

Black tea and health

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Summary

Tea is the second most consumed beverage in the world after water. Health benefits have been associated with tea drinking, including a lower risk of coronary heart disease (CHD) and cancer, and protection against dental caries and bone loss. It is likely that these benefits relate to the high polyphenol content of tea and how these polyphenols are metabolised and used by the body. In contrast, concern has arisen about the impact of tea on hydration and iron status, and the role of tea as a source of caffeine. This article updates an earlier systematic review by including more recent published evidence on the potential role of black tea in human health. While it is clear from *in vitro* and animal research that tea polyphenols act as antioxidants and have a beneficial effect on many biochemical processes in the body via a range of complex mechanisms, findings from epidemiological studies and the few available human intervention studies have been contradictory. Reasons for this are explored, including the influence of lifestyle factors other than tea consumption on cancer or CHD risk. The clearest consistent evidence points to an association between tea consumption, in excess of three cups per day, and a reduced risk of myocardial infarction. More human research is needed to draw conclusions about cancer and other markers of CHD. There was no consistent evidence pointing to a detrimental effect of tea drinking on hydration, bone health or iron status. The caffeine content of tea was modest compared with other sources and was unlikely to have an adverse effect on health within an intake range of 1 to 8 cups of tea per day.

Keywords: caffeine, heart disease, hydration, polyphenol, tea

Introduction

Tea is a beverage made from the processed leaf of *Camellia sinensis*, a plant cultivated across the world in tropical and subtropical regions. Black, oolong, green and specialty teas all originate from the same plant, but owe their unique taste to differences in processing. While black tea, the most commonly consumed tea, is extensively oxidised before being processed, green tea

undergoes a short oxidation before being steamed. This suspends the action of the oxidative enzymes.

Market data show that 135 000 tons of tea was consumed in the UK in 2006/2007, 95% of which was black tea (International Tea Committee 2007). Oolong, green and other specialty teas (*e.g.* Earl Grey, lapsang souchong) made up the remainder. Herbal, or fruit tea, is not strictly tea as it originates from plants other than *Camellia sinensis*. The proper term is a herbal or fruit infusion.

Tea is the most commonly consumed beverage in the world, after water. It has been drunk in the UK for 350 years and in Asia for more than 4000 years. Data

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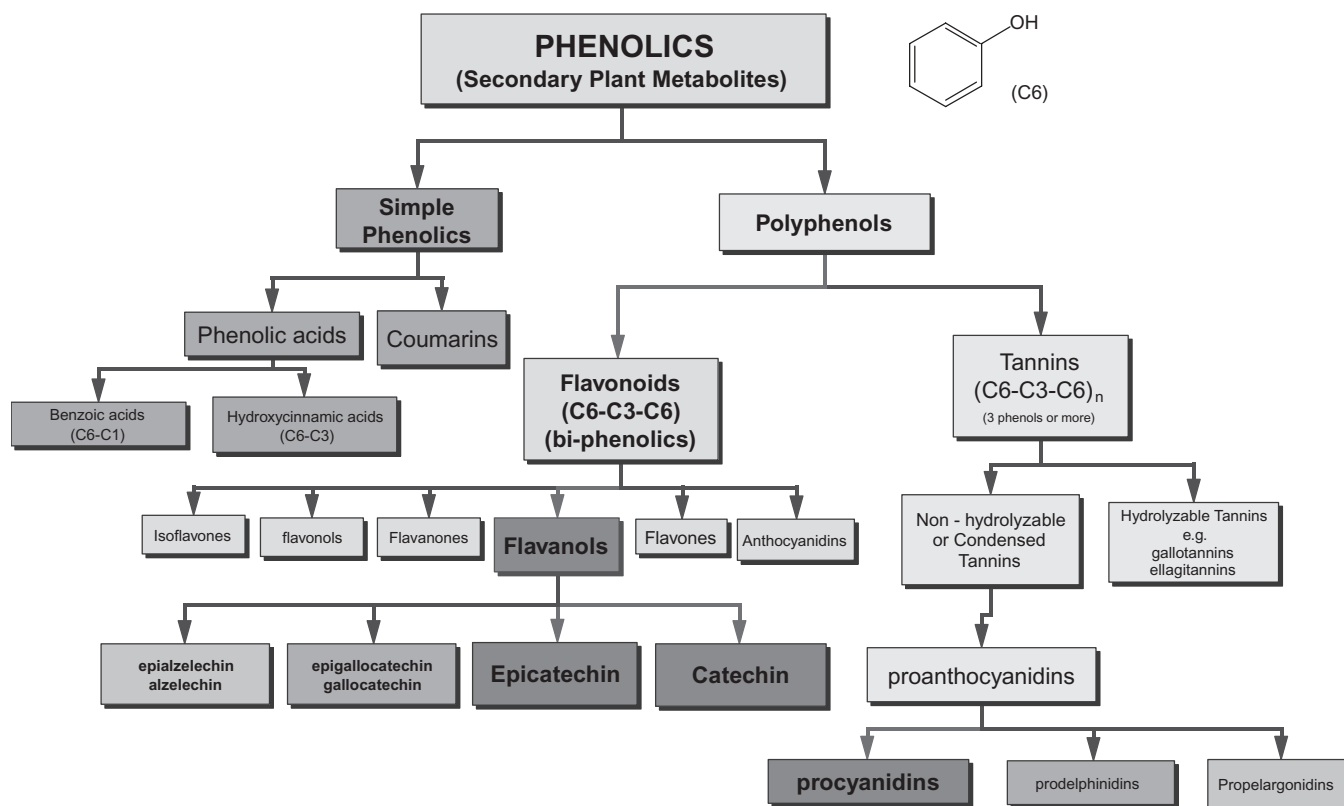


