

REVIEW

Black tea – helpful or harmful? A review of the evidence

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Objective: To consider whether consumption of black tea has a positive or negative impact on health.

Design: Databases were searched for relevant epidemiological and clinical studies published between 1990 and 2004.

Results: Clear evidence was found for coronary heart disease (CHD), where an intake of ≥ 3 cups per day related to risk reduction. The mechanism could involve the antioxidant action of tea polyphenols. While experimental models have suggested that flavonoids attenuated cancer risk, epidemiological studies failed to demonstrate a clear effect for tea, although there is moderate evidence for a slightly positive or no effect of black tea consumption on colorectal cancer. Studies on cancer were limited by sample sizes and insufficient control of confounders. There is moderate evidence suggestive of a positive effect of black tea consumption on bone mineral density although studies were few. There is little evidence to support the effect of tea on dental plaque inhibition but evidence to support the contribution of tea to fluoride intakes and thus theoretical protection against caries. There was no credible evidence that black tea (in amounts typically consumed) was harmful. Normal hydration was consistent with tea consumption when the caffeine content was < 250 mg per cup. A moderate caffeine intake from tea appeared to improve mental performance, although sample sizes were small. There was no evidence that iron status could be harmed by tea drinking unless populations were already at risk from anaemia.

Conclusions: There was sufficient evidence to show risk reduction for CHD at intakes of ≥ 3 cups per day and for improved antioxidant status at intakes of one to six cups per day. A maximum intake of eight cups per day would minimise any risk relating to excess caffeine consumption. Black tea generally had a positive effect on health.

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Introduction

Tea is the most consumed beverage in the world after water, drunk in the UK for 350 years and in Asia for more than 4000 years. Data on over 7000 adults from the UK National Diet and Nutrition Survey (NDNS) Henderson *et al.* (2002), indicate that 77% of people drink tea, with a mean consumption of 2.3 mugs (540 ml) per day. Men and women drink similar amounts, while 46% drink unsweetened tea (52% women, 39% men). Those aged 50–64 years consume

more black tea than 19- to 24-year olds (mean consumption 644 vs 298 ml).

Antioxidants play an important role in the prevention of chronic diseases. Fruits and vegetables are frequently cited as good sources but mean European intakes remain below the recommended 5-a-day (Naska *et al.*, 2000). Other useful sources are tea and red wine, which are rich in flavonoids, a group of polyphenols which possess considerable antioxidant power and have been shown to impede the actions of free radicals (Dufresne and Farnworth, 2001). Tea makes a significant contribution to dietary intakes of flavonoids; one UK study estimated a figure of 82% (Hertog *et al.*, 1997). This impacts significantly on plasma antioxidant capacity when up to six cups per day are consumed (Rietveld and Wiseman, 2003), although interpretation of the eight studies reporting this was hampered by differences in assay methods and how results were expressed.

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Black tea, as typically brewed in the UK, contains about 200 mg flavonoids per cup (Wiseman *et al.*, 1997). Table 1 shows the key flavonoids and their components as a percentage of dry weight. Green tea leaves contain more catechins, while black tea leaves, which undergo oxidation during manufacturing, contain more complex thearubigins and theaflavins. As yet, these have been more difficult to identify than the single catechins but they also exhibit antioxidant activity (Rietveld and Wiseman, 2003). Flavonoids in tea are absorbed from the upper intestine, more rapidly in the case of the catechins present in green tea (Rietveld and Wiseman, 2003), although plasma antioxidant potential is similar after consuming green vs black tea (Leenen *et al.*, 2000). The uptake of theaflavins found in black tea is relatively low compared with catechins, but this may be masked by imprecise methodologies that do not detect all of the theaflavin metabolites (Mulder *et al.*, 2001). Drinking three cups of tea per day for 2 weeks (at a strength of 2 g dry tea per cup) increased the concentration of flavonoids in the blood by 25% (Hollman *et al.*, 1997).

Consumption of flavonoids could lower the risk of coronary heart disease (CHD) via a number of mechanisms. Firstly, the antioxidant capacity of flavonoids may improve endothelial function by lowering oxidative stress. Better endothelial function impacts on vasomotor tone, platelet activity, leukocyte adhesion and vascular smooth muscle cell function. Human studies have shown that black tea flavonoids improve coronary circulation (Hirata *et al.*, 2004) and attenuate endothelial dysfunction (Duffy *et al.*, 2001), although the latter may be influenced by individual variation in flavonoid metabolism (Hodgson *et al.*, 2006). Secondly, tea flavonoids have also been shown to reduce low-density lipoprotein (LDL) cholesterol by 11.1% (Davies *et al.*, 2003). Thirdly, *in vitro* and animal studies have revealed effects that go beyond antioxidant capacity, for example, reduced expression of endothelial adhesion molecules (Ludwig *et al.*, 2004), stimulation of an anti-inflammatory response (Lin *et al.*, 1999), and gene expression favouring improved smooth muscle function (Kim and

Moon, 2005). Turning to the role of flavonoids in cancer, free radical activity seems to increase the number of DNA mutations, an early stage in cancer pathogenesis (Duthie, 2000). Antioxidants are known to protect DNA and cell membranes from oxidation and this has led to an emphasis on antioxidant-rich foods in public health nutrition.

It is common practice in the UK to consume tea with milk and, as yet, there is no consensus on whether proteins in milk might bind to flavonoids and reduce their activity. One study of 9 healthy volunteers found that the addition of milk reduced antioxidant power (Langley-Evans, 2000), while a slightly larger study ($n=21$) found no difference in antioxidant status when volunteers consumed tea with and without milk (Leenen *et al.*, 2000). Further research on 18 healthy adults showed no effect on antioxidant status (as measured by catechin availability) when milk was added to black tea (Van het Hof *et al.*, 1999). Work from the same group showed that the bioavailability of the flavonols, quercetin and kaempferol, were not affected by the addition of milk (Hollman *et al.*, 2001). From these studies, it would appear on balance that milk is unlikely to reduce the bioactivity of tea flavonoids.

The potential antioxidant power of black tea means it could have a role in helping consumers reduce their risk of CHD and cancer. Yet it is frequently suggested in the media that tea has an adverse effect on fluid balance, cognitive function, bone health, dental health and iron status, often related to its caffeine content. This review will use the available published literature to investigate whether black tea consumption is helpful or harmful in relation to health.

Methods

The Cochrane Library and MEDLINE were searched for epidemiological evidence and clinical trials relating to Seven key areas of tea and health. These were coronary heart disease, cancer, mood and cognitive performance, hydration, iron status, dental health and bone health (see Appendix A for search terms). The search was limited to black tea as this represents 78% of the tea produced worldwide and is the most common variety drunk in Western countries. Many previous reviews have not differentiated between types of tea and this can lead to ambiguity in the conclusions.

Dates of publication were restricted to January 1990 to September 2004. Inclusion criteria were: (a) studies on black tea, (b) adults as subjects, (c) set in Western countries (to focus on tea 'as drunk' in the Western style). Where more than one type of tea was considered, results from black tea had to be clearly differentiated before the study was included. The SIGN 50 Guidelines (SIGN, 2001), which inform literature gathering for reviews, were simplified and adapted to include epidemiological evidence. These were used to guide the weight placed on studies when forming conclusions, that is, randomized controlled trials and meta-analyses were given more weight than epidemiological studies (see Appendix B).

Table 1 Main components of black tea Dufresne and Farnworth (2001)

	Components	% Dry weight
Catechins	Epigallocatechin gallate [EGCG]	10–12
Theaflavins	Resulting from oxidation of catechins during black tea processing	3–6
Thearubigins		12–18
Flavonols	Quercetin	6–8
	Keampferol	
	Rutin	
Methylxanthines	Caffeine	8–11 ^a
Phenolic acids	Caffeic acid	Not available
	Quinic acid	
	Gallic acid	
Amino acids	Theanine	Not available

^aThis provides a mean caffeine level of 40 mg of caffeine per 235 ml cup FSA (2004).

Results

Coronary heart disease (CHD)

CHD is still the most common cause of death in the UK. Consumption of black tea has been associated with a lower incidence of heart disease/cardiac death and a reduction in risk factors. Searches were limited to studies that examined an effect or association (either positive, negative or null association) between black tea intake and heart health/disease in adults. The approach adopted by some studies was to evaluate the effect of additional tea or flavonoids on the factor under investigation. This can provide useful supplementary data and insight into likely mechanisms, but only if there are good data on total tea or flavonoid intake (both background and added). Thus, two studies which failed to report some background data on habitual nutrient intake or at least flavonoid intake were excluded because it would be impossible to assess whether tea/flavonoids or another dietary variables were responsible for associations. Other exclusion criteria were studies using extreme test diets, those that did not separate stroke events, those that only assessed cardiac risk factors (20 studies) and those studies with duplicate cohorts (five studies). Two meta-analyses on black tea consumption were located (Peters *et al.*, 2001; Huxley and Neil, 2003) and relevant findings from these were incorporated into the present review with two exceptions. In the review by Huxley and Neil, 2003, one study contained no data on tea intake, while in the review by Peters *et al.*, 2001, one study used only green tea. In total, 21 studies met our inclusion criteria and are described in Table 2.

