British Journal of Cancer (2011) 105, S14–S18

www.bjcancer.com

3.
Cancers attributable to consumption of alcohol in the UK in 2010

DM Parkin[*,1]

[1]*Centre for Cancer Prevention, Wolfson Institute of Preventive Medicine, Queen Mary University of London, Charterhouse Square, London EC1M 6BQ, UK*

British Journal of Cancer (2011) **105,** S14–S18; doi:10.1038/bjc.2011.476 www.bjcancer.com

In 1988, the International Agency for Research on Cancer (IARC) Monograph on the carcinogenic risk to humans of alcohol drinking concluded that the occurrence of malignant tumours of the oral cavity, pharynx, larynx, oesophagus and liver was causally related to the consumption of alcoholic beverages. In an updated review (Baan *et al*, 2007; Secretan *et al*, 2009), they noted the consistent finding of an increased risk of breast cancer with increasing alcohol intake, and that an association between alcohol consumption and colorectal cancer had been reported by more than 50 prospective and case–control studies, with no difference in the risk for colon and rectal cancers (Baan *et al*, 2007). The World Cancer Research Fund report (WCRF, 2007) considered that the evidence for an association of alcohol intake with these sites was convincing and, for liver cancer, probable.

METHODS

Quantitative risk of alcohol

Table 1 shows the increase in risk associated with consumption of 1 g per day of alcohol. The estimates in these studies had been adjusted for major confounders, notably smoking.

With respect to breast cancer, the estimate was derived from a meta-analysis of 53 studies, conducted by the Collaborative Group on Hormonal Factors in Breast Cancer (Hamajima *et al*, 2002), which found that the risk was increased by 7.1% for every 10 g of daily alcoholintake. The values observed in subsequent studies are not substantially different. A pooled analysis of six cohort studies with data on alcohol and dietary factors found that the risk of breast cancer increased monotonically with increasing intake of alcohol; the multivariate relative risk (RR) for a 10-g per day increase in alcohol was 1.09 (95% CI = 1.04–1.13; Smith-Warner *et al*, 1998). The EPIC study (Tjønneland *et al*, 2007) found that the risk was 1.03 (95% CI = 1.01–1.05) per 10-g per day recent alcohol intake, whereas in the Million Women Study the increase in risk associated with 10 g per day intake was 12% (Allen *et al*, 2009).

With respect to cancers of the colorectum, a pooled analysis of eight cohort studies reported a borderline statistically significant 16% risk increase for people drinking 30–45 g per day of alcohol and a significant 41% risk increase for people drinking $\geqslant$45 g per day (Cho *et al*, 2004). A more recent meta-analysis of cohort studies found a 15% increase in the risk of colon or rectal cancer for an increase of 100 g alcohol intake per week (Moskal *et al*, 2007), with no difference between men and women. In the EPIC study (Ferrari *et al*, 2007), the effect was a bit weaker, with alcohol intake at study baseline increasing colorectal cancer risk by 9% per 15 g per day, a risk greater for rectal cancer than for cancer of the distal colon, which in turn was greater than the risk for cancer of the proximal colon. In the WCRF (2007) report, a meta-analysis of eight studies of colon cancer yielded a combined RR of 1.09 (1.03–1.14) per 10 g intake per day, and a meta-analysis of nine studies of rectal cancer yielded an RR of 1.06 (1.01–1.12) per 10 g intake per day.

The means in the meta-analyses of Cho *et al* (2004), Moskal *et al* (2007), the EPIC study (Ferrari *et al*, 2007) and WCRF (2007) are 0.75% per gram alcohol per day for colon cancer and 0.85% per gram per day for rectal cancer. As these estimates are similar, the global figure of 0.8% per gram (increase of 0.008 per gram per day) was used for colorectal cancer as a whole (Table 1).

For the remaining cancers, the meta-analysis of Corrao *et al* (2004) was used to estimate the RRs. They present RRs associated with a mean intake of 0, 25, 50 and 100 g of alcohol per day. The RR per gram of alcohol intake was estimated by assuming a log–linear relationship between exposure and risk, so that:

$$\text{Relative risk}\,(x) = \exp(\ln(\text{risk per unit}) \times \text{exposure level}\,(x))$$

where x is the exposure level (in grams per day).

