	n.d.–1.35	n.d.–107	n.d.–99.5	n.d.–30.5	n.d.–67	n.d.–88
Mozzarella	n.d.–1.31	n.d.–1.06	n.d.–1.37	n.d.–2.34	n.d.–11.3	n.d.–41	n.d.–10
Parmesan	0.07–0.09	n.d.–0.15	n.d.–4.3	n.d.–9.8	n.d.–27.2	n.d.–29	n.d.–1.7
Provolone	0.07–0.97	n.d.–2.38	n.d.–8.7	n.d.–111	n.d.–8.2	n.d.–10.9	n.d.–1.08
Swiss	—	—	—	—	n.d.–250	n.d.–180	n.d.–1.6

Quantities are mg/L for beer and wine; mg/100g for cheese.
n.d. = not detectable; — = not determined.
Source: derived from Gloria (2003).

The depression and irritability that may result after drinking substantial quantities of alcohol are a result of temporary cerebral damage, and the hypoglycaemia (low blood sugar) that results from the liver's decreased ability to release sugar into the bloodstream and because the pancreas increases its production of insulin (Arky *et al.* 1968). This also accounts for the heavy sweating that occurs despite dehydration, and also contributes to headaches and dizziness.

Although sparkling wines and spirits were more frequently associated with migraine attacks than were other types of alcoholic beverages, including beer, it seems that stressful events were of more signi cance (Nicolodi & Sicuteri 1999). Chocolate, cheese and even citrus fruits are cited more frequently than alcohol as triggers of migraine attacks (Costa & Gloria 2003). Even so, clearly a substantial proportion of people have a migraine response to alcoholic drinks. Apart from the vasoactive amines, ethanol itself may be implicated. It is a vasodilator, probably as a consequence of an effect on the central vasomotor centres rather than directly on the blood vessels. Ethanol also inhibits monoamine oxidase, thus causing a build up of amines.

Excess consumption of alcohol leads to alcoholic dementia with dif culties in memory recall and lateral thinking skills (Dreyfuss 1979). Reversible cerebral atrophy or shrinkage of the brain may also result. The two conditions of chief signi cance in this context are Korsakoff's syndrome and Wernicke's encephalopathy (Lieber 2001).

Korsakoff's syndrome (Bowden & McCarter 1993) is characterised by a loss of memory and a loss of limb sensation and is believed to be due to a shortage of thiamine and perhaps other B vitamins. Wernicke's encephalopathy (Jolliffe *et al.* 1941), characterised by haemorrhaging in the brain, is also due to a shortage of B vitamins. Again it must be stressed that these conditions are only to be found in abusers who are not taking a well-balanced diet.

Marchiafava-Bignami disease, which is a brain disease with symptoms that include loss of balance and irrational behaviour, also probably relates to malnutrition in excessive consumers (Neiman 1998). Likewise, peripheral neuropathy (the damage to nerves around the body) is probably more a function of vitamin de ciency than alcohol *per se* (Todd *et al.* 1999).

The classic phenotypic condition associated in the popular consciousness with retreat from alcoholic excess is delirium tremens (the 'DTs'), a confused state in which the drinker is agitated, suffers delusions (especially auditory) and shakes (Saitz 1998).

The effects described above are associated with *excessive* consumption of alcohol. Are there any bene ts to the nervous system from moderate intake of alcohol?

In a study of 4739 sets of twins born between 1917 and 1927, a J-shaped curve was demonstrated between alcohol consumption and cognitive function (Christian *et al.* 1995). Moderate drinkers performed signi cantly better than abstainers or heavy drinkers. A study in the Aichi Prefecture of Japan by the National Institute for Longevity Sciences, reported in *New Scientist* of 9 December 2000, claimed that those who drink in moderation register a higher IQ than abstainers – by 3.3 points for men and 2.5 points for women. This is irrespective of the preferred drink. There appears to be no conclusive explanation for the observation – and it may well be another example of secondary correlation: those drinking alcohol in moderation may adopt other lifestyle behaviours that are commensurate with greater intellectual capacity.

Baum-Baicker (1985b) identi ed ve bene ts on the mind and intellect associated with moderate drinking. The claim is that moderate drinkers (a) are more outgoing and enthusiastic about life, (b) are less stressed and enjoy all aspects of their life more, (c) perform some tasks better after a drink, (d) enjoy fewer incidences of depression, (e) fare better when elderly, including better cognitive function.

