

Nutritional aspects of beer—a review

Charles W. Bamforth*

Department of Food Science & Technology, University of California, Davis, CA 95616-8598, USA

Received 31 August 2001; accepted 20 September 2001

Abstract

Much has been written about the favourable impact on the body of moderate consumption of red wine. Critical assessment of the literature, however, indicates that beer appears to be just as beneficial in countering diseases such as coronary heart disease. Additionally beer can make a substantial contribution to the diet in respect of certain B vitamins, minerals, antioxidants and perhaps fiber. © 2002 Elsevier Science Inc. All rights reserved.

Keywords: Beer; Coronary heart disease; Diet; Vitamins; Antioxidants

1. Introduction

Egyptian legend has it that Osiris, god of earth and vegetation, was the inventor of beer [1]. Whilst tracing such origins is of course a matter of faith, there is evidence that beer in those far off days not only comprised a valuable constituent of the diet, but also served a diversity of medicinal functions, including mouthwash, enema, vaginal douche and applicant to wounds [2].

Nowadays the use of beer is rather more restricted. It may occasionally feature in shampoos and constitutes a useful ingredient in a range of cooking. However for the most part it simply serves its main purpose, as a drink. In ancient Egypt and subsequently in many other cultures, beer was deemed the beverage of choice for all members of the family, young and old [1]. In modern society, however, beer consumption is restricted to those over a certain age, ranging from 16 in several European nations to 21 in, *inter alia*, the United States (see <http://www.icap.org/icapreport4.html>).

* Tel.: +1-530-752-1467; fax: +1-530-752-4759.

E-mail address: cwbamforth@ucdavis.edu (C.W. Bamforth).

Table 1
Harmful effects of alcohol (modified from Cooper [26])

-
- Traffic 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
-

It is accepted that indiscriminate drinking of beer (and all other alcoholic beverages) is unsociable, dangerous, and injurious to health (Table 1). However might there be a positive dimension to the consumption of alcohol, including in the form of beer? A recent survey of the bookshelves at one of our university libraries revealed a plethora of volumes on alcoholism and the undisputed health hazards of excessive alcohol consumption –see for example Watson & Watzl [3]. And yet there is an expanding body of knowledge pointing to moderate consumption of beer as being a beneficial component of the diet. The state of play in this area is reviewed in this paper.

2. The gross composition of beer in relation to nutritional need

It seems that on Captain Cook's ships beer contributed as many calories to the sailors' diets as biscuits and meat combined [4]. Perhaps this balance is tilted rather differently nowadays, yet beer can still offer significant contributions to the dietary intake (Table 2) even leaving aside its role as a thirst quencher.

It is difficult to generalise on the relationship between beer and relative impact on nutrient intake, for the composition of beers will range quite considerably depending on raw materials and mode of production. Thus the alcohol content may range down from in excess of 10% (v/v) in beers produced in Trappiste 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 respectively. Additionally, conventionally fermented beers may retain some 25% of the starch in a partially degraded, non-fermentable form which will contribute to the calorie count. By contrast so-called Light beers generally contain minimal levels of carbohydrate.

Forsander [5] has eloquently reviewed the literature that firmly points to ethanol's ability to replace other foodstuffs as a source of calories in the diet, with voluntary alcohol intake depressing food consumption in proportion to its calorie contribution. In particular it would appear that the inverse correlation is with carbohydrate rather than protein or fat. The converse applies equally—such that the consumption of candies is one part of the armoury to combat alcoholism. Carbohydrate provided in this form reduces the desire to consume alcohol.

It is evident from the data presented in Table 2 that significant quantities of several B vitamins are available via moderate consumption of beer. For example, Mayer et al [6] have demonstrated the worth of beer as a source of folic acid, leading to a decreased homocysteine

