TEA

1. Production and Use

1.1 Introduction

The origin of tea is lost in ancient history, although legend dates it at about 2700 BC (Woodward, 1980). The first generally accepted reference to tea is found in a Chinese dictionary from 350 AD which stated that the beverage was used as a medicine for various ills (Schapira *et al.*, 1975). Tea achieved popularity in the west only during the late seventeenth and eighteenth centuries, although it was brought to Europe in 1559 (Wickremasinghe, 1978). The first public sale of tea in England was held in 1657. The beverage's popularity grew, and its trade became an economic mainstay. Today, tea is arguably the most popular beverage in the world (Graham, 1984).

Black and green tea are the two main types, defined by their respective manufacturing techniques. Green tea is consumed mostly in Japan, China, North Africa and the Middle East; the remainder of the world uses black tea. Oolong tea, found in some regions of China, is an intermediate variant between black and green tea (Graham, 1983). Black and green teas lightly flavoured with other botanicals are sometimes seen; these include jasmine tea, scented with jasmine blossoms, and Earl Grey tea, flavoured with bergamot, a type of citrus fruit (Graham, 1984).

1.2 Production processes

A general description of tea manufacture and terms used is provided by Eden (1976) and, more briefly, by Graham (1984) and by Millin (1987).

(*a*) *Botany and culture*

Tea was first cultivated in China and then in Japan. With the opening of ocean routes to the east by European traders during the fifteenth, sixteenth and seventeenth centuries, commercial cultivation gradually expanded to Indonesia and then to the Indian subcontinent, including Sri Lanka. Tea is now grown

commercially in tropical and subtropical regions of Asia, Africa and South America. Major exporting countries include Argentina, China, India, Kenya and Sri Lanka (Forrest, 1985).

All varieties and cultivars of tea belong to a single species, *Camellia sinensis* (L.) O. Kuntze (Cloughley, 1983), formerly called *Thea sinensis* (Graham, 1984). The plant is a tender evergreen that can grow to heights of 12-14 m (Forrest, 1985). The bushes are usually kept about 1 m tall by pruning and periodic harvesting of new growth, called flush. Flush is used for the manufacture of finished tea (Graham, 1984).

Throughout many of the world's tea growing regions, harvesting is done by hand as it has been for centuries. Mechanical harvesting is practised to some extent; however, uniformity and, therefore, quality are considered to be superior with the precise selection of leaf that is achievable only by hand selection (Graham, 1984; Forrest, 1985).

(*b*) *Manufacture*

Following harvest, fresh tea flush is subjected to a series of treatment steps that result in the manufacture of either black, Oolong or green tea. Black tea results from promoting enzymic oxidation of fresh leaves; the process was originally called 'fermentation' but does not involve microbial action; for the production of green tea 'fermentation' is prevented. The third type, Oolong, is produced by short fermentation (Graham, 1984).

(i) *Black tea*

Withering: After transport to the tea factory, freshly harvested leaves are spread in layers on trays and belts for up to 18 h in order to reduce the initial moisture content to approximately 60% of the leaf weight (Graham, 1983). Warm or ambient air may be circulated through the beds of tea leaves depending on local climatic conditions. A uniform moisture distribution is dependent on the uniformity of the flush and is important in maintaining the quality of the finished tea. Chemical changes, including an increase in caffeine content, begin during this step (Cloughley, 1983).

Rolling: The leaves are crushed and macerated, partially destroying cell structure, to allow enzymic oxidation of the flavanols in the presence of oxygen from the atmosphere. Orthodox rolling involves use of traditional devices that impart a characteristic curl to the leaf. Other types of maceration equipment are now becoming popular, for example, crush, tear, curl (CTC) is gradually replacing orthodox processing in some growing regions (Graham, 1983).

'Fermentation': During 'fermentation', tea undergoes significant compositional changes leading to the characteristic colour and flavour of black tea

(Sanderson, 1972). 'Fermentation' actually begins during rolling when endogenous enzymes are freed to comingle with other leaf components, primarily flavanols. This enzymatically catalysed aerobic oxidation and subsequent reactions constitute the 'fermentation' process. After rolling, the tea is spread in layers to optimize temperature, moisture and air circulation. The time for which the macerated leaf is allowed to ferment varies according to temperature and other local conditions but ranges from 45 min to 3 h. During this step, the tea begins to develop its characteristic aroma and copper-coloured appearance. Duration is judged subjectively and the reaction is stopped by the next step in manufacture (Graham, 1983). Some caffeine is lost during 'fermentation' (Cloughley, 1983).

Firing: Passing the tea on trays through hot air driers halts the enzymic fermentation step. Moisture content is reduced to 3% in about 20 min. During firing, nonenzymic chemical changes, resulting in further flavour and aroma development, continue. The tea takes on the black colour characteristic of black tea (Graham, 1983). Small amounts of caffeine are lost through sublimation (Cloughley, 1983).

Grading: The last step is to sort the black tea into appropriate grades. The dried leaves are passed through a series of screens with varying mesh sizes to yield tea corresponding to particular grades such as Orange Pekoe, Pekoe, broken Orange Pekoe, fannings and dust. Traditionally, bulk tea has been shipped in aluminium foil-lined plywood chests (Millin, 1987) holding 45-60 kg, depending on the tea's density (Graham, 1983). More recently, tea 'sacks', which are also foil-lined, have begun to replace the chests.

Specification: The International Organization for Standardization (ISO) (1981) has established a standard for black tea (ISO 3720-1981), which includes the following specifications:

"*Definition*: Tea derived solely and exclusively, and produced by acceptable processes, notably 'fermentation' and drying, from the leaves, buds and tender stems of varieties of the species *Camellia sinensis* (Linnaeus) O. Kuntze known to be suitable for making tea for consumption as a beverage.

"*General requirements*: The tea shall be clean and reasonably free from extraneous matter.

"*Chemical requirements*: (1) The tea shall comply with the requirements specified in Table 1, in which all the figures given are calculated on the basis of the material oven-dried to constant mass at 103 ± 2°C.

"Table 1. Chemical requirements for black tea

Characteristic	Requirement	Test method
Water extract, % (w/w) minimum	32	ISO 1574
Total ash, % (w/w)		
maximum	8	ISO 1575
minimum	4	
Water-soluble ash, as percentage of total ash, minimum	45	ISO 1576
Alkalinity of water-soluble ash (as KOH), % (w/w)		
minimum	1.0[a]	ISO 1578
maximum	3.0	
Acid-insoluble ash, % (w/w) maximum	1.0	ISO 1577
Crude fibre, % (w/w) maximum	16.5	Annex

[a] When the alkalinity of water-soluble ash is expressed in terms of milliequivalents per 100 g of ground sample, the limits are: minimum, 17.8; maximum, 53.6.

"(2) No limit is specified for the 'moisture' content of the tea. If desired, the actual loss in mass at 103°C of the sample under test may be determined and the result recorded in the test report. The determination shall be carried out by the method described in ISO 1573."

(ii) *Green tea*

Green tea is made from the same species as black tea, although the varieties used are suited to the specific climatic conditions prevailing in the growing region and local taste preferences. Green tea is not allowed to ferment. Harvesting is similar to that for black tea, but the fresh leaves are quickly subjected to heat in order to inactivate enzymes, thus preventing any oxidative fermentation from occurring. This is accomplished by either steaming the fresh leaves (Japanese type) or roasting in pans with dry heat (Chinese type) (Yamanishi, 1986). Prior to final drying, the leaves are pressed and rolled, which develops their characteristic shape and sizes. After drying, the leaf fragments are sorted into various grades. International standards have not been finalized for green tea (Graham, 1984).

(iii) *Oolong tea*

Oolong teas are only partially oxidized and retain a considerable amount of the original polyphenolic material. Manufacture is usually a cottage industry; the teas are prepared by a series of withering, gentle rolling and drying steps, which vary greatly from facility to facility. Sun drying is often utilized as the first step. The

appearance of the leaf is considered an important aspect of quality, and a significant amount of hand labour is often utilized. The colour of Oolong tea is intermediate between that of green and black tea (Graham, 1984).

(iv) *Instant tea*

Instant tea is used almost entirely to prepare iced tea. It is manufactured by a fairly exhaustive extraction of black tea with hot water. After separation of leaf matter from the extract, the latter is usually stripped of volatile substances (aroma) and concentrated. Drying of such a concentrate without further processing would result in a product incompletely soluble in cold water, so the extract is precooled to precipitate cold water-insoluble fractions, known as 'cream'. These may be processed to improve solubility and then added to the main extract. The preserved aroma fraction is added back to the total extract concentrate before spray or freeze drying (Graham, 1984).

In the USA approximately 15% of tea is used in the instant form. Production in 1981 was about 6000 tonnes; Kenya, India and Sri Lanka together manufactured about 1000 tonnes, much of which was exported to the USA (Graham, 1984).

(v) *Decaffeinated tea*

The most prevalent process for decaffeinating tea is extraction using supercritical carbon dioxide as the solvent. Conditions of temperature and pressure are chosen to favour the selective extraction of caffeine. Carbon dioxide is removed by allowing it to vapourize (Graham, 1984).

1.3 Preparation of tea beverage

(*a*) *Traditional*

Tea beverage is prepared by steeping tea leaves in water at 90-100°C in teapots or cups. Additional hot water may be added to residual leaf in teapots to produce more but often weaker beverage. In Japan, different varieties of green tea are steeped in water at the temperature considered appropriate for the tea used.

(*b*) *Tea bags*

Tea is confined in porous bags chosen to retain solids but allow free diffusion of water and beverage without imparting taste to the tea. In the USA, tea bags now account for well over 95% of home use. Their use is increasing throughout the world.

(*c*) *Brick tea*

In China, Outer Mongolia and the USSR, tea is sometimes compressed into bricks, pieces of which are used to prepare the infusion (Graham, 1984).

(*d*) *Iced tea*

This beverage may be prepared by cooling traditionally brewed tea, but it is sometimes prepared by the prolonged (8-24 h) steeping of tea at room temperature or in chilled water. Cold water-soluble instant teas, which may be sweetened and flavoured, are also used. Instant tea products are usually used at levels of 0.6-1.0 g of tea solids per 100 ml water. Iced tea beverage is also available in canned form.

(*e*) *Tea-gruel*

Tea leaves packed in a cotton pouch are boiled in water in an iron pan for several minutes. Washed or unwashed rice is then added and the mixture is reboiled. The product is usually eaten burning hot (Anon., 1974).

1.4 Production, trade and consumption

(*a*) *Production*

World production of manufactured tea in 1988 was nearly 2.5 million tonnes (Table 2). Four of the top five producing areas are in Asia. Green tea comprises about 21% of the total (International Tea Committee, 1989).

Table 2. World tea production in 1983-88 (in thousand tonnes)[a]

Continent or country	1983	1984	1985	1986	1987	1988
Asia (including Oceania)	1630.3	1748.0	1820.3	1817.0	1921.6	2026.5
Africa	224.9	236.1	271.8	260.3	263.9	282.3
USSR	145.6	151.1	152.1	146.6	120.0	120.0
South America	53.1	57.6	44.6	55.9	50.0	50.0
Total	2054.0	2192.8	2288.8	2279.8	2355.5	2478.8

[a]From International Tea Committee (1989)

Eight countries account for 86% of world production (Table 3); six of these eight account for 95% of green tea production. Virtually all tea produced in Japan and about 60% of that produced in China is green tea. India is the largest tea producer, nearly all of which is black tea.

Table 3. Tea production by country in 1988 (in thousand tonnes)[a]

Country	All tea	Green tea
India	701.1	8.0
China	545.4	338.5
Sri Lanka	228.2	1.2
Kenya	164.0	-
Turkey	153.2	-
Indonesia	135.6	30.0
USSR	120.0	24.0
Japan	89.8	89.8
Other	341.6	28.1

[a]From International Tea Committee (1989)

(*b*) *Exports*

About 40% of total tea production is exported, and five countries account for over 80% of these exports (Table 4). Of the eight most important producing countries, China is the only significant exporter of green tea. In Japan, Turkey and the USSR, nearly all the production is consumed within the country.

Table 4. Tea exports by country in 1988 (in thousand tonnes)[a]

Country	All tea	Green tea
India	221.5	2.0
Sri Lanka	219.7	1.3
China	198.3	78.6
Kenya	138.2	-
Indonesia	92.7	0.1
Other	183.1	12.5

[a]From International Tea Committee (1989)

(*c*) *Imports*

Total tea imports (adjusted for re-export) in 1988 were approximately 1030 thousand tonnes. The 15 leading importing countries in 1988 accounted for 80% of all imports. Imports over the last six years from these countries are shown in Table 5.

Table 5. Tea imports for consumption by country in 1983-88 (in thousand tonnes)[a]

Country	1983	1984	1985	1986	1987	1988
UK	155.2	184.2	155.4	171.1	142.6	162.7
USSR	55.8	70.0	95.8	109.9	134.8	140.0
Pakistan	86.7	85.7	89.1	84.8	90.1	85.5
USA	77.1	88.2	79.1	89.5	77.3	90.1
Egypt	65.5	75.0	76.2	72.9	64.9	76.4
Iran	27.4	29.1	32.6	25.5	28.4	40.3
Iraq	37.8	45.5	34.6	44.7	41.8	57.7
Poland	25.9	25.5	34.7	29.9	32.1	33.6
Japan	12.0	15.6	22.9	26.3	26.3	27.3
Morocco	16.6	22.6	22.3	20.4	23.4	30.0
Saudi Arabia	18.0	20.5	20.6	17.6	19.0	19.0
Australia	21.8	20.6	20.7	20.6	18.2	19.4
Germany, Federal Republic of	14.1	17.1	15.5	15.5	15.2	13.6
Canada	17.5	18.4	15.7	17.5	14.2	14.1
Sudan	12.9	10.7	14.0	11.1	13.0	13.0

[a]From International Tea Committee (1989)

(*d*) *Consumption*

Consumption data based on import, export and production statistics provide a sound estimate for economic purposes; however, determination of actual human consumption or ingestion must take into account the methods of beverage preparation and varying levels of extraction of tea leaves into finished beverages. In addition to the nature of the manufactured leaf, brewing variables, such as leaf to water ratio, temperature and time, all affect the amount of solid extracted.