Because vascular disease is associated with cognitive impairment and dementia, Ruitenberg *et al.* (2002) hypothesised that alcohol consumption might also affect the risk of dementia. They found that light to moderate drinking (1–3 drinks per day) is signi cantly associated with a lower risk of dementia in those who are aged 55 years or older. It did not depend on the type of alcoholic beverage.

There is some evidence that people respond to what they *think* is the strength of the beer. For example, performance deteriorates after high consumption of an alcohol-free drink if the consumer *believes* it contained alcohol (Mortensen *et al.* 2001).

Alcohol encourages sleep initially, but reduces overall sleep and induces restlessness by reducing REM sleep time (Vitiello 1997).

Feelings of pleasure have been linked to levels of dopamine in the body (Berridge & Robinson 1998). Schmidt *et al.* (2001) report how alcohol-dependent patients display a reduced sensitivity in their central dopamine receptors.

Kidney and urinary tract

Alcohol dehydrates the whole body (except the brain, which swells) because of a diuretic impact on the kidney (Olson 1979). This explains the merit of drinking plenty of water before retiring to bed after drinking.

Buday and Denis (1974) and Piendl and Wagner (1985) have researched the diuretic effect of beer. Beer is rather more diuretic than is water, and several components, including organic acids and other yeast fermentation products and polyphenols, are said to contribute.

A category of persons at risk from consuming beer is those with gout (Eastmond *et al.* 1995). Gout is an arthritic condition caused by high levels of uric acid circulating in the blood. There is an attendant deposition of crystals of urate in connective tissue and this stimulates an acute in ammatory reaction. Alcohol is certainly not the only cause, others being gluttony and excessive eating, diuretic pills, infection, and even aspirin. Alcohol is more of a risk if its intake is not accompanied by proper eating. There is a major genetic impact on an individual's predisposition to gout. Susceptible people need to avoid purine-rich foods such as liver, kidney and shell sh. Some beers may be a particular problem when compared to other alcoholic drinks because they may contain signi cant quantities of purines (see Chapter 5).

There is good evidence that beer is superior to water alone in ' ushing out' the kidneys and protecting the kidney against stones (Curhan *et al.* 1998; Shuster *et al.* 1985; Krieger *et al.* 1996). Hirvonen *et al.* (1999) observed that beer consumption was inversely correlated with a risk of kidney stones (urolithiasis), with each bottle of beer consumed daily being estimated to reduce risk by 40%. The authors say that high intakes of calcium, potassium and water are associated with lowered risk of kidney stones. Magnesium intake, too, may have a role. Curhan *et al.* (1996) also found that beer, as well as wine, tea and coffee, reduced the risk of kidney stones. Apple juice and grapefruit juice increase the risk.

In a curious study, Nagao *et al.* (1999) presented evidence to suggest that the older and more stale the beer, the less was its tendency to promote urination. Adding materials to beer that detracted from its quality by introducing unpleasant tastes also lowered urination rate.

Age

Over and above the demonstration that moderate alcohol intake (as beer or wine) in the elderly appears to be associated with signi cantly longer survival in men aged 60–74 years and in all elderly women (Simons *et al.* 2000), there is evidence that moderate alcohol consumption may be associated with better cognitive function in old age (Dufouil *et al.* 1997; Cervilla *et al.* 2000). Moderate consumption of wine and beer reduces the odds of developing age-induced macular degeneration (Obisesan *et al.* 1998). Light to moderate consumption of alcohol reduced the risk of those 65 and older from dying of cardiovascular disease (Scherr *et al.* 1992; see earlier). Low doses of alcohol, including beer, stimulate appetite and promote bowel function in the elderly (Dufour *et al.* 1992).

Cupples *et al.* (2000) showed that regular consumers of alcohol, including those drinking above and below the recommended limits of one drink per day (women) and two drinks per day (men) had a signi cantly lower risk of Alzheimer's disease as compared to non-drinkers.