Prevalence of exposure to alcohol

The latent period or interval between 'exposure' to alcohol and the appropriate increase in risk of these cancers is not known. We chose to assume that this would be, on average, 10 years, and thus examine the effects on cancers occurring in 2010 from non-optimal levels of alcohol consumption in the year 2000.

There are two main ways of measuring the amount of alcohol consumed: asking people how much alcohol they drink or counting how much alcohol is sold. As the estimates of the effect of past alcohol drinking on cancer risk are based on epidemiological studies in which alcohol intake is estimated from questionnaire data, it is most appropriate to base the exposure prevalence on data from a similar source.

We have used data from the National Diet and Nutrition Survey, a survey of the diet and nutrition of a representative sample of adults in the age group of 19–64 years living in private households in Great Britain, carried out between July 2000 and June 2001

*Correspondence: Professor DM Parkin; E-mail: d.m.parkin@qmul.ac.uk

(Henderson *et al*, 2003). For the age group >65 years, we used data on the proportion of non-drinkers, and average alcohol consumption from the General Household Survey (for England) (Goddard, 2006). From these tables, an estimate was prepared of the proportions of individuals (by age group and sex) consuming different quantities of alcohol in terms of grams per day, assuming that 1 unit of alcoholic beverages contains 8 g of pure alcohol (Table 2).

The same data are shown in Figure 1, as the cumulative percentages of men and women of different ages with different levels of alcohol intake in 2000, as grams per day of alcohol.

Estimation of population attributable fractions (PAFs)

For the six cancer types, PAFs were calculated for each sex–age group according to the usual formula:

$$\mathrm{PAF} = \frac{\Sigma(p_x \times \mathrm{ERR}_x)}{1 + \Sigma(p_x \times \mathrm{ERR}_x)}$$

where p_x is the proportion of the population in consumption level x ($x = 1-12$) and ERR_x the excess relative risk ($\mathrm{RR}_x - 1$) in consumption level x ($x = 1-12$).

The ERR of alcohol consumption for each level x of alcohol consumption given in Table 2 was calculated as follows:

$$\mathrm{ERR}_x = \exp(R_g \times G_x) - 1$$

where R_g is the increase in risk per gram of alcohol intake (Table 1) and G_x the intake of alcohol (grams per day) in consumption category x (Table 2).

Table 1 Increase in risk of cancer associated with 1 gram of alcohol per day

Cancer type	Studies	Increase in risk per gram alcohol per day
Oral cavity and pharynx	Corrao *et al* (2004)	0.0185
Larynx	Corrao *et al* (2004)	0.0136
Oesophagus	Corrao *et al* (2004)	0.0129
Colorectal cancer	Cho *et al* (2004) Moskal *et al* (2007) Ferrari *et al* (2007) WCRF (2007)	0.0080
Breast	Collaborative Group (Hamajima *et al*, 2002)	0.0071
Liver	Corrao *et al* (2004)	0.0059

RESULTS

Table 3 shows for each sex and age group the numbers of cases of the six alcohol-related cancers in the UK in 2010, the PAFs due to alcohol consumption 10 years earlier (2000–2001) and the corresponding number of excess cases (calculated as (observed × PAF)).

Because of the high risk of upper aero-digestive tract cancer associated with alcohol drinking, cancers of the mouth and pharynx, as well as larynx, had the highest percentages of alcohol-attributable cases (30.4% of cancers of the oral cavity and pharynx, 24.6% of laryngeal cancers). Although the fractions of colorectal (11.6%) and breast (6.4%) cancers were much lower, the actual numbers of alcohol-attributable cases were much greater – together, they account for about 7700 alcohol-attributable cases in 2010 (or 62% of all alcohol-related cancers).