The estimates of the International Tea Committee of actual consumption take into account imports, exports and, when possible, locally grown tea. Tables 6 and 7 show total and per-caput consumption, respectively.

Table 6. Total average tea consumption by country (in thousand tonnes)[a]

Country	Consumption		Country or region	Consumption	
	1984-86	1985-87		1984-86	1985-87
India	415.10	430.00	Ireland	10.77	10.95
China	~ 350.00 (1988)		Netherlands	9.34	9.51
USSR	236.35	NA	Hong Kong	9.22	9.14
UK	166.97	160.03	France	9.21	9.45
Turkey	130.81	139.42	New Zealand	5.76	5.61
Japan	113.49	120.28	Algeria	5.18	4.90
Pakistan	86.56	88.02	Kuwait	4.37	4.16
USA	85.61	81.97	Jordan	3.92	4.07
Egypt	74.70	73.03	Tanzania, United Republic of	4.39	4.80
Iran	50.20	NA			
Iraq	43.20	41.40	Italy	3.43	3.55
Poland	30.06	32.25	Sweden	2.98	2.99
Sri Lanka	22.70	23.00	German Democratic Republic	2.72	2.60
Morocco	21.75	22.03			
South Africa	20.96	20.23	Denmark	2.36	2.30
Australia	20.64	19.87	Czechoslovakia	2.25	2.20
Saudi Arabia	19.54	19.05	Switzerland	1.89	1.80
Canada	17.22	15.79	Belgium/Luxembourg	1.36	1.34
Kenya	16.36	17.35	Austria	1.23	1.15
Germany, Federal Republic of	16.03	15.40	Qatar	1.16	0.99
			Norway	0.87	0.93
Syria	14.72	13.83	Finland	0.85	0.94
Tunisia	13.10	13.56	Bahrain	0.64	0.62
Sudan	11.89	12.68	Thailand	0.55	0.54
Afghanistan	11.33	NA	Greece	0.30	NA
Chile	11.01	11.43	Portugal	0.22	NA
			Spain	0.71	NA

[a] From International Tea Committee (1989)
NA, not available

Table 7. Average tea consumption *per caput*[a]

Country or region	Amount (kg)		Country or region	Amount (kg)	
	1984-86	1985-87		1984-86	1985-87
Qatar	3.74	3.21	Afghanistan	0.63	NA
Ireland	3.03	3.09	South Africa	0.56	0.53
United Kingdon	2.94	2.81	Sudan	0.55	0.56
Iraq	2.72	2.51	India	0.55	0.55
Turkey	2.65	2.72	Denmark	0.46	0.45
Kuwait	2.55	2.23	Sweden	0.36	0.35
Tunisia	1.81	1.82	USA	0.36	0.34
New Zealand	1.77	1.71	China	~ 0.35 (1988)	
Hong Kong	1.69	1.63	Switzerland	0.29	0.27
Saudi Arabia	1.69	1.40	Germany, Federal Republic of	0.26	0.25
Egypt	1.54	1.44			
Bahrain	1.52	1.45	Algeria	0.24	0.22
Sri Lanka	1.43	1.41	Norway	0.21	0.22
Syria	1.43	1.26	Tanzania, United Republic of	0.20	0.21
Australia	1.31	1.22			
Jordan	1.12	1.12	Finland	0.17	0.19
Iran	1.05	NA	France	0.17	0.17
Morocco	0.99	0.97	German Democratic Republic	0.16	0.16
Japan	0.94	0.99			
Chile	0.91	0.93	Austria	0.16	0.15
Pakistan	0.90	0.86	Czechoslovakia	0.14	0.14
USSR	0.85	NA	Belgium/Luxembourg	0.13	0.13
Poland	0.81	0.86	Italy	0.06	0.06
Kenya	0.80	0.76	Portugal	0.02	NA
Canada	0.68	0.62	Spain	0.02	NA
Netherlands	0.65	0.65	Thailand	0.01	0.01

[a] From International Tea Committee (1989)
NA, not available

2. Chemical Composition

2.1 Compounds present in black tea and its beverage

The precise composition of black tea is markedly influenced by the nature of the green shoots used and by procedures in their subsequent processing which take place in the producing countries. Differences in chemical composition are reflected in the various flavour grades and origins offered on the market, which are from mixed seedling populations with characteristics intermediate between two extreme genotypes, *Camellia sinensis* var. assamica (larger leaves) and *C. sinensis* var. sinensis (small leaves) (Millin, 1987).

The flavour aspects of black and green tea have been described (Millin, 1987), and a review of tea volatiles is available (Bokuchava & Skobeleva, 1986). A listing of the volatile compounds identified in black, Oolong and green tea has been provided by Maarse and Visscher (1986); 404 volatile compounds are listed for black tea, 48 in Oolong tea and 230 in green tea. Groups and subgroups of volatile compounds in black tea leaves are shown in Tables 8 and 9. Table 10 gives a broad tabulation of the components of fresh leaf (Millin, 1987); the structures of some of the components are given in Figure 1. Table 11 gives the composition of black tea beverage.

Table 8. Classification of volatile compounds in black tea leaf[a]

Group/subgroup	Numbers of compounds			
	Total	Aliphatic	Benzenoid	Alicyclic
Hydrocarbons	22			
Saturated		1	11	0
Unsaturated		4	1	5
Alcohols	39			
Saturated		12	3	0
Unsaturated		19	0	5
Aldehydes	54			
Saturated		11	4	0
Unsaturated		30	4	3
Hydroxy-		0	1	0
Methoxy-		0	1	0

Table 8 (contd)

Group/subgroup	Numbers of compounds			
	Total	Aliphatic	Benzenoid	Alicyclic
Ketones	48			
Mono-				
Saturated		10	9	1
Unsaturated		11	0	12
Hydroxy-		1	0	1
Di-				
Saturated		2	0	0
Unsaturated		0	0	1
Hydroxy-		0	0	0
Acids	72			
Saturated		38	2	0
Unsaturated		28	0	0
Hydroxy-		2	1	0
Oxo-		1	0	0
Esters	52			
Saturated		16	12	2
Unsaturated		19	1	2
Acetal	1	1	0	-
Nitrogen-containing	19			
Nitriles		4	1	0
Amides		2	0	0
Amines				
Primary		5	2	0
Secondary		0	3	0
Aza-, Diaza-		2	0	0
Sulfur-containing	5			
Thiols		3	0	0
Thioether		1	0	0
Other		1	0	0
Phenols	11			
Monohydroxy-		-	8	-
Alkoxy- (ethers)		-	3	-
Totals	323	224	67	32

[a] From Maarse & Visscher (1986)

0, none found

-, not applicable

Table 9. Classification of heterocyclic (oxygen-, nitrogen- and sulfur-containing) volatile compounds in dry black tea[a]

Group/ subgroup	Epoxides	Furans	Pyrans	Lactones	Pyrroles	Benzo-pyrrole (indole)	Pyrazines	Pyridines	Benzo-pyridines (quinolines)	Thiophene	Thiazoles	Benzo-xazoles	Total
Simple	–	1	–	–	–	1	–	1	–	1	–	1	
Hydrogenated	–	3	2	1	–	–	–	–	–	–	–	–	
Alkyl-	6	4	–	13	–	–	11	11	7	–	5	–	
Alkoxy-	–	1	–	–	–	–	–	1	–	–	–	–	
Aldehydes	–	2	–	–	–	–	–	–	–	–	–	–	
Alcohols	–	1	–	–	–	–	–	–	–	–	–	–	
Acyl-	–	–	–	–	3	–	–	1	–	–	–	–	
Aryl-	–	–	–	–	–	–	–	2	–	–	2	–	
Totals	6	12	2	14	3	1	11	16	7	1	7	1	81

[a] From Maarse & Visscher (1986)

–, not reported

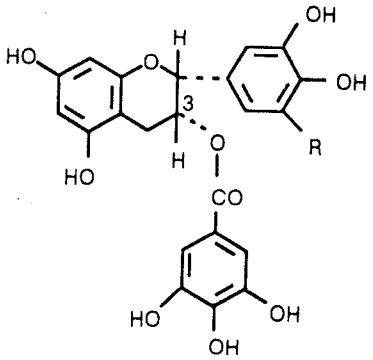
Table 10. Composition of fresh tea leaf, var. assamica[a]

Substance	% dry weight
Flavanols	25
Epi-gallocatechin gallate	9-13
Epi-catechin gallate	3-6
Epi-gallocatechin	3-6
Epi-catechin	1-3
Others	1-2
Flavonols and flavonol glycosides	3-4
Flavanediols	2-3
Polyphenolic acids and depsides	5
Other polyphenols	3
Caffeine	3-4
Theobromine	0.2
Theophylline	0.04
Amino acids	4
Organic acids	0.5
Monosaccharides	4
Polysaccharides	13-14
Protein	15
Cellulose	7
Lignin	6
Lipids	3
Chlorophyll and other pigments	0.5
Ash	5
Volatiles	0.01-0.02

[a] Adapted from Sanderson (1972), Graham (1984) and Millin (1987)

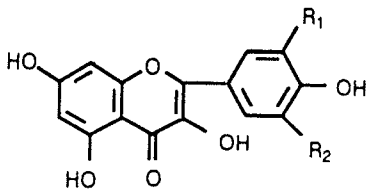
Black tea beverage differs in composition from fresh leaf in that most of the flavanols and some of the other phenolic materials are converted to the oxidized forms known as theaflavins and thearubigins. The total flavanol level is reduced to 10%, and theaflavins may be present at a level of 1-3% and thearubigins at a level of 10-40% (Graham, 1984; Ullah *et al.*, 1984). Changes in pigmentation and aroma also take place. All other components are virtually unchanged (Millin, 1987).

Fig. 1. Structures of some important tea components (From Millin, 1987)



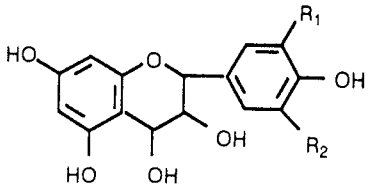
Flavanols

(-) epi-gallocatechin gallate, R = OH
(-) epi-catechin gallate, R = H
(-) epi-gallocatechin, R = OH
(without galloyl ester group, position 3)
(-) epi-catechin, R = H
(without galloyl ester group, position 3)



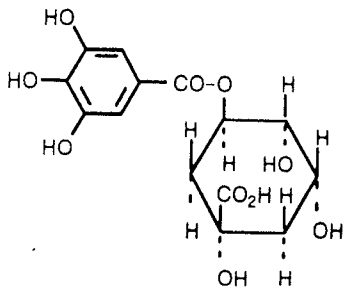
Flavonols

Kaempferol, $R_1 = R_2 = H$
Quercetin, $R_1 = OH$, $R_2 = H$
Myricetin, $R_1 = R_2 = OH$



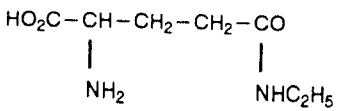
Flavan - 3,4 - diols

(leucoanthocyanins)
R_1, R_2 = H or OH



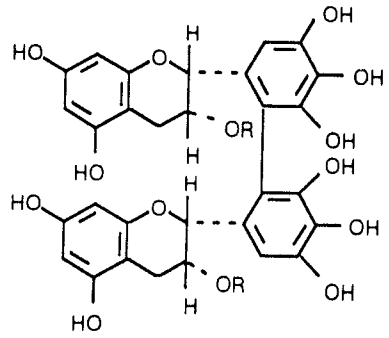
Theogallin

(3-galloylquinic acid)

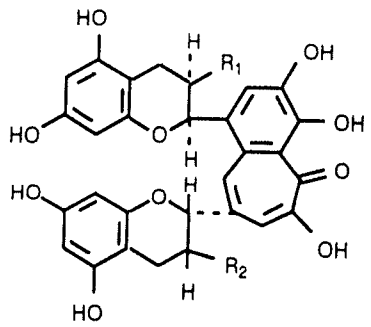


Theanine

(γ-N-ethylglutamine)



Bisflavanols



Theaflavins

Theaflavin ($R_1 = R_2 = OH$)
Theaflavin monogallates (R_1 = galloyl, R_2 = OH) or R_1 = OH, R_2 = galloyl)
Theaflavin digallate ($R_1 = R_2$ = galloyl)

Table 11. Composition of a black tea beverage[a]

Substance	% dry weight
Epi-gallocatechin gallate	4.6
Epi-gallocatechin	1.1
Epi-catechin gallate	3.9
Epi-catechin	1.2
Flavonol glycosides	trace
Bisflavanols	trace
Theaflavins	2.6
Theaflavic acid	trace
Thearubigins	35.9
Caffeine	7.6
Theobromine	0.7
Theophylline	0.3
Gallic acid	1.2
Chlorogenic acid	0.2
Oxalic acid	1.5
Malonic acid	0.02
Succinic acid	0.1
Malic acid	0.3
Aconitic acid	0.01
Citric acid	0.8
Lipids	4.8
Monosaccharides	6.9
Pectin	0.2
Polysaccharides	4.2
Peptides	6.0
Theanine	3.6
Other amino acids	3.0
Potassium	4.8
Other minerals	4.7
Volatiles	0.01

[a] Adapted from Graham (1984)

The quantitative data given below generally refer to the content in dry black tea. In order to provide representative values for the content in hot beverage prepared by steeping loose tea (or in tea bags), it is convenient and realistic to assume usage of 13.5 g black tea per litre of hot water, providing six 150-ml cups of consumable brew (or 6.67 cups in all). Thus, 200 cups are available from 450 g black tea, giving 2.25 g black tea per cup. Spiller (1984) assumed an average of 2.27 g per teabag in a cup for US usage.