Figure 1 Flavonoids within the polyphenol family.

on over 2000 UK adults from the most recent National Diet and Nutrition Survey (NDNS) suggest that 77% of adults in the UK drink tea (Henderson *et al.* 2003). The mean consumption was 2.3 mugs (540 ml) per day, with older adults drinking more tea than younger adults (644 ml *vs.* 298 ml per day). Just under half of the tea drunk was unsweetened. Similar consumption figures were reported by the National Drinks Survey, an independent annual market research survey (Taylor Nelson Sofres 2007).

Polyphenols and tea

Tea is an important source of polyphenols, plant-derived antioxidants that are believed to explain some of the health benefits associated with fruits, vegetables, cocoa and red wine (Arts & Hollman 2005). The various polyphenols found in tea form part of the flavonoid group (see Fig. 1) and include catechin, epicatechin (EC), epigallocatechin (EGC) (collectively known as flavanol monomers), epicatechin gallate, epigallocatechin gallate (EGCg) (also called flavanol gallates), quercetin glycosides, theaflavins and thearubigins. Theaflavins and thearubigins are formed from flavanol

monomers and flavanol gallates during black tea manufacture. Table 1 compares the polyphenol content of black and green teas. While differences exist in the types of flavonoids present, due to the degree of oxidation during processing, it has been postulated that the total polyphenol content of green and black tea is similar (Stangl *et al.* 2006). An average cup of tea contains around 200 mg of total flavonoids per cup (Wiseman *et al.* 1997).

Bioavailability is an important issue as all flavonoids are poorly absorbed and, if bioactivity or intakes are also low, this can limit their usefulness in practice. Determining bioavailability is no easy task as little is known about the metabolic pathways of the myriad of flavonoids (over 6000 have been described). Also, the forms of flavonoids found in food can differ from those analysed in blood because of the rapid conjugation and metabolism that follows absorption. Gut bacteria metabolise flavonoids into other compounds that can then be absorbed via the gut circulatory system (Clifford & Brown 2006), adding another dimension. All of these factors can make measurements of flavonoids in blood, faeces or urine blunt markers of consumption or bioavailability (Williamson & Manach 2005).