Epidemiological data linking black tea consumption to a reduced CHD risk appeared robust. The meta-analysis by Peters *et al.* (2001) reported that the incidence rate of MI was estimated to decrease by 11% with an increase in black tea consumption of three cups per day (one cup = 237 ml) with a fixed-effects relative risk (RR) estimate of 0.89 (95% confidence interval: 0.79, 1.01). Two case control studies provided additional evidence of the RR estimate for 3 cups/day. Sesso *et al.* (1999) reported a RR of 0.31 (95% CI: 0.09, 1.02), while Gramenzi *et al.* (1990) reported a RR of 0.29 (95% CI: 0.01, 0.81). In contrast, two UK epidemiological studies have found positive associations with tea or flavonols. Hertog *et al.* (1997) reported that flavonol intake was positively associated with CHD, while Woodward and Tunstall-Pedoe (1999), in the Scottish Heart Study, revealed a modest positive relationship between tea consumption and all-cause mortality, including CHD. However, on closer examination, these studies failed to control for confounders associated with tea consumption and CHD risk, for example, lower socio-economic status, long-term smoking and higher dietary fat intakes.

While associations cannot prove cause and effect, particularly where other dietary components are likely to be at work, the case for black tea was strengthened by the existence of experimental data suggesting a plausible mechanism. Phenolics, such as polyphenols and the sub-

group flavonoids, are powerful antioxidants capable of impacting favourably on CHD risk factors. Flavonoids are shown to prevent the oxidation of LDL (Davies *et al.*, 2003), reduce clotting and improve coronary vasodilation (Mojžišová and Kuchta, 2001); while plant polyphenols, such as those in tea and cocoa, increase plasma antioxidant levels (Weisburger, 2001). Black tea appears to have a greater impact on *ex vivo* lipoprotein oxidation than green tea (Hodgson *et al.*, 2000). It has been hypothesized that manganese in black tea could impact positively on heart disease risk, via the role of manganese superoxide dismutase in supporting cardiac muscle function and attenuating lipid peroxidation. One study examined the effect of tea drinking on markers of Mn status, finding no significant association (Hope *et al.*, 2006).

Cancer

It has been suggested that plant antioxidants, such as those found in tea, red wine and cocoa, can help prevent and control cancer development. This has arisen from experimental (mainly animal) work on green tea catechins, although polyphenols in black tea are increasingly being studied in both animals and humans. Flavonoids may also exert other effects unrelated to their antioxidant capacity, for example, anti-inflammatory effects (Aneja *et al.*, 2004) and inhibition of tumorigenesis (Ju *et al.*, 2005).

Epidemiological and other evidence on black tea and cancer risk in adults were reviewed. Studies that examined only flavonoid intakes were excluded as these could potentially come from a variety of food sources. Also excluded was a paper reviewing tea and bladder cancer (Lu *et al.*, 1999) because it combined results from oolong, black and green teas. In all, 26 studies were located and are described in Table 3.

In cancer sites other than colorectal cancer, the number of studies including data on black tea were extremely limited and, at times, conflicting. Thus, it is not possible to draw conclusions until further human studies are published. Prospective studies relating to colorectal cancers were more numerous and indicated either no relationship or a protective effect of tea at mean intakes of 1.5 cups per day or more. However, other authors dispute a relationship between tea and colorectal cancer. Arab and Il'yasova, 2003, reviewed 30 studies (most of which were included in our review) and suggested that differences in food habits, lifestyle, heredity, age, gender and environment made the data on colorectal cancer difficult to interpret. They report that, in some studies, confounding factors created more variation in cancer outcomes than the tea consumption itself. Studies from the Far East and Italy have attempted to correct for any effects of confounders but the numbers of black tea consumers in these were too low to be of use here (Tavani *et al.*, 1997; Inoue *et al.*, 1998). Only two studies implied harm and these were Far Eastern studies where black tea consumption was associated with an *increase* in colorectal

Table 2 Tea and coronary heart disease

Study	Subjects/population	Methods	Outcomes
<i>Cohort studies</i>			
Sesso et al. (2003)	Women free of CVD ($n = 38\,445$)	Prospective study; FFQ; Mean FU = 6.9 years.	Flavonoid intake not strongly associated with a reduced risk of CVD Mean flavonoid intake very modest; over 25% of sample consuming ≥ 1 cup tea/day RR of MI lower in tea drinkers with a daily intake of > 375 ml (RR: 0.57; 95% CI: 0.33, 0.98) than in non-tea drinkers Intake of flavonols and flavones inversely associated with non-fatal MI. Weaker inverse association with coronary death. Intakes of tea low
Geljeijnse et al. (2002)	Rotterdam; men ($n = 1836$), women ($n = 2971$); ≥ 55 years	Baseline semi-quantitative FFQ. Mean FU = 5.6 years	
Hirvonen et al. (2001)	Finland; male smokers $n = 25\,372$; 50–69 years	Baseline assessment with validated Q. Median FU = 6.1 years	
Arts et al. (2001a)	Netherlands; men $n = 806$, 65–84 years	Baseline DH with dietitian; FU = 10 years Outcome = mortality from CVD	Inverse association between tea intake and RR of mortality failed to reach significance ($P = 0.056$)
Arts et al. (2001b)	US; Post-menopausal women; $n = 34\,492$	Baseline FFQ, validated by 28 days UWR; mean FU = 13 years	Tea catechins not associated with CHD death Tea intake positively associated with healthy diet
Woodward and Tunstall-Pedoe (1999)	Scotland; men ($n = 5645$), women ($n = 5800$); 40–59 years	FFQ; mean FU = 7.7 years	Increasing tea consumption associated with coronary mortality and morbidity. Social class differences in tea consumption not controlled for
Hertog et al. (1997)	Wales; men; $n = 1900$; 45–59 years	Semi quantitative FFQ. Representative sub-sample ($n = 665$ men) completed 7 days WR	Tea consumption positively associated with IHD Average tea intake well in excess of mean UK intake
Rimm et al. (1996)	US; male health professionals; $n = 34\,789$; 40–75 years	Two 7 days Q kept by sub-sample ($n = 127$ men)	No strong inverse association between intake of flavonoids and total CHD. Contribution of tea to flavonoid intakes modest (25%)
Stensvold et al. (1992)	Norway; men ($n = 9856$), women ($n = 10\,233$); 35–49 years	Q; mean FU 12 years	Mortality rate higher (NS) among non-tea consumers or those drinking < 1 cup/day vs those drinking > 1 cup/day. 74% drank no tea at all
<i>Case control studies</i>			
<i>Study</i>			
Hakim et al. (2003)	Saudi Arabia; $n = 3430$; 30–70 years	Q	<i>Outcomes</i> Subjects drinking > 480 ml tea/day had lower prevalence of CHD vs non-tea drinkers ($P < 0.001$) even when risk factors accounted for. Dose-response between tea and lower CHD risk ($P < 0.001$) No significant association between tea and MI (OR = 1.0). No details on numbers of tea consumers.
Tavani et al. (2002)	Italy; 507 cases with first episode of non-fatal MI vs 478 controls admitted for acute diseases	Hospital based case control study; Q administered by investigators	
Sesso et al. (1999)	USA; men, $n = 680$; > 76 years	Outcome = MI	Tea drinking associated with lower risk of MI (OR for > 1 cup/day = 0.56; 95% CI 0.35–0.9).
Gramenzi et al. (1990)	Italy; women; $n = 936$; 21–69 years	Outcome = MI	No significant association between tea and MI
<i>Clinical trials evaluating effects on coronary risk factors</i>			
Davies et al. (2003)	USA; seven men, eight women; hypercholesterolaemic	x-over RCT; five servings of tea/day vs water/cafeine placebo on top of low fat diet	Total cholesterol reduced by 3.8% in tea group compared to placebo (with or without caffeine) LDL reduced by 7.5% ($P = 0.01$)
<i>Epidemiological data on coronary risk factors</i>			
<i>Study</i>			
Mennen et al. (2004)	France; men ($n = 1005$), women ($n = 1286$); 45–60 years	Methods x-sectional analysis of SU.VI.MAX Study; FU = 8 years	<i>Outcomes</i> RR for CVD in tea drinkers: Females 0.31 (95% CI, 0.14, 0.68) Males 1.38 (95% CI, 0.96, 2.00) Lower BP in women only ($P = 0.005$) Mean tHcy lower when > 2 cups tea/day drunk ($P < 0.05$) vs 1 cup/month
Jacques et al. (2001)	USA; $n = 1960$; 28–82 years	Analysis of Framingham Offspring Study prior to folic acid fortification	

Table 2 Continued

Study	Subjects/population	Methods	Outcomes
De Bree <i>et al.</i> (2001)	Netherlands; n = 3025; 20–65 years	x-sectional study on lifestyle impact on non-fasting tHcy	tHcy inversely associated with tea intake, especially in females
Rasmussen <i>et al.</i> (2000)	Denmark; women (n = 578) either young (25–30 years) or elderly (60–65 years)	Q on vitamin supplements; RBC measured in n = 204, additional UDR in n = 258	tHcy inversely associated with tea intake in elderly women (P < 0.05) but not young women
Woodward and Tunstall-Pedoe (1999)	Scotland; men (n = 5724), women (n = 5483); 40–59 years	Scottish Heart Health Study	Total cholesterol and HDL inversely associated with tea intake in men (P < 0.001) but not women
Nygard <i>et al.</i> (1997)	Norway; men (n = 7589), women (n = 8585); 40–67 years	Effect of beverage consumption on tHcy reported from baseline data	tHcy inversely associated with tea intake. Difference in tHcy between lowest and highest tea drinkers was 0.44 µmol/l. Total cholesterol inversely associated with tea intake. Difference was 0.22 mmol/l
Stensvold <i>et al.</i> (1992)	Norway; men (n = 9586), women (n = 10 233); 35–49 years	x-sectional study.	Inverse linear trend between tea intake and total cholesterol in men and women

Abbreviations: MI, myocardial infarction; FFQ, food frequency questionnaire; FU, follow-up; Q, simple dietary questionnaire; DH, diet history; UWR, unweighed dietary record; WR, weighed dietary record; RCT, randomized controlled trial; tHcy, serum homocysteine; CVD, coronary heart disease; OR, odds ratio; RR, relative risk; NS, non-significant; LDL, low density lipoproteins; HDL, high density lipoproteins; BP, blood pressure.

cancer (Kato *et al.*, 1990; Inoue *et al.*, 1998). However, Arab and Il'yasova, 2003, suggested that the tea consumers in these studies adopted other Western habits that may have increased their risk of colorectal cancer, for example, high-saturated fat, low-fibre diets.

