Medicinal Plants of the World

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of the

World

Volume 3

Chemical Constituents, Traditional and Modern Medicinal Uses

By

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Preface

This volume of the series *Medicinal Plants of the World: Chemical Constituents, Modern and Traditional Medicinal Uses* contains information on 16 plant species and follows the same format as volumes 1 and 2. Some of the plants discussed in volume 3 may be considered controversial in their classification as "medicinal." However, the Paracelsian dictum that "sola dosis fecit venenum" has been appreciated since ancient times, and throughout the ages many highly toxic materials used for lethal purposes have also found applications in modern medicine. It has been recognized that plants contain substances that are either harmful or toxic. However, it is wrong to think that there are plant toxins that are known or that are likely to have adverse effects on any and every form of life. A common feature of most toxic plants is that they are also known for their curative properties, and although they may provide the cure for an individual's disease at one dose, they may cause the death of the same individual at another.

Poisons are widespread in plants, and humans have tried to either get rid of them or convert them to their own advantage. By their very nature, poisons are biodynamic substances because they affect, or are intended to affect, the functioning of the victims' body. This also means that they have been, and are, important sources of medicine. With such potentially dangerous substances, it also means that care in medication is essential, and it raises the question of the relationship between the toxic dose and the therapeutic dose. For full advantage to be taken of their properties, a combination of reliable sources of materials and effective methodologies is required to enable not only isolation of the substances responsible, but also the investigation of their mechanisms of action. As more sophisticated methods are evolved to elucidate their chemical and pharmacological natures, it will be possible to target more precisely the use of these substances as possible templates to produce medicinal agents.

I am very grateful to a number of individuals for their valuable cooperation in this work. I owe sincere appreciation to Professor Ron Olowin of St. Mary's College of California for granting me permission to use his photograph of *Plantago ovata* and Mr. Gary Monroe of Reno, Nevada for sharing his picture of *Larrea tridentata*.

In work of this nature there is always room for improvement. Suggestions from readers are welcome and will be gratefully received.

Ivan A. Ross

Acknowledgments

I am very grateful to Dr. Diana E. Dyrda of the University of Agriculture, Lublin, Poland for her contribution in collecting data and working on the manuscript, and to Yvonne Gordon for editing this work. Also, to our families for enduring our absence in their lives.

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Abbreviations Used in Chemical Constituents Sections

Aer	Aerial parts
An	Anther
As	Ash
Bd	Bud
Bk	Bark
Bu	Bulb
Call Tiss	Callus tissue
Cr	Crown
Ct	Coat
Cx	Calyx
Cy	Cotyledon
Em	Embryo
EO	Essential oil
Ep	Epidermis
Fl	Flower
Fr	Fruit
Gel	Jell
Hu	Hull
Ju	Juice
Lf	Leaf
Lx	Latex
Pc	Pericarp
Pe	Peel
Pl	Plant
Pn	Panicle
Pt	Part
Pu	Pulp
Rh	Rhizome
Rt	Root
Sd	Seed
Sh	Shoot
St	Stem
Tr	Trunk
Tu	Tuber
Tw	Twig

1 Camellia sinensis

L.

Common Names

Aisiksikimi	United States	Te	Denmark
Caj	Albania	Te	Faroe Islands
Caj	Croatia	Te	France
Caj	Czech Republic	Te	Italy
Caj	Hawaii	Te	Norway
Caj	Serbia	Te	Spain
Cay	Turkey	Te	Surinam
Ceai	Romania	Te	Switzerland
Cha	Brazil	Te	Wales
Cha	China	Tea plant	England
Cha	Hawaii	Tea	Australia
Cha	Japan	Tea	England
Cha	Pacific Islands	Tea	Guyana
Cha	Portugal	Tea	Hungary
Chai	Bulgaria	Tea	United States
Chai	Mozambique	Tebusk	Denmark
Chai	Russia	Tebuske	Sweden
Chai	Tanzania	Tee	Finland
Chai	Ukraine	Tee	Germany
Chai	Zaire	Tee	Netherlands
Chaj	Macedonia	Tee	South Africa
Chayna roslina	Ukraine	Teepensas	Finland
Chinesischer tea	Germany	Tey	The Isle of Man (Manx)
Cunuc yacu	Ecuador	Teye	Northern Sotho
Eaj	Czech Republic	The	France
Eajovnik	Czech Republic	The	Indonesia
Herbata	Poland	The	Malaysia
Icayi	Rwanda	Thee	Netherlands
Ilitye	Africa	Theesoort	Netherlands
Itiye	Africa	Theestrauch	Germany
Oti	United States	Theestruik	Netherlands
Taa	Germany	Theler	France
Tae	Ireland	Ti	Congo
Te	Cornwall	Ti	Samoa

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Ti	Scotland	Tra	Vietnam
Tii	Greenland	Tsa	Philippines
Tii	New Zealand	Yaku-q'oniwan	Ecuador
Tii	Northwest Territories, Canada	Zaya	Turkmenistan

BOTANICAL DESCRIPTION

Camellia sinensis is an evergreen tree or shrub of the THEACEAE family that grows to 10–15 m high in the wild, and 0.6–1.5 m under cultivation. The leaves are short-stalked, light green, coriaceous, alternate, elliptic-obovate or lanceolate, with serrate margin, glabrous, or sometimes pubescent beneath, varying in length from 5 to 30 cm, and about 4 cm wide. Young leaves are pubescent. Mature leaves are bright green in color, leathery, and smooth. Flowers are white, fragrant, 2.5–4 cm in diameter, solitary or in clusters of two to four. They have numerous stamens with yellow anthers and produces brownish-red, one- to four-lobed capsules. Each lobe contains one to three spherical or flattened brown seeds. There are numerous varieties and races of tea. There are three main groups of the cultivated forms: China, Assam, and hybrid tea, differing in form. *Camellia sinensis assamica*, the source of much of the commercial tea crop of Ceylon is a tree that, unpruned, may attain a height of 15 m and has proportionally longer, thinner leaves than typical species.