At a yield of 23-28% w/w soluble solids in black tea from, say, a 3-5 min brew or first withdrawal, 2.25 g per cup would provide 0.3% w/w soluble solids per cup.

(*a*) *Nonvolatile compounds*

Considerable information is given by Yamanishi (1986) on the changes in composition of both nonvolatile and volatile components during storage under both normal and accelerated conditions.

(i) *Caffeine and other purines*

Because brewing techniques vary widely according to cultural customs around the world (Woodward, 1980), estimation of caffeine intake from tea is subject to considerable variation (Stavric *et al.*, 1988). There is little published information on extraction efficiency under household conditions, but examination of the caffeine contents of brewed tea (Bunker & McWilliams, 1979) allowed the Working Group to calculate an extraction efficiency of total solids in the range of 20-30%.

Caffeine has been reported to be present in dry black tea at 3-4% (Millin, 1987), depending upon the type of leaf used (e.g., there is more in fresh shoots). More detailed information is available from Cloughley (1983). In five samples of commercial blended black tea available in the UK, Kazi (1985) found caffeine contents ranging from 2.7-3.2% by a high-performance liquid chromatography procedure.

Table 12 provides estimates of probable caffeine contents per cup of brewed tea, together with directly obtained data. Bunker and McWilliams (1979) provided data on the caffeine content of black tea brews after various brewing times (Table 13). Caffeine tends to form complexes with oxidized polyphenols in black tea (especially theaflavins and thearubigins); when the latter possess gallate ester groupings, such complexes are poorly soluble in cold water.

Kazi (1985) found theobromine at 0.09-0.28% and theophylline at 0.02-0.06% in dry black tea, which were calculated by the Working Group to correspond to 2.6-8.4 mg and 0.5-1.8 mg per cup.

(ii) *Flavanols and their gallic acid esters*

Four flavanols and their gallic acid esters occur in large amounts among the polyphenols present in tea shoots (34% on a dry basis), i.e., epi-gallocatechin and epi-catechin and their corresponding gallates. Normally, only 5-10% of these flavanols survive the processing of black tea, e.g., 1-3% in dry black tea and 3-8% in its total soluble solids (Millin, 1987). Minimal amounts of other flavanols have been reported (Millin, 1987; Hashimoto *et al.*, 1989). Flavandiols, which are originally present in small quantities, disappear totally on processing (Millin, 1987).

Table 12. Caffeine content of black tea brews

Reference	Type of brew	Caffeine content (mg/cup)	
		Average	Range
Kazi (1985)	-		[57-67][a]
Barone & Roberts (1984)	Bag	42	-
	Leaf tea	41	30-48
	Bag	-	28-44
	Instant tea	28	24-31
	NS	27	8-91
US Food and Drug Administration (1984)	NS (US brands)	40	20-90
	NS (imported brands)	60	25-110

[a]Estimates based upon average caffeine content in dry black tea of 3.0% (Kazi, 1985) and range given for 85-100% extraction efficiencies on addition of 150 ml boiling water; 2.25 g tea per cup

NS, not specified; -, not reported

Table 13. Mean caffeine content by brand and brewing time of black and Oolong teas[a]

Type of tea	Weight of tea leaf (g)	Caffeine content (mg/140 ml; mean ± SD) with brewing time of:		
		1 min	3 min	5 min
Bagged				
Black				
Brand A	NS	33±0.4	46±7.0	50±5.0
Brand B	NS	29±0.2	44±6.0	48±4.8
Brand C	NS	21±1.0	35±1.8	39±2.4
Oolong	NS	13±2.9	30±1.7	40±1.6
Leaf				
Black, Brand A	3	31±1.1	38±1.9	40±6.7
Black, Brand E	1.7	19±2.7	25±1.7	28±3.3
Oolong	2	17±0.3	20±0.6	24±0.2

[a]From Bunker & McWilliams (1979)

NS, not stated; bag used as purchased, one bag per cup of beverage

(iii) *Flavonols and their glycosides*

Three flavonols are present: kaempferol (see IARC, 1983a), quercetin (see IARC, 1983b) and myricetin, predominantly as their 3-glycosides. A portion survives the processing stages unchanged and is present in the final product (Millin, 1987).

(iv) *Phenolic acids and depsides*

A depside is an ester formed by the condensation of two naturally occurring hydroxy acids. Gallic acid is the most important phenolic acid, while theogallin (3-galloylquinic acid) is the major depside, up to 4% occurring in dry black tea, and is substantially water soluble. The depsides are often referred to as hydrolysable tannins and are the gallo-equivalent of chlorogenic acid in coffee. They are virtually unchanged by processing (Millin, 1987).

(v) *Theaflavins and their gallates*

These are of major significance in determining the quality and flavour of tea. They are formed in black tea by oxidation of quinones derived from the epicatechins. They are present to the extent of 1-2% in dry black tea and are substantially water extractable (Millin, 1987).

(vi) *Bisflavanols*

Bisflavanols result from the condensation of *ortho*-quinones, derived from the gallocatechins. They are present at low levels (2-4%) in black tea and are largely water extractable (Millin, 1987).

(vii) *Thearubigins*

Thearubigin is a collective name for the largely unidentified, highly coloured flavanol oxidation products. They are highly heterogeneous in molecular weight and molecular structure and comprise a significant proportion of non-dialysable material. They are often structurally linked to small quantities of peptides or proteins. Their quantity in dry black tea has been given as 10-20% (Ullah *et al.*, 1984), and they are substantially water extractable.

(viii) *Amino acids and peptides*

These compounds are present to a significant extent in black tea (5% on a dry basis); among the amino acids, theanine (γ-*N*-ethylglutamine) is a major component (Millin, 1987).

(ix) *Other organic acids*

These comprise only a small proportion of black tea (0.5%) and are water extractable (see Table 10).

(x) *Trace elements*

Minerals including fluoride and potassium are present in black and green teas. The tea plant is known to accumulate aluminium and manganese (Graham, 1984).

(xi) *Other nonvolatile compounds*

The remaining compounds consist of partially soluble proteins, polysaccharides, lignins and sugars (monosaccharides).

(*b*) *Volatile substances*

van Straten *et al.* (1983) compiled quantitative data on 56 volatile substances in black tea; 404 such compounds were listed by Maarse and Visscher (1986). Volatile essences (obtained by steam distillation) were reported to account for only 0.02% by weight of black tea, i.e., 200 mg/kg (Sanderson, 1972). It is notable that different groups of workers emphasize different groups of substances as being important to the flavour of tea.

(i) *Carbonyls*

van Straten *et al.* (1983) listed quantatitive data for nine aliphatic and two aromatic aldehydes. The quantities reported are generally very small, except that for *trans*-2-hexenal (1.6-25 mg/kg) derived from lipids in the mature leaf; this compound is generally recognized as being undesirable with regard to flavour.

van Straten *et al.* (1983) listed only one ketone and one diketone, both aliphatic, present in very small quantities (0.004-0.2 mg/kg). β-Ionone, a mixed alicyclic-aliphatic ketone, is regarded as important for flavour and has been quantified by Skobeleva *et al.* (1979) at 1.3-4.4 mg/kg (0.13-0.44 mg %) in a range of black teas. 2,3-Butanedione (diacetyl) is reported to be present (Wickremasinghe & Swain, 1965) at 0.01-0.2 mg/kg [corresponding to 0.02-0.45 μg per cup (2.25 g/cup black tea)]. Small quantities (0.05 mg/l) of methylglyoxal were reported in black tea by Nagao *et al.* (1986); 2.4 mg/kg (0.7 μg per serving) were reported in instant tea (Hayashi & Shibamoto, 1985).

(ii) *Alcohols*

Quantitative data are reported by van Straten *et al.* (1983) for 15 aliphatic alcohols, including citronellol and geraniol. Higher quantities of linalool and its oxides, citronellol and geraniol are present in more 'flavourful' teas (e.g., from India) than in lower grades (e.g., from Georgia) (Skobeleva *et al.*, 1979). The listing by von Straten *et al.* (1983) included three other alcohols: benzyl alcohol, 2-phenylethanol and α-terpineol. Of the simpler aliphatic alcohols, 1-butanol is reported to be present in the largest quantity (12-89 mg/kg); of the others, linalool is reported at 1-29 mg/kg.

(iii) *Volatile acids*

Maarse and Visscher (1986) listed 72 volatile acids in black tea. van Straten *et al.* (1983) gave quantitative data for only three of these: formic, acetic and butanoic acids were reported at levels of 0.4, 5.3 and 1.0 mg/kg, respectively, in one sample.

(iv) *Esters*

Quantitative data on five aliphatic and three aromatic esters were listed by van Straten *et al.* (1983). The largest reported amount is for hexyl benzoate, at 4-22 mg/kg; methyl salicylate is present at 4.8-4.9 mg/kg.

(v) *Nitrogen compounds*

Two amines have been reported to be present in substantial quantities: ethylamine at 288 mg/kg and propylamine at 20-29 mg/kg (van Straten *et al.*, 1983). Although a number of *N*, *N/S* and *N/O*-heterocyclic compounds have been reported (see Table 9), none has been quantified. Yamanishi (1986) reported the occurrence of benzyl cyanide and indole in black tea.

(vi) *Furans*

Two complex furans were listed by van Straten *et al.* (1983): one, *cis*-5-(2-hydroxyisopropyl)-2-methyl-2-vinyltetrahydrofuran, was reported to occur at 4-20 mg/kg.

(vii) *Sulfur compounds*

Methylthiomethane was reported to be present in black tea at 0.05-0.1 mg/kg (van Straten *et al.*, 1983)

(viii) *Phenols*

Eleven phenols were listed as present (Maarse & Visscher, 1986); only phenol was quantified and found at 7-15 mg/kg (Skobeleva *et al.*, 1979).

(ix) *Epoxides*

cis- and *trans*-Linalool oxides were reported to be present in small amounts (Saijo & Kuwabara, 1967). Yamanishi (1986) additionally identified pyranoid and furanoid forms of these two substances. In conjunction with linalool itself, they are regarded as being important for flavour.

(x) *Hydrocarbons*

Twenty-two hydrocarbons have been reported in black tea (Maarse & Visscher, 1986). Ruschenburg (1985) reported quantities of polycyclic aromatic hydrocarbons ranging from 0.5 to 3.12 µg/kg in 11 samples of black tea. Four other

samples of black teas had levels ranging from 13.3 to 18.7 μg/kg; 51.5-64.6 μg/kg were found in five samples of smoked tea. In a 5-min black tea brew, the quantities were less than 0.01 μg/l.

(xi) *Hydrogen peroxide*

The hydrogen peroxide content of tea brews was found to increase with the length of incubation and the concentration of tea: for example, a solution of 1 mg/ml tea contained 11.8 nmol/ml [0.4 μg/ml] hydrogen peroxide 1 min after it was prepared; a solution of 0.5 mg/ml tea contained 270.4 nmol/ml [9.2 μg/ml] hydrogen peroxide after standing at 30°C for about 24 h (Ariza *et al.*, 1988).

(xii) *Summarized data*

Table 14 gives estimates of the contents per cup of the groups of volatile compounds considered above. The approximate calculated total is 570 mg/kg (0.06%) in black tea, which is higher than the figure obtained for essence weight (0.02%). [The Working Group suggested that the determined quantity of amines had been overestimated.]

Table 14. Estimated content of various groups of volatile compounds in brewed black tea

Group	Number identified[a]	Number quantified[b]	Total average amount (mg/cup)[c]
Carbonyls	102	13	0.115 (mainly *trans*-2-hexanol and hexanal)
Alcohols	39	18	0.31 (mainly 1-butanol, linalool, 2-phenyl ethanol)
Acids	72	3	0.013 (mainly acetic acid)
Ester	52	8	0.074 (mainly hexyl benzoate)
Amines	12	2	0.68 (substantially ethylamine)
Sulfur compounds	13	1	0.0002
Phenols	11	1	0.015-0.034
Furans	12	3	0.05
Epoxides/lactones	20	2	0.014
Hydrocarbons	22	3	0.0001
Others	49	-	-
Total	404	54	1.3 (578 mg/kg)

[a] From Maarse & Visscher (1986)
[b] From van Straten *et al.* (1983)
[c] Calculated by the Working Group assuming 100% extraction from 2.25 g of dry black tea

Table 15 provides quantitative data on the most abundant aroma compounds in a high-quality black Darjeeling tea (Schreier & Mick, 1984).

Table 15. Principal aroma components in a dry Darjeeling tea[a]

Component	Quantity (mg/kg)
Linalool oxides	23
Linalool	18
Geraniol and benzyl alcohol	7.5
Methyl salicylate	5.5
cis-3-Hexen-1-ol	4.2
2-Phenylethanol	3.3
trans-2-Hexenal	2.5
Hexanal	1.7
1-Penten-3-ol	1.6
trans-2-penten-1-ol	1.3
Phenylacetaldehyde	1.3
trans,trans-2,4-Heptadienal	1.2
trans-2-Hexen-1-ol	1.2

[a]From Schreier & Mick (1984)

(d) Additives and contaminants

Allowable levels of pesticide residues are given by the US Department of Agriculture (1989). Most teas in international trade comply with these regulations.