The epidemiological evidence does not appear consistent enough to enable firm conclusions about associations (although it is evident that black tea is not harmful). The area of colorectal cancer, where there was moderate evidence for a slightly positive or no effect of black tea consumption, merits further study but requires better control of confounders to differentiate any associations of tea consumption from those linked to other lifestyle factors.

Dental health

The pathogenesis of dental caries involves the fermentation of carbohydrates by plaque bacteria, a byproduct of which is acid. This causes demineralization of tooth enamel over time (Kandelman, 1997). Fluoride – both systemic and topical – attenuates the risk of demineralization.

The tea plant naturally accumulates fluoride from the soil and can contain 196 µg per 2 g dry tea (around one teabag) (Panya-ngarm, 1988), although the fluoride content of a cup of tea can exceed this if fluoridated water is used during brewing. The FSA Total Diet Study (FSA, 2000) estimated that 1 l of tea (four to five cups) prepared with fluoridated water would make a significant contribution to fluoride intake, that is, 0.03 mg/kg body weight (2.2 mg/day for a 70 kg adult based on usual methods of preparation by consumers). If made from non-fluoridated water, the concentration would be 0.34–3.71 mg/l (mean = 1.5 mg/l) (Chan and Koh, 1996). Decaffeinated teas in America have been shown to contain higher levels of fluoride ranging from 1.01 to 5.2 mg/l (mean = 3.19 mg/l) (Chan and Koh, 1996), possibly due to the use of fluoridated water during the decaffeination process. A systematic review has suggested that fluoride from tea may benefit dental health (NHS CRD, 2000).

Studies that examined the impact of tea on the various stages of caries development (including those considering bactericidal effects on plaque bacteria) were included in this review. Five studies were found to fulfil the inclusion criteria, details of which are in Table 4. Studies were excluded if they were based on animal experiments or interventions in children, while others were excluded because they used green tea, or the semi-fermented oolong tea, as test substances. Catechin levels are higher in these types of teas and may have an anti-cariogenic effect by inhibiting oral bacterial growth (Hamilton-Miller, 2001).

Few trials in adults were found and they provided varying results which were limited by small sample sizes. The most positive evidence was reported by Zhang and Kashket, 1998, who suggested that brews of black tea suppress salivary amylase activity. This, in turn, can reduce the cariogenic potential of starch which acts as a slow-release source of fermentable carbohydrate. Other studies showed that black

Table 3 Tea and cancer

Study	Participants	Methods	Outcomes
<i>Multi-site</i> Arts et al. (2002)	US; women; n = 34 650; 55–69 years	Longitudinal FU from 1998 Baseline information on diet, medical history and lifestyle. Cancer cases obtained from registry	Catechins derived primarily from tea inversely associated with rectal cancer (RR across quartiles: 1.00, 0.56, 0.66 and 0.39; P = 0.02)
Zheng et al. (1996)	US; post-menopausal women; n = 35 369	Mailed survey of tea drinking and lifestyle factors collected at baseline; FU = 8 years; 2936 cancer cases.	Tea intake related to modest significantly lower incidence of combined cancers. No correlation with cancers of pancreas, lung or breast. For those drinking ≥ 2 cups/day, RR for digestive tract cancers = 0.68 (95% CI 0.47–0.98).
Goldbohm et al. (1996)	Netherlands; cohort study; men (58 279), women (62 573); 55–69 years	Self-administered baseline Q on diet and risk factors; FU = 4.3 years; FU Q used to classify black tea consumption in 2265 cancer cases vs 3500 random sub-group	No association between tea intake and risk of colorectal cancer. Inverse association with risk of stomach and lung cancers which became NS when smoking and diet taken into account.
<i>Lung Study</i> Mendilaharsu et al. (1998)	Uruguay; men; n = 855; cigarette smokers	<i>Methods</i> 427 lung cancer cases matched with 428 hospitalized controls	<i>Outcomes</i> Tea associated with lower risk of lung cancer. RR for intake of ≥ 2 cups/day = 0.34 (95% CI 0.14–0.84)
<i>Colorectal</i> Il'yasova et al. (2003a)	Moscow; 663 cases vs 323 controls	Q on history of tea consumption. Included three measures of tea exposure (beverage, tea concentrate, dry tea)	Tea associated with lower risk of rectal cancer. Dose-response with higher concentrations of tea related to stronger associations
Il'yasova et al. (2003b) Su and Arab (2002)	US; 630 cases vs 1040 matched controls US; NHANES I & NHEFS; Period I: 2359 tea consumers vs 6498 non-tea. Period II: 7656 tea consumers vs 4514 non-tea consumers	Lifestyle Q including tea consumption. Two cohort periods examined (I = baseline, II = first FU)	No significant association between tea and colon cancer Period I – no significant association with tea. Period II – RR for colon cancer = 0.57 (95% CI 0.42–0.78) for tea consumers compared with non-tea consumers, suggesting inverse association between colon cancer risk and habitual tea consumption
Woolcott et al. (2002)	Canada; 927 bladder cancer cases, 991 colon cancer cases, 875 rectal cancer cases, 2118 population controls US; 685 colon cancer cases vs 655 rectal cancer cases vs 2434 controls Sweden; women; n = 61 463 women. 460 incidents of colorectal cancer	Q at baseline	Tea consumption not related to any cancer site
Cerhan et al. (2001)	US; 1993 cases of colon cancer, 2410 population based controls	Mailed Q on tea consumption and other dietary data. Q at baseline; mean FU 9.6 years	No significant association between tea and colon or rectal cancer
Terry and Walk (2001)	Argentina; 190 colorectal cancer cases <80 years vs 393 controls with acute non-neoplasms	DR for previous two years on food and fluid intake, plus lifestyle information. Q at baseline.	No association between tea intake and colorectal cancers in age- or multivariate- adjusted models No significant association with tea consumption
Inoue et al. (1998)	Japan; n = 1706 digestive tract cancer cases vs 21 128 non-cancer outpatients; ≥ 40 years	Q on diet and lifestyle recalled when patient was healthy	No significant association between tea and colorectal cancer
Hartman et al. (1998)	Finland; middle-aged smokers; 11 colon cancer cases vs 83 rectal cancer	Q at baseline. Median FU = 9 years	Less than 10% of subjects drank black tea daily. Daily intake of black tea positively associated with colon cancer risk (OR = 1.59, CI = 1.06–2.37) No significant association between tea and incidence of rectal cancer.
Baron et al. (1997)	US; Patients with at least one recent large bowel adenoma	FFQ and colonoscopy at baseline. FU of colonoscopy at 4 years	Positive association between tea and colon cancer No association between regular tea intake and risk of recurrent colorectal adenomas

Table 3 Continued

Study	Participants	Methods	Outcomes
Tavani et al. (1997)	Italy; cancer patients; n = 985, 45–70 years	Q on diet and lifestyle at baseline	Tea consumption limited to < 1 cup/day. 20% of cancer cases and 17% of controls were tea drinkers. No impact of tea on risk of colon or rectal cancers
Fredriksson et al. (1995)	Sweden; 312 cases vs 623 controls; 30–75 years	Mailed FFQ	Daily tea intake of ≥ 2 cups/day gave a reduced Mantel-Haenszel OR ^a of 0.61
Baron et al. (1994)	Sweden; 352 colon cancer cases vs 217 rectal cancer cases vs 512 controls	Self-completed Q on diet, exercise, tobacco use, personal characteristics	No significant association between tea and colon cancer risk. OR for rectal cancer = 0.56 (95% CI, 0.34–0.9) for those drinking ≥ 2 cups/day compared with non-tea drinkers
Olsen and Kronborg (1993)	Denmark; 49 colorectal cancer cases, 171 with adenoma and 177 high risk vs 362 matched controls; 45–74 years	Blind telephone interview	No significant association between tea and risk of adenoma
Kato et al. (1990)	Japan; 221 colorectal cancer cases vs 525 adenoma cases vs 578 controls	Mailed FFQ on diet, drinking habits and lifestyle factors	Daily tea drinking associated with an increased risk of colon cancer (RR = 2.50, 95% CI: 1.19–5.26). No details about numbers of habitual tea drinkers
<i>Bladder/kidney</i> Bianchi et al. (2000)	USA; 1452 bladder cancer cases vs 406 kidney cancer cases vs 2434 controls	Q at baseline	No significant association between tea and risk of kidney cancer. Tea consumption of > 5 cups/day (> 90th percentile) linked to reduced severity of bladder cancer. No evidence of a dose–response
<i>Prostate</i> Ellison (2000)	Canada; 145 male incident cases from larger study of 3400; 50–84 years	Q at baseline	No significant difference in risk between subjects who drank > 500 ml per d vs non-tea consumers
Jain et al. (1998)	Canada; 617 incident cases vs 637 population controls	DH of beverage intake during personal interview.	Decrease in risk associated with tea intake of > 500 ml/day (OR = 0.7)
<i>Skin</i> Hakim et al. (2000); Hakim and Harris (2001)	USA; Anglo and Hispanic whites; n = 450 cases with skin squamous cell carcinoma vs 566 controls; ≥ 30 years	Structured interview on diet, skin characteristics, lifestyle, family history by trained interviewer	No significant association between occasional or regular tea intake and risk of skin cancer Controls more likely to report drinking strong tea vs cases (OR = 0.33; 95% CI, 0.12–0.87)
<i>Oral</i> Li et al. (1999)	China; 59 oral mucosa leukoplakia patients; 6 m trial.	Double blind RCT; treated group (n = 29) given 3 g/d mixed tea oral administration and topical treatment) vs control group (n = 30) given placebo and glycerine	In tea group, size of oral lesion decreased in 37.9% of cases and increased in 3.4%. In control group, size of oral lesion decreased in 10% of cases and increased in 6.7%
<i>Pancreas</i> Bueno de Mesquita et al. (1992)	Netherlands; 176 pancreatic carcinoma cases vs 487 controls	Interviewer administered Q on diet, lifestyle, intake of alcohol, tea and coffee	Lifetime consumption of tea not significantly associated with risk

^aNote – Mantel Haenszel OR is a statistical procedure that calculates odds ratios by stratification on sex, age and individual job-related physical activity.