ORIGIN AND DISTRIBUTION

The cultivation and enjoyment of tea are recorded in Chinese literature of 2700 BC and in Japan about 1100. Through the Arabs, tea reached Europe about 1550. Native to Assam, Burma, and the Chinese province of Yunnan, it is highly regarded in southern Asia and planted in India, southern Russia, East Africa, Java, Ceylon, Sumatra, Argentina, and Turkey. China, India, Indonesia, and Japan produced about a half of the total world production.

TRADITIONAL MEDICINAL USES

India. Decoctions of the dried and fresh buds and leaves are taken orally for headache and fever[CS145]. Powder or decoction of the dried leaf is applied to teeth to prevent tooth decay[CS146]. Fresh leaf juice is taken orally for abortion[CS155], and as a contraceptive and hemostatic[CS147].

Mexico. Hot water extract of the leaf is taken orally by nursing mothers to increase milk production[CS148].

Turkey. Leaves are taken orally to treat diarrhea[CS149].

China. Hot water extract of the dried leaf is taken orally as a sedative, an antihypertensive, and anti-inflammatory[CS108].

Guatemala. Hot water extract of the dried leaf is used as eyewash for conjunctivitis[CS154].

Kenya. Water extract of the dried leaf is applied ophthalmically to treat corneal opacities[CS150]. The infusion is used for chalzion and conjunctivitis[CS151.]

Thailand. Hot water extract of the dried leaf is taken orally as a cardiotonic and neurotonic[CS152]. Hot water extract of the dried seed is taken orally as an antifungal[CS153].

CHEMICAL CONSTITUENTS

(ppm unless otherwise indicated)

Acetaldehyde, phenyl: Sh 1.52–1.78%[CS100]
Acetaldehyde: Lf[CS073]
Acetamide, *N*-ethyl: Lf[CS027]
Acetic acid: Lf[CS086]
Acetoin: Lf[CS068]
Acetone: Lf[CS091]
Acetophenone, 2-4-dimethyl: Lf[CS027]
Acetophenone, 3-4-dimethoxy: Lf[CS027]
Acetophenone, para-ethyl: Lf[CS027]

Acetophenone: Headspace volatile[CS044]
Actinidiolide, dihydro: Lf EO[CS132]
Afzelechin, epi, (–): Lf 350[CS084]
Afzelechin, epi, 3-*O*-gallate (–): Lf 37[CS005]
Afzelechin, epi, 3-*O*-gallate (4-b-6)-epi, gallocatechin-3-*O*-gallate: Lf 5.6[CS008]
Allantoic acid: Pl[CS033]
Allantoin: Pl[CS033]
Aluminium inorganic: Lf[CS028]
Amyrin, α: Sd oil[CS095]
Amyrin, β: Sd oil 76[CS095]
Aniline, *N*-ethyl: Lf[CS027]
Aniline, *N*-methyl: Lf[CS027]
Aniline: Lf[CS027]
Apigenin: Lf[CS108]
Apigenin-6-8-di-*C*-β-D-arabinopyranosyl: Lf 20[CS156]
Apigenin-6-8-di-*C*-glucoside: Sh[CS096]
Arbutin: Lf 0.2[CS078]
Aromadenrin: Sh[CS094]
Ascorbic acid: Sh[CS038], Lf 0.257%[CS048]
Assamicain A: Lf 58.2[CS007]
Assamicain B: Lf 76.6[CS007]
Assamicain C: Lf 33.6[CS007]
Assamsaponin A: Sd 0.01%[CS021]
Assamsaponin B: Sd 28.3[CS021]
Assamsaponin C: Sd 36.5[CS021]
Assamsaponin D: Sd 26.1[CS021]
Assamsaponin E: Sd 11.1[CS021]
Assamsaponin F: Sd 14.1[CS021]
Assamsaponin G: Sd 79.1[CS021]
Assamsaponin H: Sd 13.4[CS021]
Assamsaponin I: Sd 98.5[CS021]
Astragalin: Lf[CS139]
Avicularin: Lf[CS058]
Barrigenol, A-1: Pl[CS118]
Barringtogenol C, 3-*O*-β-D-galacto-pyranosyl(1-2) β-D-xylopyranosyl (1-2)α-l-arabinopyranosyl(1-3)β-D-glucuronopyranosyl-21-*O*-cinnamoyl-16-22-di-*O*-acetyl: Lf[CS013]
Benzene, 1-2-3-trimethoxy: Lf[CS077]
Benzene, 1-2-3-trimethoxy-5-ethyl: Lf[CS077]
Benzene, 1-2-3-trimethoxy-5-methyl: Lf[CS077]
Benzene, 1-2-4-trihydroxy: Lf[CS003]
Benzene, 1-2-5-trihydroxy: Lf[CS003]
Benzene, 1-2-dimethoxy: Lf[CS077]
Benzene, 1-2-dimethoxy-4-ethyl: Lf[CS077]
Benzene, 1-2-dimethoxy-4-methyl: Lf[CS077]
Benzene, 1-3-diacetyl: Lf[CS027]
Benzene, 1-4-diacetyl: Lf[CS027]
Benzoic acid: Headspace volatile[CS044]
Benzothiazole, 2-methyl: Lf[CS036]
Benzothiazole: Lf[CS036]
Benzoxazole: Lf[CS036]
Benzyl alcohol: Lf EO 1.01–1.6%[CS136], Headspace volatile[CS044], Lf[CS091], Sh 0.09–0.14%[CS100]
Benzyl butyrate: Lf[CS002]
Benzyl ethyl ketone: Lf[CS002]
Benzylaldehyde, 2-methyl: Lf[CS002]
Benzylaldehyde, 4-methoxy: Lf[CS002]
Benzylaldehyde: Headspace volatile[CS044], Lf[CS077], Sh 0.21–0.23%[CS100]
Benzylamine: *N*-*N*-dimethyl: Lf[CS027]
Bicyclo(4.3.0)non-8-en-7-one, 1-5-5-9-tetramethyl: Lf[CS002]
Brassicasterol: Sd oil[CS134]
Brassinolide, 28-homo, 6-keto: Lf[CS135]
Brassinolide, 28-nor, 6-keto: Lf[CS135]
Brassinolide, 28-nor: Lf[CS135]
Brassinolide, 6-keto: Lf[CS135]
Brassinolide: Lf 0.0046 ppb[CS089]
Brassinone, 24(*S*)-ethyl: Lf 30 ng/65 kg[CS110]
Brassinone, 24-ethyl: Lf[CS089]
Brassinone: Lf 130 ng/65 kg[CS110]
Butan-2-ol: Lf[CS068]
Butyrate, ethyl-3-hydroxy: Lf[CS068]
Butyroin: Lf[CS068]
Butyrospermol: Sd oil[CS095]
Caffeine: Lf 0.381–9.9%[CS114,CS049], Sh[CS038], Pl, Call Tiss[CS050], Sd[CS093], Sd Ct, Peduncle, Pc[CS102], Fl bud, Stamen, Pistil, Fl[CS107], An 0.05–6.77 ppt, Stem call 0.64 ppt[CS099], Petal[CS117], Fr[CS037]
Camellia galactoglucan: Lf[CS067]
Camellia polysaccharide: Lf[CS122]
Camellia saponin B, deacyl: Lf[CS081]
Camellia sinensis polysaccharide TSA: Lf[CS012]
Camellianin A: Lf[CS108]