Table 2
The composition of beer relative to recommended dietary intakes

Parameter	Daily adult (age 25–50) requirement		Range in beer (per litre)
	Male	Female	
Energy (Kcal)	2550	1940	150–1100
Protein (g)	63	50	3–5
Carbohydrate (g)	^a	^a	0–61
Fat (g)	^a	^a	Neg.
Vitamin A (μg)	1000	800	Neg.
Vitamin D (μg)	5	5	Neg.
Vitamin E (mg)	10	8	Neg.
Vitamin K (μg)	80	65	Neg.
Vitamin C (mg)	60	60	Up to 30
Thiamine (mg)	1.5	1.1	0.003–0.08
Riboflavin (mg)	1.7	1.3	0.02–0.8
Niacin (mg)	19	15	3–8
Vitamin B ₆ (mg)	2.0	1.6	0.07–1.7
Folate (μg)	200	180	40–600
Vitamin B ₁₂ (μg)	2	2	3–30
Biotin (μg)	30–100	20–100	2–15
Calcium (mg)	800	800	40–140
Phosphorus (mg)	800	800	90–400
Magnesium (mg)	350	280	60–200
Potassium (mg)			330–1100
Sodium (mg)			40–230
Iron (mg)	10	15	0.1–0.5
Zinc (mg)	15	12	0.01–1.48
Selenium (μg)	70	55	<0.4–7.2

Sources of dietary recommendations: Food and Nutrition Board, National Academy of Sciences; British Nutrition Foundation (<http://www.nutrition.org.uk>).

^a 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.

Sources of analytical data: references [90–92].

content in blood (hyperhomocysteinemia is a significant risk factor for vascular diseases). Chronic alcoholism leads to the obverse effect, although beer drinkers had significantly lower serum concentrations of homocysteine than did those consuming wine or spirits [7]. Of the B vitamins it is thiamine which is notably deficient in beer, although Agranoff [8] hypothesizes that it wasn't ever thus. The yeast present in 18th century beer will have provided vitamins to the diet and might have been 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 did gin. There is no robust scientific evidence that the present day naturally-conditioned bottle beers, with their sizeable charge of yeast, are more valuable components of the diet than their filtered counterparts. 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 depending on alcohol as a source of calories runs the risk of vitamin shortage. In this context beer, with its finite vitamin content, would be a wiser beverage than other alcoholic drinks (though, of course, still used in moderation). However it is firmly

documented that thiamine deficiency stimulates alcohol consumption [5]: thiamine shortages interfere with glucose metabolism, so perhaps the same causal inverse link between intake of alcohol and carbohydrate is at play.

Beer is frequently cited as being a significant dietary source of selenium. The relatively high potassium to sodium ratio (typically 4:1) is consistent with a low sodium diet. Related to this, beer has a significantly greater diuretic effect than has water [9].

Beer has been shown to contain between 0.4 and 6.2 g/L of dietary fibre [10]. The British Nutrition Foundation targets 18g per day as being a desirable level for adults. Low doses of alcohol, including as beer, stimulate appetite and promote bowel function in the elderly [11].

I might also mention in passing that beer does not support the growth of pathogenic organisms.

3. Moderate consumption of beer: a positive or a negative?

3.1. Coronary heart disease

There have been many reports demonstrating a causal inverse relationship between moderate alcohol consumption and coronary heart disease, with 1–3 units per day offering the lowest risk [12,13,14,15]. (One unit of alcohol in the form of an averaged strength beer, e.g. 5% alcohol by volume, equates to approximately one half pint.) Many authors have referred to a U-shaped curve depicting the relationship between all causes of mortality and alcohol consumption, with moderate intake offering the lowest risks [16]. One study in Japan referred to a J-shaped curve [17]. It is believed that alcohol increases the concentration in serum of high-density lipoprotein cholesterol –i.e. it lessens accumulation of this material in blood vessels [18,19]). Furthermore it has been suggested that alcohol influences platelet aggregation [20] and clotting/fibrinolysis mechanisms [21,22]. Components other than alcohol may also combat coronary heart disease [23]), including polyphenols [24]. The impact which moderate consumption of alcohol has in decreasing stress may also play out as a factor in reduced heart problems [25]. Beer may be particularly valuable as compared to other alcoholic beverages, as the hop-derived bittering agents are said to have sedative and hypnotic impact [26].

Of the alcoholic drinks, red wine has received the most favourable press, and producers of this style of beverage have not been reticent in making claims in support of its role as a beneficial component of the diet - see for example <http://www.wired.com/news/topstones/0,1287,17770,00.html>. But what of beer?