Table 4 sums the excess numbers of cases at the six sites, caused by alcohol consumption, and expresses these numbers as a fraction of the total burden of (incident) cancer. The estimates are 4.6%

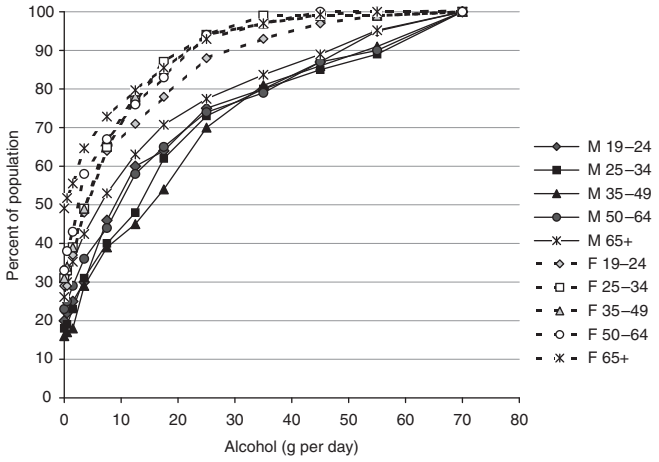

Figure 1 Cumulative percentage of population with different alcohol intakes.

Table 2 Estimated percentage of the population at 12 levels of alcohol consumption

		% of population consuming the specified grams per day alcohol in Great Britain during 2000–2001											
Alcohol consumption		Men by age (years)						Women by age (years)					
Level	Grams per day	19–24	25–34	35–49	50–64	65+	All 19+	19–24	25–34	35–49	50–64	65+	All 19+
1	0	20	18	16	23	26	20	29	31	31	33	49	35
2	0.5	2	1	1	2	4	2	0	2	3	5	3	3
3	1.5	3	4	1	4	5	3	8	6	5	5	4	5
4	3.5	5	8	11	7	7	8	11	10	10	15	9	11
5	7.5	16	9	10	8	11	10	16	16	18	9	8	13
6	12.5	14	8	6	14	10	10	7	12	11	9	7	9
7	17.5	4	14	9	7	8	9	7	10	7	7	6	7
8	25	11	11	16	9	7	11	10	7	9	11	7	9
9	35	5	7	11	5	6	7	5	5	3	3	4	4
10	45	7	5	5	8	5	6	4	0	2	3	2	2
11	55	8	4	5	3	6	5	2	0	0	0	1	0
12	70	5	11	9	10	5	8	1	1	1	0	0	0
Mean grams per day		20.4	22.2	23.1	21.1	12.6	23.6	11.4	9.1	9.2	8.6	7.7	11.6

Data for 19–64-year-olds from Henderson (2003); data for >65-year-olds from Goddard (2006).

Table 3 Cancer cases diagnosed in 2010 attributable to alcohol consumption in 2000–2001