Table 1 Polyphenol content of black and green tea (mg per 100 ml consumed)

Polyphenol	Black tea brewed [mean (range)]	Green tea brewed [mean (range)]	Black tea (decaffeinated) brewed [mean (range)]	Green tea (decaffeinated) brewed [mean (range)]
Epicatechin	2.33 (0.48–8.74)	8.47 (1.90–26.00)	0.49 (0.34–0.87)	6.16 (5.31–7.01)
Epigallocatechin	10.43 (0.29–31.04)	17.08 (1.00–54.40)	0.55 (0.36–1.01)	16.02 (15.56–16.48)
Catechin	1.52 (0.35–4.79)	2.73 (0.00–44.4)	NR	NR
Gallocatechin	1.26 (0.56–2.78)	NR	NR	NR
Theaflavins	1.58 (0.36–5.27)	0.05 (0.02–0.08)	0.35 (0.08–0.86)	0.12 (0.04–0.20)
Kaempferol [†]	1.34 (0.25–2.41)	1.42 (0.67–3.31)	1.25 (1.00–1.84)	1.00 (0.81–1.18)
Myricetin [†]	0.45 (0.17–0.90)	1.10 (0.52–1.60)	0.33 (0.26–0.49)	1.00 (0.89–1.11)
Quercetin [†]	2.07 (0.41–4.75)	2.69 (1.40–4.10)	2.84 (2.46–3.38)	2.77 (2.40–3.13)

Source: US Department of Agriculture (2003) (data for thearubigins were not presented).

[†]Not present in tea but found after hydrolysis of the naturally occurring glycosides.

NR, not reported.

Table 2 Bioavailability of selected polyphenols

Author	Food source	Sample size	Dose	Plasma concentration (µmol/l)	Urinary excretion (% of intake)	Elimination half-life (hours)
Erlund <i>et al.</i> (2001)	Orange juice	8	126 mg eq hesperetin	2.2	5.3	2.2
Leenen <i>et al.</i> (2000)	Black tea	21	140 mg total flavanols	0.34	NR	NR
Van het Hof <i>et al.</i> (1998)	Black tea and milk	12	300 mg total flavanols	0.18	NR	8.6
Bell <i>et al.</i> (2000)	Red wine (120 ml)	9	35 mg flavanol monomers	0.077	NR	3.2
Van het Hof <i>et al.</i> (1999)	Green tea	21	640 mg total flavanol monomers and gallates	1.8	NR	NR
Zhang <i>et al.</i> (1999)	Soya milk	14	0.49 mg Da/kg BW 0.59 mg Ge/kg BW 0.10 mg Gly/kg BW	1.14 at 6 h 1.74 at 6 h 0.21 at 6 h	48.6 27.8 55.3	NR

BW, bodyweight; eq, equivalents; Da, diadzein; Ge, genistein; Gly, glycitein; NR, not reported.

Table 2 shows the reported bioavailability of key polyphenols. Comparison is virtually impossible as a result of differences in dosage. However, it can be seen that changes in plasma levels reflect polyphenol consumption, suggesting at least some bioavailability in each case. Manach *et al.* (2005), in a review of 97 bioavailability studies, reported that gallic acid and isoflavones were the most bioavailable polyphenols, followed by flavanol monomers, flavanones and quercetin glycosides. The least bioavailable polyphenols were proanthocyanidins, the flavanol gallates and anthocyanins. Various authors have examined the bioavailability of tea flavonoids during normal consumption. Warden *et al.* (2001) asked subjects to consume four cups of black tea over 24 hours following a 5-day low-flavonoid diet. Blood, urine and faecal samples were then monitored over 7 hours. Plasma levels of EC, EGC and EGCg

increased significantly and peaked at 5 hours, prompting the authors to conclude that black tea flavanol monomers and flavanol gallates were bioavailable. Another study (Widlansky *et al.* 2005) examined total plasma flavanols (monomers and gallates) before and after acute (450 ml bolus) and chronic black tea consumption (900 ml per day for 4 weeks). Acute intake significantly increased the flavanols (monomers and gallates) by 33%, while the figure for chronic intake was 29%. A limitation of both of these studies is that the flavanols measured in plasma are similar but not identical to the flavanols present in tea. This is because tea flavanols are extensively metabolised into other polyphenols as soon as they are absorbed by the body and generally do not show up in plasma in their original form. Comparisons between the bioavailability of black and green tea flavonoids suggest that flavanols in black

tea are less well absorbed than the same flavanols in green tea (Rietveld & Wiseman 2003).