Cleophas [27] and Rimm et al [28] reviewed the literature on the relative impact of beer, wine or spirits on coronary heart disease. The latter authors highlight the difficulties inherent in so-called “ecological studies”, which are based on existing data collected as part of census and surveillance programs. Such studies have tended to report that wine has beneficial impacts, but not beer or spirits. In particular they note Renaud et al’s [20] observation that wine drinking may be associated with other habits that are actually responsible for the benefits (i.e. a secondary correlation). Thus in certain countries (e.g. the United States) wine drinkers tend to belong to socio-economic categories enjoying a healthier lifestyle and better

health care [29]. Wine is associated with the consumption of healthier foods than is beer [30]. Burke et al [31] illustrate the same point from the obverse aspect, noting that Australian men who preferred beer to wine also drank larger volumes, smoked more and selected a generally less healthy diet. Comparable findings are reported by Galobardes et al [32]. In analysing dietary patterns, beer was bracketed with “convenience food” [33]. Watten [34] reported that males and females that smoked daily tended to drink more beer –but also more coffee, less tea and more marihuana! Osler [35] even observed that a partner’s smoking promotes one’s consumption of various foodstuffs, including beer. Rogers and Greenfield [36] claim that hazardous drinking (defined as occasions when five or more drinks are consumed daily) is associated more with beer than with other types of alcoholic beverage and that, in turn, this correlates with younger, male, unmarried males. Mortensen et al [37] in a study of young Danish adults, found that wine drinking was associated with higher IQ, higher parental educational attainment and higher socioeconomic status when compared with those who drank beer. Furthermore on scales of psychiatric and health-related behavior beer drinkers fared less well than wine drinkers. Mortensen and co-workers concluded that the apparent superior health benefits of wine were related to better social and psychological performance. All of this surely asks more about culture and peer pressure than it does about the relative merits of beer and other drinks on a comparable unit of alcohol basis. A further criticism of studies based on surveys concerns the reliability of individuals’ reporting of alcohol intake [38,39]. Yet another complication arises from the unsurprising observation that those engaging in team sports (one assumes leading to increasing fitness) consume more beer and liquor [40].

Rimm et al [28] highlight the greater reliability of case-control studies, in which controlled studies are made of ailments such as coronary heart disease and consumption of a specific alcoholic beverage. From such investigations (see also [27]) it is clear that wine, beer and spirits all confer a reduction in coronary heart disease. Or, as Rimm and co-workers [28] put it: *we conclude that if any type of drink does provide extra cardiovascular benefit apart from its alcohol content, the benefit is likely to be modest at best or possibly restricted to certain sub-populations.*

Nevertheless, there are several reports of selective benefits of one type of alcoholic beverage as opposed to another. Rimm et al [28] suggest that this relates to aspects of lifestyle associated with consuming drinks of a certain style. Thus Klatsky et al [29] showed that wine consumers were less likely to develop coronary heart disease than spirit drinkers were, whereas beer drinkers were more prone. However it was shown that adjustment of the data for parameters such as sex, race, cigarette smoking and consumption of coffee eliminated the correlations. That is, differences in benefit or risk associated with different beverages may be associated with other lifestyle parameters within the population studied. Furthermore if there is an abiding preference for one type of beverage within a population, then studies across that population that relate moderate alcohol consumption to health benefits tend to emphasise the advantages of that particular drink. Thus, in a study in Honolulu where only a minority of the population consume wine, there was a significant inverse population between coronary heart disease and beer drinking [41]. Comparable results were obtained in a German study [42] and the Czech Republic [43]. Conversely in rural Italy, where wine was the drink of choice, the benefits were seen for wine [44].

In a controlled study Hendricks et al [45] demonstrated that alcohol in the form of wine, beer or spirits taken at dinner (40g alcohol) impacted beneficially on the plasminogen system, justifying why moderate consumption of alcohol in any of these forms decreased the risk of coronary heart disease. Comparable results were reported by Rimm et al [46], who monitored changes in a range of parameters related to coronary heart disease. They concluded that 30g of alcohol per day would offer the drinker a 24.7% reduced risk of this affliction. Van der Gaag et al [47] report that alcohol taken in the form of red wine and spirits leads to an increase in the level of homocysteine in serum, an event associated with heart disease. By contrast, the consumption of alcohol as beer did not lead to such an increase, perhaps because of the presence of vitamin B₆ in the beer. Dawson [48] stresses that alcohol dependence nullifies any benefit from moderate drinking.