Age (years)		Cases attributable to alcohol consumption for each cancer																	
		Oral cavity and pharynx			Oesophagus			Colon–rectum			Liver			Larynx			Breast		
At exposure	At outcome (+10 years)	PAF	Obs.	Excess attrib. cases	PAF	Obs.	Excess attrib. cases	PAF	Obs.	Excess attrib. cases	PAF	Obs.	Excess attrib. cases	PAF	Obs.	Excess attrib. cases	PAF	Obs.	Excess attrib. cases
Men																			
15–24	25–34	0.36	55	19.6	0.25	11	2.8	0.16	133	20.8	0.12	19	2.1	0.26	2	0.5			
25–34	35–44	0.39	244	96.1	0.28	86	24.0	0.17	397	69.2	0.13	43	5.5	0.29	35	10.3			
35–49	45–59	0.40	1591	631.8	0.28	970	274.9	0.18	2921	521.1	0.13	351	46.4	0.30	407	121.3			
50–64	60–74	0.38	1888	709.1	0.26	2535	668.2	0.16	9481	1548.0	0.12	1011	121.4	0.28	914	253.9			
⩾65	⩾75	0.32	768	249.1	0.22	2108	473.7	0.14	9162	1262.7	0.10	828	83.7	0.24	444	105.3			
Total			4571	1705.9		5713	1443.5		22 127	3421.8		2270	259.1		1803	491.3			
%				37.3			25.3			15.5			11.4			27.3			
Women																			
15–24	25–34	0.23	50	11.4	0.16	4	0.6	0.10	136	13.0	0.07	12.11	0.9	0.17	2	0.3	0.08	715.1	60.7
25–34	35–44	0.18	131	23.2	0.12	27	3.3	0.07	402	29.9	0.05	29.48	1.6	0.13	12	1.5	0.07	3857	254.0
35–49	45–59	0.18	622	113.5	0.12	303	37.9	0.08	2292	174.8	0.06	142.8	8.0	0.13	99	13.1	0.07	14 628	987.4
50–64	60–74	0.17	855	146.3	0.12	922	108.5	0.07	6116	440.7	0.05	453	23.9	0.12	168	20.9	0.06	17 194	1096.9
⩾65	⩾75	0.16	666	105.2	0.11	1560	166.8	0.06	8810	568.8	0.05	642.4	30.2	0.11	101	11.4	0.06	11 952	681.3
Total			2359	399.7		2819	317.2		17 787	1227.3		1298	64.6		386	47.3		48 385	3080.3
%				16.9			11.3			6.9			5.0			12.2			6.4
Persons																			
15–24	25–34		105	31.1		15	3.4		269	33.8		31	3.0		4	0.9		715	60.7
25–34	35–44		375	119.3		113	27.3		799	99.1		72	7.1		47	11.8		3857	254.0
35–49	45–59		2213	745.4		1273	312.8		5213	695.9		494	54.3		506	134.4		14 628	987.4
50–64	60–74		2743	855.5		3457	776.7		15 597	1988.8		1464	145.4		1082	274.8		17 194	1096.9
⩾65	⩾75		1434	354.3		3668	640.5		17 972	1831.5		1470	113.9		545	116.7		11 952	681.3
Total			6930	2105.6		8532	1761		39 914	4649		3568	324		2189	539		48 385	3080
%				30.4			20.6			11.6			9.1			24.6			6.4

Abbreviations: attrib. = attributable; Obs. = observed cases; PAF = population-attributable fraction.

Table 4 Estimated total numbers of cancers in the UK in 2010, PAFs due to alcohol consumption 10 years earlier (2000–2001), and the corresponding number and percentage of excess cases, by age group and sex

Age (years)		All cancers[a]		
Exposure	Outcome (+10 years)	Observed cases	Excess attributable cases	PAF (%)
Men				
15–24	25–34	2109	46	2.2
25–34	35–44	4124	205	5.0
35–49	45–59	22 388	1596	7.1
50–64	60–74	68 043	3301	4.9
⩾65	75+	60 149	2175	3.6
Total		158 667	7322	4.6
Women				
15–24	25–34	3284.1	87	2.6
25–34	35–44	8619.2	313	3.6
35–49	45–59	31 631	1335	4.2
50–64	60–74	54 966	1837	3.3
⩾65	75+	55 437	1564	2.8
Total		155 584	5136	3.3
Persons				
15–24	25–34	5393	133	2.5
25–34	35–44	12 743	519	4.1
35–49	45–59	54 019	2930	5.4
50–64	60–74	123 009	5138	4.2
⩾65	75+	115 586	3738	3.2
Total		314 251	12 458	4.0

Abbreviations: PAF = population-attributable fraction. [a]Excluding non-melanoma skin cancer.

cancers in men and 3.3% in women due to alcohol consumption, or 4.0% cancers overall.

DISCUSSION

The estimates of the RR of alcohol consumption for various cancers are an 'average' taken from widely cited meta-analyses; more extreme values can be found in specific studies.

Table 5 compares the excess RRs of 1 g of alcohol consumption per day as used in this study with those from the Million Women Study (Allen *et al*, 2009) and the EPIC study (Ferrari *et al*, 2007; Tjonneland *et al*, 2007), as well as with those derived from various meta-analyses by WCRF (2007). The values for cohort studies are shown for cancers of the breast, colon, rectum and liver. For upper aero-digestive and oesophageal cancers, meta-analyses were based on case–control studies only.