Some black tea has traditionally been flavoured with various natural agents; the most famous is the 'Earl Grey' blend, prepared by the addition of oil of bergamot (main constituent, linalool) (Millin, 1987). Another popular additive is jasmine flowers, added at the time of drying to both black and green tea. Lapsang Souchong teas are smoked during processing (Graham, 1984).

2.2 Compounds present in green tea and its beverage

The flavour of the green tea beverage is considered to depend upon a suitable balance between the largely unoxidized polyphenols and amino acids, especially theanine (Graham, 1984). The volatile fraction is derived from the original volatiles present in the fresh leaf and pyrolysis products produced during firing. Like black tea, the most important desirable flavour characteristics are associated with higher-boiling terpenid and aromatic substances (Millin, 1987). A total of 230 volatile compounds has been identified in green tea (Maarse & Visscher, 1986).

The quantitative data presented below refer to the content in dry green tea, assuming that the quantity of green tea used per cup is similar to that for black tea, i.e., 2.25-3.0 g.

(*a*) *Nonvolatile compounds*

(i) *Caffeine*

The caffeine content of green tea is similar to that of black tea (Table 16).

Table 16. Mean caffeine content by brand and brewing time of green tea[a]

Type of tea	Weight of tea leaf (g)	Caffeine content (mg/140 ml; mean ± SD) with brewing time of:		
		1 min	3 min	5 min
Bagged				
Brand A	NS	19±1.0	33±2.7	36±2.7
Brand B	NS	9±0.2	20±0.2	26±0.2
Leaf				
Brand A	2.7	28±1.5	33±5.8	35±1.6
Brand C	1.2	15±0.1	-	20±1.8
Pan-fired	1.7	14±0.9	20±2.7	21±3.5

[a]From Bunker & McWilliams (1979)
NS, not stated; bag used as purchased, one bag per cup of beverage

(ii) *Flavanols, flavonols and their glycosides*

As no 'fermentation' is involved, there is very little polyphenol oxidation; polyphenols amount to 38% of the total soluble solids of dry extract (Graham, 1984).

(iii) *Phenolic acids and their depsides*

Depsides are present in the green tea shoots and are largely unchanged by processing (Millin, 1987).

(iv) *Theaflavins and thearubigins*

Green tea has little or none of these transformation products.

(v) *Ascorbic acid*

Ascorbic acid (vitamin C) is present in green tea at an average level of 2.0-2.5 g/kg (Yamanishi, 1986).

(vi) *Amino acids and peptides*

Theanine (γ-*N*-ethylglutamine) is the most important constituent of green tea, constituting some 4.70% of the dry weight of extract. Other free amino acids are present, in particular glutamic acid (0.50%), aspartic acid (0.50%) and arginine (0.74%); others are present to a total of 0.74% (Graham, 1984).

(*b*) *Volatile compounds*

van Straten *et al.* (1983) listed data on 113 volatile compounds in green tea. The total volatile compound content is reported to be one-third to one-quarter of that in black tea, and quantitative data are available for a large number of compounds.

(i) *Carbonyls*

van Straten *et al.* (1983) reported quantitative data for three aliphatic aldehydes, one aromatic aldehyde, β-cyclocitral and safranal. Only *trans*-2-hexenal was reported to be present in a significant quantity, i.e., 10 mg/kg. These authors also reported quantitative data for 13 complex ketones and diketones, all present in very small quantity, except β-ionone at 0.4-6.4 mg/kg. Traces of methylglyoxal have been reported in green tea (Nagao *et al.*, 1986).

(ii) *Alcohols*

van Straten *et al.* (1983) gave quantitative data for 26 alcohols, including geraniol, nerol and linalool. The concentration of geraniol ranged from 0.2 to 13.8 mg/kg, and that of linalool from 0.4 to 50 mg/kg.

(iii) *Acids*

van Straten *et al.* (1983) reported that six aliphatic acids up to decanoic occurred at low levels.

(iv) *Esters*

van Straten *et al.* (1983) reported data for 11 mainly aliphatic esters, including methyl jasmonate (0.2 mg/kg).

(v) *Nitrogen compounds*

van Straten *et al.* (1983) reported ethylamine at 210-457 mg/kg and diphenylamine at 1.5 mg/kg. They also reported data on four pyrroles, two indoles and three pyrazines, presumably arising from the 'firing' stage, but in small quantities, except for indole at 1.2-9.7 mg/kg.

(vi) *Furans*

The same furans as in black tea are reported to be present in very small quantities.

(vii) *Others*

van Straten *et al.* (1983) reported figures for five lactones, benzylcyanide, three phenols, 1,2,4-trichlorobenzene and three epoxides. They also reported figures for 20 hydrocarbons, of which δ-cadinene occurred in the largest amount (23.5 mg/kg).

(viii) *Summarized data*

Belitz and Grosch (1986) listed the percentages of volatile compounds, ranging from linalool (19.9%) and δ-cadinene (9.4%) down to heptanol (0.1%). Table 17 gives estimates of the contents per cup of the groups of volatile compounds considered above.

Table 17. Estimated content of various groups of volatile compounds in brewed green tea

Group	Number identified[a]	Number quantified[b]	Total average amount (mg/cup)[c]
Carbonyls	55	19	0.11
Alcohols	34	15	1.1
Acids	15	6	0.007
Esters	20	11	0.0018
Amines	3	2	0.9 (mainly ethylamine)
Pyroles and indoles	10	6	0.03
Pyrazines	23	3	0.0018
Phenols	14	3	0.0039
Furans	8	7	0.048
Lactones	5	5	0.006
Epoxides	6	3	0.018
Hydrocarbons	30	20	0.15
Others	7	28	0.009
Total	230	128	2.4

[a]From Maarse & Visscher (1986)
[b]From van Straten *et al.* (1983)
[c]Calculated by the Working Group assuming 100% extraction from 3 g green tea

Kosuge *et al.* (1981) determined the aroma composition in high-quality pan-fired green Japanese teas. One example is shown in Table 18.

Table 18. Volatile compounds in a Japanese pan-fired green tea[a]

Compound	%
Geraniol	17.9
Linalool oxides	16.1
Linalool	9.5
Nerolidol	8.8
cis-Jasmone	7.5
2,6,6-Trimethyl-2-hydroxycyclohexane-1-one	7.0

Table 18 (contd)

Compound	%
β-Tonone	5.5
Benzyl alcohol	4.7
cis-3-Hexenyl hexanoate	3.5
5,6-Epoxy-β-ionone	2.7
1-Penten-3-ol	2.7
α-Terpineol	2.2
cis-3-Hexen-1-ol	2.0
Acetylpyrrole	1.8
2-Phenylethanol	1.3
cis-2-Penten-1-ol	1.1
Pentanol	0.7
2,5-Dimethylpyrazine	0.6

[a] From Kosuge *et al.* (1981)

3. Biological Data Relevant to the Evaluation of Carcinogenic Risk to Humans

3.1 Carcinogenicity studies in animals

(*a*) *Subcutaneous administration*

Rat: Groups of 15 male and 15 female NIH Black rats, one to two months of age, received weekly subcutaneous injections of 12 mg of the total aqueous extract or 8 mg of the tannin fraction of Assam tea leaf alternately into each flank for 69-70 weeks (extract) or 45-77 weeks (fraction). A group of 15 male and 15 female controls received injections of saline. Whereas a high number of local tumours (malignant fibrous histiocytomas) developed in the rats receiving the tannin fraction (10/15 males and 11/15 females), a nonsignificant number of local tumours developed in rats treated with the aqueous extract (1/15 males and 1/15 females). No local tumour developed in the controls (Kapadia *et al.*, 1976).

(*b*) *Administration with known carcinogens*

(i) *Polycyclic aromatic hydrocarbons*

Mouse: A group of 15 young Swiss mice [sex unspecified] received a single skin application in the neck region of a 1% solution of benzo[*a*]pyrene in acetone,

followed by applications of black tea solution (1 g tea brewed for 5 min in 155 ml boiling water) on alternate days for 55 paintings. A control group of 15 mice received benzo[*a*]pyrene alone. [The duration of the study was not stated]. In the group treated with benzo[*a*]pyrene and tea, 6/15 mice developed epithelial-cell carcinomas in the neck region. Other mice in this group developed various preneoplastic lesions of the squamous epithelium. No skin lesion was observed in the group treated with benzo[*a*]pyrene alone (Kaiser, 1967). [The Working Group noted the absence of an appropriate control group and the inadequate reporting of histological findings.]

Two groups of 25 male and 25 female Charles River CD-1 random-bred albino mice, six weeks old, were treated with a single application of 50 µl of a 1% solution of benzo[*a*]pyrene (0.53-0.6 mg benzo[*a*]pyrene) in acetone on the shaved interscapular area. One group received no further treatment and served as controls. The other group received 80 applications three times per week of an infusion of black Chinese (Keemun) tea containing 1% tannin. Mice were observed for 567-580 days, at which time all survivors were killed. Survival was similar in both groups. The incidence of hepatomas was 10% in the group receiving benzo[*a*]pyrene alone and 6% in the group also given tea. Skin tumour occurrence in the interscapular area was similar in both groups: the incidences of benign and malignant (carcinomas) tumours in the group given benzo[*a*]pyrene were 15/50 and 2/50, respectively; those in animals also given tea were 15/50 and 3/50, respectively. In the group treated with benzo[*a*]pyrene plus tea, skin tumours occurred significantly earlier than in those given benzo[*a*]pyrene alone (Bogovksi *et al.*, 1977).

Eight groups of 20 female Sencar mice, six weeks old, were pretreated with plant phenols (tannic acid or quercetin; 3000 nmol), green tea polyphenols (24 mg/mouse) or acetone for seven days, after which they received a single topical application of 200 nmol (±)-7β,8α-dihydroxy-9α,10α-epoxy-7,8,9,10-tetrahydrobenzo[*a*]pyrene as the initiating agent or acetone. Beginning seven days after initiation, animals received applications of 3.24 nmol 12-*O*-tetradecanoylphorbol 13-acetate three times per week until termination of the experiment at 18 weeks. The formation of skin tumours [probably papillomas] > 1 mm in diameter and persisting for two weeks or more was recorded weekly and included in the cumulative total. Tannic acid and green tea phenols afforded significant protection ($p < 0.01$) against the induction of skin tumours; quercetin gave only moderate protection (Khan *et al.*, 1988).

Four groups of 20 female BALB/c mice, six weeks of age, were painted on shaved dorsal skin as follows: Group 1 received 0.2 ml acetone daily for seven days then 0.2 ml acetone twice weekly for 16 weeks; Group 2 received acetone daily for seven days and then 3-methylcholanthrene twice weekly for 16 weeks; Group 3 received 1.2 mg green tea polyphenols in acetone daily for seven days and then

3-methylcholanthrene in acetone 1 h following the green tea polyphenols twice weekly for 16 weeks; Group 4 received green tea polyphenols daily for seven days and then green tea polyphenols 1 h following acetone twice weekly for 16 weeks, at which time the experiment was terminated. The number of skin tumours > 1 mm in diameter and persisting for two weeks or more was 11.6 ± 0.5 in mice receiving 3-methylcholanthrene (Group 2) as compared with 5.8 ± 0.9 in those pretreated with green tea polyphenols followed by 3-methylcholanthrene (Group 3). In a parallel experiment, protection by green tea polyphenols was obtained in female Sencar mice pretreated by topical application or in the drinking-water against initiation by 7,12-dimethylbenz[*a*]anthracene or promotion by 12-*O*-tetradecanoyl phorbol 13-acetate; i.e., there was a longer latent period and 28.8 ± 1.7 or 29.1 ± 3.7 *versus* 51.3 ± 3.6 tumours per animals. Green tea polyphenols did not initiate skin tumours. Furthermore, when administered topically or orally, they significantly inhibited polycyclic aromatic hydrocarbon-DNA adduct formation in epidermis after topical administration of ^{3}H-7,12-dimethylbenz[*a*]anthracene or ^{3}H-benzo[*a*]-pyrene (Wang *et al.*, 1989a).

(ii) N-*Nitroso compounds*

Mouse: Thirty-one male C57Bl/6 mice, eight weeks of age, received 100 mg/l *N*-ethyl-*N'*-nitro-*N*-nitrosoguanidine in the drinking-water for four weeks; 15 animals subsequently received 0.005% epi-gallocatechin gallate (the main polyphenolic constituent of green tea infusion) in the drinking-water for 11 weeks; 16 animals received tap-water only for 11 weeks. All mice were sacrificed in the 16th week after the start of treatment with the nitrosamide. The incidences of duodenal tumours were 3/15 (20%) in the group receiving epi-gallocatechin gallate and 10/16 (63%; $p < 0.001$) in the group treated with the nitrosamide alone. Similar results were obtained when the experiment was repeated (Fujita *et al.*, 1989).

3.2 Other relevant data

(*a*) *Experimental systems*

(i) *Absorption, distribution, metabolism and excretion*

No data were available to the Working Group.

(ii) *Toxic effects*

Green and black teas have been reported to decrease significantly the activity of transketolase in whole blood of rats. The activity of liver transketolase was decreased by green tea only. Neither type of tea had any effect on the activity of intestinal transketolase. The activity of lactate dehydrogenase was not affected by the teas, while both green and black teas decreased the activity of lactate

dehydrogenase in whole blood. Neither tea had an effect on intestinal alkaline phosphatase, but thiamine diphosphatase activity was decreased by both teas (Ali *et al.*, 1989).