Camellianin B: Lf[CS108]
Camelliaside A: Sd 656–2733.3[CS011,CS119]
Camelliaside B: Sd 291–3026.6 [CS011,CS119]
Camelliaside C: Sd 2.5[CS011]
Campesterol: Sd oil[CS134]
Carvacrol: Lf[CS002]
Castasterone: Lf 7.2 mg/65 kg[CS110]
Catechin-(4-α-8)-epi-gallocatechin: Lf 45.4[CS008]
Catechin-(4-α-8)-epi-gallocatechin-3-gallate: Lf 45.4[CS008]
Catechin-(4-β-8)-epi-gallocatechin-3-gallate, epi: Lf 20.8[CS008]
Catechin, (+): Lf 0.0017–2.9%[CS053,CS049], Call Tiss[CS060]. St call[CS099], Sh[CS038], An, St[CS099]
Catechin, epi (–): Lf 0.004–6.8%[CS053,CS049], Call Tiss[CS060], Sh[CS038], St call 0.07 ppt[CS099]
Catechin, epi, 3-*O*-para-hydroxy-benzoate (–): Lf 3.6[CS005]
Catechin, epi, epi-gallo-catechin(4-β-8)-3-*O*-galloyl: Lf 50[CS092]
Catechin, epi-gallo (–), 3-*O*-para-coumaroate: Lf 83.3[CS140]
Catechin, epi-gallo (–): Lf 3269[CS140]
Catechin, epi-gallo, 3-3'-di-*O*-gallate(–): Lf[CS140]
Catechin, epi-gallo, 3-4'-di-*O*-gallate(–): Lf[CS140]
Catechin, epi-gallo, 3-*O*-gallate (–): Lf 0.8718%[CS140]
Catechin, epi-gallo, gallate(–): St call 0.02 ppt[CS099], Lf[CS109]
Catechin, epi-gallo: Lf[CS140]
Catechin-3-*O*-(3'-*O*-methyl)-gallate, epi(–): Lf 0.08%[CS010]
Catechin-3-*O*-(3-*O*-methyl)-gallate, epi(–): Lf 70.6-96.2[CS005,CS140]
Catechin-3-*O*-(4-*O*-methyl)-gallate, epi(–): Lf 16[CS005]
Catechin-3-*O*-gallate-(4-β-6)-epi-gallocatechin-3-*O*-gallate, epi: Lf 5.8[CS008]
Catechin-3- *O*-gallate-(4-β-8)-epi-gallocatechin-3-*O*-gallate, epi: Lf 3.6[CS008]
Catechin-3-*O*-gallate, (+): Lf 0.011%[CS084]
Catechin-3-*O*-gallate, epi(–): Lf 0.0086–6.6%[CS053,CS049]
Catechin-gallate, (+): Lf[CS082]
Catechin-gallate: Lf[CS042]
Catechol, (+): Pl[CS138]
Catechol, epi(–): Sh[CS128], Pl[CS138]
Catechol, epi, gallate(–): Sh[CS128]
Catechol, epi-gallo(–): Sh[CS128]
Catechol, epi-gallo, gallate(–): Sh[CS128]
Catechol, gallo, (+): Sh[CS128]
Chasaponin: Pl[CS035]
Chlorogenic acid: Call Tiss[CS087], Lf[CS139]
Chondrillasterol: Sd oil[CS130]
Citric acid: Lf[CS086]
Cresol, meta: Lf[CS003]
Cresol, ortho: Lf[CS003]
Cresol, para: Lf[CS003]
Cyclocitral, β: Sh 0.08–0.1%[CS100], Lf[CS002]
Cyclohex-2-en-1-4-dione, 2-6-6-trimethyl: Lf[CS002]
Cyclohex-2-en-1-one, 2-6-6-trimethyl: Lf[CS002]
Damascenone, β: Lf[CS002]
Damascone, α: Lf[CS002]
Damascone, β: Lf[CS002]
Dammaridienol: Sd oil 30[CS095]
Deca-*trans*-2-*cis*-4-dien-1-al: Lf[CS002]
Deca-*trans*-2-en-1-al: Lf[CS002]
Dehydrogenase, NADP-dependent-alcohol: Sd[CS031]
Demmarenol, 24-methylene: Sd oil[CS095]
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Gadoleic acid: Sd oil[CS134]
Gallic acid: Lf[CS051]
Gallocatechin gallate, (–): Lf 0.188%[CS112]
Gallocatechin gallate, (+): Lf[CS082]
Gallocatechin gallate, epi(–): Lf[CS125]
Gallocatechin gallate, epi(+): Lf[CS123]
Gallocatechin-(4-α-8)-epi-catechin: Lf 36.6[CS008]
Gallocatechin, (–): Lf[CS056]
Gallocatechin, (+): Lf 0.01–12.8%[CS053,CS049]
Gallocatechin, epi(+): Lf 1.1%[CS083]
Gallocatechin, epi, (–): Lf 0.088-16.8%[CS005,CS049], Sh[CS038]
Gallocatechin, epi, (4-β-8)-epi-catechin-3-)-gallate: Lf 27.6[CS008]
Gallocatechin, epi, 3-*O*-cinnamate(–): Lf 13.2[CS005]
Gallocatechin, epi, 3-3'-di-*O*-gallate(–): Lf 9[CS005]
Gallocatechin, epi, 3-4'-di-*O*-gallate(–): Lf 9[CS005]
Gallocatechin, epi, 3-*O*-gallate(–): Lf 0.714%[CS005]
Gallocatechin, epi, 3-*O*-gallate-(4-β-6)-epi-catechin-3-*O*-gallate: Lf 4.2[CS008]
Gallocatechin, epi, 3-*O*-gallate-(4-β-8)-epi-catechin-3-*O*-gallate: Lf 44[CS008]
Gallocatechin, epi, 3-*O*-para-coumaroate(–): Lf 38.4[CS005]
Gallocatechin, epi, 8-*C*-ascorbyl-3-*O*-gallate: Lf 11.2[CS008]
Gallocatechin, epi: Lf 1.0867%[CS101]
Gallocatechin-3-5'-di-*O*-gallate, epi(–): Lf 0.06%[CS008]
Gallocatechin-3-*O*-(3'-*O*-methyl)-gallate, epi(–):Lf 38[CS084]
Gallocatechin-3-*O*-gallate (–): Lf[CS079]
Gallocatechin-3-*O*-gallate (+): Lf[CS157]
Gallocatechin-3-*O*-gallate (4-β-8) epi-catechin-gallate, epi: Lf 0.06%[CS010]
Gallocatechin-3-*O*-gallate, epi(–): Lf 0.0328–21.3%[CS053,CS049], Sh[CS038]
Gallocatechin-3-*O*-para-coumaroate, epi (–): Lf[CS010]
Gallocatechin-gallate, (–): Lf[CS042]
Gallocateuchin-3-*O*-gallate, epi (–): Lf 5.33%[CS010]
Galloyl-β-D-glucose, 1-4-6-tri-*O*: Lf 0.01%[CS010]
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Germanicum inorganic: Lf[CS120]
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Gibberellin A-19: Endosperm[CS004]
Gibberellin A-20: Endosperm[CS004]
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Gibberellin A-3: Endosperm[CS004]
Gibberellin A-38: Endosperm[CS004]
Gibberellin A-44: Endosperm[CS004]
Gibberellin A-8: Endosperm[CS004]
Gibberellin A-S: Endosperm[CS004]
Glucogallin, β: Lf 28.4[CS008]
Glucose, β-D, 1-0-galloyl-4-6-(–)-hexahyroxy-diphenoyl: Lf 30[CS092]
Glucose, β-D, 1-4-6-tri-*O*-galloyl: Lf 5[CS092]
Glutamic acid: *N*-para-coumaryl: Lf[CS133]
Heptan-1-al: Sh 0.02–0.03%[CS100]
Heptan-2-ol: Lf[CS068]
Heptan-2-one, 5-iso-propyl: Lf[CS002]
Heptan-2-one: Lf[CS002]
Heptan-3-ol: Lf[CS068]
Hepta-*trans*-2-*trans*-4-dien-1-al: Sh 0.06–0.1%[CS100]
Hept-*trans*-2-en-1-al: Lf[CS002]
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Hexadecane, N: Lf[CS091]
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Hexan-2-ol: Lf[CS068]
Hexa-*trans*-2-*cis*-4-dien-1-al: Lf[CS002]
Hex-*cis*-3-en-1-al: Lf 370[CS034]
Hex-*cis*-3-en-1-ol acetate: Lf[CS091]
Hex-*cis*-3-en-1-ol butyrate: Lf[CS091]
Hex-*cis*-3-en-1-ol caproate: Lf[CS091]