Commenting on the findings of Bobak et al [43] concerning the beneficial effects of moderate beer drinking on the health of Czechians (see earlier), Dennington [49] wonders whether the effects are not necessarily due to ethanol, but rather to copper. He cites in support of this conjecture the observation that brewing vessels in the Czech republic are still fabricated from copper, whereas elsewhere they are increasingly fashioned from stainless steel. Whilst this is an intriguing possibility, most brewers actually endeavour to minimise the level of copper in beer as it promotes staling [50]. Entering into the same chain of correspondence, Morrell [51] suggests that it is rather more likely that the beneficial effects of alcohol consumption are a consequence of the impact of drinking on relaxation. Factors and societal habits that lead to relaxation (e.g. siestas) are associated with less stress, which in turn is associated with lower incidence of cardiovascular disease. Cleophas [27] also concludes that there is a significant psychological component in the beneficial relationship between moderate alcohol consumption and mortality.

Klatsky et al [29] decided that there might be minor additional benefits linked to drinking both beer *and* wine, and not especially red wine.

Excessive consumption of alcohol has clear detrimental effects [52]. Thus Kauhanen et al [53] demonstrated the adverse impact of binge drinking of beer (six bottles or more per session as compared to three) on all causes of mortality, including myocardial infarction.

3.2. Ulcers

It has been reported that alcohol protects against infection by *Helicobacter pylori* [54,55], in fact countering the effect of coffee.

3.3. Cancer

Beer, being produced from cereals, is at risk from contamination with ochratoxins, which are teratogenic, immunotoxic, genotoxic, mutagenic and carcinogenic [56]. However the vast majority of beers, which are produced from sound, uncontaminated grain, are devoid of significant levels of ochratoxin [57].

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 [58] means that levels of nitrosamines these days are extremely low, at one-fifteenth the level

of twenty years ago [59]. When considering nitrates and nitrites as potential carcinogens, then the levels originating from beer are vastly lower than those in vegetables and cured meat products [60]. For moderate beer drinkers the levels of nitrosamines are unlikely to constitute a hazard [61]. A more recently highlighted concern has been the possible presence of chlororopropanols in some of the more intensely heated grist materials used for brewing, yet it seems that these substances do not carry forward into beer [57].

Remarkably, it has been concluded that non-drinkers are at increased risk of lung cancer [62].

Freudenheim et al [63] and Zhang et al [64] report that alcohol consumption seems not to be related to the risk of breast cancer, but Ferraroni et al [65] deduce the opposite, more so for wine than beer, while Hebert et al [66] claim that there is an increased risk of recurrence of breast cancer in beer drinkers. However, to illustrate the difficulties in relating disease to specific dietary components, we should consider the findings of Jacobsen [67] 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 job [68].

It has been suggested that there is an increased risk of carcinoma in the upper digestive tract in beer and spirit drinkers [69].

3.4. Other impacts of beer on health

There is evidence that moderate alcohol consumption may be associated with better cognitive function in old age [70,71]. Moderate consumption of wine and beer reduces the odds against age-induced macular degeneration [72]. Gruenewald and Ponicki [73] reported a link between cirrhosis and excessive consumption of liquor, but not beer or wine. Beer is said to be associated with higher blood pressure [74]. Beer drinking, as well as a low fat or weight reduction diet, relates to substantial reduction in the risk of urolithiasis [75]. Beers containing significant levels of residual sugar and unusually low pH (<4.0) have potentially harmful effects on teeth [76]. 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 significance [77].

Lapcik et al [78] demonstrated that beer contains a range of health-promoting isoflavonoids (phytoestrogens) (see also [79]). There have been concerns that such materials may adversely modify the hormonal status of men, but Promberger et al [80] have concluded that the risk is negligible owing to the extremely low levels of these substances found in beer. Equally this does bring into question whether such low levels have any beneficial effects either: the levels of the principle isoflavonoid, isoxanthohumol, found in beer (1.5mg/L or less) is about 20-fold less than the effective human dose for anti-cancer treatments [81]. Piendl and Zeuch [82] quantified the level of beneficial "ballast" substances provided by beer. Beer contains useful levels of antioxidants, including the phenolics [83], and indeed it has been demonstrated that ferulic acid in beer is readily assimilated by the body [84]. The silicic acid provided by beer has benefits in promoting the renal excretion of aluminium [85].

Beer can contain biogenic amines, notably tyramine [86], which presents a risk to those

taking monoamine oxidase inhibitors [87]. Others at risk from consuming beer are those with gout [88] and sensitivity to cereal proteins [89].