(iii) *Effects on reproduction and prenatal toxicity*

No data were available to the Working Group.

(iv) *Genetic and related effects*

The genetic and related effects of tea have been reviewed (Sugimura, 1982; Sugimura & Sato, 1983; Nagao *et al.*, 1986).

Tea has been studied in experimental genetic and related systems following preparation by various methods (Nagao *et al.*, 1979; Uyeta *et al.*, 1981), including adding water at various temperatures to tea leaves, followed by decanting or filtering the solution. Variations included the use of different types of tea, water:leaf ratios and steeping times. The tea beverage was evaporated and the residue redissolved in a solvent (e.g., dimethylsulfoxide) for testing. The doses are expressed either as the original weight of tea leaves from which the extract was obtained or as the original volume of the tea beverage (infusion). Tea preparations were hydrolysed by various methods (e.g., acid, enzymes or bacteria), the hydrolysates extracted with organic solvents, the solvents evaporated and the residue redissolved in a solvent (e.g., dimethylsulfoxide) suitable for genetic assays.

Several studies addressed the nature of the components of green and black tea that are mutagenic to bacteria. Mutagenic activity suppressed by catalase may be attributed in part to hydrogen peroxide (Ariza *et al.*, 1988) and dicarbonyls present in tea (Nagao *et al.*, 1986). In addition, tea has been shown to contain precursors (flavonoid glycosides) of mutagenic flavonols (e.g., kaempferol, quercetin and myricetin) which are released when the tea is treated to produce hydrolysates. In these studies, treatment of dried teas with acid, glycosidase enzymes (e.g., hesperidinase) or bacteria (e.g., human intestinal bacteria) increased their mutagenic activity in *Salmonella typhimurium* TA98 and TA100, especially when an exogenous metabolic activation system was added (Nagao *et al.*, 1979; Uyeta *et al.*, 1981).

The results described in this section are listed at the end, in Table 19, with the evaluation of the Working Group, as positive, negative or inconclusive, as defined in the footnotes. The results are tabulated separately for the presence and absence of an exogenous metabolic system. The lowest effective dose (LED), in the case of positive results, or the highest ineffective dose (HID), in the case of negative results, are shown, together with the appropriate reference. The studies are summarized briefly below.

Black tea: Black teas were mutagenic to *S. typhimurium*. Oolong, Lapsang Souchong and jasmine teas inhibited DNA synthesis in cultured lymphocytes (Yang *et al.*, 1979).

Green tea: Green teas were mutagenic to *S. typhimurium*. An antioxidant fraction of green tea did not affect gap-junctional intercellular communication in cultured mouse hepatocytes and human keratinocytes.

Japanese green tea was found to contain a considerable amount of epi-gallocatechin gallate; this tannin effectively reduced the spontaneous mutation rate in NIG 1125 *Bacillus subtilis* carrying a mutation in DNA-polymerase III, but failed to lower the frequency of chemically or ultra-violet radiation-induced reverse mutations in *S. typhimurium* or *Escherichia coli* (Kada *et al.*, 1985).

Unspecified teas: Tea (Horniman's brand, puchased in Córdoba, Spain [unspecified as black or green, but probably black]) was mutagenic to *S. typhimurium* TA104 in the histidine reversion assay and to *S. typhimurium* BA13 in the arabinose-resistance forward mutation assay.

Tea in combination with other agents: Both green and black teas decreased the mutagenic activity of *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine in *E. coli* and *S. typhimurium* TA100, both *in vitro* and in an in-vivo/in-vitro assay in which the gastric contents of rats were sampled and tested 1 h after oral administration of the nitrosamide either alone or with tea extracts (Jain *et al.*, 1989a).

Green tea and black tea decreased the mutagenic activity of nitrosation products of methylurea and salted fish in *S. typhimurium* TA1535 (Stich *et al.*, 1982a,b) and decreased the mutagenic activity of benzo[*a*]pyrene in *S. typhimurium* TA100 (Joner & Dommarsnes, 1983). Both Oolong and green teas similarly decreased the mutagenicity induced by benzo[*a*]pyrene in *S. typhimurium*; and the frequency of chromosomal aberrations induced by benzo[*a*]pyrene in Chinese hamster lung (CHL) cells was decreased by the addition of Oolong tea (Kojima *et al.*, 1989). Oolong tea also decreased the mutagenicity induced in *S. typhimurium* by gasoline vehicle exhaust, cooked salmon, 1,6-dinitropyrene, 3-amino-1-methyl-5*H*-pyrido[4,3-*b*]indole (Trp-P-2) and 2-amino-3-methylimidazo[4,5-*f*]quinoline (IQ) (Kojima *et al.*, 1989).

The frequency of chromosomal aberrations in rat bone marrow following intraperitoneal injection of aflatoxin B_1 was reduced by hot-water extracts of green tea but not of black tea. This effect was observed when the teas were administered 24 h before the aflatoxin, and was attributed to green tea tannins (Ito *et al.*, 1989).

An antioxidant fraction from green tea containing catechins prevented the inhibition of gap-junctional intercellular communication induced by paraquat, glucose oxidase and phenobarbital in mouse hepatocytes, and by 12-*O*-tetradecanoylphorbol 13-acetate in human keratinocytes (Ruch *et al.*, 1989).

Green tea and polyphenols from green tea inhibited the mutagenicity of benzo[*a*]pyrene, aflatoxin B_1, 2-aminofluorene and coal-tar pitch in *S. typhimurium* TA100 and/or TA98. The polyphenols from green tea also inhibited aflatoxin B_1-induced mutations, decreased sister chromatid exchange and chromosomal aberrations in V79 cells and inhibited benzo[*a*]pyrene-induced mutations in V79 cells (Wang *et al.*, 1989b).

In one study, green tea and black tea reduced the induction of anchorage-independent growth of mouse epidermal JB6 cells by 12-*O*-tetradecanoylphorbol 13-acetate (Jain *et al.*, 1989b).

(*b*) *Humans*

(i) *Absorption, distribution, metabolism and excretion*

No data were available to the Working Group.

(ii) *Toxic effects*

In a number of studies in which coffee drinking was associated with increased serum cholesterol levels, participants who consumed tea showed either no association or a negative correlation with serum cholesterol levels (Arab *et al.*, 1983; Haffner *et al.*, 1985; Klatsky *et al.*, 1985; Curb *et al.*, 1986; Green & Jucha, 1986; Little *et al.*, 1986; Tuomilehto *et al.*, 1987).

Several studies, including the Boston Collaborative Drug Surveillance Program (1972), that showed a correlation between coffee consumption and the risk of coronary heart disease (see p. 104) showed no difference between patients and controls for tea drinking (Jick *et al.*, 1973; Rosenberg *et al.*, 1980).

Cases of asthma due to sensitivity to tea dust have been reported in workers who process tea in the tea industry (Lewis & Morgan, 1989).

A positive association between the consumption of tea and other caffeine-containing beverages and the premenstrual syndrome was suggested (Rossignol, 1985; Rossignol *et al.*, 1989). [The Working Group noted the limitation of the methodology and the small number of subjects.]

(iii) *Effects on reproduction and prenatal toxicity*

No association was found between tea consumption during pregnancy and the occurrence of malformations in offspring (Rosenberg *et al.*, 1982) (for a description of the study, see the monograph on coffee, p. 105).

In a study by Berkowitz *et al.* (1982), described on p. 109, tea drinking was compared among women who had had preterm infants and women who had had full-term infants. Drinking four or more cups daily was more frequent among cases than controls (odds ratios, 1.5-2.0 in the three trimesters), but these differences were

Table 19. Genetic and related effects of black, green and unspecified teas

Test system	Results		Dose[a] LED/HID	Reference
	Without exogenous metabolic activation	With exogenous metabolic activation		
Black tea				
SA0, Salmonella typhimurium TA100, reverse mutation	+	-	7500.0000[b]	Nagao et al. (1979)
SA0, Salmonella typhimurium TA100, reverse mutation	-	+	0.0000 (hydrolysate)	Nagao et al. (1979)
SA0, Salmonella typhimurium TA100, reverse mutation	+	+	0.0000 (hydrolysate)	Uyeta et al. (1981)
SA9, Salmonella typhimurium TA98, reverse mutation	-	-	0.0000 (hydrolysate)	Nagao et al. (1979)
SA9, Salmonella typhimurium TA98, reverse mutation	+	+	0.0000 (hydrolysate)	Uyeta et al. (1981)
SA9, Salmonella typhimurium TA98, reverse mutation	-	+	0.0000 (hydrolysate)	Nagao et al. (1979)
Green tea				
SA0, Salmonella typhimurium TA100, reverse mutation	+	-	8000.0000[b]	Nagao et al. (1979)
SA0, Salmonella typhimurium TA100, reverse mutation	-	+	0.0000 (hydrolysate)	Nagao et al. (1979)
SA0, Salmonella typhimurium TA100, reverse mutation	+	+	0.0000 (hydrolysate)	Uyeta et al. (1981)
SA9, Salmonella typhimurium TA98, reverse mutation	-	-	0.0000 (hydrolysate)	Nagao et al. (1979)
SA9, Salmonella typhimurium TA98, reverse mutation	+	+	0.0000 (hydrolysate)	Uyeta et al. (1981)
SA9, Salmonella typhimurium TA98, reverse mutation	-	+	0.0000	Nagao et al. (1979)
ICR, Cell communication, mouse hepatocytes in vitro	-	0	50.0000[c]	Ruch et al. (1989)
ICH, Cell communication, human keratinocytes in vitro	-	0	50.0000[c]	Ruch et al. (1989)
Tea unspecified				
SAF, Salmonella typhimurium BA13, forward mutation ara[R]	+	0	220.0000	Alejandre-Duran et al. (1987)
SA2, Salmonella typhimurium TA102, reverse mutation	-	0	880.0000	Alejandre-Duran et al. (1987)
SA4, Salmonella typhimurium TA104, reverse mutation	+	0	880.0000	Alejandre-Duran et al. (1987)
SA5, Salmonella typhimurium BA13, reverse mutation	-	0	880.0000	Alejandre-Duran et al. (1987)

[a] Expressed as dry weight of extract
[b] Weight of material from which extract was prepared
[c] Green tea antioxidant fraction

not significant. The odds ratio was 1.6 (95% confidence interval (CI), 0.7-3.7) after adjustment for smoking and alcohol and coffee consumption.

Brooke *et al.* (1989), in a study described on p. 110, found an association between tea consumption and low birth weight in their crude data. The birthweight ratios, adjusted to 40 weeks' gestation, were 1.050, 1.043, 1.034 and 1.012 in babies born to mothers drinking 0, 1-14, 15-43 and more than 43 cups of tea per week. While the difference among the groups and the linear trend were statistically significant, no significance remained when the data were additionally adjusted for smoking.

In the papers of Watkinson and Fried (1985) and Martin and Bracken (1987) tea drinkers were included but data were not given separately. The studies of Furuhashi *et al.* (1985) and Caan and Goldhaber (1989) showed no association between tea drinking and low birth weight. (These studies are discussed on pp. 109, 110, 106 and 111.)

In a study described on p. 111, late spontaneous abortions were studied by Srisuphan and Bracken (1986) in relation to caffeine intake from various beverages including tea. Tea was apparently not associated with spontaneous abortions (crude relative risk (RR), 1.1).

(iii) *Genetic and related effects*

No data were available to the Working Group.

3.3 Case reports and epidemiological studies of cancer in humans

In comparison with coffee, less attention has been paid to the possible relationship between tea and cancer. No study has been specifically designed to study this issue, but data on tea have been published in several studies in which coffee and other possible risk factors for cancer were considered.

(*a*) *Descriptive epidemiology*

In the study of Phelps and Phelps (1988), described on p. 114, no association was found with breast cancer; no distinction was made between tea and coffee.

An ecological study examining the association between annual tea consumption, estimated from trade data, and age-adjusted cancer mortality rates for 1964-65 from 20 different countries (Stocks, 1970) noted that mean death rates from cancers at the following sites were significantly associated with tea consumption: intestine ($p < 0.01$), breast (women; $p < 0.01$), larynx (women; $0.01 < p < 0.05$), lung and bronchus (women; $0.01 < p < 0.05$). There was no association with oesophageal cancer.

A comprehensive examination of the correlation between cancer incidence (27 cancers in 23 countries) and mortality (14 cancers in 32 countries) and a variety of dietary and other environmental variables (Armstrong & Doll, 1975) showed weak positive correlations between tea consumption and cancers of the colon and rectum, although the adjusted correlation coefficients for tea were not noted. Lung cancer incidence in women was significantly associated with tea consumption ($p < 0.01$).

(i) *Pancreatic cancer*

In an early Japanese paper (Ishii *et al.*, 1973), information on diet, smoking and occupation was collected from a case series of 311 male patients with pancreatic cancer. A comparison group of the same age [but unspecified sex] was identified from a separate prospective study. In a ridit analysis, patients had a nonsignificantly higher prevalence of drinking hot tea.

(ii) *Oesophageal cancer*

A number of the studies investigating the association between oesophageal cancer and tea consumption have focused on the Caspian littoral of Iran, where oesophageal cancer rates are the highest in the world (Hormozdiari *et al.*, 1975; Joint Iran-IARC Study Group, 1977; Mahboubi & Aramesh, 1980; Ghadirian, 1987). Within this region, incidence rates for oesophageal cancer vary by 30 fold in women and by six fold in men (Hormozdiari *et al.*, 1975). A household survey showed that in the villages in the areas with the highest cancer rates there was higher tea consumption than in the lowest incidence villages, although the intermediate incidence region reported the highest tea consumption overall. These data were confirmed in a subsequent report (Joint Iran-IARC Study Group, 1977).