As mentioned previously, the value of a polyphenol reflects not only the bioavailability but *in vivo* bioactivity plus habitual intake. While flavanol monomers and gallates from black tea are not well absorbed, they are bioactive and in plentiful supply in the UK diet. Williamson and Manach (2005) reviewed 93 human intervention studies, reporting that consumption of flavanol monomers and gallates was associated with increased plasma antioxidant activity, decreased plasma lipid peroxide and improved resistance of low density lipoprotein (LDL) cholesterol to oxidation. For example, Henning *et al.* (2004) asked 30 healthy subjects to consume a single bolus of black or green tea in a randomised crossover study. Plasma antioxidant levels rose significantly in each case, with a peak at around 1–2 hours. The largest rise was seen with green tea consumption. Other studies have identified bioactivity of black tea flavonoids *in vitro* (Leung *et al.* 2001) and *in vivo* (Ishikawa *et al.* 1997).

Tea flavanols are consumed in large amounts, compensating for their relatively low bioavailability. Manach *et al.* (2005) estimated that the average daily intake of flavanols and proanthocyanidin dimers and trimers in the US was 18–50 mg out of an estimated total polyphenol intake of up to 1 g per day (Scalbert *et al.* 2005). The majority of this came from black tea, chocolate, apples, pears, grapes and red wine. Higher flavanol intakes of 72 ± 47.8 mg per day were reported in a study of elderly Dutchmen, the main sources being black tea, apples and chocolate (Arts *et al.* 2001). In the UK, it is estimated that 82% of dietary flavonoids come from tea (Hertog *et al.* 1997). Using the average tea consumption of 540 ml per day from the NDNS (Henderson *et al.* 2003) and tea flavanol composition figures derived by Khokhar and Magnusdottir (2002), the average flavanol (monomers and gallates) intake from black tea in the UK could be around 83 g per day. However, the low habitual consumption of green tea in Western countries makes it a relatively unimportant contributor to flavonoid intakes.

There has been concern about whether adding milk to black tea adversely affects the bioavailability and bioactivity of flavonoids. This is because experimental data suggest that milk proteins can bind to flavanols, especially flavanol gallates. Stanner (2007) reviewed the evidence, finding that studies were evenly split, with some reporting a significant reduction in bioavailability or bioactivity while others found no effect. A recent human clinical study (Kyle *et al.* 2007) concluded that addition of milk did not influence plasma concentrations of fla-

vonoids or reduce antioxidant capacity, following consumption of 400 ml of black tea. Given the small data set on this issue, it may be that variations in brewing time (and thus flavonoid content of the resulting beverage) are an important factor (Kyle *et al.* 2007). Certainly more research is required, considering that 98% of black tea in the UK is consumed with milk (Hertog *et al.* 1997).

Tea and chronic disease risk

A number of studies have investigated the potential health benefits associated with tea consumption. Gardner *et al.* (2007) conducted a systematic review of epidemiological and experimental studies on black tea consumption. The findings are summarised below, supplemented by recent studies not included in the Gardner review that met similar inclusion criteria (*i.e.* studies in adult humans, intervention or epidemiological design, consumption of black tea).

Coronary heart disease

Studies investigating the potential impact of tea drinking on coronary heart disease (CHD) risk fall into three broad categories: (1) epidemiological or observational studies that attempt to correlate habitual tea drinking with CHD risk factors or mortality; (2) human intervention studies that examine the impact of tea consumption on markers of CHD risk or function; and (3) experimental studies where human tissues are exposed to tea polyphenols. While the epidemiological data associate tea consumption with a lower risk of CHD and experimental studies provide evidence of likely mechanisms of action, results from human intervention studies are variable.

A meta-analysis of 10 cohort and seven case-control studies by Peters *et al.* (2001) found that the risk of myocardial infarction was 11% lower when tea consumption reached three cups per day (1 cup = 237 ml). Two case-control studies included in the meta-analysis reported a 70% reduction in risk at three cups per day (Gramenzi *et al.* 1990; Sesso *et al.* 1999). Five recent epidemiological studies that were not included in the Peters *et al.* (2001) meta-analysis or the Gardner *et al.* (2007) review are shown in Table 3. These suggested a fairly consistent benefit for tea consumption. While one reported no association, the other four found significant correlations between tea consumption and a lower risk of CHD mortality, hypertension, carotid artery plaques and raised plasma homocysteine (a marker for CHD risk).