Hex-*cis*-3-en-1-ol formate: Lf[CS002]
Hex-*cis*-3-en-1-ol hexanoate: Sh 0.02–0.03%[CS100]
Hex-*cis*-3-en-1-ol hex-*trans*-2-enoate: Lf[CS002]
Hex-*cis*-3-en-1-ol propionate: Lf[CS002]
Hex-*cis*-3-en-1-ol, β-D-glucoside: Lf[CS076]
Hex-*cis*-3-en-1-ol: Lf[CS025], Lf EO 2.15–15%[CS136], Sh 0.09–0.13%[CS100]
Hex-*trans*-2-en-1-al: Lf[CS065], Lf EO 1.13–25.48%[CS136], Sh 2.09–3.1%[CS100]
Hex-*trans*-2-en-1-ol: Sh 0.04–0.06%[CS100]
Hex-*trans*-2-enyl acetate: Lf[CS002]
Hex-*trans*-2-enyl butyrate: Lf[CS002]
Hex-*trans*-2-enyl formate: Lf[CS002]
Hex-*trans*-2-enyl hexanoate: Lf[CS002]
Hex-*trans*-2-enyl propionate: Lf[CS002]
Hex-*trans*-3-enyl butyrate: Lf[CS002]
Hex-*trans*-3-enyl hex-*cis*-3-enoate: Lf[CS002]
Hex-*trans*-3-enyl propionate: Lf[CS002]
Hex-*trans*-3-enyl-2-methyl butyrate: Lf[CS002]
Hexyl butyrate: Lf[CS002]
Hexyl formate: Lf[CS002]
Hyperoside: Lf[CS058]
Indole: Lf[CS109]
Indole-3-methyl-ethanolate: Lf[CS015]
Inositol, myo, 2-O-β-L-arabinopyranosyl: Lf 0.4%[CS116]
Inositol, myo, 2-O-β-L-arabinopyranoside: Lf 0.4%[CS106]
Inositol, myo, 2-O-β-L-arabinoside: Lf[CS077]
Ionone, α: Sh 0.03–0.05%[CS100], Lf[CS068]
Ionone, β, 1'-2'-dihydro, 1'-2'-epoxy: Lf EO[CS132]
Ionone, β, 1'-2'-dihydroxy, 1'-2'-threo: Lf EO[CS132]
Ionone, β, 3'-oxo: Lf EO[CS132]
Ionone, β: Lf EO 0.02–0.31%[CS136], Sh 0.17–0.29%[CS100]
Jasmonate, dihydro, methyl-*trans*: Lf[CS002]
Jasmone, *cis*: Lf EO 0.05–0.2%[CS136]
Jasmone: Lf[CS091]
Jasmonic acid, (1R, 2R), (–): Lf[CS080]
Jasmonic acid, (1R, 2S), (+): Lf[CS080]
Jasmonic acid: Pollen[CS137], An[CS137], Lf[CS091]
Kaempferitin: Lf[CS139]
Kaempferol: Lf[CS026], Sh[CS094]
Kaempferol-3-O-galactosyl-rhamnosyl-glucoside: Lf[CS058]
Kaempferol-3-O-glucosyl(1-3)rhamnosyl (1-6)galactoside: Lf[CS009]
Kaempferol-3-O-glucosyl-rhamnoside: Lf[CS058]
Kaempferol-3-O-glucosyl-rhamnosyl-galactoside: Lf[CS058]
Lauric acid: Sd oil[CS134]
Ligustrazine: Lf[CS027]
Limonene: Lf[CS068]
Linalool β-D-glucopyranoside: Sh[CS113]
Linalool oxide A: Lf[CS091]
Linalool oxide B: Lf[CS091]
Linalool oxide C: Lf[CS091]
Linalool oxide I: Lf[CS077]
Linalool oxide II: Lf[CS077]
Linalool oxide III: Lf[CS077]
Linalool oxide IV: Lf[CS077]
Linalool oxide: Headspace volatile[CS044]
Linalool, (R): Lf[CS074]
Linalool, *cis*, oxide (furanoid): Lf[CS074]
Linalool, *cis*, oxide (pyranoid): Lf[CS074]
Linalool, *cis*, oxide: Sh 0.06–0.16%[CS100]
Linalool, *trans*, oxide (furanoid): Lf[CS074]
Linalool, *trans*, oxide (pyranoid): Lf[CS074]
Linalool, *trans*, oxide: Lf EO 3.18–4.23%[CS136], Sh 0.15–0.43%[CS100]
Linalool: Lf[CS121], Headspace volatile[CS044], Sh[CS113], Lf EO 8.2–19.84[CS136]
Linoleic acid: Sd oil[CS134], Lf[CS069]
Linolenic acid: Lf[CS069]
Loliolide: Lf EO[CS132]
Lupeol: Sd oil[CS062]
Malic acid: Lf[CS086]
Malonic acid: Lf[CS086]
Menthol: Lf[CS068]
Methionine, *S*-methyl: Lf 7–24.5 mg%[CS158]
Methylamine: Lf 50[CS141]
Morine: Lf[CS045]
Myrcene: Lf[CS091]
Myricetin: Lf[CS026]
Myristic acid: Sd oil[CS134], Lf[CS069]
Naringenin: Sh[CS094]
Naringenin-fructosyl-glucoside: Lf[CS063]