Aluminium has been cited as an agent promoting Alzheimer's disease (Martyn 1990), although more recent evidence is rather that those with Alzheimer's are more sensitive to the impacts of aluminium, rather than it being a causative agent *per se* (Roberts *et al.* 1998). Approximately 1% of the aluminium in the diet is accumulated in the liver, spleen, brain, muscles and bone (Fahal *et al.* 1993). Concerns from some quarters about beer kegs being a source of aluminium have been refuted by Williams (1996) who observed from a study of a diversity of beers that there is far less aluminium in beer than many other foods and beverages. Not only that, but Williams reminds us that this and other metals may only exert an effect when they are in a free, accessible state, whereas the low levels of aluminium in a product such as beer are in sequestered forms (e.g. with silicate) that do not assimilate into the body. Silicon is believed to be a key agent in eliminating aluminium from the body (Bellia *et al.* 1996).

A study in Massachusetts (Volpe & Kastenbaum 1967) used beer among other factors to enhance the environment in a ward nursing elderly psychogeriatric men. The impact was a decrease in the need for medication, including anti-psychotic drugs, a regaining of continence and a reduced need for physical restraint. The patients became more sociable. It seems that the beer itself (approximately half a pint a day) was key. There was a sizeable psychological in uence at play – the beer was seemingly symbolic of a trust in the patients that they could look after themselves and be responsible.

As Professor Robert Kastenbaum says:

> There is by now suf cient information available to indicate that moderate use of alcoholic beverages is pleasurable and bene cial for older adults. The lower

> ethanol beverages such as beer and wine may have their place in the lifestyles of some older adults.
>
> Stuttaford (1997)

Thomas and Rockwood (2001) and Liberto *et al.* (1992) sound a cautionary note, however. Alcohol abuse can of course occur in the elderly just as it does in younger people, leading to an increased prevalence of all types of dementia, except Alzheimer's disease.

An obvious risk of excess alcohol consumption in the elderly is the increased tendency to fall. However, there is very little evidence to support claims that alcohol consumption increases the risk of osteoporosis. Osteoporosis means 'porous bones', and is caused by a depletion of calcium, phosphorus and other minerals. In fact bone mineral density is higher in social or moderate drinkers than in abstainers or heavy drinkers. 8-Prenylnaringenin is claimed to counter osteoporosis (Miyamoto *et al.* 1998).

Rico *et al.* (2000) suggest that silicic acid is readily absorbed from beer and that this may protect against osteoporosis. Jugdaosingh *et al.* (2002) stress how beer and bananas can be the richest source of silicon for men, although string beans replace beer in this context for women.

Mukherjee and Sorrell (2000) show that moderate alcohol consumption has positive effects on bone mineral density in elderly women, and say that this is probably mediated by a decrease in bone remodelling. Feskanich *et al.* (1999) found that women who consumed 75 g or more alcohol per week had signi cantly higher bone densities when compared to women who did not drink. The authors adjusted for age, body mass index, age at menopause, the use of postmenopausal oestrogens, and whether or not the woman smoked. The authors suggested that moderate alcohol consumption might help to maintain bone density in postmenopausal women by increasing endogenous oestrogens or alternatively by promoting the secretion of calcitonin. However, Grainge *et al.* (1998) found that bone mineral density was particularly related to smoking habits, with smokers having signi cantly lower bone mineral densities. Neither lifetime alcohol consumption nor current alcohol consumption displayed an independent association with bone mineral density. The authors did say, however, that the heaviest beer drinkers had a lower bone density.

Tobe *et al.* (1997) found that xanthohumol and humulone inhibited bone resorption. Humulone in particular had very strong inhibitory activity. In reminding us that vitamin D mobilises calcium stores from bone by inducing the dissolution of bone mineral and matrix, Honma *et al.* (1998) showed that the hop α-acid humulone inhibits bone resorption. The authors point out that vitamin D also inhibits proliferation (and induces differentiation) of myelomonocytic leukaemia cells, but that its clinical use for this purpose is limited by the adverse effect of hypercalcaemia. Humulone alone inhibited the growth of monoblastic leukaemia U937 cells and effectively enhanced the

differentiation-inducing action of vitamin D. Other myelomonocytic leukaemia cells were induced to differentiate by vitamin D and this was also enhanced by humulone. It seems that a combination of humulone and vitamin D might be useful in therapy for myelomonocytic leukaemia.

Cancer

The World Health Organization takes the stance that alcoholic beverages are carcinogenic to humans. However researchers from the University of Louisville in 1993 (cited by Stuttaford 1997) doubted that there was evidence to support the contention of a relationship between alcohol and cancers.