4. Conclusions

It appears that beer is at least on a par with wine for the potential benefits that it may confer on the human body when taken in moderation. However, there probably remains a need for more case-control studies to fully evaluate the favourable aspects of beer drinking. Beers contain materials that are associated with positive impacts on the body (antioxidants, certain minerals, some vitamins, fibre, as well as relatively low levels of ethanol). Brewers probably don't need to enhance this composition, but might do more to position their products as part of an overall healthy and balanced diet.

Acknowledgments

The author thanks Bob Feeney and Linda Harris for supplying references.

References

- [1] Bamforth CW. A brief history of beer. *Proceedings of the 26th Conv Inst Brew Asia Pac Sect*, 2000. p. 5–12.
- [2] Darby WJ, Ghalioungi P, Grivetti L. *Food: the gift of Osiris*. London: Academic Press, 1977.
- [3] Watson RR, Watzl B. *Nutrition and Alcohol*. Boca Raton: CRC Press, 1992.
- [4] Feeney RE. *Polar journeys: the role of food and nutrition in early exploration*. Fairbanks: University of Alaska Press, 1997.
- [5] Forsander OA. Dietary influences on alcohol intake: a review. *J Stud Alc* 1998;59:26–31.
- [6] Mayer O, Simon J, Roslova H. A population study of beer consumption on folate, and homocysteine concentrations. *Eur J Clin Nut* 2001;55:605–9.
- [7] Cravo ML, Gloria LM, Selhub J, Nadeau MR, Camilo E, Rosendi MP, Cardoso JN, Leitao CN, Mira FC. Hyperhomocysteinemia in chronic alcoholism: correlation with folate, vitamin B12 and vitamin B6. *Am J Clin Nut* 1996;63:220–4.
- [8] Agranoff BW. William Hogarth, unwitting neurochemist? *Neurochem Res* 2000;25:1431–4.
- [9] Buday AZ, Denis G. The diuretic effect of beer. *Brew Dig* 1974;49(6):56–8.
- [10] Gromes R, Zeuch M, Piendl A. Further investigations into the dietary fibre content of beers. *Brau Int* 2000;18:24–8.
- [11] Dufour MC, Archer L, Gordis E. Alcohol and the elderly. *Clin Ger Med* 1992;8:127–41.
- [12] Moore RD, Pearson TA. Moderate alcohol consumption, and coronary heart disease. A review. *Medicine* 1986;65:242–67.
- [13] Maclure M. Demonstration of deductive meta-analysis: ethanol intake and risk of myocardial infarction. *Epid Rev* 1993;15:328–51.
- [14] Klatsky AL, Armstrong MA, Friedman GD. Alcohol, and mortality. *Ann Int Med* 1992;117:646–54.
- [15] Verschuren PM. *Health issues related to alcohol consumption*. Washington DC: ILSI Press, 1993.
- [16] Doll R, Peto R, Hall E, Wheatley K, Gray R. Mortality in relation to consumption of alcohol: 13 years' observations on male British doctors. *Brit Med J* 1994;309:911–8.
- [17] Tsugane S, Fahey MT, Sasaki S, Baba S. Alcohol consumption and all-cause and cancer mortality among middle-aged Japanese men: seven-year follow-up of the JPHC study cohort I. *Am J Epid* 1999;150:1201–7.