Neral: Lf[CS002]
Nerolidol: Lf[CS109], Sh 0.08–0.12%[CS100]
NH_3 inorganic: Lf 400[CS141]
Nicotiflorin: Lf[CS133]
Nicotine: Lf 15.5 ng/g[CS047]
Nonal-1-al: Sh 0.04–0.06%[CS100]
Nonal-2-ol: Lf[CS068]
Nonan-2-one: Lf[CS002]
Nona- *trans*-2-*cis*-4-dien-1-al: Lf[CS002]
Nona- *trans*-2-*cis*-6-dien-1-al: Lf[CS002]
Nona-*trans*-2-en-1-al: Lf[CS002]
Nona-*trans*-2-*trans*-4-dien-1-al: Lf[CS002]
Oct-1-en-3-ol: Lf[CS068]
Octa-1-5-7-trien-3-ol, 3(*S*)-7-dimethyl: Lf EO[CS132]
Octa-1-5-diene-3-7-diol, 3(*S*)-7-dimethyl, (+): Lf EO[CS132]
Octan-2-one: Lf[CS002]
Octan-3-ol: Lf[CS068]
Octanoate, ethyl: Lf[CS002]
Octanoate, methyl: Lf[CS002]
Octa-*trans*-2-*cis*-4-dien-1-al: Lf[CS002]
Octa-*trans*-2-*trans*-4-dien-1-al: Lf[CS002]
Octa-*trans*-3-*cis*-5-dien-2-one: Lf[CS002]
Oct-*trans*-2-enoic acid: Lf[CS002]
Oleic acid: Sd oil[CS134], Lf[CS069]
Oolonghomobisflavan A: Lf 10.6[CS008]
Oolonghomobisflavan B: Lf 7.2[CS008]
Oolongtheanin: Lf 1.8[CS006]
Oxalic acid: Lf 1.0%[CS144]
Palmitic acid: Sd oil[CS134], Lf[CS069]
Pedunculagin: Lf[CS041]
Pent-1-en-3-ol: Lf[CS068], Sh 0.21–0.23%[CS100]
Pent-2-en-1-al, 4-methyl-2-phenyl: Lf[CS002]
Pentadecane, 2-6-10-14-tetramethyl: Lf[CS091]
Pentan-1-ol: Sh 0.06–0.11%[CS100]
Pentan-2-ol, methyl: Lf[CS068]
Pentan-3-ol, methyl: Lf[CS068]
Pentanoic acid: 2-amino-5-(*N*-ethyl-carboxamido): Lf 120[CS105]
Pent-*cis*-2-en-1-ol: Sh 0.1-0.14%[CS100]
Pent-*cis*-3-en-1-al: Lf[CS002]
Phenol: Lf[CS003]
Phenyl, acetate, ethyl: Lf[CS002]
Phenyl, acetate, hexyl: Lf[CS002]
Phenylacetic acid: Lf[CS002]
Phenylethanol, 2: Lf[CS091]
Phenylethyl alcohol, 2: Sh 0.1–0.13%[CS100]
Phenylethyl alcohol: Headspace volatile[CS044]
Pheophytin A: Lf[CS088]
Pheophytin B: Lf[CS088]
Pinene, a: Lf[CS068]
Pipecolic acid, L: Fr[CS030]
Pipecolic acid: Lf[CS037], Fr[CS037]
Polysaccharide T-B: Lf[CS111]
Procyanidin B-2 3'-*O*-gallate: Lf 166.7[CS140]
Procyanidin B-2, 3-3'-di-*O*-gallate: Lf 0.00084–0.13%[CS008,CS010]
Procyanidin B-2: Lf 5.8[CS008]
Procyanidin B-3, 3-*O*-gallate: Lf[CS070]
Procyanidin B-3: Lf 0.21%[CS010]
Procyanidin B-4, 3'-*O*-gallate: Lf 141[CS140]
Procyanidin B-4: Lf 46.6[CS008]
Procyanidin B-5, 3-3'-di- *O*-gallate: Lf 2.6[CS008]
Procyanidin C-1: Lf[CS010]
Prodelphinidin A-2, 3'-*O*-gallate: Lf 4.4[CS008]
Prodelphinidin B-2, 3'-*O*-gallate: Lf 238[CS008]
Prodelphinidin B-2, 3-3'-di-*O*-gallate: Lf 18.4[CS008]
Prodelphinidin B-2,3'-*O*-gallate: Lf 147.4[CS140]
Prodelphinidin B-4, 3'-*O*-gallate: Lf 63.8–1200[CS008,CS010]
Prodelphinidin B-4: Lf 56.8–800[CS008,CS010]
Prodelphinidin B-5, 3-3'-di-*O*-gallate: Lf 29.8[CS008]
Proline, hydroxy: Lf[CS037], Fr[CS037]
Propionamide, *N*-ethyl: Lf[CS027]
Propiophenone, 2-4-dimethyl: Lf[CS027]
Propiophenone, para-ethyl: Lf[CS027]
Prunasin: Lf[CS059]
Pyrazine, 2-3-dimethyl: Lf[CS027]
Pyrazine, 2-5-dimethyl: Lf[CS027]
Pyrazine, 2-6-dimethyl: Lf[CS027]
Pyrazine, 2-ethyl-3-5-dimethyl: Lf[CS027]
Pyrazine, 2-ethyl-3-6-dimethyl: Lf[CS036]
Pyrazine, 2-ethyl-5-methyl: Lf[CS027]
Pyrazine, 2-ethyl-6-methyl: Lf[CS027]
Pyrazine, ethyl: Lf[CS027]
Pyrazine, methyl: Lf[CS027]
Pyrazine, trimethyl: Lf[CS027]
Pyridine, 2-5-dimethyl: Lf[CS027]