> The WHO statement needs to be analysed carefully, as it may have been in uenced by international politics and religious belief. *The Journal of Cancer Education* commenting on the statement, said 'the scienti c literature extant in 1992 provides only weak support for that nding'. Yet despite its critical reception by detached authorities, the WHO opinion is frequently quoted by opponents of alcohol.
>
> […]
>
> Of the 441 articles published by 1992 in the medical press about the links between drinking and cancer, only 29 were judged to meet the requirements of even an acceptable meta-analysis. There is … nothing in the literature about alcohol and cancer that comes near to paralleling the research ndings that linked cigarette smoking with cancer.
>
> Stuttaford (1997)

The 1989 report of the Committee on Diet and Health of the National Academy of Sciences concluded that one-third of cancers are linked to diet and that the strongest causal links were between stomach and colon cancer and diets high in fats and low in fresh fruits and vegetables. This led to the recommendation that no more than 30% of dietary calories should be in the form of fat. The recommendation is also to avoid excessive consumption of cured and smoked foods, but to ensure an intake of fresh vegetables and fruit. They also caution against *excessive consumption of alcohol*.

It is well understood that a signi cant factor in bodily tissue damage is exerted through the action of free radicals. At higher concentrations ethanol is metabolised not by the alcohol dehydrogenase system but by the cytochrome P450 system and this leads to signi cant radical formation (Lieber 1994; Nordmann 1994). Alcohol is also claimed to decrease the production of, and increase loss of, glutathione from the liver and to decrease the levels of other antioxidants, vitamin E and vitamin C (Speisky *et al.* 1985). The overall impact is a reduced resistance to oxidation.

We might compare these observations with those of Gasbarrini *et al.* (1998) that a beer-containing diet (as opposed to an ethanol-containing one) reduced the prevalence of oxidative markers in rats.

One of the first reports of a link between alcohol and cancer was the observation in France nearly a century ago that 80% of the cancers of the oesophagus and stomach occurred in absinthe-drinking alcoholics (Lamy 1910). There is, however, no evidence that ethanol itself is carcinogenic, although its oxidation product acetaldehyde may be (Prival 2003).

Beer, being produced from cereals, is at risk from contamination with ochratoxins, which are teratogenic, immunotoxic, genotoxic, mutagenic and carcinogenic (Creppy 1999). However, the vast majority of beers, which are produced from sound, uncontaminated grain, are devoid of significant levels of ochratoxin (Long 1999; and see Chapter 5).

Most widely publicised of the potential carcinogens in beer are the nitrosamines, but a concerted effort by maltsters and brewers ever since the problem was first mooted in 1978 (Spiegelhalder *et al.* 1979) means that levels of nitrosamines these days are extremely low, at one-fifteenth the level of 20 years ago (Sen *et al.* 1996). When considering nitrates and nitrites as potential carcinogens, the levels originating from beer are vastly lower than those in vegetables and cured meat products (Dich *et al.* 1996). For moderate beer drinkers the levels of nitrosamines are unlikely to constitute a hazard (Tricker & Preussmann 1991). A more recently highlighted concern has been the possible presence of chloropropanols in some of the more intensely heated grist materials used for brewing, yet it seems that these substances do not carry forward into beer (Long 1999).

Acrylamide, another material recently highlighted as a concern for many products, perhaps least of all beer (http://www.europa.eu.int/comm/food/fs/sc/scf/out131_en.pdf) has been shown not to correlate with cancer of the large bowel, kidney or bladder at current rates of consumption (Mucci *et al*. 2003).

It is claimed that heavy drinkers have a greatly increased risk of oropharyngeal and lower oesophageal cancer compared to light drinkers or abstainers (Day *et al.* 1993; Kabat *et al.* 1993; Kune *et al.* 1993). Ishii *et al.* (2001) observe that the risks are greater with 'stronger' alcoholic beverages and heavy smoking, but also for those people with an atypical alcohol dehydrogenase phenotype (ADH2). Seemingly this enzyme protects by preventing heavy drinking.