- [18] Hulley SB, Gordon S. Alcohol, and high density lipoprotein cholesterol. Causal inference from diverse study designs. *Circulation* 1981;64:57–63.
- [19] Thornton J, Symes C, Heaton K. Moderate alcohol intake reduces bile cholesterol saturation and raises HDL cholesterol. *Lancet* 1983;ii:819–22.
- [20] Renaud SC, Beswick AD, Fehily AM, Sharp DS, Elwood PC. Alcohol and platelet aggregation: the Caerphilly prospective heart disease study. *Am J Clin Nut* 1992;55:1012–7.
- [21] Ridker PM, Vaughan DE, Stampfer MJ, Glynn RJ, Hennekens CH. Association of moderate alcohol consumption and plasma concentration of endogenous tissue-type plasminogen activator. *J Am Med Ass* 1994;272:929–33.
- [22] Kluff C, Veestra J, Schaafsma G, Pikaar NA. Regular moderate wine consumption for five weeks increases plasma activity of the plasminogen activator inhibitor-1 (PAI-1) in healthy young volunteers. *Fibrinolysis* 1990;4(Suppl 2):69–70.
- [23] Klatsky AL. Moderate drinking, and reduced risk of heart disease. *Alc Res Health* 1999;23:15–23.
- [24] Halpern MJ, Dahlgren AL, Laakso I, Seppanen-Laakso T, Dahlgren J, McAnulty PA. Red-wine polyphenols and inhibition of platelet aggregation: possible mechanisms and potential use in health promotion and disease prevention. *J Int Med Res* 1998;26:171–80.
- [25] Pohorecky LA. Interaction of alcohol and stress at the cardiovascular level. *Alcohol* 1990;7:537–41.
- [26] Cooper TJ. Medical considerations of moderate alcohol consumption. *Proceedings of the 23rd Conv Inst Brew Aust NZ Sect* 1994. p. 32–7.
- [27] Cleophas TJ. Wine, beer, and spirits, and the risk of myocardial infarction: a systematic review. *Biomed Pharm* 1999;53:417–23.
- [28] Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine or spirits? *Brit Med J* 1996;312:731–6.
- [29] Klatsky AL, Armstrong MA, Friedman GD. Red wine, white wine, liquor, beer and risk for coronary artery disease hospitalisation. *Am J Card* 1997;80:416–20.
- [30] Tjonneland A, Gronbaek M, Stripp C, Overvad K. Wine intake and diet in a random sample of 48763 Danish men and women. *Am J Clin Nut* 1999;69:49–54.
- [31] Burke V, Puddey IB, Beilin LJ. Mortality associated with wines, beers and spirits. *Brit Med J* 1995;311:1166a.
- [32] Galobardes B, Morabia A, Bernstein MS. Diet and socioeconomic position: does the use of different indicators matter? *Int J Epid* 2001;30:334–40.
- [33] Pryer JA, Nichols R, Elliott P, Thakrar B, Brunner E, Marmot M. Dietary patterns among a national random sample of British adults. *J Epid Comm Health* 2001;55:29–37.
- [34] Watten RG. Smokers and non-smokers: differences in alcohol consumption and intake of other health-related substances in Norway – a general population study. *Eur J Pub Health* 1999;9:306–8.
- [35] Osler M. The food intake of smokers and non-smokers: the role of partner's smoking behaviour. *Prev Med* 1998;27:438–43.
- [36] Rogers JD, Greenfield TK. Beer drinking accounts for most of the hazardous alcohol consumption reported in the United States. *J Stud Alc* 1999;60:732–9.
- [37] Mortensen EL, Jensen HH, Sanders SA, Reinisch JM. Better psychological functioning and higher social status may largely explain the apparent health benefits of wine—a study of wine and beer drinking in young Danish adults *Arch Int Med* 2001;161:1844–8.
- [38] McCann SE, Marshall JR, Trevisan M, Russell M, Muti P, Markovic N, Chan AWK, Freudenheim JL. Recent alcohol intake as estimated by the health habits and history questionnaire, the Harvard semiquantitative food frequency questionnaire and a more detailed alcohol intake questionnaire. *Am J Epid* 1999; 150:334–40.
- [39] Dawson DA. Volume of ethanol consumption: effects of different approaches to measurement. *J Stud Alc* 1998;59:191–7.
- [40] Watten RG. Sports, physical exercise, and use of alcohol. *Scan J Med Sci Sports* 1995;5:364–8.
- [41] Yano K, Rhoads GG, Kagan A. Coffee, alcohol, and risk of coronary heart disease among Japanese men living in Hawaii. *New Eng J Med* 1977;297:405–9.