For the most part, the risks associated with consumption of alcohol used in the present study are similar to those in the three comparative studies listed in Table 5. The ERRs reported in the Million Women Study (Allen *et al*, 2009) are rather higher than those in Table 1 for cancers of the oesophagus, liver and larynx, although the values used in the current analysis (Table 1) lie within the relevant 95% confidence intervals; for colon cancer, however, the value is considerably lower.

With respect to cancer of the oesophagus, some of the differences may relate to the differing proportions of squamous cell and adenocarcinomas in the series of cancers in various studies. Although squamous cell carcinomas are clearly related to alcohol exposure, the risk of adenocarcinoma is much lower, or nil (Lagergren *et al*, 2000; Wu *et al*, 2001; Lindblad *et al*, 2005; Pandeya *et al*, 2009). Currently, adenocarcinomas comprise

Table 5 Estimates of excess relative risk associated with 1 gram alcohol intake per day

Cancer	Excess relative risk (ERR)			
	This study	MWS 2009[a]	WCRF/AICR 2007[b]	EPIC[c]
Breast	0.0071	0.0114	0.0095	0.0030[d]
Colon	0.0081	0.0010	0.0086	0.0045
Rectum	0.0081	0.0096	0.0058	0.0070
Liver	0.0059	0.0217	0.0095	
Oesophagus	0.0129	0.0201	0.0183[e]	
Oral cavity and pharynx	0.0185	0.0258		
Larynx	0.0136	0.0371	0.0138[e]	

[a]Million Women Study, Allen *et al* (2009). [b]WCRF (2007). [c]European Prospective Investigation into Cancer and Nutrition, Ferrari *et al* (2007). [d]Tjonneland *et al* (2007). [e]Based on meta-analysis of case–control studies only.

Table 6 UK alcohol consumption per adult

Year	General Household Survey[a]		HM Revenue and Customs[b]	
	Units per week	Litres of pure alcohol per year	Units per week	Litres of pure alcohol per year
1990	10.8	5.3	19.2	10
2000	12.0	6.2	20.2	10.5
2005	10.8	5.6	21.9	11.4

[a]General Household Survey (Goddard, 2006). [b]HM Revenue and Customs (HMRC, 2008).

approximately 70% of oesophageal cancers in men in the UK, and 40% in women (see section 8, in Cancers attributable to overweight and obesity). However, the studies currently used to estimate the RR of oesophageal cancer in relation to alcohol do not distinguish between the histological subtypes, and no correction to the estimate for the UK has been made on this basis.

We chose to use the estimates of alcohol consumption in the UK based on population survey data (the National Diet and Nutrition Survey). However, it is well known that surveys produce figures far lower than would be expected from alcohol sales. Alcohol sales are estimated based on clearance data produced by HM Revenue and Customs (HMRC). Not all alcohol that is cleared is actually consumed; for example, it is conceivable that some of it may be thrown away when it passes its best-before date. Conversely, not all alcohol that is consumed in the UK is cleared by HMRC; for example, home brew and illegally imported alcohol.

Table 6 compares consumption as estimated by the General Household Survey (Goddard, 2006) and from clearance data produced by HM Revenue and Customs (HMRC, 2008). The large difference between the two sets of data is unlikely to be due to large amounts of purchased alcohol not being consumed. Both the General Household Survey and the Government's alcohol strategy (HMG, 2007) believe that many people underestimate the amount of alcohol they drink. However, as estimates of risk are generally based on responses to questionnaires, they are likely to overestimate the risk in relation to actual alcohol consumption. It is more appropriate, therefore, to use estimates of alcohol intake from (self-reported) survey data than the more accurate clearance data.