Remarkably, it has been concluded that non-drinkers are at increased risk of lung cancer (Woodson *et al.* 1999). However, although the authors claim that the study is inconclusive, Bandera *et al.* (2001) suggest that beer increases the risk of lung cancer. In reviewing the literature they observe that of 11 prior studies on beer in relation to lung cancer, five suggested a positive association, two indicated possibly weak support, but four found no association.

Freudenheim *et al.* (1995) and Zhang *et al.* (1999) report that alcohol consumption seems not to be related to the risk of breast cancer, but Ferraroni *et al.* (1998) deduce the opposite, more so for wine than beer, while Hebert *et al.* (1998) claim that there is an increased risk of recurrence of breast cancer in beer drinkers. However, to illustrate the dif culties in relating disease to speci c dietary components, we should consider the ndings of Jacobsen (1996) that there is an inverse relationship between a woman's age when she last gives birth and her tendency to drink beer! Conversely the correlation is positive with consumption of vegetables and white bread.

Excessive beer drinking is claimed to be associated with an increased risk of colon cancer, though so too is eating red meat more than twice daily and having a white-collar occupation (Hsing *et al.* 1998). Riboli *et al.* (1991) suggested a link between beer drinking and rectal cancer but not colon cancer. Pelucchi *et al.* (2002) could nd no link between alcohol intake and incidence of bladder cancer.

It has been said that there is an increased risk of carcinoma in the upper digestive tract for beer and spirit drinkers (Gronbaek *et al.* 1998). Conversely, Albertsen and Gronbaek (2002) and Tavani *et al.* (1994) say that there is no apparent link between alcohol consumption (or type) and prostate cancer. Indeed Kampa *et al.* (2000) draw a positive correlation between antioxidant polyphenol levels and the inhibition of prostate cancer cell lines.

In fact there is strong evidence that the consumption of alcoholic beverages in moderation may actually *counter* cancer. An international panel of epidemiologists, toxicologists and pharmacologists penned a letter to the *British Journal of Cancer* in 1993, which rmly suggested that moderate intake of alcohol protected rather than caused certain forms of cancer (Stuttaford 1997). The writers of the letter had re-examined published data on cancers of the mouth and gastrointestinal tract and concluded that men taking two drinks per day had half the risk of cancer as those who abstained. Drinking two to four drinks daily reduced the risk by two-thirds and only those taking a dozen or more drinks each day had a greater risk of cancer than did abstainers.

There is substantial evidence for the presence in beer of materials that might counter cancer. Components of beer derived from barley or hops have been studied in isolation and shown to display a number of promising effects. Several unique avonoid compounds have been isolated from hops and shown to be present in beer. They have cancer chemopreventive properties at least in part due to the inhibition of cytochrome P450 enzymes that activate carcinogens (Henderson *et al.* 2000). The compounds include xanthohumol, 8-prenylnaringenin and isoxanthohumol.

Miranda *et al.* (1999) showed that *in vitro* proliferation of a range of cancer cells was inhibited by prenylated avonoids of the type derived from hops, especially xanthohumol. Normal cells were not affected. These molecules may inhibit the activation of the cytochrome P450 enzymes that catalyse the conversion of procarcinogens into carcinogens. The material is very effective in stimulating the slow aggregation of breast

cancer cells, indicating that it may inhibit metastasis (Rong *et al.* 2001). And so hop extracts in very low concentration are very ef cient inhibitors of breast cancer cells (Zava *et al.* 1998).

8-Prenylnaringenin is claimed to counter enzymes involved in the development of prostate cancer and to inhibit the formation of new blood vessels important in tumour growth (De Keukeleire *et al.* 2001). Small wonder that this molecule has been renamed *hopein,* from hop and hope. Tagashira *et al.* (1995) showed that the hop alpha and beta acids have potent ability to suppress lipid peroxidation.

Kondo and Arimoto reported to the Pharmaceutical Society of Japan and the US National Cancer Institute (see *Japan Today* www.japantoday.com, 10 May 2002 – Kondo & Arimoto 2002) that they gave beer to rats that had been injected with carcinogens and showed that there was a 50% lowered incidence of cancer as compared to control animals not receiving the beer. The authors suggest that this level of beer intake equates to 250–500 mL for a 60-kg man. It was suggested that the active ingredients are pseudouridine and betaine.