Abbreviations: FFQ, food frequency questionnaire; FU, follow-up; Q, simple dietary questionnaire; DH, diet history; RCT, randomized controlled trial; DR, diet recall; RR, relative risk; OR, odds ratio; NS, non significant.

Table 4 Tea and dental health

Study	Participants	Methods	Outcomes
Simpson <i>et al.</i> (2001)	10 healthy subjects; 21–23 years	Tooth surface pH assessed <i>in situ</i> , 100 ml of tea solution (40 g tea leaves in 4 l deionised water)	Only small decreases in pH detected with high inter-subject variation. Resting pH restored within approximately 2 min. No impact of tea.
Lingstrom <i>et al.</i> (2000)	10 healthy subjects	Crossover RCT varying number of times rinsed with tea (5 vs 10 times/day) with water as control	Rinsing with black tea infusion 10 times/day resulted in attenuated fall in pH ($P < 0.05$ or < 0.01), lower plaque index ($P < 0.05$), higher fluoride concentrations in plaque and saliva vs water.
Zhang and Kashket (1998)	15 healthy subjects	Consumption of salt crackers followed by rinse with black tea vs green tea vs tap water. Maltose release monitored	Black tea infusion significantly more effective than green tea
Rasheed and Haider (1998)	40 patients with caries (28 male, 12 female), 18–45 years	Bacteria isolated from samples of dental plaque and incubated with discs impregnated with tea extracts (black and green) and three designated antibiotics	No impact of black tea extracts on any types of bacteria
Attin <i>et al.</i> (1995)	30 healthy subjects	3 days x-over trial rinsing with black tea vs Meridol (positive control) vs tap water (negative control) 3 times/day after meals. Plaque surface area tested. No oral hygiene during trial	Decrease in plaque surface area after rinsing with Meridol (plaque score 15%) but no difference seen with tea or water (plaque score 22–24%). Tea ineffective against plaque.

Abbreviation: RCT, randomized controlled trial.

tea decreased tooth surface pH (Simpson *et al.*, 2001), and suppressed the growth and virulence of periodontal pathogens *in vitro* (Wei and Wu, 2001). Studies that tested the impact of black tea on plaque demonstrated no significant fall in pH or lowered plaque index except when used as a rinse 10 times per day. It was suggested that the anti-cariogenic properties of black tea were most likely mediated by its contribution to fluoride intakes, rather than as a plaque inhibitor. No studies indicated that tea was detrimental to adult dental health, although there were no specific studies on the effects of sweetened tea. A study on sweetened liquids showed that seven exposures per day did not result in net demineralization when fluoride was present so this may indicate that sweetened tea is unlikely to be harmful to dental health when consumed at current levels (Duggal *et al.*, 2001).

Bone health

There have been suggestions that bone mineral density (BMD) may be influenced by chemical compounds in tea such as caffeine, fluoride and phytoestrogens. Studies looking at BMD, fractures and black tea consumption were searched. This yielded five epidemiological studies that met the inclusion criteria, details of which are in Table 5. Studies that looked at the effects of caffeine in isolation, rather than as a component of black tea were excluded as were studies on tea consumption and BMD in the Far East (as these covered a variety of teas).

The available evidence suggested that black tea consumption had a moderately positive effect on BMD, particularly in older women. There was a significant increase in BMD with higher levels of tea consumption (four or more cups per day) (Chen *et al.*, 2003). Black tea was also identified as an independent protective factor for the risk of hip fractures in men in the Mediterranean Osteoporosis Study (Johnell *et al.*, 1995; Kanis *et al.*, 1999; Hegarty *et al.*, 2000) reported that this effect was independent of the addition of milk to tea. In the UK, black tea consumption increases the overall calcium intake of middle-aged women by around 3% of the Reference Nutrient Intake due to the routine addition of milk (Harland, 2004).

Impact of caffeine

Data based on 400 samples of tea from family homes, workplaces and retail outlets from 10 areas across the UK suggest that the caffeine content of an average cup of tea is 17 mg/100 ml (40 mg per 235 ml cup with a range of 1–90 mg) (FSA, 2004). In comparison, coffee supplies 75–100 mg per cup (FSA, 2001b). There is controversy about the effects of caffeine on health. Some authors claim that excessive intakes of caffeine are related to hypertension, dehydration, anxiety, insomnia and birth defects (Green and Suls, 1996; Neuhauser-Berthold *et al.*, 1997; Nuriminen *et al.*, 1999; FSA, 2001b; Smith, 2002). Others suggest positive effects on cognitive performance, physical endurance, fatigue and alertness at intakes of 60–400 mg caffeine per

Table 5 Tea and bone health

Study	Participants	Methods	Outcomes
Chen <i>et al.</i> (2003)	US; women; <i>n</i> = 91 465; 50–59 years	Structured lifestyle Q, and history of fractures from medical records. BMD measured in sub-sample (<i>n</i> = 4979)	BMD positively correlated with tea drinking (<i>P</i> < 0.05) No significant association between tea drinking and risk of fractures at hip and forearm/wrist Only 9% of sample drank black tea. No significant difference in BMD between drinkers of different types of tea. Lumbar spine BMD higher in subjects with habitual tea consumption of 6–10 years. BMD in general higher in those with habitual tea consumption > 10 years
Wu <i>et al.</i> (2002)	Taiwan; men (<i>n</i> = 497), women (<i>n</i> = 540); ≥ 30 years	Structured Q on lifestyle including tea consumption (green vs oolong vs black tea). <i>N</i> = 502 habitual tea drinkers. BMD measured	Tea drinkers had significantly greater (5%) mean BMD measurements, adjusted for age, BMI and smoking Multivariate analysis showed that low consumption of black tea remained independent risk factor in men and women
Hegarty <i>et al.</i> (2000)	UK; women; <i>n</i> = 1256; 65–76 years	Self-administered Q. Sample classified into tea (90%) and non-tea drinkers (10%)	
1. Kanis <i>et al.</i> (1999) 2. Johnell <i>et al.</i> (1995)	Europe multi-centre; <i>n</i> = 730; > 50 years with hip fracture	Lifestyle Q. Sample compared with matched healthy controls	

Abbreviations: Q, simple dietary questionnaire; BMD, bone mineral density.

day (Warburton, 1995; Graham, 2001; Smith, 2002). The majority of adverse studies have considered caffeine alone, or in coffee, and have used experimental intakes far in excess of what would be reasonably ingested (i.e. 300–600 mg per day equating to 9–18 average cups of tea in a single bolus). We examined studies addressing the impact of caffeine from tea on mood, performance and hydration.

Mood and mental performance

Six studies were reviewed and are shown in Table 6. The effects of tea were in a positive or neutral direction overall, although the low sample sizes must be taken into account. Black tea ingestion seemed to produce a rapid increase in alertness and self-reported improvements in mood. The capacity to process information was also increased, while adverse effects on sleep duration or quality were not evident. When taken in regular amounts throughout the day, black tea appeared to prevent the diurnal pattern of performance reduction (Hindmarch *et al.*, 1998). As tea is not a high-caffeine drink, factors other than caffeine may be influencing these results, for example, a specific psychological response to tea drinking or other constituents in tea (Hindmarch *et al.*, 1998; Quinlan *et al.*, 2000). One example is an amino acid found in tea (theanine) which could act as a neurotransmitter. A study in rats found that theanine modulated serotonin and dopamine levels and appeared to improve memory and learning ability (Unno *et al.*, 1999). Work on humans is needed to confirm this finding.

Hydration

Fluid balance is vital for physical and mental performance. The National Drinks Survey, 2003, suggests that tea contributes significantly to fluid intakes, particularly in those aged 65 years and over where it represents 85% of beverage consumption. It is a common perception is that caffeine-containing drinks cause a net loss in fluid and may lead to dehydration. Again, many of the studies investigating this have used high doses of caffeine, often as a bolus. When caffeine is given in this way, there is indeed evidence of a diuretic effect but this is not relevant to normal use of caffeine-containing beverages where the caffeine would be consumed with 200–250 ml of fluid.

An extensive review of the scientific literature by Maughan and Griffin (2001), attempted to separate out those studies using ‘experimental’ caffeine doses from those considering ‘real life’ consumption. They concluded that ‘there is no evidence base for the assumption that all caffeine-containing drinks should be avoided in situations where fluid balance is, or might become, precarious’. It was found that tea consumption did not produce a diuretic effect unless the amount of tea consumed at one sitting contained more than 300 mg of caffeine (equivalent to six or seven cups of tea).