Table 3 Recent studies reporting associations between black tea consumption and cardiovascular disease

Study	Subjects/population	Methods	Outcomes
CVD risk/mortality			
Mukamal <i>et al.</i> (2002)	N = 1900 adults; post-MI; US	Prospective cohort study. Habitual tea intake assessed by FFQ	Total and CVD mortality adjusted for age and sex significantly lower in those consuming ≥ 14 cups tea per week
Sesso <i>et al.</i> (2003)	N = 17 228; mean age 60 years; US	Prospective longitudinal study with median follow-up of 15 years	Median tea intake low at one cup/day. No significant difference in CVD risk between those consuming <1, 1, 2, 3, ≥ 4 cups tea/day
Mukamal <i>et al.</i> (2007)	N = 28; age ≥ 55 years; at risk of CVD; US	6-month randomised intervention. CVD risk markers before and after exposure to 3 cups of tea/day	No significant effect on lipid levels, lipid oxidation, inflammatory markers, BP, thrombosis risk
Platelet aggregation			
Hodgson <i>et al.</i> (2002)	N = 20 healthy adults; Australia	One-day cross-over intervention. Platelet aggregation and lipids before and 4 hours after three cups of tea + high-fat meal or water + high-fat meal	Urinary flavonoids increased, suggesting bioavailability of tea polyphenols. Tea exposure had no effect on platelets or lipids compared with the water control
Wolfram <i>et al.</i> (2002)	N = 12 adults; Austria	4-week intervention. Subjects asked to consume 500 ml of tea daily containing 2 mg of quercetin. CHD markers measured at baseline and 4 weeks.	Plasma markers of inflammation reduced following tea. Platelet aggregation reduced in women only (N = 6).
Homocysteine			
Hodgson <i>et al.</i> (2006)	N = 232 women; age ≥ 70 years; Australia	Cross-sectional observational study. Tea intake assessed by 24-hour recall. Plasma tHcy and red cell folate measured	Higher tea consumption significantly associated with lower tHcy. Effect seen at >2 cups/day. Red cell folate not associated with tea
Hodgson <i>et al.</i> (2007)	N = 20; age 45–70 years; existing CHD; Australia	One-day cross-over intervention comparing tea vs. water (with and without meal). Plasma tHcy measured before and after. Tea bolus 230 ml. Meal was high-fat	Tea alone significantly increased tHcy. Tea or water + meal reduced tHcy. Implications for CHD risk unclear
Hodgson <i>et al.</i> (2003a)	N = 22 healthy adults; Australia	Randomised controlled intervention. Subjects asked to consume 1250 ml of tea daily (5 cups) for 4 weeks. Plasma tHcy measured before and after	No impact on tHcy. However, evidence of responders and non-responders. Rise in tHcy seen in subjects with a high excretion of methylated polyphenols. Drop in tHcy seen in those with a low excretion.
Other risk factors			
Hodgson <i>et al.</i> (2005)	N = 218 women; age >70 years; Australia	Cross-sectional observational study. Tea intake assessed by 24-hour recall	Mean tea intake 525 ml/day. Higher tea consumption significantly associated with lower BP
Hodgson <i>et al.</i> (2003b)	N = 20 adults; existing CHD; Australia	One-day intervention. Brachial artery dilatation measured before and after exposure to 3 cups of tea with or without high-fat meal	Systolic BP increased following tea alone. Dilatation improved following tea + high-fat meal. Implications for CHD risk unclear
Ardalan <i>et al.</i> (2007)	N = 15; adult renal transplant patients; mean age 37 years; US	One-day cross-over intervention. Brachial artery dilatation before and after 500 ml of tea or water	Dilatation significantly improved following tea compared with the water control
Debette <i>et al.</i> (2007)	N = 6597 adults; age = 65 years; France	Prospective cohort study. Carotid artery intima-media thickness	Tea consumption ≥ 3 cups/day associated with lower prevalence of carotid artery plaques in women only
Jochmann <i>et al.</i> (2007)	N = 21 healthy women; Germany	Cross-over intervention. FMD compared before and after green or black tea, compared with water control	FMD significantly higher after tea consumption. Green and black teas equally effective

MI, myocardial infarction; FFQ, food frequency questionnaire; CHD, coronary heart disease; CVD, cardiovascular disease; tHcy, plasma homocysteine; BP, blood pressure; FMD, flow-mediated dilatation.