Humulone can inhibit growth of skin tumours in mice (Yasukawa *et al*. 1995) and it can inhibit the growth of leukaemia cells (Honma *et al*. 1998). The polyphenol quercetin can inhibit synthesis of DNA of human leukaemia cells (Uddin & Choudhry 1995), block synthesis of a protein that leads to development of colon cancer in humans (Hosokawa *et al*. 1990), inhibit propagation of stomach cancer cells in humans (Yoshida *et al*. 1990) and inhibit growth of squamous cell carcinoma of throat and head in rats (Castillo *et al*. 1989). Quercetin may suppress growth of breast cancer cells (Stangl 2001). The phenolic acids can inhibit formation of carcinogenic nitrosamines from nitrite and secondary amines while xanthohumol and isoxanthohumol (as well as beer *per se*) can inhibit the mutagenic effect of heterocyclic amines (Arimoto-Kobayashi *et al*. 1999; Miranda 2000b). Incidentally, humulone inhibits ear oedema in mice (Yasukawa *et al.* 1993). Pignatelli *et al.* (1983) showed that beers with the highest content of polyphenol were the ones most able to block nitrosation events in the rat. Yoshikawa *et al.* (2002) demonstrated the presence of pseudouridine in a diversity of beers and demonstrated its ability to counter mutagens.

So how are we to reconcile the con icting observations? I believe it is essential to recognise that there is a substantial variance between studies based on the dosing of isolated materials, perhaps in atypically high concentrations, whether it be alcohol or a purported protectant, and those investigations that are closer to the 'real world' in which the various materials are together in better balanced quantities, e.g. in the form of beer.

The reader must appreciate that beer (and, indeed, wine) is not unique as a source of antioxidants. The essential point that I wish to convey is that beer is comprised of the self-same type of molecules that are found in other foodstuffs. It is a responsible attitude to regulate the intake of these materials so as to obtain a well-balanced diet (see Chapter 4). Where beer (and other alcoholic beverages) offers a relatively unique proposition is for

the plausible direct impact of alcohol in reducing risks of atherosclerosis and for the bene cial impact on the mind and spirit. The presence of antioxidants, phytoestrogens, etc. is less well understood but offers promise.

Allergy

Allergic and asthmatic reactions to alcoholic drinks were recently reviewed by Vally and Thompson (2003). They say that most reported cases of intolerance to alcoholic beverages occur with wine. However, beer can contain biogenic amines, notably tyramine (Izquierdo-Pulido *et al*. 2000), which presents a risk to those taking monoamine oxidase inhibitors to counter stress and depression (Shulman *et al*. 1997) and can cause migraines and hypertensive crises (Crook, 1981; Zee *et al*. 1981). Tyramine is a 'pressor amine' which can cause a rise in blood pressure by constricting the vascular system and increasing the heart rate (Gloria 2003). Histamine acts as a neural transmitter and thus has a psychoactive in uence. However, it can directly stimulate the heart, impact on smooth muscle and control gastric secretion. Gorinstein *et al*. (1999) measured levels of tyramine in the range 3.6–7.4 mg/L and histamine in the range 3–3.2 mg/L in beers. Other amines are also present (Table 6.4). However, it will be seen that other foodstuffs, notably cheese, are even richer sources. Not listed here is chocolate, which contains high levels of 2-phenylethylamine. There is a belief that these various amines are a signi cant source of migraines in those partaking of foodstuffs such as chocolate, red wine and larger quantities of beer. The suggested upper limit for alcoholic beverages (per litre) is 2–8 mg histamine and 8 mg tyramine.

Ehlers *et al.* (2002) report a direct allergic reaction of individuals to ethanol itself.

Some beers may contain added sulphite, which may trigger reactions in people sensitive to this agent, which is commonly used in sodas and other non-alcoholic drinks as a preservative. However, sulphite is sparingly used in beer, also when compared to wine, and in many markets (e.g. the US) beers will contain much less than 10 mg/L SO_2. Nonetheless Gall *et al.* (1996) indicate that one patient was sensitive to just 3–4 mg/L SO_2.