The effect of differences in the temperature at which the tea was consumed was reported (Ghadirian, 1987), and this was related to incidence rates in high- and low-risk regions of the littoral. In the low-risk region, tea was consumed at an average temperature of 50.1°C compared with an average temperature of 61.3°C ($p < 0.0001$) in the high incidence area.

In an ecological study by Segi (1975) in Japan, a geographical correlation was found between rates for cancer of the oesophagus and consumption of tea-gruel.

(*b*) *Cohort studies*

These studies are summarized in Table 20.

(i) *All sites*

In the study of Heilbrun *et al.* (1986), 7833 Hawaiian Japanese men were interviewed about their consumption of black tea in 1965-68, and incident cases of

cancer were identified. The RRs for rectal cancer, adjusted for age at examination and alcohol consumption, showed a significant dose-response relationship (p for trend = 0.0007) when the tea consumption category 'almost never' was used as the standard. The excess risk for rectal cancer was confined to men aged 58 years or over at examination. The authors also reported that none of the examined variables that were associated with tea intake (height, weight, pack-years of smoking and physical activity level) was associated with rectal cancer risk. Prostate cancer risk, adjusted for age at examination, showed a significant negative association with tea consumption, although there was no clear dose-response relationship. There was a nonsignificant, steady inverse association between the risk for liver cancer and tea consumption.

Kinlen *et al.* (1988) examined the relationship between tea consumption and rectal and other cancers in a mortality study of 14 453 men in London, UK, aged 45-60, who were administered a questionnaire about diet and smoking in 1967; 97% were traced to the end of 1986 (14 085 men). Only deaths that occurred after the first 18 months of follow-up were included in analyses. Expected numbers of deaths were calculated using age-specific mortality rates for men in England and Wales for the appropriate time periods. Rectal cancer showed no trend with tea consumption, while colon cancer showed a negative trend with increasing consumption; the risk associated with tea consumption was similar among older and younger men. The authors reported positive trends between increasing tea consumption and the risk for stomach, lung and kidney cancers. The association with stomach and lung cancer persisted after limited adjustment for social class and smoking. [The Working Group noted that there were inconsistencies in the paper in reporting the number of rectal cancer deaths.]

(ii) *Pancreatic cancer*

In the study by Hiatt *et al.* (1988; described on p. 117), cancer patients had drunk 0.32 cup of tea per day, while the controls from within the cohort had drunk 0.62 cup per day (p = 0.10). These figures were adjusted for age and smoking.

The nested case-control study of Whittemore *et al.* (1983; described on p. 116) found a RR of 0.6 for drinking ≥2 cups per day. The authors stated that adjustment for smoking at college did not alter the findings. The reduction remained after adjustment for coffee drinking.

Table 20. Summary of results of cohort studies on cancer and tea consumption

Reference, location, site	Subjects	Tea consumption	RR (95% CI)	Comments
Heilbrun *et al.* (1986)	7833 Japanese men			
Hawaii	76 cases	Almost never	1.0	*p* for trend = 0.0007;
Rectum		Twice/week	1.3	adjusted for age at
		2–4×/week	2.0	examination and
		Almost daily	2.1 significant	alcohol consumption
		>Once daily	4.2 significant	
Pancreas	30 cases	Almost never	1.0	*p* for trend = 0.87;
		Twice/week	0.6	adjusted for age at
		2–4×/week	1.4	examination and
		Almost daily >Once daily	0.9	pack-years of smoking
Prostate	149 cases	Almost never	1.0	*p* for trend = 0.02;
		Twice/week	0.8	adjusted for age at
		2–4×/week	0.4	examination
		Almost daily >Once daily	0.6	
Bladder	57 cases	Almost never	1.0	*p* for trend = 0.68;
		Twice/week	1.4	adjusted for age at
		2–4×/week	1.0	examination and
		Almost daily >Once daily	0.8	pack-years of smoking
Liver	25 cases	Almost never	1.0	*p* for trend =
		Twice/week	0.8	0.134; adjusted for
		2–4×/week Almost daily >Once daily	0.6	age at examination and alcohol consumption
Rectum	37 cases	Almost never	1.0	*p* for trend = 0.67; ad-
	<58 years old	Twice/week	1.4	justed for age at
	at examination	2–4×/week	1.4	examination and
		Almost daily	1.1	alcohol consumption
		>Once daily	1.4	
Rectum	39 cases	Almost never	1.0	*p* for trend < 0.0001;
	≥58 years old	Twice/week	1.0	adjusted for age at ex-
	at examination	2–4×/week	2.9	amination and alcohol
		Almost daily	3.5 significant	consumption
		>Once daily	8.7 significant	

Table 20 (contd)

Reference, location, site	Subjects	Tea consumption	RR (95% CI)	Comments
Kinlen *et al.* (1988) London, UK	14 085 men		Observed:expected ratios	Expected no. of deaths calculated using national age-specific data
Rectum	62 cases	0–3 cups/day	0.5	p for trend = 0.94
		4–6 cups/day	0.9	
		7–9 cups/day	0.8	
		≥10 cups/day	0.5	
Pancreas	70 cases	0–3 cups/day	0.6	p for trend = 0.28
		4–6 cups/day	0.8	
		7–9 cups/day	1.1	
		≥10 cups/day	0.9	
Prostate	88 cases	0–3 cups/day	0.6	p for trend = 0.30
		4–6 cups/day	0.8	
		7–9 cups/day	1.0	
		≥10 cups/day	0.8	
Bladder	71 cases	0–3 cups/day	1.0	p for trend < 0.13
		4–6 cups/day	0.7	
		7–9 cups/day	1.2	
		≥10 cups/day	1.4	
Stomach	172 cases	0–3 cups/day	0.6	p for trend < 0.0005
		4–6 cups/day	0.8	
		7–9 cups/day	1.2	
		≥10 cups/day	1.4	
Colon	79 cases	0–3 cups/day	1.0	p for trend = 0.066
		4–6 cups/day	0.8	
		7–9 cups/day	0.5	
		≥10 cups/day	0.7	
Lung	718 cases	0–3 cups/day	0.6	p for trend = 0.0001
		4–6 cups/day	0.8	
		7–9 cups/day	1.1	
		≥10 cups/day	1.4	
Kidney	26 cases	0–3 cups/day	0.4	p for trend = 0.04
		4–6 cups/day	0.7	
		7–9 cups/day	1.2	
		≥10 cups/day	1.8	

Table 20 (contd)

Reference, location, site	Subjects	Tea consumption	RR (95% CI)	Comments
Whittemore *et al.* (1983) USA	50 000 men			
Pancreas	126 cases	Nondrinkers	1.00	Adjusted for age, college and class year
		Drinkers	0.5 (0.3–0.9)	
		< 2 cups/day	1.0	Adjusted for age, college and class year
		≥2 cups/day	0.6 (0.3–1.1)	

(c) *Case-control studies*

(i) *Bladder and urinary tract*

These studies are summarized in Table 21, at the end of this section.

Bladder: In a Canadian case-control study of patients with histologically confirmed bladder cancer (Morgan & Jain, 1974; described on p. 122), there was no association with tea intake (overall RR for drinkers *versus* non-drinkers adjusted for sex, 0.7).

In the US study of Simon *et al.* (1975; described on p. 122), no relation between tea drinking and cancer of the lower urinary tract (renal pelvis, ureter, bladder and urethra) was revealed.

In the study by Miller *et al.* (1978; described on p. 123) of cases of bladder cancer in Ottawa, Canada, the RR for drinking tea (users *versus* nonusers) was close to unity for people of each sex. No quantitative data were provided on doses.

In the Canadian study of Howe *et al.* (1980; described on p. 123), the RRs for tea drinkers *versus* nondrinkers were 1.0 for men and 0.5 for women.

In the largest case-control study on bladder cancer carried out in the USA (Hartge *et al.*, 1983; described on p. 124), the relation between tea consumption and bladder cancer risk was examined among subjects who drank no more than seven cups of coffee per week. No evidence of an association was found for people of either sex, and the trends in risk with dose were nonsignificant.

In a case-control study conducted in 1976-78 in metropolitan Nagoya, Japan (Ohno *et al.*, 1985; described on p. 124), no relation was evident with consumption of black tea.

In the study by Jensen *et al.* (1986); described on p. 126) on bladder cancer in Denmark, a significant, direct relationship with dose of tea emerged among men, but not among women. The overall trend in risk associated with tea drinking in the

two sexes combined, adjusted for age, sex, smoking, coffee and soft drinks, was significant. The authors noted that the association with tea might be related to the unexpected finding of a positive association between total beverage consumption and bladder cancer risk in this study.

In a case-control study of bladder cancer in the Federal Republic of Germany (Claude *et al.*, 1986; described on p. 126), the estimated RRs for drinking black tea were above unity in men and women, but the trends in risk with dose were not significant. In this study, a positive association was found with total daily fluid intake, with a particularly high RR in men. [The Working Group noted the possible influence of the use of urological controls on these estimates.]

In the study by Iscovich *et al.* (1987; described on p. 127) in La Plata, Argentina, the RR for tea drinking was above unity. However, only four cases drank three or more cups per day, and the trend in risk with dose was not significant.

In a population-based case-control study from Utah, USA (Slattery *et al.*, 1988; described on p. 127), data for tea consumption were presented separately for people who had never smoked and those who had ever smoked. The authors stated that the risk increased in nonsmokers with number of cups of tea per week. [The Working Group noted that, when the data were examined globally, however, there was no appreciable association with tea. The RRs, as calculated by the Working Group, were 1.0 for one to three cups per week and 1.2 for more than three cups per week.]

In the study by Risch *et al.* (1988; described on p. 128), on bladder cancer in Canada, no association was found with various measures of tea consumption: the RR for 100 cup-years, derived from a multiple logistic model, was 1.0 for men and women.

In a study from northern Italy (La Vecchia *et al.*, 1989; described on p. 128), univariate analysis suggested a reduced bladder cancer risk among tea drinkers (RR, 0.5; [95%, 0.3-0.8]), but the risk rose to 0.8 (nonsignificant) after multivariate analysis.

Renal pelvis and ureter: The etiology and pathogenesis of transitional-cell cancer of the renal pelvis and ureter are in several aspects similar to those of bladder cancer, although the frequency of cancers at these sites is much lower and, hence, the studies are based on small data sets.

A matched hospital-based study of 33 cases of cancer of the renal pelvis and 33 controls in the UK (Amstrong *et al.*, 1976) found no association with tea drinking, although no risk estimate was reported.

Table 21. Summary of results of case–control studies of bladder cancer and tea consumption

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Morgan & Jain (1974) Canada	Men and women (232, 232)	0 < 1 cup/day 1–< 3 cups/day 3–< 5 cups/day ≥5 cups/day	1 [0.5] [0.6] [1.1] [1.0]	Nonsignificant; adjusted for sex only
Simon *et al.* (1975) USA	Men and women (132, 384)	Nonuser < 1 cup/day 1–2 cups/day ≥3 cups/day	1 1.0 1.3 0.8	Nonsignificant; unadjusted
Miller *et al.* (1978) Canada	Men (183, 366) Women (72, 144)	Users *versus* nonusers	1.1 0.9	Nonsignificant; unadjusted Nonsignificant
Howe *et al.* (1980) Canada	Discordant pairs[a] Men (80/79) Women (12/26)	Users *versus* nonusers	1.0 (0.7–1.4) 0.5 (0.2–1.0)	Nonsignificant; unadjusted estimates from matched analysis Nonsignificant
Hartge *et al.* (1983) USA	Men (455, 1106)	Nonuser < 7 cups/week 7–14 cups/week > 14 cups/week	1 1.1 1.1 1.0	Nonsignificant; adjusted for age, race, geographical area, tobacco and coffee
	Women (204, 452)	Nonuser < 7 cups/week 7–14 cups/week > 14 cups/week	1 1.1 1.7 1.2	Nonsignificant
Ohno *et al.* (1985) Japan	Men (227, 443) Women (65, 146)	Users *versus* nonusers	1.0 (0.7–1.3) 0.6 (0.3–1.0)	Nonsignificant; adjusted for age and smoking Nonsignificant
Jensen *et al.* (1986) Denmark	Men and women (371, 771)	0 < 0.5 l/day 0.5–0.99 l/day ≥1 l/day	1 0.8 2.0 1.5	$p = 0.03$; adjusted for age, sex, smoking (never/current and lifetime pack-years), coffee and soft drinks

Table 20 (contd)

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Claude *et al.* (1986) Federal Republic of Germany	Men (340, 340)	0 1–2 cups/day 3–4 cups/day >4 cups/day	1 1.0 1.5 1.9	Nonsignificant; adjusted for smoking (never/ever and lifetime packs)
	Women (91, 91)	0 1–2 cups/day 3–4 cups/day >4 cups/day	1 1.9 1.3 1.9	Nonsignificant
Iscovich *et al.* (1987) Argentina	Men and women (99, 99)	0 1 cup/day 2 cups/day ≥3 cups/day	1 1.5 1.2 1.4	Nonsignificant; adjusted for smoking
Slattery *et al.* (1988) USA	Men and women (413, 886)	0 1–3 cups/week ≥4 cups/week	1 [1.0] [1.2]	Nonsignificant; adjusted for smoking (never/ever) from published data; calculated by the Working Group
Risch *et al.* (1988) Canada	Men and women (826, 792)	Per 100 cup-years	1.0 (0.9–1.1) 1.0 (0.8–1.2)	Adjusted for smoking (cumulated pack-years) and history of diabetes
La Vecchia *et al.* (1989) Italy	Men and women (163, 181)	Users *versus* nonusers	0.8 [0.4–1.3]	Adjusted for age, area of residence, social class and selected indicator foods

[a]Patient ever used, control never used/patient never used, control ever used

A population-based case-control study of 74 cases (50 men and 24 women) of cancer of the renal pelvis and 697 controls from the USA (McLaughlin *et al.*, 1983) showed a positive trend with tea drinking in women only. The RRs, adjusted for smoking, were 2.6 for two cups per day, based on three cases, and 18.8 for three cups or more, based on nine cases. [The Working Group noted that the same control group was used in this study and in that on renal-cell cancer described below.]