Experimental studies have attempted to explain the potential benefits of tea consumption on CHD risk. An initial interpretation was that tea flavonoids behaved as antioxidants, preventing the oxidation of LDL cholesterol by free radicals (Arts *et al.* 2001; Davies *et al.* 2003; Rietveld & Wiseman 2003). However, this has been superseded by other suggested mechanisms. These include the impact of tea metabolites on cell signalling, and the possibility that tea flavonoids have a prebiotic effect and may stimulate gut flora to convert flavonoids to metabolites that reduce cholesterol synthesis in the liver (Clifford & Brown 2006). According to *ex vivo* data (Hodgson *et al.* 2000), black tea may have a greater impact on lipoprotein oxidation than green tea. Other theories on mechanisms address the impact of flavonoids and their metabolites on clotting and vasodilatation (Mojžišová & Kuchta 2001) or on inflammatory processes (Stangl *et al.* 2006).

Eight further recent intervention studies (not included in the Gardner *et al.* 2007 review) were found (see Table 3). These examined a variety of CHD risk factors, including lipid levels, lipid oxidation, inflammatory markers, blood pressure, platelet aggregation, vasodilatation and plasma homocysteine. Three studies reported improvements in blood vessel dilatation, but one also found an unexpected rise in systolic blood pressure. One study found increases in plasma homocysteine following acute tea exposure (Hodgson *et al.* 2007), while another reported improvements in inflammatory markers and platelet aggregation, the latter seen in women only (Wolfram *et al.* 2002). One study compared green and black teas (Jochmann *et al.* 2007), finding that they were equally effective at improving vasodilatation in a small sample of women, while three studies reported no impact of tea consumption on a variety of CHD markers (Hodgson *et al.* 2002; 2003a; Mukamal *et al.* 2007). The evidence around homocysteine is unclear as epidemiological studies found consistently that tea drinkers had lower homocysteine levels, yet experimental work suggests that polyphenols may elevate homocysteine in the short term. However, it is worth noting that experts remain undecided as to whether plasma homocysteine contributes to CHD risk or is simply a marker of another underlying risk factor.

In attempting to explain the lack of uniformity between observational and human intervention studies, Stangl *et al.* (2006) cited inadequate control of confounders (*e.g.* previous lifestyle, genetic predisposition) as a problem, as well as differences in methodology (*e.g.* tea preparation, CHD markers, length of exposure) across the few available human intervention trials. As

animal, experimental and epidemiological studies tend to provide promising results, and taking into account the results of the meta-analysis of Peters *et al.* (2001), it remains likely that drinking tea contributes to a lower risk of CHD. However, more studies are needed to understand individual differences in response (as explored by Hodgson *et al.* 2003a) and how tea polyphenols behave *in vivo* rather than in a test tube.

Cancer

The long-term nature of cancer aetiology and the variety of cancer sites makes the study of individual dietary predictors of cancer challenging. Early animal studies demonstrated that tea polyphenols were anti-inflammatory (Aneja *et al.* 2004), inhibited tumorigenesis (Ju *et al.* 2005) and stimulated the death of cancer cells. They also showed that tea polyphenols acted as antioxidants, protecting human DNA from free radical attack (Siddiqui *et al.* 2006).

Twenty-six studies were reviewed by Gardner *et al.* (2007), 16 on colorectal cancer and 10 on other cancers. Most were case-control or prospective observational studies. It was concluded that the evidence was too contradictory to suggest a benefit for tea consumption, despite promising work from experimental studies on likely mechanisms of action. Indeed, in a review of tea consumption and colorectal cancer incidence, Arab and Il'yasova (2003) reported that confounding factors, such as diet, lifestyle, heredity, age, gender and environment, created more variation in cancer outcomes than the variation because of tea consumption *per se*.