This position was confirmed by a study (Scott *et al.*, 2004) that compared regular tea consumers with non-consumers in

Table 6 Tea, mood and cognitive performance

Study	Participants	Methods	Outcomes
Scott <i>et al.</i> (2004)	Expedition members (9 male, 4 female) at Mount Everest base camp.	Tea vs non-tea control in 2 × 24-h interventions. No other caffeinated beverages or alcohol taken during study	Subjects reported reduced fatigue when tea was included in diet ($P = 0.005$)
Hindmarch <i>et al.</i> (2000)	30 healthy subjects	RCT; 5-way x-over design. Equal volumes of tea (37.5 mg or 75 mg caffeine) vs coffee (75 mg or 150 mg caffeine) vs water.	Day-long consumption of tea improved cognitive and psychomotor function. Caffeinated beverages had a dose dependent inverse effect on all aspects of sleep quality ($P < 0.001$).
Quinlan <i>et al.</i> (2000)	Study 1, $n = 17$ Study 2, $n = 15$ Overnight caffeine abstinence	Performance (psychomotor and cognitive) tested and sleep quality monitored RCT; x-over design to manipulate caffeine exposure: 1. Tea or coffee prepared at different strengths vs water vs no drink controls 2. Caffeine level alone manipulated	Tea group showed mild autonomic stimulation, elevation in mood, increased systolic and diastolic BP, and skin conductance vs water. Also lowered HR and skin temp vs water.
Stephoe and Wardle (1999)	18 male and 31 females from two stressful occupations	Participants completed daily records of drink consumption alongside ratings of anxious and positive moods for 8 weeks	Associations between beverage and mood inconsistent. Tea not consistently related to mood across entire sample. Women (not men) who enjoyed high social support at work felt more relaxed when tea was drunk
Hindmarch <i>et al.</i> (1998)	19 healthy subjects	Five-way x-over design. Caffeine alone (100 mg) vs water vs tea vs coffee (containing 100 mg caffeine)	Consumption of tea vs water associated with transient improvements in performance (alertness, information processing capacity)
Quinlan <i>et al.</i> (1997)	16 healthy caffeine-withdrawn subjects	x-over design: 400 ml hot tea or coffee consumed with/without 100 mg caffeine and milk vs water control. Outcomes = BP, skin conductance and temp, mood	Mood improved and anxiety decreased when caffeine and/or milk added to hot beverages. Tea related to increase in skin temp

Abbreviations: RCT, randomized controlled trial; BP, blood pressure.

a crossover study of fluid balance during extreme physiological stress. Participants were members of an expedition at Mount Everest Base Camp. Even when tea was drunk at high altitude, where the risk of dehydration is considerable, there was no evidence that tea produced a diuretic effect when consumed by habitual tea drinkers.

It would appear that a moderate intake of caffeine from tea is not harmful and could be helpful. However, it is acknowledged that the upper extremes of consumption could pose some risk. Nawrot *et al.*, 2003, have suggested that a safe maximum daily caffeine intake is 300 mg for pregnant women and 400 mg for other adults. Caffeine consumption for most tea drinkers in the UK is within the range of 300–400 mg/day, based on the available data on mean intakes (NDNS, 2002). While the Food Standards Agency (FSA) promotes the 300 mg/day limit for pregnant women (FSA, 2001a), there is currently no official advice on daily caffeine intakes for the rest of the population.

Iron status

It has been suggested that phenolic compounds in black tea could have an adverse effect on iron uptake in the diet, particularly in vulnerable groups such as children, elderly, pregnant women and those with low iron stores. A systematic review described 35 studies (published 1980–2002) on the impact of tea drinking on iron status in the UK (Nelson and Poulter, 2004). The authors concluded that, while tea drinking limited the absorption of non-haem iron from the diet, there was insufficient evidence to conclude what effect this would have on indicators of overall iron status. Whether or not milk was added made little difference to the findings. It was suggested that healthy people with a minimal risk of iron deficiency had no cause to restrict tea consumption while, for groups at risk of iron deficiency, tea drinking should be avoided at mealtimes.

An earlier review on tea consumption and iron status (Temme and Van Hodonck, 2002) concluded that tea drinking did not pose a risk to iron status in Western populations as the overall risk of iron deficiency is low. Both of these reviews included studies on children as well as on adults. To be included in the present review, the conclusions for adults had to be clearly differentiated and this was indeed the case. A Medline search revealed no additional studies on this topic up to September 2004.

Discussion

A summary of the results from this review is given in Table 7. Evidence for positive effects were found to relate to consumption of black tea, most consistently in the case of CHD. It is likely that the mechanism for this involves the antioxidant properties of tea polyphenols as these can protect cells from oxidation by free radicals (Mojžišová and Kuchta, 2001). Certainly the majority of human studies

Table 7 Summary of results of systematic review of the effect of black tea on health

Disease	Strength and amount of evidence in humans	References
Coronary heart disease	<p>Strong evidence from meta-analysis and cohort studies concerning a reduction in MI. Supported by evidence from epidemiology, case control studies and one RCT.</p> <p>A weak positive association found in two studies (Woodward and Tunstall-Pedoe (1999); Hertog <i>et al.</i> (1997)) but control of confounding factors, for example, social class, appeared inadequate. Moderate evidence for a slightly positive or no effect of black tea consumption on colorectal cancer.</p>	<p>Arts <i>et al.</i> (2001a); Davies <i>et al.</i> (2003); Geleijnse <i>et al.</i> (2002); Hakim <i>et al.</i> (2003); Hirvonen <i>et al.</i> (2001); Huxley and Neil (2003); Jacques <i>et al.</i> (2001); Mennen <i>et al.</i> (2004); Peters <i>et al.</i> (2001); Rasmussen <i>et al.</i> (2000); Sesso <i>et al.</i> (1999).</p>
Colorectal cancer ^a	<p>A weak positive association found in 4 studies with various types of cancers and, in one case, an unknown number of people consuming tea. Weak evidence suggesting increased mood and improved cognitive performance when black tea consumed. Numbers in studies were low.</p> <p>Evidence that tea had no diuretic effect unless the caffeine content of tea consumed at one sitting exceeded 300 mg. No evidence that tea was dehydrating at altitude. Little evidence to support effect of tea on plaque inhibition. Evidence supporting the contribution of tea to fluoride intakes and, thus, theoretical protection against caries. Moderate evidence suggestive of a positive effect of black tea consumption on bone mineral density. Low number of studies.</p>	<p>Arts <i>et al.</i> (2002); Baron <i>et al.</i> (1997); Cerhan <i>et al.</i> (2001); Goldbohm <i>et al.</i> (1996); Il'yasova <i>et al.</i> (2003a); Il'yasova <i>et al.</i> (2003b); Munoz <i>et al.</i> (1998); Olsen and Kronborg (1993); Slattery <i>et al.</i> (1999); Terry and Wolk (2001); Woolcott <i>et al.</i> (2002). Hartman <i>et al.</i> (1998); Kato <i>et al.</i> (1990); Su and Arab (2001); Baron <i>et al.</i> (1994). Scott <i>et al.</i> (2004); Hindmarch <i>et al.</i> (2000); Quinlan <i>et al.</i> (2000); Steptoe and Wardle (1999); Hindmarch <i>et al.</i> (1998). Maughan and Griffin (2001)</p>
Mood and cognitive performance Hydration	<p>Evidence that tea had no diuretic effect unless the caffeine content of tea consumed at one sitting exceeded 300 mg. No evidence that tea was dehydrating at altitude. Little evidence to support effect of tea on plaque inhibition. Evidence supporting the contribution of tea to fluoride intakes and, thus, theoretical protection against caries. Moderate evidence suggestive of a positive effect of black tea consumption on bone mineral density. Low number of studies.</p>	<p>Scott <i>et al.</i> (2004). Lingstrom <i>et al.</i> (2000) FSA (2000); NHS CRD (2000).</p>
Dental health	<p>Little evidence to support effect of tea on plaque inhibition. Evidence supporting the contribution of tea to fluoride intakes and, thus, theoretical protection against caries. Moderate evidence suggestive of a positive effect of black tea consumption on bone mineral density. Low number of studies.</p>	<p>Hegarty <i>et al.</i> (2000); Chen <i>et al.</i> (2003).</p>
Bone health	<p>Moderate evidence suggestive of a positive effect of black tea consumption on bone mineral density. Low number of studies.</p>	<p>Hegarty <i>et al.</i> (2000); Chen <i>et al.</i> (2003).</p>

^aNote that there are an insufficient number of studies to make any meaningful comment on cancer risk or protection, related to black tea consumption, for the other sites reviewed (lung, bladder/kidney, prostate, skin, oral and pancreas).

supported this interpretation (Rimm *et al.*, 1996; Hirvonen *et al.*, 2001; Arts *et al.*, 2001a), while work on animals indicated that black tea improved plasma lipid profiles and reduced the oxidation of LDL and VLDL following a high-cholesterol diet (Vinson and Dabbagh, 1998).

For cancer, benefits relating to tea consumption were far less clear. Experimental studies have certainly shown benefits for tea components but these have not been mirrored in the epidemiological data. The area of colorectal cancer has received the most attention and there is a growing body of mechanistic and animal work. It has been postulated that tea polyphenols act in the gastrointestinal tract by modulating the composition of the gut microflora (Weisburger and Chung, 2002). A high content of clostridia and a low percentage of bifidobacteria have been observed in the intestinal microflora of patients with colon cancer (Siddiqui *et al.*, 2004). Animal studies on green tea show that polyphenols selectively inhibit the growth of clostridia and promote bifidobacteria colonisation, leading to a drop in faecal pH (Yamamoto *et al.*, 1997). As yet, no studies of this type have been carried out using black tea in humans. Other proposed mechanisms include roles for tea flavonoids and other polyphenols in (a) protecting colonic cell membranes from free radical damage; (b) regulating cell growth and apoptosis; (c) promoting detoxifying enzymes (Weisburger and Chung, 2002).