One of the most prevalent sensitivities to products based on cereals is coeliac disease (Campbell 1992), a reaction to gluten and related proteins. Ellis *et al.* (1994) demonstrated the presence in barley malt of coeliac-activating material, seemingly derived from the hordein storage proteins. Of course, the amounts of such material might be expected to vary considerably between beers, depending on the extent to which the protein is hydrolysed and denatured in the malting and brewing processes, the levels of protein in the grist, and so on. It has been suggested that other proteins in beer may also have an allergic impact. One such protein is a lipid transfer protein (Asero *et al.* 2001), which is believed to be important for the foaming of beer. The claims were made on the basis

of the reactivity of a single patient to various foodstuffs, the common denominator of which was lipid transfer protein. However, curiously, not all beers caused a reaction.

Kortekangassavolainen *et al.* (1993) report on allergic reactions to brewing yeast.

The common cold

Might we anticipate other ailments for which claims for helpful impacts of alcoholic beverages are made? And will these continue sometimes to favour wine over beer? To illustrate, Takkouche *et al.* (2002), from a study of faculty and staff of ve Spanish universities, found that consumption of wine, especially red wine, led to a reduced risk of suffering from the clinical common cold. No such bene t was observed for beer or spirits. It will be interesting to see whether such a study performed in a primarily beer-drinking country might lead to another conclusion. Certainly, Cohen *et al.* (1993) found that smokers were more prone to the cold than were non-smokers, and that among the latter, those taking 3 to 4 drinks per day were the least susceptible of all.

7 Conclusion

Beer-drinking is a cultural phenomenon, spanning the whole spectrum from absolute abstention to the drinking ritual. It can comprise an integral and prized part of the very fabric of life, for example in the Czech Republic or within the tribes of the Kofyar or Tiriki. Elsewhere it represents a growing trend, such as in China where economic and physiological factors will most likely self-regulate the rate of growth.

Surely beer is a very metaphor for the toleration of other cultures?

For a vast part of society's history, and in a great number of countries, beer has been a staple part of the diet, more so than any other alcoholic beverage. It is the drink of moderation for the general classes.

For the longest time ale was an integral contributor to the nutrition of the masses, young and old. It would be idiosyncratic today to champion beer as still being the cheapest and most appropriate source of key ingredients of the diet, especially for the young. However, the point needs to be stressed that brewers have not digressed from, but rather continuously improved, their practices in respect of selection of the best raw materials and adoption of the most consistent and reliable brewing procedures. Beer has never been more wholesome. Indeed, the progressive shift towards ever more hygienic operating protocols means that beer has never been so consistently good in every respect. Thus it still does *add* to the dietary intake of useful materials, even though for many it is more frequently regarded as an item purchased for its hedonic and social attributes.

Taken to excess, of course, any alcoholic beverage presents adverse and negative impacts, which are focused upon by those who would, in the extreme, ban the sale of alcoholic beverages or at least tax them so as to make them prohibitively expensive. The latter approach is surely ill conceived, insofar as beer is still very much the 'drink of the masses' and the vast majority of those who buy and consume it do not present a risk either to society or to themselves. And those who would advocate this type of solution to the supposed disease of alcoholism are surely naive in their supposition that simply by raising the cost the addictive pressures will be held at bay. If they genuinely believe that this is a solution, then what better argument can there be that there is no physiological basis for addiction to alcohol?

There is still no unequivocal answer to the question of whether alcoholism is anything other than an addictive tendency to be listed alongside the many others that humankind is subject to. The search for a genetic basis for the 'disease' continues – it will be

interesting to see whether any such genotypic foundation for alcohol abuse is also the causal factor in determining other compulsive phenotypes.

It is unavoidably the case that sustained and excessive consumption of alcoholic beverages is damaging to the body. However, the diseases generally associated by the general populace with alcohol are not suffered by the greatest number of people who drink moderately. Diseases such as cirrhosis are developed by those who take alcoholic drinks in relatively huge amounts.

Indeed we see that a beer intake of the order of 2 pints per day has, on balance, a beneficial impact on the body, particularly in lowering the risk of cardiovascular disease.

Yet despite the growing evidence, it is still a message that sits uncomfortably at the highest levels. Thus in March 2003 the Alcohol and Tobacco Tax and Trade Bureau of the US Department of the Treasury issued its final rule on 'Health Claims and other Health-related Statements in the Labelling and Advertising of Alcohol Beverages'. The particular concern is with the marketing of alcoholic drinks from a health perspective. It should be realised by the reader that brewers have tended not to do this overtly since the days of generalised advertisements of the Guinness and Mackeson variety. I have detected no tendency within beer companies to shift overtly from this stance, unlike the case for certain other types of alcoholic beverage. Indeed the Tax and Trade Bureau (TTB) document (which runs to 142 pages) says:

> TTB recognises that based on the administrative record, it does not appear that distillers and brewers are interested in using health claims or health-related statements in the labelling or advertising of alcohol beverages.