Additional studies on cancer are presented in Table 4. As with CHD, there were varying associations with tea intake. Baker *et al.* (2007) showed that two or more cups per day reduced the risk of ovarian cancer by 30%, while Sun *et al.* (2007) found no association between tea consumption and colorectal cancer. In a small intervention trial, Henning *et al.* (2006) demonstrated inhibition of prostate cancer cell proliferation in men exposed to four cups of tea per day for 5 days. A study by Friedman *et al.* (2007) confirmed that tea polyphenols could destroy cancer cells in the laboratory, although this does not guarantee a similar effect when tea polyphenols are consumed by humans.

Three meta-analyses and an expert review by the World Cancer Research Fund (WCRF) have also recently been published. One examined 13 studies on tea and breast cancer risk but reported conflicting results (Sun *et al.* 2006a): while the combined data from the eight case-control studies showed a weak association between high tea intake and lower risk of breast

Table 4 Recent studies reporting associations between black tea consumption and cancer

Study	Subjects/population	Methods/cancer site	Outcomes
Sun <i>et al.</i> (2007)	N = 60 000 adults; healthy at baseline; Singapore	Prospective cohort study of colorectal cancer. Tea intake estimated by diet interview. Average follow-up of 8.9 years	845 cancer cases were identified. Tea consumption was not significantly associated with cancer risk, RR 0.92
Baker <i>et al.</i> (2007)	N = 414 female patients matched with 868 healthy controls; US	Case-control study of ovarian cancer. Tea intake estimated by FFQ	Tea consumption was significantly correlated with a lower cancer risk. Women drinking more than two cups per day had a 30% lower risk
Henning <i>et al.</i> (2006)	N = 20 men pre surgery; US	Intervention. Prostate cancer. Men randomised to receive 4 cups of black tea or control for 5 days prior to surgery	Prostate cells of men consuming tea contained a higher concentration of polyphenols and were less likely to proliferate when cultured <i>in vitro</i>
Friedman <i>et al.</i> (2007)	Not available	<i>In vitro</i> experimental study. Various human cancer cells exposed to tea flavonoids or tea extract and cell death examined	Breast, colon, liver and prostate cancer cells were inhibited by exposure to tea. Lung cancer cells were unaffected. Tea may have anti-carcinogenic properties

FFQ, food frequency questionnaire; RR, relative risk.

cancers (odds ratio = 0.91, 95% confidence interval: 0.84–0.98), data from the five cohort studies suggested the opposite (*i.e.* a weak association between high tea intake and higher risk of breast cancer). However, it is worth pointing out that none of the odds ratios were statistically significant, meaning that a true association between tea intake and breast cancer is unlikely. A second meta-analysis looked at 25 studies on tea and colorectal cancer (Sun *et al.* 2006b). Again, the data for black tea varied widely, giving a summary odds ratio of 0.99, indicating no significant association. The authors noted that while *in vitro* and animal studies looked promising, the available epidemiological data were insufficient to conclude that any type of tea could protect against colorectal cancer in humans. Furthermore, Zhou *et al.* (2007) analysed data on tea and ovarian cancer from two cohort and seven case-control studies, finding no significant associations. Finally, the WCRF expert report concluded that black tea was probably unrelated to cancers of the stomach, pancreas and kidney (WCRF/AICR 2007).

Other health aspects

Bone health

It was previously suggested that certain constituents of tea (*i.e.* caffeine and fluoride), might adversely affect bone mineral density (BMD). Interestingly, research studies now focus on whether tea consumption could have a *positive* impact on bone health. Five studies reviewed by Gardner *et al.* (2007) suggested that tea had a modest beneficial effect on BMD, particularly in older women, where significant increases in BMD were seen at intakes in excess of four cups per day (Chen *et al.* 2003).

An additional study (Devine *et al.* 2007) used observational data on 1500 women aged 70–85 years to investigate associations between tea drinking and BMD. The cross-sectional data suggested that BMD in tea consumers was 3% higher than in non-consumers, while the longitudinal data showed that bone mineral loss over a 4-year period was 1.6% in tea drinkers but 4% in

non-tea drinkers ($P < 0.05$), indicating a protective effect of tea drinking.