- [42] Keil U, Chambless LE, Doring A, Filipiak B, Stieber J. The relation of alcohol intake to coronary heart disease and all-cause mortality in a beer-drinking population. *Epidemiology* 1997;8:150–6.
- [43] Bobak M, Skodova Z, Marmot M. Effect of beer drinking on risk of myocardial infarction: population based case-control study. *Brit Med J* 2000;320:1378–9.
- [44] Farchi G, Fidanza F, Giampaoli S, Mariotti S, Menotti A. Alcohol and survival in the Italian rural cohorts of the Seven Countries Study. *Int J Epid* 2000;29:667–71.
- [45] Hendriks HFJ, Veenstra J, Velthuis-Te Wierik EJM, Shaafsma G, Kluit C. Effect of moderate dose of alcohol with evening meal on fibrinolytic factors. *Brit Med J* 1994;308:1003–6.
- [46] Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *Brit Med J* 1999;319:1523–8.
- [47] Van der Gaag MS, Ubbink JB, Sillanaukee P, Nikkari S, Hendriks HFJ. Effect of consumption of red wine, spirits and beer on serum homocysteine. *Lancet* 2000;355:1522.
- [48] Dawson DA. Alcohol consumption, alcohol dependence and all-cause mortality. *Alc Clin Exp Res* 2000;24:72–81.
- [49] Dennington S. Does copper in beer protect the heart? *Brit Med J* 2000;320:1378–9.
- [50] Bamforth CW, Parsons R. New procedures to improve the flavor stability of beer. *J Am Soc Brew Chem* 1985;43:197–202.
- [51] Morrell P. Re: does copper in beer protect the heart? *Brit Med J* 2000;320:1378–9.
- [52] Poikolainen, K. Alcohol and overall health outcomes. *Ann Med* 1996;28:381–4.
- [53] Kauhanen J, Kaplan GA, Goldberg DE, Salonen JT. Beer bingeing and mortality: results from the kuopio ischaemic heart disease risk factor study, a prospective population based study. *Brit Med J* 1997;315:846–51.
- [54] Brenner H, Rothenbacher D, Bode G, Adler G. Relation of smoking and alcohol and coffee consumption to active *Helicobacter pylori* infection: cross sectional study. *Brit Med J* 1997;315:1489–92.
- [55] Brenner H, Rothenbacher D, Bode G, Adler G. Inverse graded relation between alcohol consumption and active infection with *Helicobacter pylori*. *Am J Epid* 1999;149:571–6.
- [56] Creppy EE. Human ochratoxicosis. *J Toxic Toxin Rev* 1999;18:277–93.
- [57] Long DE. From cobalt to chloropropanol: de tribulationibus aptis cervisiis imbibendis. *J Inst Brew* 1999;105:79–84.
- [58] Spiegelhalter B, Eisenbrand G, Preussmann R. Contamination of beer with trace quantities of N-nitrosodimethylamine. *Food Cosm Tox* 1979;17:29–31.
- [59] Sen NP, Seaman SW, Bergeron C, Brousseau R. Trends in the levels of N-nitrosodimethylamine in Canadian and imported beers. *J Ag Food Chem* 1996;44:1498–1501.
- [60] Dich J, Jarvinen R, Knekt P, Penttila PL. Dietary intakes of nitrate, nitrite and NDMA in the Finnish Mobile Clinic health examination survey. *Food Add Contam* 1996;13:541–52.
- [61] Tricker AR, Preussmann R. Volatile and non-volatile nitrosamines in beer. *J Canc Res Clin Onc* 1991;117:130–2.
- [62] Woodson K, Albanes D, Tangrea JA, Rautalahti M, Virtamo J, Taylor PR. Association between alcohol and lung cancer in the alpha-tocopherol, beta-carotene cancer prevention study in Finland. *Canc Causes Cont* 1999;10:219–26.
- [63] Freudenheim JL, Marshall JR, Graham S, Laughlin R, Vena JE, Swanson M, Ambrosone C, Nemoto T. Lifetime alcohol consumption and risk of breast cancer. *Nutr Canc* 1995;23:1–11.
- [64] Zhang YQ, Kreger BE, Dorgan JF, Splansky GL, Cupples LA, Ellison RC. Alcohol consumption and risk of breast cancer: the Framlingham study revisited. *Am J Epid* 1999;149:93–101.
- [65] Ferraroni M, Decarli A, Franceschi S, La Vecchia C. Alcohol consumption and risk of breast cancer: a multicentre Italian case-control study. *Eur J Canc* 1998;34:1403–9.
- [66] Hebert JR, Hurley TG, Ma YS. The effect of dietary exposures on recurrence and mortality in early stage breast cancer. *Breast Canc Res Treat* 1998;51:17–28.
- [67] Jacobsen BK. Relationships between childbearing and some food and alcohol habits: the Nordland health study. *Eur J Epid* 1996;12:327–30.