While tea did not appear to be harmful and plausible mechanisms for cancer prevention existed, there was insufficient evidence from human research as yet to claim benefits in relation to cancer prevention. Reasons for this may be the high doses of tea components used experimentally, insufficient control of confounders in epidemiological research and small samples sizes (i.e. 60–90 tea consumers per study). Epidemiological data that suggest tea consumption contributes to cancer prevention do exist, however, these failed to differentiate between green, black or oolong tea and would not have met the inclusion criteria for this review. Studies of colorectal cancer suggested either a slightly positive effect or null effect; although an important review concluded that data published before 2002 were not sufficiently persuasive (Arab and Il'yasova, 2003). Future epidemiological studies need larger numbers of black tea consumers and better control of confounders to throw further light on this area.

The number of studies on bone health and dental caries was small and indicated a positive effect of tea, less convincingly in the case of dental health due to the lack of large human studies. For bone health, several suggestions have been put forward concerning likely mechanisms. These include the contribution of tea to dietary fluoride, which could alleviate osteoporotic progression, (Hillier *et al.*, 2000), the impact of flavonoids (including phytoestrogens) on bone mineral content, inhibition of bone resorption by tea extracts (Delaisse *et al.*, 1986), and involvement in bone mineral metabolism (Zeyuan *et al.*, 1998). These may work

independently or together, but certainly require further investigation.

Turning to areas where tea consumption could be potentially harmful, we found no consistent evidence that normal tea drinking impacted adversely on mood, mental performance, hydration or iron status in Western populations. In the case of mental performance, five out of the six studies demonstrated positive effects of tea, although sample sizes were low. Some risks to iron status and hydration were apparent at high levels of consumption and, based on estimates of healthy caffeine consumption; it would be wise to encourage intakes of tea to remain below eight cups per day. For high-caffeine beverages, this figure would be lower.

One challenge in interpreting the evidence concerning tea and health is the diversity of the beverage as drunk by the population. In this review, studies were limited to black tea in Western countries to attempt to control for this. However, this leaves other sources of variation such as grams of dry tea per cup, brew time and temperature, tea growing conditions, processing and blending. Future studies would be more helpful if they defined tea preparation and consumption patterns, and attempted to control or adjust for confounders known to impact on disease. While the addition of milk to tea does not seem to interfere with flavonoid absorption or activity (NHS CRD, 2000; Hollman *et al.*, 2001), it is not clear if other factors do, for example, the frequency and timing of tea intake in relation to meals, the addition of sucrose or lemon, and variations in gut microflora.

Conclusion

It is always difficult to translate diverse scientific findings into public health messages, yet it is a logical step. Assuming a minimal variation in tea preparation between individuals, the evidence points towards an intake of at least three cups a day for CHD prevention, one to six cups per day for significant increases in plasma antioxidant capacity and less than eight cups of tea per day for the avoidance of adverse effects on hydration and iron status (to manage intakes of caffeine and phenolic compounds from tea). For cancer, bone health and dental health, there was insufficient evidence to make any recommendations about intakes.

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References

- Aneja R, Odoms K, Denenberg AG, Wong HR (2004). Theaflavin, a black tea extract, is a novel anti-inflammatory compound. *Crit Care Med* **32**, 2097–2103.
- Arab L, Il'yasova D (2003). The epidemiology of tea consumption and colorectal cancer incidence. *J Nutr* **133**, 3310S–3319S.
- Arts ICW, Hollman PCH, Feskens EJM, Bueno de Mesquita HB, Kromhout D (2001a). Catechin intake might explain the inverse relation between tea consumption and ischemic heart disease: the Zutphen Elderly Study. *Am J Clin Nutr* **74**, 227–232.
- Arts ICW, Jacobs Jr DR, Harnack LJ, Gross M, Folsom AR (2001b). Dietary catechins in relation to coronary heart disease death among postmenopausal women. *Epidemiology* **12**, 668–675.
- Arts ICW, Jacobs Jr DR, Gross M, Harnack LJ, Folsom AR (2002). Dietary catechins and cancer incidence among postmenopausal women: the Iowa Women's Health Study (United States). *Cancer Causes Control* **13**, 373–382.
- Attin T, Zimmermann C, Kielbassa AM, Hellwig E (1995). Plaque surface area after rinsing with a low-level fluoride-containing Darjeeling tea. *Eur J Oral Sci* **103**, 416–418.
- Baron JA, Gerhardsson de Verdier M, Ekblom A (1994). Coffee, tea, tobacco and cancer of the large bowel. *Cancer Epidemiol Biomarkers Prev* **3**, 565–570.
- Baron JA, Greenberg ER, Haile R, Mandel J, Sandler RS, Mott L (1997). Coffee and tea and the risk of recurrent colorectal adenomas. *Cancer Epidemiol Biomarkers Prev* **6**, 7–10.
- Bianchi GD, Cehan JR, Parker AS, Putnam SD, See WA, Lynch CF et al. (2000). Tea consumption and risk of bladder and kidney cancers in a population-based case-control study. *Am J Epidemiol* **151**, 377–383.
- Bueno de Mesquita HB, Maisonneuve P, Moerman PCJ, Runia S, Boyle P (1992). Lifetime consumption of alcoholic beverages, tea and coffee and exocrine carcinoma of the pancreas: a population based case study in the Netherlands. *Int J Cancer* **50**, 514–522.
- Cerhan JR, Putnam SD, Bianchi GD, Parker AS, Lynch CF, Cantor KP (2001). Tea consumption and risk of cancer of the colon and rectum. *Nutr Cancer* **41**, 33–40.
- Chan JT, Koh SH (1996). Fluoride content in caffeinated, decaffeinated and herbal teas. *Caries Research* **30**, 88–92.
- Chen Z, Petinger MB, Ritenbaugh C, LaCroix AZ, Robbins J, Caan BJ et al. (2003). Habitual tea consumption and risk of osteoporosis: a prospective study in the Women's Health Initiative Observational Cohort. *Am J Epidemiol* **158**, 772–781.
- Davies MJ, Judd JT, Baer DJ, Clevidence BA, Paul DR, Edwards AJ et al. (2003). Black tea consumption reduces total and LDL cholesterol in mildly hypercholesterolemic adults. *J Nutr* **133**, 3298S–3302S.
- de Bree A, Verschuren WM, Blom HJ, Kromhout D (2001). Lifestyle factors and plasma homocysteine concentrations in a general population sample. *Am J Epidemiol* **154**, 150–154.
- Delaisse JM, Eeckhout Y, Vaes G (1986). Inhibition of bone resorption in culture by (+) catechin. *Biochem Pharmacol* **35**, 3091–3094.
- Duffy SJ, Keane J, Holbrook M, Gokce N, Swerdloff PL, Frei B et al. (2001). Short- and long-term black tea consumption reverses endothelial dysfunction in patients with coronary artery disease. *Circulation* **104**, 151–156.
- Dufresne CJ, Farnworth ER (2001). A review of the latest findings on the health promotion properties of tea. *J Nutr Biochem* **12**, 404–421.
- Duggal MS, Toumba KJ, Amaechi BT, Kowash MB, Higham SM (2001). Enamel demineralization *in situ* with various frequencies of carbohydrate consumption with and without fluoride toothpaste. *J Dent Res* **80**, 1721–1724.
- Duthie GG (2000). Vitamin E and its antioxidant role in relation to other dietary components. In: Garrow JS, James WPT, Ralph A (eds). *Human Nutrition and Dietetics*. Churchill Livingstone: London. pp 226–236.
- Ellison LF (2000). Tea and other beverage consumption and prostate cancer risk; A Canadian retrospective cohort study. *Eur J Cancer Prev* **9**, 125–130.
- Food Standards Agency (2000). *Total Diet Study – Fluorine, Bromine and Iodine*. FSA Surveillance Unit: London.
- Food Standards Agency (2001a). Advice for pregnant women on caffeine consumption. www.food.gov.uk/news/pressreleases/2001/oct/caffeinepregnant (accessed 5/8/05).
- Food Standards Agency (2001b). Statement on the reproductive effects of caffeine. www.food.gov.uk/science/ouradvisors/toxicity/statements/cotstatements.
- Food Standards Agency (2004). Survey of caffeine levels in hot beverages. FSA Surveillance Unit.
- Fredriksson M, Hardell L, Bengtsson NO, Axelsson O (1995). Colon cancer and dietary habits—a case controlled study. *Int J Oncol* **7**, 133–141.
- Geleijnse JM, Launer LJ, Van der Kuip DA, Witteman J (2002). Inverse association of tea and flavonoid intakes with incident myocardial infarction: the Rotterdam Study. *Am J Clin Nutr* **75**, 880–886.
- Goldbohm RA, Hertog MG, Brants HA, van Poppel G, van der Brand PA (1996). Consumption of black tea and cancer risk: a prospective cohort study. *J Natl Cancer Inst* **88**, 93–100.
- Graham TE (2001). Caffeine and exercise: metabolism, endurance and performance. *Sports Med* **31**, 785–807.
- Gramenzi A, Gentile A, Fasoli M, Negri E, Parazzini F, La Vecchia C (1990). Association between certain foods and risk of acute myocardial infarction in women. *Br Med J* **300**, 771–773.
- Green PJ, Suls J (1996). The effects of caffeine on ambulatory blood pressure, heart rate, and mood in coffee drinkers. *J Behav Med* **19**, 111–128.
- Hakim IA, Harris RB, Weisgerber UM (2000). Tea intake and squamous cell carcinoma of the skin: Influence of type of tea beverages. *Cancer Epidemiol Bio Prev* **9**, 727–731.
- Hakim IA, Harris RB (2001). Joint effects of citrus peel use and black tea intake on squamous cell carcinoma of the skin. *BMC Dermatol* **1**, 3–14.
- Hakim IA, Alsaif MA, Alduwaihy M, Al-Rubeaan K, Al-Nuaim AR, Attas OS (2003). Tea consumption and the prevalence of coronary heart disease in Saudi adults: results from a Saudi national study. *Prev Med* **36**, 64–70.
- Hamilton-Miller JM (2001). Anti-cariogenic properties of tea (*Camellia sinensis*). *J Med Microbiol* **50**, 299–302.
- Harland J (2004). Personal Communication.
- Hartman TJ, Tangrea JA, Pietinen P, Malila N, Virtanen M, Taylor PR et al. (1998). Tea and coffee consumption and risk of colon and rectal cancer in middle-aged Finnish men. *Nutr Cancer* **31**, 41–48.
- Hegarty VM, May HM, Khaw KT (2000). Tea Drinking and bone mineral density in older women. *Am J Clin Nutr* **71**, 1003–1007.
- Henderson L, Gregory J, Swan G (2002). *National Diet and Nutrition Survey: adults aged 19 to 64 years*. FSA: London.
- Hertog MG, Sweetnam PM, Fehily AM, Elwood PC, Kromhout D (1997). Antioxidant flavonols and ischemic heart disease in a Welsh population of men: the Caerphilly Study. *Am J Clin Nutr* **65**, 1489–1494.
- Hillier S, Cooper C, Kellingray S, Russell G, Hughes H, Coggon D (2000). Fluoride in drinking water and risk of hip fracture in the UK: a case control study. *Lancet* **355**, 265–269.
- Hindmarch I, Quinlan PT, Moore KL, Parkin C (1998). The effects of black tea and other beverages on aspects of cognition and psychomotor performance. *Psychopharmacology* **139**, 230–238.
- Hindmarch I, Rigney U, Stanley N, Quinlan P, Rycroft J, Lane J (2000). A naturalistic investigation of the effects of day-long consumption of tea, coffee and water on alertness, sleep onset and sleep quality. *Psychopharmacology* **149**, 203–216.
- Hirata K, Shimada K, Watanabe H, Otsuka R, Tokai K, Yoshiyama M et al. (2004). Black tea increases coronary flow rate velocity reserve in healthy male subjects. *Am J Cardiol* **93**, 1384–1388.