Pyridine, 2-6-dimethyl: Lf[CS027]
Pyridine, 2-acetyl: Lf[CS036]
Pyridine, 2-ethyl: Lf[CS027]
Pyridine, 2-ethyl-5-methyl: Lf[CS036]
Pyridine, 2-ethyl-6-methyl: Lf[CS036]
Pyridine, 2-methyl: Lf[CS027]
Pyridine, 2-phenyl: Lf[CS027]
Pyridine, 3-ethyl: Lf[CS027]
Pyridine, 3-methoxy: Lf[CS036]
Pyridine, 3-methyl: Lf[CS027]
Pyridine, 3-*N*-butyl: Lf[CS036]
Pyridine, 3-phenyl: Lf[CS027]
Pyridine, 4-methyl: Lf[CS027]
Pyridine, 4-vinyl: Lf[CS036]
Pyridine: Lf[CS027]
Quercetin: Lf[CS026], Sh[CS094]
Quercetin-3-glucosyl(1-3)rhamnosyl (1-6)galactoside: Lf[CS009]
Quercetin-fructosyl-glucoside: Lf[CS063]
Quercimeritrin: Lf[CS026]
Quercitrin, iso: Lf[CS133]
Quercitrin: Lf[CS058]
Quinic acid, (–): Lf[CS104]
Quinoline, 2-4-dimethyl: Lf[CS027]
Quinoline, 2-6-dimethyl: Lf[CS027]
Quinoline, 2-methyl: Lf[CS036]
Quinoline, 3-*N*-butyl: Lf[CS027]
Quinoline, 3-*N*-propyl: Lf[CS027]
Quinoline, 4-8-dimethyl: Lf[CS027]
Quinoline, 6-methyl: Lf[CS036]
Rutin: Lf[CS058]
Safranal: Lf[CS002]
Safrole: Lf[CS002]
Salicylic acid: Headspace volatile[CS044]
Sesquiphelandrene, b: Lf[CS109]
Sitosterol, β: Sd oil[CS134]
Spinasterol, 22-23-dihydro: Sd oil[CS131]
Spinasterol, α, β-D-glucoside: Rt[CS039]
Spinasterol, α: Rt[CS039]
Spinasterol: Sd oilCS131
Spinasterone, 22-23-dihydro: Sd oil[CS131]
Spinasterone: Sd oil[CS131]
Stearic acid: Sd oil[CS134]
Stigmasterol: Sd oil[CS134]
Strictinin: Lf 0.01%[CS010]
Succinic acid: Lf[CS086]
Tannic acid: Lf [CS126]
Tannin: Lf[CS024]
Taraxasterol, Pseudo: Sd oil[CS095]
Taraxerol: Sd oil 20[CS095]
Tartaric acid: Lf[CS086]
Tea polysaccharides: Lf[CS055]
Teasaponin B-1: Lf[CS057]
Teasaponin B-2: Lf[CS040]
Teasaponin B-3: Lf[CS040]
Teasaponin B-4: Lf[CS040]
Teasterone: Lf[CS090]
Tectoquinone: Rt[CS039]
Terpineol, 4: Lf[CS002]
Terpineol, α: Sh 0.07–0.1%[CS100], Lf[CS068]
Theacitrin A: Lf 0.08%[CS016]
Theaflagallin, epi, 3-*O*-gallate: Lf 17[CS008]
Theaflagallin-3-*O*-gallate, epi: Lf 0.02%[CS010]
Theaflavate B: Lf[CS019]
Theaflavic acid, epi, gallate: Lf[CS064]
Theaflavic acid, epi: Lf[CS064]
Theaflavin, digallate: Lf[CS071]
Theaflavin, iso: 3'-*O*-gallate: Lf 25[CS019]
Theaflavin, monogallate A: Lf[CS071]
Theaflavin, monogallate B: Lf[CS071]
Theaflavin, monogallate: Lf[CS124]
Theaflavin, neo: 3-*O*-gallate: Lf 30[CS019]
Theaflavin: Lf[CS046], Sh 1.12–1.40%[CS100], Fl[CS159]
Theaflavin-3'-gallate: Lf[CS046]
Theaflavin-3'-*O*-gallate: Fl[CS159], Lf 18.6–800[CS008,CS010]
Theaflavin-3-3'- digallate: Fl[CS159]
Theaflavin-3-3'-di-*O*-gallate: Lf 18.2–300[CS008,CS010]
Theaflavin-3-gallate: Lf[CS046]
Theaflavin-3-*O*-gallate: Fl[CS159], Lf 6-700[CS008,CS010]
Theaflavin-monogallate A: Lf[CS085]
Theaflavin-monogallate B: Lf[CS085]
Theaflavonin, degalloyl: Lf 17.5[CS010]
Theaflavonin: Lf 11.5[CS010]
Theanaphthoquinone: Lf[CS023]
Theanine: Lf[CS052], Call Tiss[CS066], Seedling Rt 109, Sh 63, Cy 577 mg%[CS097], St Call 0.37 ppt, An 1.6-2.9%, St 34.9 ppt[CS099]
Thearubigin: Sh 13.56–15.74%[CS100], Lf[CS139]