If we study the TTB document we find statements such as this:

> In view of the undisputed health risks associated with alcohol consumption, we and our predecessors have always taken the position that statements attributing positive effects on health to the consumption of alcohol beverages are misleading unless such statements are appropriately qualified and properly balanced.

This one sentence suggests that the writers have set out their stall with an unquestioning acceptance of the negatives associated with alcohol but an inherent suspicion of those who suggest that there may be positives.

They go on to say that:

> TTB view statements that make substantive claims regarding health benefits associated with alcohol beverage consumption (e.g. 'moderate alcohol consumption is good for your health') as making curative or therapeutic claims. Claims that set forth only a partial picture or representation might be as likely to mislead the

> consumer as those that are actually false. A claim that is supported by scienti c evidence might still mislead the consumer without appropriate quali cation and detail. Any such claim is considered misleading unless it is properly quali ed and balanced, suf ciently detailed and speci c and outlines the categories of individuals for whom any positive effects on health would be outweighed by numerous negative effects on health.

However, might it not equally be said that it is wrong to imply that the consumption of an alcoholic beverage will perforce be injurious to health? Most people do not take alcohol to excess. The present warning that is mandatory on beer packages in the US is vague in respect of the latter part of its second statement ('…may cause health problems') and is certainly not suf ciently detailed along the lines described in the above extract from the TTB document. Would it be no less vague to say '…may afford health bene ts'?

The TTB document presents testimony from medical experts who span the divide. Dr Michael Gough says that:

> …with the exception of those well-de ned groups of people who should avoid alcohol, there is clearly convincing evidence for the health bene ts of moderate alcohol consumption … based on understanding of the biological basis for the protective effects of alcohol, it is likely that moderate alcohol consumption in the 20s and 30s is important to the bene cial effects seen in later years.

On the other hand, the National Council on Alcoholism and Drug Dependence commented that:

> … while most people who choose to drink do so without negative health or life consequences, there are 13.8 million Americans over the age of 18 who have problems with drinking, including 8.1 million who are alcoholic.

There is no comparative information concerning other forms of addiction.

Dr Michael Criqui is cited as saying that 'half of all the alcohol consumed in the United States is consumed by the 10% of men and the 5% of women who are alcohol-dependent'. But then Dr Curt Ellison says:

> Science clearly indicates that moderate drinkers have much lower risk of coronary heart disease and ischaemic stroke. Because these are the number one and number three causes of death, it is not surprising that moderate drinkers will live longer in the United States. If I am withholding from a patient information that may reduce that individual's risk of a heart attack by 30 or 40 per cent and do not tell him about it, I am doing him a disservice.

It is not my intention or desire to end this book on a political note. It is certainly not my intent to have positioned this volume in any way other than as an exercise in balanced scholarship. I certainly have not sought to use the book to persuade those who don't already drink to start, or to hector them into drinking more, or to switch their consumption from other forms of alcohol to beer. Rather I have attempted to present an appreciation of beer as it stands within the fabric of society and, it is hoped, correcting some misunderstandings but not shying away from the detrimental impact that excessive alcohol consumption can have.

I don't believe that any producer of alcohol-containing beverages should overtly market a product on the basis of health bene t. I do believe that beer and other alcoholic drinks can form a rich and pleasurable aspect of a ful lling lifestyle. Advertising of alcohol should be responsible and honest. Images of wholesome raw materials and moderate drinking patterns are good. Depiction of raucous and irresponsible behaviour is bad. Taken to excess, drinking can cause as much misery as all the many other addictive behaviours, whether an over-dependence on sex, spending, smoking, gambling or the religious fervour that can lead too readily to terrorism and war.

The solution to problems with alcohol is not to try to destroy the industry, any more than it would be logical to ban a religion because its existence is an affront to others. The answer rather is to lay out the factors 'for' and 'against' a drink such as beer, and to educate on the basis of reasoned discussion and a presentation of all the facts. I hope that this volume has made a worthwhile contribution to the debate.

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