The current estimate (3.6% of new cancers in 2010 related to alcohol) is similar to the figure published by Doll and Peto (2003) – that around 6% of UK cancer deaths could be avoided if people did not drink. The estimation is based on the attribution to alcohol of 2/3 deaths from alcohol-related cancers (mouth, pharynx, larynx, oesophagus) in men and 1/3 in women, plus 'a small proportion' of liver cancer deaths. A recent publication, based on the risks of alcohol consumption observed in the EPIC study, estimates a rather higher fraction of cancers attributable to alcohol in the UK – especially in men: 8% of cancer in men and 3% in women (Schütze *et al*, 2011). The difference appears to be mainly because of the rather higher level and prevalence of alcohol consumption that were used to estimate attributable fractions (an average intake of 35.2 g per day in men and 17.6 g per day in women, cf. Table 2). These were calculated from data available on the World Health Organisation website, which appear to be derived from clearance data, with levels of consumption equivalent to those in Table 6 (on average, annually 13.4 l of alcohol per capita in 2003–5). As noted above, it would seem more appropriate to use self-reported consumption, even though this is an underestimate of the true situation, as the RR estimates in EPIC (as in other cohort studies) are also based on questionnaire data.

See acknowledgements on page Si.

Conflict of interest

The author declares no conflict of interest.

REFERENCES

Allen NE, Beral V, Casabonne D, Kan SW, Reeves GK, Brown A, Green J (2009) Moderate alcohol intake and cancer incidence in women. *J Natl Cancer Inst* **101:** 296–305

Baan R, Straif K, Grosse Y, Secretan B, El Ghissassi F, Bouvard V, Altieri A, Cogliano V (2007) Carcinogenicity of alcoholic beverages. *Lancet Oncol* **8:** 292–293

Cho E, Smith-Warner SA, Ritz J, van den Brandt PA, Colditz GA, Folsom AR, Freudenheim JL, Giovannucci E, Goldbohm RA, Graham S, Holmberg L, Kim DH, Malila N, Miller AB, Pietinen P, Rohan TE, Sellers TA, Speizer FE, Willett WC, Wolk A, Hunter DJ (2004) Alcohol intake and colorectal cancer: a pooled analysis of 8 cohort studies. *Ann Intern Med* **140:** 603–613

Corrao G, Bagnardi V, Zambon A, La Vecchia C (2004) A meta-analysis of alcohol consumption and the risk of 15 diseases. *Prev Med* **38:** 613–619

Doll R, Peto R (2003) Epidemiology of Cancer. In *Oxford Textbook of Medicine*, Warrell DA, Cox TM, Firth JD, Benz Jr EJ (eds), 4th edn. Oxford University Press: Oxford, UK

Ferrari P, Jenab M, Norat T, Moskal A, Slimani N, Olsen A, Tjønneland A, Overvad K, Jensen MK, Boutron-Ruault MC, Clavel-Chapelon F, Morois S, Rohrmann S, Linseisen J, Boeing H, Bergmann M, Kontopoulou D, Trichopoulou A, Kassapa C, Masala G, Krogh V, Vineis P, Panico S, Tumino R, van Gils CH, Peeters P, Bueno-de-Mesquita HB, Ocké MC, Skeie G, Lund E, Agudo A, Ardanaz E, López DC, Sanchez MJ, Quirós JR, Amiano P, Berglund G, Manjer J, Palmqvist R, Van Guelpen B, Allen N, Key T, Bingham S, Mazuir M, Boffetta P, Kaaks R, Riboli E (2007) Lifetime and baseline alcohol intake and risk of colon and rectal cancers in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Int J Cancer* **121:** 2065–2072

Goddard E (2006) *General Household Survey, 2006. Smoking and Drinking Among Adults, 2006.* Office of National Statistics, 2006: London. http://www.parliament.uk/deposits/depositedpapers/2008/DEP2008-2395.pdf

Hamajima N, Hirose K, Tajima K, Rohan T, Calle EE, Heath Jr CW, Coates RJ, Liff JM, Talamini R, Chantarakul N, Koetsawang S, Rachawat D, Morabia A, Schuman L, Stewart W, Szklo M, Bain C, Schofield F, Siskind V, Band P, Coldman AJ, Gallagher RP, Hislop TG, Yang P, Kolonel LM, Nomura AM, Hu J, Johnson KC, Mao Y, De Sanjosé S, Lee N, Marchbanks P, Ory HW, Peterson HB, Wilson HG, Wingo PA, Ebeling K, Kunde D, Nishan P, Hopper JL, Colditz G, Gajalanski V, Martin N, Pardthaisong T, Silpisornkosol S, Theetranont C, Boosiri B, Chutivongse S, Jimakorn P, Virutamasen P, Wongsrichanalai C,