Theasapogenol A, 22-O-angeloyl: Sd[CS020]
Theasapogenol B, 22-O-angeloyl: Sd[CS020]
Theasapogenol E, 22-O-angeloyl: Sd[CS020]
Theasaponin B-1: Lf[CS081]
Theasaponin E-1: Sd 75[CS017]
Theasaponin E-2: Sd 10[CS017]
Theasaponin, gluco: Sd[CS061]
Theasaponin: Sd[CS127], Lf[CS142]
Theasinensin A: Lf 0.01866–4.8718%[CS006,CS140]
Theasinensin B: Lf 128.2–600[CS140,CS010]
Theasinensin C: Lf 70.2[CS006]
Theasinensin D: Lf 17.6[CS006]
Theasinensin E: Lf 14.4[CS006]
Theasinensin F: Lf 19.6[CS006]
Theasinensin G: Lf 8[CS006]
Theaspirane, dihydro, 6-7-epoxy: Lf[CS002]
Theaspirane, dihydro, 6-hydroxy: Lf[CS002]
Theaspirane: Lf[CS002]
Theaspirone: Lf EO[CS132]
Theobromine: Lf[CS029], Call Tiss[CS050], Sd, Pc[CS102], Fl Bd, Fl[CS107], Petal, Pistil, Stamen[CS117], An, St, St Call[CS099], Pl[CS033], Seedcoat[CS102]
Theogallin: Lf 6-55.5[CS008,CS010]
Theophylline: Sd[CS093]
Thiazole, 2-4-5-trimethyl: Lf[CS036]
Thiazole, 2-4-dimethyl: Lf[CS036]
Thiazole, 2-4-dimethyl-4-ethyl: Lf[CS036]
Thiazole, 2-5-dimethyl: Lf[CS036]
Thiazole, 5-methyl: Lf[CS036]
Thymol: Lf[CS002]
Tirucalla-7-24-dien-3-β-ol, 5-α: Sd oil 12[CS062]
Tirucalla-7-24-dien-3-β-ol: Sd oil[CS095]
Tirucallol: Sd oil[CS095]
Toluidine, ortho: Lf[CS027]
Triacontan-1-ol: Lf[CS075]
Tricetin: Sh[CS094]
Tricetinidin: Lf[CS139]
Trifolin: Lf[CS058]
Tr-saponin A: Rt 2.2[CS022]
Tr-saponin B: Rt 5.9[CS022]
Tr-saponin C: Rt 2.8[CS022]
Typhasterol: Lf[CS090]
Umbelliferone: Lf[CS032]
Undeca-2-one, 6-10-dimethyl: Lf[CS002]
Undeca-*trans*-2-en-1-al: Lf[CS002]
Urea: Pl[CS033]
Vitamin K-1: Lf 3.1-16.5[CS072],
Vitexin, iso, 2''-O-glucoside: Lf[CS103]
Vitexin: Sh[CS096]
Vomifeliol, dehydro: Lf EO[CS132]

PHARMACOLOGICAL ACTIVITIES AND CLINICAL TRIALS

Antibacterial activity. Alcohol extract of black tea, assayed on *Salmonella typhi* and *Salmonella paratyphi* A, was active on all strains of Salmonella paratyphi A, and only 42.19% of Salmonella typhi strains were inhibited by the extract[CS048]. Hot water extract of the dried entire plant and the tannin fraction, on agar plate, were active on *Escherichia coli*, *Pseudomonas aeruginosa*, and *Staphylococcus aureus*[CS160].

Anticancer activity. Catechin, administered to pheochromocytoma cells in cell culture, was active. The cells were incubated with different concentrations of catechin at short-term (2 days) and long-term (7 days) in Dulbecco's modified Eagle medium. The activity of superoxide dismutase was measured and its mRNA assayed by Northern blotting. After incubation for 2 days, catechin significantly increased the activity of copper/zinc superoxide dismutase. However, it did not produce significant effect at 7 days. The magnesium superoxide dismutase activity produced significant changes in both short- and long-term treatment groups. The amount of mRNA also showed similar changes[CS040].