Kidney: Among the studies in which tea was considered in the epidemiology of renal-cell cancers, four (Armstrong *et al.*, 1976; Goodman *et al.*, 1986, ≥2 cups/day; RR, 1.4 (0.9-2.2); Yu *et al.*, 1986, RR for daily consumption, 0.6; McCredie *et al.*, 1988) found no association, and one (McLaughlin *et al.*, 1984) found an association with drinking ≥3 cups/day in women only (RR, 3.4; 95% CI, 1.4-8.9), although the trend in risk was largely inconsistent.

(ii) *Pancreas* (see Table 22)

In the study of Kinlen and McPherson (1984), described on p. 136, a positive trend for tea consumption was found, which was not significant.

In several other case-control studies (MacMahon *et al.*, 1981; Mack *et al.*, 1986; La Vecchia *et al.*, 1987; Raymond *et al.*, 1987; Cuzik & Babiker, 1989; described on pp. 135, 137, 138, 139), no association was found between tea consumption and pancreatic cancer.

Table 22. Summary case–control studies of tea consumption and pancreatic cancer

Reference, location	Subjects (cases, controls)	Tea consumption (cups/day)	RR	Comments
MacMahon *et al.* (1981) USA	Men (216, 306)	0	1.0	$\chi^2 = 1.4$
		1–2	0.7	
		≥3	0.8	
	Women (150, 336)	0	1.0	$\chi^2 = 1.9$
		1–2	0.7	
		≥3	0.6	
Kinlen & McPherson (1984) UK	Men and women (216, 432)	0–2	1.0	
		3–4	2.2	Significant
		5–9	2.3	Significant
		≥10	2.5	
	Men and women	0–2	1.0	Adjusted for smoking and coffee
		3–4	2.3	Significant
		5–9	2.3	Significant
		≥10	2.6	
Mack *et al.* (1986) USA	Men and women (490, 490)	0	1.0	Crude odds ratio
		1–4	0.7	
		≥5	0.7	
La Vecchia *et al.* (1987) Italy	Men and women (150, 605)	0	1.0	
		1	0.9	
		≥2	1.3	
	Men and women	0	1.0	Adjusted for smoking and occupation
		1	0.8	
		≥2	1.1	

Table 22 (contd)

Reference, location	Subjects (cases, controls)	Tea consumption (cups/day)	RR	Comments
Raymond *et al.* (1987) Switzerland	Men and women (88, 336)	0 <910 ml/week >910 ml/week	1.0 2.2 (1.1–4.3) 1.0 (0.5–2.0)	90% CI
Cuzik & Babiker (1989) UK	Men and women (216, 279)	0–2 3–4 5–6 ≥7	1.0 0.8 0.9 0.9	Adjusted for smoking; χ^2 = 0.01
	Men and women	0–2 3–4 5–6 ≥7	1.0 1.2 1.1 1.5	Consumption ~10 years previously; adjusted for smoking; χ^2 = 1.0

(iii) *Breast*

Case-control studies of breast cancer and tea consumption are summarized in Table 23.

A study by Lawson *et al.* (1981), in which coffee and tea drinking were grouped, is described on p. 144.

In the hospital-based case-control study in Israel of Lubin *et al.* (1984, 1985; described on p. 144), no association between tea consumption and breast cancer risk was found.

In the study of Rosenberg *et al.* (1984, 1985; described on p. 145), using data from a case-control study in eastern USA, the consumption of tea was similar among all cases and controls and among those cases and controls who did not drink caffeine-containing coffee.

In the study by La Vecchia *et al.* (1986; described on p. 145), the RRs for drinkers of one and two or more cups per day were 1.3 and 1.0.

In the study by Schairer *et al.* (1987; described on p. 146), results were given on consumption of non-herbal tea: RRs were 0.9, 0.8, 1.3, 1.0 and 0.6 for increasing levels of consumption.

In the study of Mabuchi *et al.* (1985a; see p. 147), on risk factors for male breast cancer in a large number of hospitals in five US metropolitan areas, 42% of cases and 54% of controls drank one cup or more of tea per day; the difference was no significant.

Table 23. Summary of results of case–control studies of breast cancer and tea consumption

Reference, location	Subjects (cases, controls)	Tea consumption (cups/day)	Relative risk (95% confidence interval)	Comments
Lubin *et al.* (1984, 1985) Israel	Women (731, 731) surgical controls	0 1 2–3 ≥4	1.0 1.0 (0.6–1.4) 0.9 (0.5–1.2) 0.9 (0.4–1.8)	Matched for age, country of origin, length of residence in Israel
	(804, 804) neighbourhood controls	0 1 2–3 ≥4	1.0 0.8 (0.6–1.4) 0.8 (0.5–1.2) 0.8 (0.4–1.8)	
Rosenberg *et al.* (1985) USA	Women (2651, 1501) noncancer controls	0 1–2 3–4 ≥5	1.0 1.0 (0.8–1.1) 0.8 (0.6–1.0) 0.6 (0.5–0.9)	Adjusted for age within five years
	(2651, 385) cancer controls	0 1–2 3–4 ≥5	1.0 0.9 (0.7–1.2) 1.1 (0.7–1.6) 0.8 (0.5–1.4)	
La Vecchia *et al.* (1986) Italy	Women (616, 616)	0 1 ≥2	1.0 1.3 1.0	
Schairer *et al.* (1987) USA	Women (1510, 1882)	0 ≤1 2 3 4 ≥5	1.0 0.9 (0.8–1.1) 0.8 (0.6–1.1) 1.3 (0.8–2.1) 1.0 (0.5–2.3) 0.6 (0.2–1.9)	
Mabuchi *et al.* (1985a) USA	Men (52, 52)	<1 ≥1		42% *versus* 54%; nonsignificant; matched on age, race, marital status

(iv) *Ovary* (see Table 24)

In the study by Byers *et al.* (1983; described on p. 150), there was no significant association with any consumption category or dose trend of tea among ovarian cancer patients in Buffalo, NY, USA.

In the North American multicentre study of Miller *et al.* (1984, 1987; described on p. 150), no association was found between tea consumption and ovarian cancer.

Table 24. Summary of case–control studies of ovarian cancer (common epithelial tumours) and tea intake

Reference, location	Subjects (cases, controls)	Tea consumption (cups/day)	Relative risk (95% confidence interval)	Comment
Byers *et al.* (1983) USA	274, 1034	0	1.0	No significant association with any consumption category or trend
		<3	1.1	
		≥3	0.8	
Miller *et al.* (1984, 1987) Several cities in the USA and Canada	290 cases 480 noncancer controls	0	1.0	
		1–2	0.8 (0.6–1.2)	
		3–4	0.8 (0.5–1.4)	
		≥5	0.5 (0.2–1.0)	
	376 cancer controls	0	1.0	
		1–2	0.7 (0.5–1.0)	
		3–4	1.1 (0.6–2.1)	
		≥5	0.7 (0.3–1.6)	
	856 combined controls	0	1.0	
		1–2	[0.8]	
		3–4	[1.0]	
		≥5	[0.6]	

(v) *Cancers of the digestive tract*

Large bowel: Case-control studies on colorectal cancer and tea consumption are summarized in Table 25.

In the study of Higginson (1966; described on p. 152), no significant association was found between tea consumption and colorectal cancer in patients in Kansas City, USA.

In the study by Watanabe *et al.* (1984; described on p. 155), drinking black tea was negatively associated with the risk for rectal cancer in cases in Kyoto, Japan. There was a possible dose-response relationship (the RR for non-daily drinkers was 0.5 and for daily drinkers, 0.4). The positive association between rectal cancer and drinking green tea was not significant, nor were the associations between colon cancer and the consumption of black or green tea.

In the study of Tajima and Tominaga (1985; described on p. 155) in Nagoya, Japan, there was no significant association between black or green tea drinking and cancers of the colon or rectum.

In the large case-control study in Milan, Italy, conducted by La Vecchia *et al.* (1988; described on p. 156), subjects with a higher intake of tea had RRs of 1.4 for colon cancer [$p = 0.06$] and 1.5 for rectal cancer ($p < 0.05$).

Table 25. Summary of results of case–control studies on colorectal cancer and tea consumption

Reference, location, site	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Higginson (1966) USA Colon and rectum	Men and women (340, 1020)	Never/irregular	1.0	Nonsignificant
		< 1 cup/day	[1.0]	
		< 2 cups/day	[1.1]	
		≥3 cups/day	[0.5]	
Watanabe *et al.* (1984) Japan Rectum	Men and women (65, 65)	Green tea		Crude RR
		Nondrinkers	1.0	
		Drinkers	3.0 (0.4–25.8)	
		Black tea		
		Nondrinkers	1.0	
		Drinkers	0.5 (0.2–1.0)	
		Frequency		
		Never	1.0	
		Daily	0.4 (0.1–1.6)	
		Sometimes	0.5 (0.2–1.1)	
Colon	Men and women (138, 138)	Green tea		
		Nondrinkers	1.0	
		Drinkers	1.3 (0.3–5.9)	
		Black tea		
		Nondrinkers	1.0	
		Drinkers	1.1 (0.7–1.7)	
Tajima & Tominaga (1985) Japan Rectum	Men and women (51, 186)	Green tea		Adjusted for age and sex; nonsignificant
		0–3 times/day	1.0	
		≥4 times/day	0.9	
		Black tea		
		Nondrinkers	1.0	
		Drinkers	0.9	Nonsignificant
Colon	Men and women (42, 186)	Green tea		
		0–3 times/day	1.0	
		≥4 times/day	1.0	Nonsignificant
		Black tea		
		Nondrinkers	1.0	
		Drinkers	1.7	Nonsignificant
La Vecchia *et al.* (1988) Italy Rectum	Men and women (236, 778)	Low tertile	1.0	$p < 0.05$; adjusted for age, marital status, education, social class, smoking and alcohol consumption
		Intermediate/ high tertiles	1.5	

Table 25 (contd)

Reference, location, site	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Colon	Men and women (339, 778)	Low tertile	1.0	
		Intermediate/ high tertiles	1.4	Nonsignificant

Stomach: Case-control studies on stomach cancer and tea consumption are summarized in Table 26.

In the studies of Higginson (1966; see p. 152), in the Kansas City area, USA, of Graham *et al.* (1967; see p. 161) in Buffalo, NY, USA, of Tajima and Tominaga (1985; see p. 155) in Nagoya, Japan, and of Trichopoulos *et al.* (1985; see p. 161) in Piraeus, Greece, there was no significant association between drinking tea and cancer of the stomach.

Kono *et al.* (1988) studied 139 incident cases of gastric cancer in Kyushu, Japan. Two control groups were used: 2574 controls were subjects screened for gastrointestinal conditions but found to be healthy, and 278 (two per case) were population controls from ten neighbouring municipalities, matched to the cases by sex and year of birth. Individuals consuming ten or more cups of green tea per day tended to have a lower risk for gastric cancer. There was no difference, however, between individuals drinking none to four cups and five to nine cups per day.

Table 26. Summary of results of case–control studies on gastric cancer and tea consumption

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Higginson (1966) USA	Men and women (93, 279)	Never/irregular	1.0	Nonsignificant
		< 1 cup/day	[0.9]	
		< 2 cups/day	[1.3]	
		≥3 cups/day	[1.3]	
Graham *et al.* (1967) USA	Men and women (276, 2221)			No association
Tajima & Tominaga (1985) Japan	Men and women (93, 186)	Green tea		Nonsignificant; adjusted for age and sex
		0–4 times/day	1.0	
		≥5 times/day	0.6	
		Black tea		Nonsignificant
		Nondrinkers	1.0	
		Drinkers	0.8	

Table 26 (contd)

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Trichopoulos *et al.* (1985) Greece	Men and women (110, 100)	1 (low) 2 3 4 5 (high)	1.0 [1.7] [1.8] [2.7] [3.2]	Nonsignificant; adjusted for age, sex, years of schooling; p value based on chi-squared test for linear trend; coffee and tea intakes added
Kono *et al.* (1988) Japan	Men and women (139, 2574)	0–9 cups/day ≥10 cups/day	1.0 0.5 (0.3–1.1)	p = 0.10; hospital controls; adjusted for age, sex, smoking, consumption of oranges and other fruit
	Men and women (139, 278)	0–9 cups/day ≥10 cups/day	1.0 0.3 (0.1–0.7)	p = 0.007; population controls

Oesophagus: Case-control studies on oesophageal cancer and tea consumption are summarized in Table 27.

Kaufman *et al.* (1965) studied 82 cases of oesophageal cancer and 73 controls in Kazakhstan, USSR, and later added 51 cases from another area (effective numbers, 127 cases and 72 controls). Drinking very hot tea was associated with a three-fold higher risk of cancer.

In the same region, Bashirov *et al.* (1968) carried out a comparison of 301 cases of oesophageal cancer (142 men and 159 women) and 301 healthy population controls. Cases and controls had smoked for similar durations and used nass to the same extent. Oesophageal cancer was more common among those who reported drinking six or more cups of hot black tea at a single sitting than among those who did not.

