EDITORIAL

Awash with Alcohol in the World of Prevention

In line with very much of the literature, the report by Hoshiyama and co-workers in the present volume does not point to any significant link between alcohol use and colon adenoma development. In the Food, Nutrition and the Prevention of Cancer: a Global Perspective (World Cancer Fund /American Institute for Cancer Research, 1998) (see Table 1) the sites for which convincing evidence of an increased risk of neoplasia with alcohol consumption was concluded to be available were only the mouth and pharynx, larynx and oesophagus, other than the liver which clearly is complicated by the role played by cirrhosis and chronically elevated cell proliferation. Probable increase was concluded for the colon/rectum and breast and a possible risk for lung. In the latter case the lack of distinction between cancer types makes discussion very difficult, given the differences in modifying factors impacting on adenocarcinoma and squamous cell carcinoma in the lung (Moore et al., 1999), The cancers of the upper digestive and respiratory tract are very largely of squamous cell type, and for these the evidence for a contribution of combination of tobacco and ethanol is overwhelming, alcohol perhaps facilitating carcinogen exposure while itself causing lipid peroxidative damage and altered eicosanoid production (Mufti, 1998).

Table 1. Epidemiological Findings for Alcohol and Cancer

Organ	Cohort	Case-Control
Mouth and Pharynx	9/11* (82**)	17/20 (85)
Larynx	5/7 (71)	18/19 (95)
Oesophagus	6/10 (60)	17/20 (85)
Stomach	1/5 (20)	3/26 (12)
Pancreas	2/6 (33)	1/21 (5)
Colorectal	4/6 (67)	3/15 (20)
Breast	3/11 (27)	9/35 (26)
Prostate	1/4 (25)	0/4 (0)

* Positive/total. Data from Food, Nutrition and the Prevention of Cancer: a Global Perspective (World Cancer Fund /American Institute for Cancer Research, 1998) arbitrarily setting a relative risk of 2.0 or greater as positive. ** Percentage data

The probable evidence for increased risk concluded by the World Cancer Fund in the colon and breast, however, warrants close attention since these are sites of adenocarcinoma development where blood borne factors may be very important (Giovannucci, 1995; Kaaks, 1996). Clearly there are a number of complicating aspects which could impact on different tissues with consumption of alcoholic beverages. The statement that the International Agency for Research on Cancer generally identifies alcoholic drinks as carcinogenic is not in agreement with a great deal of the information that is available.

In particular it should be stressed that one cannot treat alcohol as a single factor. Table 2 summarizes details of constituents for a number of different alcoholic beverages (data from Constituents of Japanese Foodstuffs, 1996). Depending on the form, the intake of energy varies considerably and this is of paramount significance, energy balance being a very important consideration with many cancers (Gerber and Corpet, 1999). The vast majority of epidemiological studies of the influence of alcohol do not adequately take this into account.). Alcohol and a high-fat diet is known to be a combination favoring overfeeding (Trembaly et al., 1995). Furthermore, as pointed out by Kleiner in a recent review (1999), not enough emphasis is placed on the major component of alcoholic drinks, water 'an essential but overlooked nutrient'. Its ommission from the index of the Food, Nutrition and the Prevention of Cancer: a Global Perspective (World Cancer Fund /American Institute for Cancer Research, 1998), and the lack of reference to fluid intake is clearly of great interest in this context. This clearly reflects the difficulty in collection of data, but should also be borne in mind in considering the impact of consumption of other beverages like tea, Japanese or otherwise, and coffee which have been shown to have preventive potential. While other constituents might be active the possibility that the vehicle is playing an important role has unfortunately been ignored in many cases. With alcohol the situation is complicated by diuretic effects, but these do not necessarily rule out a contribution of the fluid intake, for example with beer, to modulation of tumour development.

Indeed, a number of studies have pointed to links between water intake and cancer development, the quantity of liquid consumed generally showing an inverse correlation with risk of neoplasia in the colon (Shannon et al., 1996; Slattery et al., 1999; Tang et al., 1999). Water consumption was also

Table 2. Constitutents of Alcoholic Beverages

Type	Energy (kcal)	Water	Protein	Sugar	Calcium	Alcohol %
Japanese Sake	113	81.4	0.5	5.0	4	16.5
Beer – Lager	39	92.8	0.4	3.1	35	4.5
Beer - Black	46	91.2	0.5	4.2	50	5.0
Wine – White	75	88.1	0.2	2.0	75	12
Wine – Red	73	88.4	0.2	1.5	100	12
Whiskey	225	67.5	0	0	~0	39
Brandy/Cognac	250	63.9	0	0	~0	43

negatively associated with cancer of the breast in the hospital-based pilot case-control study of Stookey and coworkers (1997) and in Hawaii, total fluid intake was found to be inversely related to lower urinary tract cancer (Wilkens et al 1996). In line with this, patients with urinary tract cancers in Israel were discovered to have consumed significantly less fluid than healthy controls (Bitterman et al., 1991). Two other epidemiological studies pointed to similar links (Cantor et al., 1987; Lu et al., 1999). With regard to the mechanism, Satoh has argued on the basis of characteristics of an enzyme often overexpressed in cancers that increasing fluid intake can aid excretion of carcinogens important for cancer in man (Satoh, 1998; Satoh et al., 1999). Clearly this is an area deserving further research attention.

The physiological effects of beer and more alcoholic beverages may be very different, for example with reference to blood pressure (Takashima et al., 1997). Even the same general type of drink may vary considerably in impact, red and white wines, for example having very different effects on lower esophageal sphincter pressure and gastroesophageal reflux (Pehl et al., 1998). This is of course to be expected from the marked differences in constituents and underlines the necessity for a non-simplistic approach to analysis of the importance of alcohol consumption for cancer and what measures are warranted in terms of preventive efforts. As argued earlier, pathophysiological epidemiology might also warrant greater exploitation in international efforts at cancer control (Moore and Tsuda, 1998).

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Conflict of Interest Statement

The author of this editorial wishes it to be known that he is an occasional supporter of the Scottish single malt whiskey industry and a very much more regular contributor to the coffers of brewers of fine beers around the world. As such, his views on the benefits or adverse effects of alcohol are clearly not totally unbiased.

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