- Hirvonen T, Pietinen P, Virtanen M, Ovaskainen ML, Hakkinen S, Albanes D et al. (2001). Intake of flavonols and flavones and risk of coronary heart disease in male smokers. *Epidemiology* **12**, 62–67.
- Hodgson JM, Puddey IB, Burke V, Croft KD (2006). Is reversal of endothelial dysfunction by tea related to flavonoid metabolism? *Br J Nutr* **95**, 14–17.
- Hodgson JM, Puddey IB, Croft KD, Burke V, Mori TA, Caccetta RA et al. (2000). Acute effects of ingestion of black and green tea on lipoprotein oxidation. *Am J Clin Nutr* **71**, 1103–1107.
- Hollman P, Tijburg LBM, Yang CS (1997). Bioavailability of flavonoids in tea. *Crit Rev Food Sc Nutr* **37**, 719–738.
- Hollman PCH, Van het Hof KH, Tijburg LBM, Katan MB (2001). Addition of milk does not affect the absorption of flavonols from tea in man. *Free Radic Res* **34**, 297–300.
- Hope SJ, Daniel K, Gleason KL, Comber S, Nelson M, Powell JJ (2006). Influence of tea drinking on manganese intake, manganese status and leucocyte expression of MnSOD and cytosolic aminopeptidase P. *Eur J Clin Nutr* **60**, 1–8.
- Huxley RR, Neil HA (2003). The relation between dietary flavonol intake and coronary heart disease mortality: a meta-analysis of prospective cohort studies. *Eur J Clin Nutr* **57**, 904–908.
- Il'yasova D, Arab L, Martinchik A, Sdvizhkov A, Urbanovich L, Weisgerber U (2003a). Black tea consumption and the risk of rectal cancer in Moscow population. *Ann Epidemiol* **13**, 405–411.
- Il'yasova D, Martin C, Sandler RS (2003b). Tea intake and risk of cancer in African-Americans and whites: North Carolina colon cancer study. *Cancer Causes Control* **14**, 767–772.
- Inoue M, Tajima K, Hirose K, Hamajima N, Takezaki T, Kuroishi T et al. (1998). Tea and coffee consumption and the risk of digestive tract cancers: data from a comparative case referent study in Japan. *Cancer Causes Control* **9**, 209–216.
- Jacques PF, Bostom AG, Wilson PW, Rich S, Rosenberg IH, Selhub J (2001). Determinants of plasma total homocysteine concentration in the Framingham Offspring cohort. *Am J Clin Nutr* **73**, 613–621.
- Jain MG, Hislop GT, Howe GR, Burch JD, Ghadirian P (1998). Alcohol and other beverage use and prostate cancer among Canadian men. *Int J Cancer* **78** (6), 707–711.
- Johnell O, Gullberg B, Kanis JA, Allander E, Elffors L et al. (1995). Risk factors for hip fracture in European women: the MEDOS study (Mediterranean Osteoporosis Study). *J Bone Miner Res* **10**, 1802–1815.
- Ju J, Hong J, Zhou JN, Pan Z, Bose M, Liao J et al. (2005). Inhibition of intestinal tumorigenesis in Apcmin/+ mice by (-)-epigallocatechin-3-gallate, the major catechin in green tea. *Cancer Res* **65**, 10623–10631.
- Kandelman D (1997). Sugar, alternative sweeteners and meal frequency in relation to caries prevention, new perspectives. *Br J Nutr* **77** (Suppl 1), S121–S128.
- Kanis J, Johnell O, Gullberg B, Allander E, Elffors L, Ranstam J et al. (1999). Risk factors for hip fracture in men from Southern Europe: the MEDOS study (Mediterranean Osteoporosis Study). *Osteoporosis Int* **9**, 45–54.
- Kato I, Tominaga S, Matsuura A, Yoshii Y, Shirai M, Kobayashi S (1990). A comparative case-control study of colorectal cancer and adenoma. *Japan J Cancer Res* **81**, 1101–1108.
- Kim CH, Moon SK (2005). Epigallocatechin-3-gallate causes the p21/WAF1-mediated G(1)-phase arrest of cell cycle and inhibits matrix metalloproteinase-9 expression in TNF-alpha-induced vascular smooth muscle cells. *Arch Biochem Biophys* **435**, 264–272.
- Langley-Evans SC (2000). Consumption of black tea elicits an increase in plasma antioxidant potential in humans. *Int J Food Sci Nutr* **51** (5), 309–315.
- Leenen R, Roodenburg AJ, Tijburg LB, Wiseman SA (2000). A single dose of tea with or without milk increases plasma antioxidant activity in humans. *Eur J Clin Nutr* **54**, 87–92.
- Li N, Sun Z, Han C, Chen J (1999). The chemoprotective effects of tea on human oral precancerous mucosa lesions. *Proc Soc Ex Biol Med* **220**, 218–224.
- Lin YL, Tsai SH, Lin-Shiau SY, Ho CT, Lin JK (1999). Theaflavin-3,3'-digallate from black tea blocks the nitric oxide synthase by down-regulating the activation of NF-kappaB in macrophages. *Eur J Pharmacol* **367**, 379–388.
- Lingstrom P, Wu CD, Wefel JS (2000). *In vivo* effects of black tea infusion on dental Plaque. *J Dent Res* **79**, 593 (Abstract No 3600).
- Lu CM, Lan SJ, Lee YH, Huang JK, Huang CH, Hsieh CC (1999). Tea consumption: fluid intake and bladder cancer risk in Southern Taiwan. *Urology* **54**, 823–828.
- Ludwig A, Lorenz M, Grimbo N, Steinle F, Meiners S, Bartsch C et al. (2004). The tea flavonoids epigallocatechin-3-gallate reduces cytokine-induced VCAM-1 expression and monocyte adhesion to endothelial cells. *Biochem Biophys Res Commun* **316**, 659–665.
- Maughan R, Griffin J (2001). *Tea Drinking And Fluid Balance: A Review*. Brooke Bond Tea Company: UK.
- Mendilaharsu M, De Stefani E, Deneo-Pellegrini H, Carzoglio JC, Ro A (1998). Consumption of tea and coffee and the risk of lung cancer in cigarette smoking men: a case control study in Uruguay. *Lung Cancer* **19**, 101–107.
- Mennen L, Saphino D, de Bree A, Arnault N, Bertrais S, Galan P et al. (2004). Consumption of foods rich in flavonoids is related to a decreased cardiovascular risk in apparently healthy French women. *J Nutr* **134**, 923–926.
- Mojžišová G, Kuchta M (2001). Dietary flavonoids and risk of coronary heart disease. *Physiol Res* **50**, 529–535.
- Mulder TP, van Platerink CJ, Wijnand Schuyt PJ, van Amelsvoort JM (2001). Analysis of theaflavins in biological fluids using liquid chromatography-electrospray mass spectrometry. *J Chromatogr Biomed Sci Appl* **760**, 271–279.
- Munoz SE, Navarro A, Lantieri MJ, Fabro ME, Peyrano MG, Ferraroni M et al. (1998). Alcohol, methylxanthine- containing beverages and colorectal cancer in Cordoba, Argentina. *Eur J Cancer Prev* **7**, 207–213.
- Naska A, Vasdekis VGS, Trichopoulou A, Friel S, Leonhäuser IU, Moreiras O et al. (2000). Fruit and vegetable availability among ten European countries: how does it compare with the 'five-a-day' recommendation? *Br J Nutr* **84**, 549–556.
- National Diet and Nutrition Survey (2002) Vol. 1. The Stationary Office: London.
- National Drinks Survey (2003): Taylor Nelson Sofres Ltd: London.
- Nawrot P, Jordan S, Eastwood J, Rotstein J, Hugenholtz A, Feeley M (2003). Effects of caffeine on human health. *Food Addit Contam* **20**, 1–30.
- Nelson M, Poulter J (2004). Impact of tea drinking on iron status in the UK: A review. *J Hum Nutr Diet* **17**, 43–54.
- Neuhauser-Berthold M, Beine S, Verwied SC, Luhrmann PM (1997). Coffee consumption and total body water homeostasis as measured by fluid balance and bioelectrical impedance analysis. *Ann Nutr Metab* **41**, 29–36.
- NHS CRD (2000). *A systematic Review of Public Water Fluoridation (CRD Report No 18)*. NHS Centre for Review and Dissemination, University of York: York, UK.
- Nurminen ML, Niittyen L, Korpela R, Vapaatalo H (1999). Coffee, caffeine and blood pressure: a critical review. *Eur J Clin Nutr* **53**, 831–839.
- Nygard O, Refsum H, Ueland PM, Stensvold I, Nordrehaug JE, Kvale G et al. (1997). Coffee consumption and plasma total homocysteine: The Hordaland Homocysteine Study. *Am J Clin Nutr* **65**, 136–143.
- Olsen J, Kronborg O (1993). Coffee, tobacco and alcohol as risk factors for cancer and adenoma off the large intestine. *Int J Epidemiol* **22**, 398–402.
- Panya-ngarm Y (1988). Fluoride in black tea. *CU Dental J* **11**, 43–52.
- Peters U, Poole C, Arab L (2001). Does tea affect cardiovascular disease? A meta-analysis. *Am J Epidemiol* **154**, 495–503.
- Quinlan P, Lane J, Aspinnall L (1997). Effects of hot tea, coffee and water ingestion on physiological responses and mood: the