- [68] Hsing AW, McLaughlin JK, Chow WH, Schuman LM, Chien HTC, Gridley G, Bielke E, Wacholder S, Blot WJ. Risk factors for colorectal cancer in a prospective study among US white men. *Int J Canc* 1998;77: 549–53.
- [69] Gronbaek M, Becker U, Johansen D, Tonnesen H, Jensen G, Sorensen TIA. Population based cohort study of the association between alcohol intake and cancer of the upper digestive tract. *Brit Med J* 1998;317: 844–8.
- [70] Cervilla JA, Prince M, Lovestone S, Mann A. Long-term predictors of cognitive outcome in a cohort of older people with hypertension. *Brit J Psych* 2000;177:66–71.
- [71] Dufouil C, Ducimetiere P, Alperovitch A. Sex differences in the association between alcohol consumption and cognitive performance. EVA study group. *Epidemiology of vascular ageing*. *Am J Epid* 1997;146:405–12.
- [72] Obisesan TO, Hirsch R, Kosoko O, Carlson L, Parrott M. Moderate wine consumption is associated with decreased odds of developing age-related macular degeneration in NHANES-1. *J Amer Geriat Soc* 1998; 46:1–7.
- [73] Gruenewald PJ, Ponicki WR. The relationship of alcohol sales to cirrhosis mortality. *J Stud Alc* 1995;56: 635–41.
- [74] Nevill AM, Holder RL, Fentem PH, Rayson M, Marshall T, Cooke C, Tuxworth W. Modelling the associations of BMI, physical activity and diet with arterial blood pressure: some results from the Allied Dunbar national fitness survey. *Ann Hum Biol* 1997;24:229–47.
- [75] Krieger JN, Kronmal RA, Coxon V, Wortley P, Thompson L, Sherrard DJ. Dietary and behavioural risk factors for urolithiasis: potential implications for prevention. *Am J Kidney Dis* 1996;28:195–201.
- [76] Nogueira FN, Souza DN, Nicolau J. In vitro approach to evaluate potential harmful effects of beer on health. *J Dent* 2000;28:271–6.
- [77] Nicolodi M, Sicuteri F. Wine and migraine: compatibility or incompatibility. *Drugs Exp Clin Res* 1999; 25:147–153.
- [78] Lapcik O, Hill M, Hampl R, Wahala K, Adlercreutz H. Identification of isoflavanoids in beer. *Steroids* 1998;63:14–20.
- [79] Walker CJ. Phytoestrogens in beer—good news or bad news? *Brau Int* 2000;18:38–9.
- [80] Promberger A, Dornstauder E, Fruhwirth C, Schmid ER, Jungbauer A. Determination of estrogenic activity in beer by biological and chemical means. *J Ag Food Chem* 2001;49:633–40.
- [81] Forster A, Koberlein A. The location of xanthohumol from hops during beer production. *Brauwelt* 1998;138:1677–9.
- [82] Piendl A, Zeuch M. On the physiological significance of ballast substances for the human being. *Monat Brauwiss* 1995;48:377–89.
- [83] McMurrough I, Delcour JA. Wort polyphenols. *Ferment* 1994;3:175–82.
- [84] Bourne L, Paganga G, Baxter D, Hughes P, Rice-Evans C. Absorption of ferulic acid from low-alcohol beer. *Free Rad Res* 2000;32:273–80.
- [85] Bellia JP, Birchall JD, Roberts NB. The role of silicic acid in the renal excretion of aluminium. *Ann Clin Lab Sci* 1996;26:227–33.
- [86] Izquierdo-Pulido M, Marine-Font A, Vidal-Carou MC. Effect of tyrosine on tyramine formation during beer fermentation. *Food Chem* 2000;70:329–32.
- [87] Shulman KI, Tailor SAN, Walker SE, Gardner DM Tap (draft) beer, and monoamine oxidase inhibitor dietary restrictions. *Can J Psych* 1997;42:310–2.
- [88] Eastmond, CJ, Garton M, Robins S, Riddoch S. The effects of alcoholic beverages on urate metabolism in gout sufferers. *Brit J Rheum* 1995;34:756–9.
- [89] Ellis HJ, Freedman AR, Ciclitira PJ. Detection and estimation of the barley prolamin content of beer and malt to assess their suitability for patients with celiac disease. *Clin Chim Acta* 1990;189:123–30.
- [90] Moll, M. *Beers and coolers*. Andover: Intercept, 1991.
- [91] Hough JS, Briggs DE, Stevens E, Young TW. *Malting and brewing science*. Volume 2. Hopped wort and beer. London: Chapman & Hall, 1982.
- [92] Donhauser S, Wagner D, Jacob F. Critical trace elements in brewing technology. 2. Occurrence of arsenic, lead, cadmium, chromium, mercury and selenium in beer. *Monat Brauwiss* 1987;40:328–33.