Ewertz M, Adami HO, Bergkvist L, Magnusson C, Persson I, Chang-Claude J, Paul C, Skegg DC, Spears GF, Boyle P, Evstifeeva T, Daling JR, Hutchinson WB, Malone K, Noonan EA, Stanford JL, Thomas DB, Weiss NS, White E, Andrieu N, Brêmond A, Clavel F, Gairard B, Lansac J, Piana L, Renaud R, Izquierdo A, Viladiu P, Cuevas HR, Ontiveros P, Palet A, Salazar SB, Aristizabel N, Cuadros A, Tryggvadottir L, Tulinius H, Bachelot A, Lê MG, Peto J, Franceschi S, Lubin F, Modan B, Ron E, Wax Y, Friedman GD, Hiatt RA, Levi F, Bishop T, Kosmelj K, Primic-Zakelj M, Ravnihar B, Stare J, Beeson WL, Fraser G, Bullbrook RD, Cuzick J, Duffy SW, Fentiman IS, Hayward JL, Wang DY, McMichael AJ, McPherson K, Hanson RL, Leske MC, Mahoney MC, Nasca PC, Varma AO, Weinstein AL, Moller TR, Olsson H, Ranstam J, Goldbohm RA, van den Brandt PA, Apelo RA, Baens J, de la Cruz JR, Javier B, Lacaya LB, Ngelangel CA, La Vecchia C, Negri E, Marubini E, Ferraroni M, Gerber M, Richardson S, Segala C, Gatei D, Kenya P, Kungu A, Mati JG, Brinton LA, Hoover R, Schairer C, Spirtas R, Lee HP, Rookus MA, van Leeuwen FE, Schoenberg JA, McCredie M, Gammon MD, Clarke EA, Jones L, Neil A, Vessey M, Yeates D, Appleby P, Banks E, Beral V, Bull D, Crossley B, Goodill A, Green J, Hermon C, Key T, Langston N, Lewis C, Reeves G, Collins R, Doll R, Peto R, Mabuchi K, Preston D, Hannaford P, Kay C, Rosero-Bixby L, Gao YT, Jin F, Yuan JM, Wei HY, Yun T, Zhiheng C, Berry G, Cooper Booth J, Jelihovsky T, MacLennan R, Shearman R, Wang QS, Baines CJ, Miller AB, Wall C, Lund E, Stalsberg H, Shu XO, Zheng W, Katsouyanni K, Trichopoulou A, Trichopoulos D, Dabancens A, Martinez L, Molina R, Salas O, Alexander FE, Anderson K, Folsom AR, Hulka BS, Bernstein L, Enger S, Haile RW, Paganini-Hill A, Pike MC, Ross RK, Ursin G, Yu MC, Longnecker MP, Newcomb P, Bergkvist L, Kalache A, Farley TM, Holck S, Meirik O, Collaborative Group on Hormonal Factors in Breast Cancer (2002) Alcohol, tobacco and breast cancer - collaborative reanalysis of individual data from 53 epidemiological studies, including 58,515 women with breast cancer and 95,067 women without the disease. *Br J Cancer* **87:** 1234 – 1245

Henderson L, Gregory J, Irving K, Swan G (2003) *The National Diet & Nutrition Survey: Adults Aged 19 to 64 Years. Vol. 2. Energy, Protein, Carbohydrate, Fat and Alcohol Intake*. HMSO: London. http://www.food.gov.uk/science/dietarysurveys/ndnsdocuments/ndnspreviousurveyreports/ndnsv2

HM Government (HMG) (2007) *Safe. Sensible. Social. The next steps in the National Alcohol Strategy*