In the study of de Jong *et al.* (1974; described on p. 163) among Singapore Chinese, there was no association between tea drinking and oesophageal cancer; however, drinking of 'burning hot' tea was associated with a three- to four-fold higher risk.

In northern Iran, Cook-Mozaffari *et al.* (1979) studied 344 patients with oesophageal cancer registered at the Caspian cancer registry between January 1975 and 1976. These comprised 54% of the patients registered during that period. Two population controls were chosen per case, matched for village of residence, age, sex and language group. A second group of 181 patients with other cancers (of whom

approximately 50% had stomach cancer) were also matched to two neighbourhood controls. Drinking hot tea was associated with a doubling in the risk of oesophageal cancer in males and females ($p < 0.01$). Hot-tea drinking was also associated with a three-fold higher risk of the other cancers studied among men, but with no excess among women.

In the study of Victora *et al.* (1987; see p. 280) in southern Brazil, cases and controls did not differ significantly in the frequency of tea intake, although no data were given.

Table 27. Summary of results of case–control studies on oesophageal cancer and tea consumption

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk	Comments
Kaufman *et al.* (1965) USSR	Men and women (127, 72)	Drinking of very hot tea		[$p < 0.001$]
		No	1.0	
		Yes	[3.2]	
Bashirov *et al.* (1968) USSR	Men (142, 142)	Glasses of hot tea at a time		[$p < 0.01$]
		<7	1.0	
		≥ 7	[2.6]	
	Women (159, 159)	Glasses of hot tea at a time		Nonsignificant
		<7	1.0	
		≥ 7	[3.0]	
de Jong *et al.* (1974) Singapore	Men (95, 465)	Not daily	1.0	Nonsignificant; adjusted for dialect group
		Daily	0.8	
		Burning hot	3.7	$p < 0.01$
	Women (36, 200)	Not daily	1.0	
		Daily	0.8	Nonsignificant
		Burning hot	3.5	$p < 0.05$
Cook-Mozaffari *et al.* (1979) Iran	Men (217, 434)	Drinking hot tea		
		No	1.0	$p < 0.01$
		Yes	1.7	
	Women (127, 254)	No	1.0	$p < 0.01$
		Yes	2.2	
Victora *et al.* (1987) Brazil	Men and women (171, 342)			No association

Other digestive tract (Table 28): In the study of Yen *et al.* (1987; described on p. 163) in eastern USA, subjects who consumed tea had half the risk for cancer of the extrahepatic bile ducts than people who had never drunk tea. This difference was marginally significant, but there was no suggestion of a dose-response relationship.

In the study of Franco *et al.* (1989; described on p. 164), in Brazil, no association was found between the frequency of tea drinking and oral cancer.

Table 28. Summary of results of case–control studies on other digestive tract cancers

Reference, location, site	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Yen *et al.* (1987) USA Extrahepatic bile duct	Men and women (67, 275)	0 Ever Occasionally 1–2 cups/day 3–4 cups/day ≥5 cups/day	1.0 0.5 (0.3–1.0) 0.4 (0.2–0.9) 0.6 (0.3–1.3) 0.5 (0.2–1.4) 0.8 (0.2–3.3)	Adjusted for age and sex
Franco *et al.* (1989) Brazil Oral cavity	Men and women (232, 464)	< 1 cup/month 1–30 cups/month > 30 cups/month	1.0 0.9 (0.6–1.3) 1.0 (0.6–1.7)	Nonsignificant; crude matched analysis; *p* level based on test for linear trend
		< 1 cup/month 1–30 cups/month > 30 cups/month	1.0 0.9 (0.6–1.3) 1.3 (0.7–2.3)	Nonsignificant; adjusted for tobacco and alcohol consumption

(vi) *Nasopharynx*

Case-control studies of nasopharyngeal cancers (NPC) and tea consumption are summarized in Table 29.

Lin *et al.* (1973) studied 343 patients with NPC and 1017 controls in Taiwan. No association was found between tea drinking and NPC, but no data were given.

In the study of Henderson *et al.* (1976; described on p. 165), tea drinking was associated with a significantly decreased risk for NPC ($p = 0.02$). Cases with other pharyngeal cancers, however, were not more likely to drink tea (RR, 1.4; $p = 0.3$).

Shanmugaratnam *et al.* (1978) investigated the association between tea drinking and NPC in Singapore. The case series consisted of 379 Chinese patients (266 men, 113 women) with histologically confirmed NPC in Singapore between 1966 and 1968. Two control groups were recruited: the first were 595 Chinese patients from the ear, nose and throat department at the same hospital, and the

second were 1044 Chinese patients in the medical, surgical and orthopaedic wards of another hospital. No difference was found regarding the frequency of consumption of Chinese tea between cases and ear, nose and throat controls; the comparison with other hospital controls showed no consistent association.

Table 29. Summary of results of case–control studies on nasopharyngeal cancers and tea consumption

Reference, location	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Henderson *et al.* (1976) USA	Men and women (156, 267)	0 Drinkers	1.0 0.5 [0.3–1.0]	$p = 0.02$; adjusted for age, sex, race, socio-economic status and place of residence
Shanmugaratnam *et al.* (1978) Singapore	Men and women (379, 595)	< once/month < once/day Daily	0.9 0.9 1.2	Nonsignificant; ear, nose and throat controls; adjusted for sex, age and interviewer
	Men and women (379, 1044)	< once/month < once/day Daily	1.1 0.7 1.3	$p < 0.05$; other hospital controls

(vii) *Other sites*

Case-control studies of cancers at other sites and tea consumption are summarized in Table 30.

In the study of Mabuchi *et al.* (1985b; described on p. 166) of carcinoma of the vulva in five US metropolitan areas, no association was found between the consumption of tea and cancer of the vulva.

Bunin *et al* (1987) studied tea drinking during pregnancy and Wilms' tumour in the offspring. Cases were white children under 15 years of age identified through the registries of the three main childhood cancer hospitals in Philadelphia, PA, during 1970-83. About 30% of eligible cases could not be interviewed. Tea consumption during pregnancy was associated with a doubling of the risk for Wilms' tumour in the offspring ($p < 0.05$).

[The Working Group noted that, unless otherwise specified, the studies did not distinguish different types of tea (green, black or herbal). Furthermore, the effect of the consumption of other hot beverages was not considered in most of the studies. Consequently, non-drinkers of tea could represent drinkers of other hot beverages, including coffee.]

Table 30. Summary of results of case–control studies on other cancers and tea consumption

Reference, location, site	Subjects (cases, controls)	Tea consumption	Relative risk (95% CI)	Comments
Mabuchi *et al.* (1985b) USA Vulva	Women (149, 149)	< 1 cup/day 1–2 cups/day 3–4 cups/day ≥5 cups/day	1.0 0.8 1.1 1.1	Nonsignificant; unmatched analysis
Bunin *et al.* (1987) USA Wilms' tumour	Boys and girls (88, 88)	Tea intake of mother during pregnancy 0 Drinkers	 1.0 2.2 (1.0–4.7)	Matched analysis $p < 0.05$

4. Summary of Data Reported and Evaluation

4.1 Exposure data

Tea is an aqueous infusion prepared from the dried leaves of *Camellia sinensis*, which has been consumed since ancient times in Asia and since the late seventeenth century in most other parts of the world. Tea is the most widely consumed beverage in the world. About 80% of world production of tea is in Asian countries. Depending on manufacturing techniques, teas can be divided into two main types: black tea, which has undergone an enzymic oxidation called 'fermentation' during processing, and green tea, which has not. Black tea represents about 80% of world production.

Annual tea consumption varies from country to country, ranging from a high level of about 3 kg *per caput* to negligible values in many countries. World consumption is approximately 0.5 kg *per caput*. Green tea is the primary form consumed in China, Japan and some Middle Eastern countries. Instant tea and decaffeinated tea consumption is small, but the latter is becoming more significant in the USA.

Over 400 volatile compounds comprising many structural categories have been identified in black teas and over 200 in green teas; these contribute to the flavour and aroma of the beverage. In addition to the expected components of leaf matter (e.g., flavonols, flavanols and phenolic acids), other nonvolatile components are present; bisflavanols, theaflavins and thearubigins are found in black tea. Average caffeine levels in both black and green teas are 3-4% on a dry weight basis, resulting

in about 30-50 mg caffeine per cup. Some black and green teas have traditionally been flavoured with natural agents such as oil of bergamot and jasmine flowers.

4.2 Experimental carcinogenicity data

Tea was tested for carcinogenicity in one study in rats by repeated subcutaneous injection of a total aqueous extract of tea leaves. A nonsignificant increase in the incidence of local tumours was observed.

In a number of studies, various known carcinogens were administered by different routes either simultaneously or sequentially with tea or its constituents by various routes. In one study in mice, skin application of black tea infusion containing 1% tannin after a single application of benzo[*a*]pyrene did not affect the incidence of skin tumours.

Administration of polyphenolic extracts of green tea in combination with known carcinogens resulted in decreased incidences of skin tumours in mice treated with benzo[*a*]pyrene diol epoxide, 3-methylcholanthrene or 7,12-dimethylbenz[*a*]anthracene and of duodenal tumours in mice treated with *N*-ethyl-*N'*-nitro-*N*-nitrosoguanidine, within a limited period of observation.

4.3 Human carcinogenicity data

Correlation studies on cancer risk associated with tea consumption have provided inconsistent reports of increased risks for cancers of the breast, intestine, larynx, lung and colon. Ecological studies of villages in the Caspian littoral have shown a broad correspondence between the occurrence of oesophageal cancer and tea consumption. An additional report found a relationship with the temperature at which the tea was drunk. A geographical study showed that in areas of Japan with high reported consumption of tea-gruel there were higher mortality rates from oesophageal cancer.

(a) Bladder and urinary tract cancer

In two cohort studies in which bladder cancer risk was examined, no association was reported.

The overall evidence from 12 case-control studies indicates no consistent association between measures of tea consumption and risk for bladder cancer. Although the data are limited, a similar pattern of trend was apparent for transitional-cell cancers of the renal pelvis and ureter.

One cohort study found a positive dose-response relationship for cancer of the kidney, but there was inadequate adjustment for confounding. Case-control studies on adenocarcinoma of the kidney are scarce and do not provide evidence of an association with tea drinking.

(*b*) *Pancreatic cancer*

The effect of tea consumption was examined in four cohort studies: three reported no association, and one documented a small protective effect.

Six case-control studies were designed to evaluate the relationship between tea consumption and pancreatic cancer: one showed a positive association.

(*c*) *Breast cancer*

None of five studies in which results on tea consumption were presented showed an association with breast cancer.

(*d*) *Ovarian cancer*

In two case-control studies, there was no association between tea consumption and ovarian cancer.

(*e*) *Cancer of the large bowel*

One cohort study found a strong positive dose-response relationship for cancer of the rectum, but another indicated no relationship with rectal cancer and a nonsignificant 'protective' effect for colon cancer.

The association between tea consumption and cancer of the colon and rectum was investigated in four case-control studies. Two showed no association. One study found a decreased risk for cancer of the rectum but not for cancer of the colon among drinkers of black tea relative to nondrinkers; another found an increased risk in the high consumption group. Taken together, these studies do not suggest the existence of an association.

(*f*) *Gastric cancer*

One cohort study found an increased risk for gastric cancer, which remained after inadequate adjustment for social class.

The role of tea drinking as a risk factor for cancer of the stomach was considered in five case-control studies. Four of these found no association. A negative association was observed in one study, but no dose-response relationship was seen.

(*g*) *Cancer of the oesophagus*

Five case-control studies were carried out, in Iran, the USSR, Brazil and Singapore, to investigate the effect of tea drinking on the frequency of cancer of the oesophagus. One study in Brazil did not show an association between tea drinking

and oesophageal cancer, but the subjects were not asked about the temperature at which they drank tea. The other four studies, three of which were conducted in the Caspian area, stressed the role of the temperature of tea. All four studies showed that ingestion of very hot tea was associated with a two- to three-fold increase in the risk for oesophageal cancer. Only one of these studies investigated the effect of frequency of tea ingestion irrespective of temperature; no association was found. Taken together, these studies suggest that the temperature may be more important than the composition of the beverage, but the results are not conclusive.

One case-control study on oral cancer and one on cancer of the extrahepatic bile ducts reported no clear association with tea drinking.

(*h*) *Nasopharyngeal cancer*

Three case-control studies showed no evidence of an association between tea drinking and nasopharyngeal cancer.

(*i*) *Cancers at other sites*

One cohort study found no association with liver cancer. Another showed a significant positive dose-response relationship for lung cancer after adjusting for age and smoking; these findings could, however, be attributed to residual confounding by smoking.

One case-control study showed no association between tea drinking and cancer of the vulva. Another indicated a possible effect of maternal tea drinking during pregnancy on the frequency of Wilms' tumour in the offspring.

4.4 Other relevant data

The few informative studies concerning the effect of tea consumption during pregnancy on the frequency of adverse reproductive effects did not show an association.

In a number of studies, no association was seen between consumption of tea and the frequency of coronary heart disease.

Black tea, green tea and several unspecified teas were mutagenic to bacteria. Teas were found to reduce the activity of known mutagens both *in vivo* and *in vitro*.

4.5 Evaluation[1]

There is *inadequate evidence* for the carcinogenicity in humans of tea drinking.

There is *inadequate evidence* for the carcinogenicity in experimental animals of tea.

Overall evaluation

Tea is *not classifiable as to its carcinogenicity to humans (Group 3).*

5. References

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[1]For definition of the italicized terms, see Preamble, pp. 27-31.

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