Dental health

Fluoride is known to protect teeth from dental caries. The tea plant naturally accumulates fluoride from the soil and can contain 196 µg per 2 g of dry tea (Panyangarm 1988). It has been estimated that one litre of tea per day would contribute 1.5–2.2 mg of fluoride to the diet of a 70-kg adult, based on figures from the Total Diet Study (FSA 2000). Despite knowledge of this, few studies have investigated the potential impact of tea on caries risk.

The review by Gardner *et al.* (2007) included five small studies. Some noted that tea inhibited plaque bacteria and suppressed salivary amylase activity, which would have the effect of slowing sugar release following starch consumption. Other studies found no effect. One small human intervention study noted that the pH of tea (at 4.9) was insufficient to cause erosion (Simpson *et al.* 2001). A large epidemiological study of diet and dental health found a significantly lower prevalence of dental caries in adolescents who drank tea, independent of the addition of sugar (Jones *et al.* 1999), although it could be that tea consumers simply drank fewer cariogenic beverages.

Mineral absorption

It has been suggested that phenolic compounds in black tea could inhibit the bioavailability of non-haem iron, and thus adversely affect the iron status of at-risk groups, such as children, vegetarians, pregnant women and the elderly. Two reviews (Temme & Van Hodonck 2002; Nelson & Poulter 2004) examined the published data and concluded that there was insufficient evidence to identify tea consumption as a predictor of iron status. Instead, it was advised that 'at risk' groups avoid tea drinking at mealtimes when iron-rich foods are likely to be consumed, while those with a minimal risk of iron deficiency could drink tea at any time of the day. One additional study on this topic was located. Mennen *et al.* (2007) assessed whether consumption of green or black tea influenced the baseline iron status of 2500 healthy French adults recruited to take part in the randomised, controlled Supplementation en Vitamines et Mineraux Antioxydants (SU. VI. MAX) trial. It was found that serum ferritin, an objective measure of iron status, was not associated with tea consumption, tea strength or brewing time.

Caffeine

Tea contains around 17 mg of caffeine per 100 ml (40 mg per 235 ml cup), with a wide range of 1–90 mg per 100 ml, reflecting different brewing times (FSA 2004). Some authors cite high caffeine consumption as a risk factor for a number of conditions (*e.g.* hypertension, dehydration, anxiety or insomnia) (Smith 2002). Others suggest positive effects on cognitive performance, physical endurance, fatigue and alertness at intakes of 60–400 mg of caffeine per day (Graham 2001; Smith 2002).

A recent review (Ruxton 2008) examined the evidence around these health issues and investigated the range of daily caffeine consumption expected to maximise benefits while minimising risk. It was found that the majority of studies reporting adverse effects used acute caffeine doses well in excess of reasonable intakes (*i.e.* 300–600 mg, equating to 9–18 average cups of tea per dose). Caffeine intakes of 38–400 mg per day, equating to 1 to 8 cups of tea, appeared to deliver benefits, such as alertness and mood elevation, without adversely affecting sleep quality or hydration. A number of studies identified a role for caffeine in enhancing sports performance, although sample sizes were small.

Conclusions

Taken together, the evidence indicates a positive role for tea in human health, although the final proof from intervention studies remains elusive. It is known from experimental research that black and green teas contain polyphenols, and that these act as antioxidants *in vitro*, although there is only weak evidence for an antioxidant effect *in vivo*. Various human studies have suggested that tea polyphenols beneficially modulate the biochemistry and physiology underpinning CHD and cancer development via a range of other mechanisms.

However, observational studies and the few available human interventions do not give uniform results. As yet, the reasons for this are unclear. It may be that research methodologies fail to reproduce the circumstances that enable tea to work. Certainly, there are wide variations in the strength of tea consumed and the frequency of consumption reported in studies. Tea drinking may simply be a marker for a healthier lifestyle, or there could be a genetic element, as suggested by the intriguing research on homocysteine and polyphenol excretion (Hodgson *et al.* 2003a). Controlling for potential confounders is important, as noted by Arab and Il'yasova (2003) in relation to colorectal cancer risk. In conclusion, it is clear that tea is worthy of further research and,

in the meantime, can be enjoyed safely within the optimal intake range of 3 to 8 cups per day.

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