HM Revenue and Customs (HMRC) (2008) *Alcohol fact sheet*, http://www.uktradeinfo.com/index.cfm?task = factalcohol

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans (1988) *Alcohol Drinking*, Vol. 44, IARC: Lyon

Lagergren J, Bergstrom R, Lindgren A, Nyrén O (2000) The role of tobacco, snuff and alcohol use in the aetiology of cancer of the oesophagus and gastric cardia. *Int J Cancer* **85:** 340 – 346

Lindblad M, Rodriguez LA, Lagergren J (2005) Body mass, tobacco and alcohol and risk of esophageal, gastric cardia, and gastric non-cardia adenocarcinoma among men and women in a nested case-control study. *Cancer Causes Control* **16:** 285 – 294

Moskal A, Norat T, Ferrari P, Riboli E (2007) Alcohol intake and colorectal cancer risk: a dose-response meta-analysis of published cohort studies. *Int J Cancer* **120:** 664 – 671

Pandeya N, Williams G, Green AC, Webb PM, Whiteman DC (2009) Australian Cancer Study. Alcohol consumption and the risks of adenocarcinoma and squamous cell carcinoma of the esophagus. *Gastroenterology* **136:** 1215 – 1224

Schütze M, Boeing H, Pischon T, Rehm J, Kehoe T, Gmel G, Olsen A, Tjønneland AM, Dahm CC, Overvad K, Clavel-Chapelon F, Boutron-Ruault MC, Trichopoulou A, Benetou V, Zylis D, Kaaks R, Rohrmann S, Palli D, Berrino F, Tumino R, Vineis P, Rodríguez L, Agudo A, Sánchez MJ, Dorronsoro M, Chirlaque MD, Barricarte A, Peeters PH, van Gils CH, Khaw KT, Wareham N, Allen NE, Key TJ, Boffetta P, Slimani N, Jenab M, Romaguera D, Wark PA, Riboli E, Bergmann MM (2011) Alcohol attributable burden of incidence of cancer in eight European countries based on results from prospective cohort study. *Br Med J* **342:** d1584

Secretan B, Straif K, Baan R, Grosse Y, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, Guha N, Freeman C, Galichet L, Cogliano V (2009) A review of human carcinogens – part E: tobacco, areca nut, alcohol, coal smoke, and salted fish. *Lancet Oncol* **10:** 1033 – 1034

Smith-Warner SA, Spiegelman D, Yaun S-S, Adami HO, van den Brandt PA, Folsom AR, Goldbohm RA, Graham S, Howe GR, Marshall JR, Miller AB, Potter JD, Speizer FE, Willett WC, Wolk A, Hunter DJ (1998) Alcohol and breast cancer in women: a pooled analysis of cohort studies. *J Am Med Assoc* **279:** 535 – 540

Tjonneland A, Christensen J, Olsen A, Stripp C, Thomsen BL, Overvad K, Peeters PH, van Gils CH, Bueno-de-Mesquita HB, Ocké MC, Thiebaut A, Fournier A, Clavel-Chapelon F, Berrino F, Palli D, Tumino R, Panico S, Vineis P, Agudo A, Ardanaz E, Martinez-Garcia C, Amiano P, Navarro C, Quirós JR, Key TJ, Reeves G, Khaw KT, Bingham S, Trichopoulou A, Trichopoulos D, Naska A, Nagel G, Chang-Claude J, Boeing H, Lahmann PH, Manjer J, Wirfält E, Hallmans G, Johansson I, Lund E, Skeie G, Hjartåker A, Ferrari P, Slimani N, Kaaks R, Riboli E (2007) Alcohol intake and breast cancer risk: the European Prospective Investigation into Cancer and Nutrition (EPIC). *Cancer Causes Control* **18:** 361 – 373

World Cancer Research Fund (WCRF) Panel (2007) *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*. World Cancer Research Fund: Washington, DC

Wu AH, Wan P, Bernstein L (2001) A multiethnic population-based study of smoking, alcohol and body size and risk of adenocarcinomas of the stomach and esophagus (United States). *Cancer Causes Control* **12:** 721 – 732