- role of caffeine, water and beverage type. *Psychopharmacology* **134**, 164–173.
- Quinlan PT, Lane J, Moore KL, Aspen J, Rycroft JA, O'Brien DC (2000). The acute physiological and mood effects of tea and coffee: the role of caffeine level. *Pharmacol Biochem Behav* **66**, 19–28.
- Rasheed A, Haider M (1998). Antibacterial activity of *Camellia sinensis* extracts against dental caries. *Arch Pharm Res* **21**, 348–352.
- Rasmussen LB, Ovesen L, Bulow I, Knudsen N, Laurberg P, Perrild H (2000). Folate intake, lifestyle factors, and homocysteine concentrations in younger and older women. *Am J Clin Nutr* **72**, 1156–1163.
- Rietveld A, Wiseman S (2003). Antioxidant effects of tea: evidence from human clinical trials. *Am Soc Nutr Sci* **133**, 3285S–3292S.
- Rimm EB, Katan MB, Ascherio A, Stampfer MJ, Willett WC (1996). Relation between intake of flavonoids and risk for coronary heart disease in male health professionals. *Ann Intern Med* **125**, 384–389.
- Scott D, Rycroft JA, Aspen J, Chapman C, Brown B (2004). The effect of drinking tea at high altitude on hydration status and mood. *Eur J Appl Physiol* **91**, 493–498.
- Sesso HD, Gaziano JM, Buring JE, Hennekens CH (1999). Coffee and tea intake and the risk of myocardial infarction. *Am J Epidemiol* **149**, 162–167.
- Sesso HD, Gaziano JM, Liu S, Buring JE (2003). Flavonoid intake and the risk of cardiovascular disease in women. *Am J Clin Nutr* **77**, 1400–1408.
- Siddiqui IA, Afaq F, Adhami VM, Ahmad N, Mukhtar H (2004). Antioxidants of the beverage tea in promotion of human health. *Antioxid Redox Signal* **6**, 571–582.
- SIGN (Scottish Intercollegiate Guidelines Network) (2001). *SIGN 50 Guidelines*. Edinburgh www.sign.ac.uk(30/9/04).
- Simpson A, Shaw L, Smith AJ (2001). Tooth surface pH during drinking of black tea. *Br Dent J* **190**, 374–376.
- Slattery ML, Caan BJ, Anderson KE, Potter JD (1999). Intake of fluids and methylxanthine-containing beverages: association with colon cancer. *Int J Cancer* **81**, 199–204.
- Smith A (2002). Effects of caffeine on human behaviour. *Food Chem Toxicol* **40**, 1243–1255.
- Stensvold I, Tverdal A, Solvoll K, Foss OP (1992). Tea consumption. Relationship to cholesterol, blood pressure, and coronary and total mortality. *Prev Med* **21**, 546–553.
- Steptoe A, Wardle J (1999). Mood and drinking: a naturalistic diary study of alcohol, coffee and tea. *Psychopharmacology* **141**, 315–321.
- Su LJ, Arab L (2001). Tea consumption and the reduced risk of colon cancer- results from a national prospective cohort study. *Pub Health Nutr* **5**, 419–425.
- Tavani A, Pregnolato A, La Vecchia C, Negri E, Talamini R, Franceschi S (1997). Coffee and tea intake and risk of cancers of the colon and rectum: a study of 3530 cases and 7057 controls. *Int J Cancer* **73**, 193–197.
- Tavani A, Bertuzzi M, Negri E, Sorbara L, La Vecchia C (2002). Alcohol, smoking and coffee and risk of non-fatal acute myocardial infarction in Italy. *Eur J Epidemiol* **17**, 1131–1137.
- Temme EH, Van Hodonck PG (2002). Tea consumption and iron status. *Eur J Clin Nutr* **56**, 379–386.
- Terry P, Wolk A (2001). Tea consumption and the risk of colorectal cancer in Sweden. *Nutr Cancer* **39**, 176–179.
- Unno T, Suzuki Y, Kakuda T, Hayakawa T, Tsuge H (1999). Metabolism of theanine, gamma-glutamylethylamide in rats. *J Agric Food Chem* **47**, 1593–1596.
- Van het Hof KH, Wiseman SA, Yang CS, Tijburg LB (1999). Plasma and lipoprotein levels of tea catechins following repeated tea consumption. *Proc Soc Exp Biol Med* **220**, 203–209.
- Vinson JA, Dabbagh YA (1998). Effect of green and black tea supplementation on lipids, lipid oxidation and fibrinogen in hamster: mechanisms for the epidemiological benefits of tea drinking. *FEBS Lett* **433**, 44–46.
- Warburton DM (1995). Effects of caffeine on cognition and mood without caffeine abstinence. *Psychopharmacology* **119**, 66–70.
- Wei GX, Wu CD (2001). Black tea extract and tea polyphenols inhibit growth and virulence factors of periodontal pathogens. *J Dent Res* **80**, 73 (Abstract No. 304).
- Weisburger JH (2001). Chemoprotective effects of cocoa polyphenols on chronic diseases. *Exp Biol Med* **226**, 891–897.
- Weisburger JH, Chung FL (2002). Mechanisms of chronic disease causation by nutritional factors and tobacco products and their prevention by tea polyphenols. *Food Chem Toxicol* **40**, 1145–1154.
- Wiseman SA, Balentine DA, Frei B (1997). Antioxidants in tea. *Crit Rev Food Sci Nutr* **37**, 705–718.
- Woodward M, Tunstall-Pedoe H (1999). Coffee and tea consumption in the Scottish Heart Health Study follow up: conflicting relations with coronary risk factors, coronary disease, and all cause mortality. *J Epidemiol Comm Health* **53**, 481–487.
- Woolcott CG, King WD, Marrett LD (2002). Coffee and tea consumption and cancers of the bladder, colon and rectum. *Eur J Cancer Prev* **11**, 137–145.
- Wu CH, Yang YC, Yao WJ, Lu FH, Wu JS, Chang CJ (2002). Epidemiological evidence of increased bone mineral density in habitual tea drinkers. *Arch Intern Med* **162**, 1001–1006.
- Yamamoto T, Juneja LR, Chu D-C, Kim M (1997). *Chemistry and Applications of Green Tea*. CRC Press LLC: Boca Raton, USA.
- Zeyuan G, Bingying T, Xiaolin L, Jinming H, Yifeng C (1998). Effect of green tea and black tea of the metabolisms of mineral elements in old rats. *Biol Trace Elem Res* **65**, 75–86.
- Zhang J, Kashket S (1998). Inhibition of salivary amylase by green and black teas and their effects on the intraoral hydrolysis of starch. *Caries Res* **32**, 233–238.
- Zheng W, Doyle TJ, Kushi LH, Sellers TA, Hong CP, Folsom AR (1996). Tea consumption and cancer incidence in a prospective cohort study of post-menopausal women. *Am J Epidemiol* **144**, 75–182.

Appendix A

Search terms

Topic	Search terms
Cancer	'black tea and cancer' black tea and antioxidants' 'fluoride and bone cancer'
CHD	'black tea and heart disease', black tea and polyphenols', 'black tea and flavonoids' 'black tea and CHD', 'black tea and cholesterol',
Mood and cognitive performance	'tea and mood', tea and cognitive function', tea and sleep', 'tea and anxiety', 'theanine and mood'
Hydration and renal health	'tea and hydration', tea and kidney stones', 'tea and renal disease', 'beverages and kidney stones'
Iron status	'tea and iron', 'tea and anaemia'
Dental health	'tea and dental caries', tea and dental health', 'tea and teeth', 'tea and dental erosion', 'tea and fluoride'
Bone health	'tea and bone health', 'tea and bone mineral density', 'tea and osteoporosis'

Appendix B

Ranked levels of evidence adapted from SIGN literature grading system (SIGN, 2001).

- 1 RCT, Meta-analysis, systematic reviews of RCT.
 - 2 Systematic reviews of case control or cohort studies, and case control or cohort studies with a moderate to high probability that the relationship is causal.
 - 3 Non-RCT interventions, epidemiology and case reports.
 - 4 Expert opinion.
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