Anticarcinogenic activity. The anticarcinogenic activity of tea phenols has been demonstrated in rats and mice transplantable tumors, carcinogen-induced tumors in digestive organs, mammary glands, hepatocarcinomas, lung cancers, skin tumors, leukemia, tumor promotion, and metastasis. The mechanisms of this effect indicated that the inhibition of tumors may be the result of both extracellular and intracellular mechanisms indicating the modulation of

metabolism, blocking or suppression, modulation of DNA replication and repair effects, promotion, inhibition of invasion and metastasis, and induction of novel mechanisms[CS002]. The association of green tea and cancer has been investigated in 8552 Japanese women 40 years of age. After 9 years of follow-up study, 384 cases of cancer were identified. There was a negative association between cancer incidence and green tea consumption, especially among females consuming more than 10 cups of tea a day. A slow down in increases of cancer incidence with age was observed among females who consumed more than 10 cups daily[CS010]. Tea, taken by lung cancer patients at a dose of two or more cups per day, reduced the risk by 95%. The protected effect was more evident among Kreyberg I tumors (squamous cell and small cells) and among light smokers[CS011]. The green tea polyphenols, epi-gallocatechin-3-gallate, applied topically to human skin, prevented penetration of ultraviolet (UV) radiation. This was demonstrated by the absence of immunostaining for cyclobutane pyrimidine dimers in the reticular dermis. Topical administration to the skin of mice inhibited UVB-induced infiltration of $CDIIb^+$ cells. The treatment also results in reduction of the UVB-induced immunoregulatory cytokine interleukin (IL)-10 in the skin and draining lymph nodes, and an elevated amount of IL-12 in draining lymph nodes[CS015]. Green tea extract, in human umbilical vein endothelial cells, did not affect cell viability but significantly reduced cell proliferation dose-dependently and produced a dose-dependent accumulation of cells in the gastrointestinal phase. The decrease of the expression of vascular endothelial growth factor receptors fms-like tyrosine kinase and fetal liver kinase-I/kinase insert domain containing receptor in the cell culture by the extract was detected with immunohistochemical and Western blotting methods[CS020]. Green and black tea, administered orally to hairless mice in the absence of any chemical initiators or promoters, resulted in significantly fewer skin papillomas and tumors induced by UVA and UVB light. Black tea however, provided better protection against UVB-induced tumors than green tea. Black tea consumption was associated with a reduction in the number of sunburn cells in the epidermis of mice 24 hours after irradiation, although there was no effect of green tea. Other indices of early damage such as necrotic cells or mitotic figures were not affected. Neutrophil infiltration as a measure of skin redness was slightly lowered by tea consumption in the UVB group[CS023]. Epigallocatechin-3-gallate, in cell culture, activated proMMP-2 in U-87 glioblastoma cells in the presence of concanavalin A or cytochalasin D, two potent activators of MT1-MMP, resulted in proMMP-2 activation that was correlated with the cell surface proteolytic processing of Mt1-MMP to it's inactive 43 kDa form. Addition of epigallocatechin-3-gallate strongly inhibited the MT1-MMP-driven migration in the cells. The treatment of cells with non-cytotoxic doses of epigallocatechin-3-gallate significantly reduced the amount of secreted pro MMP-2, and led to a concomitant increase in intracellular levels of that protein. The effect was similar to that observed using well-characterized secretion inhibitors such as brefeldin A and manumycin, indicative that epigallocatechin could also potentially act on intracellular secretory pathways[CS044]. Green tea polyphenols, at a dose of 30 mg/mL, inhibited the photolabeling of P-glycoprotein (P-gp) by 75% and increased the accumulation of rhodamine-123 in the multidrug-resistant cell line CH(R)C5. This result indicated that green tea polyphenols interact with P-gp and inhibited its transport activity. The modulation of P-gp was a reversible process. Epigallocatechin-3-gal-

late potentiates the cytotoxicity of vinblastine in CH(R)C5 cells. The inhibitory effect on P-gp was also observed in human Caco-2 cells[CS045].

Anticataract activity. Tea, administered in culture to enucleated rat lens, reduced the incidence of selenite cataract in vivo. The rat lenses were randomly divided into normal, control and treated groups and incubated for 24 hours at 37°C. Oxidative stress was induced by sodium selenite in the culture medium of the two groups (except the normal group). The medium of the treated group was additionally supplemented with tea extract. After incubation, lenses were subjected to glutathione and malondialdehyde estimation. Enzyme activity of superoxide dismutase, catalase, and glutathione peroxidase were also measured in different sets of the experiment. In vivo cataract was induced in 9-day-old rat pups of both control and treated groups by a single subcutaneous injection of sodium selenite. The treated pups were injected with tea extract intraperitoneally prior to selenite challenge and continued for 2 consecutive days thereafter. Cataract incidence was evaluated on 16 postnatal days by slit lamp examination. There was positive modulation of biochemical parameters in the organ culture study. The results indicated that tea act primarily by preserving the antioxidant defense system[CS039].

Antidiarrheal activity. Hot water extract of tea, administered orally to rats, was effective in all the models of diarrhea used. Naloxone (0.5 mg/kg, ip) and loperamide significantly inhibited the antidiarrheal activity of the extract[CS029].

Antifungal activity. Ethanol (50%) extract of the entire plant, in broth culture at a concentration of 1 mg/mL, was inactive on *Aspergillus fumigatus* and *Trichophyton mentagrophytes*[CS161]. Hot water extract of the leaf on agar plate at a concentration of 1.0% was active on *Alternaria tenuis*, *Pythium aphanidermatum*, and *Rhizopus stolonifer*[CS162]. Saponin fraction of the leaf on agar plate was active on *Microsporum audonini*, minimum inhibitory concentration (MIC) 10 mg/mL; *Epidermophyton floccosum* and *Trichophyton mentagrophytes*, MICs 25 μg/mL[CS165].

Antihypercholesterolemic activity. Tea supplemented with vitamin E, administered to male Syrian hamsters, reduced plasma low-density lipoprotein (LDL) cholesterol concentrations, LDL oxidation, and early atherosclerosis compared to the consumption of tea alone by the hamsters. The antioxidant action of vitamin E is through the incorporation of vitamin E into the LDL molecule. The hamsters were fed a semipurified hypercholesterolemic diet containing 12% coconut oil, 3% sunflower oil, and 0.2% cholesterol (control), control and 0.625% tea, control and 1.25% tea or control and 0.044% tocopherol acetate for 10 weeks. The hamsters fed the vitamin E diet compared to the different concentrations of tea significantly lower plasma LDL cholesterol concentrations, –18% ($p < 0.007$), –17% ($p < 0.02$), and –24% ($p < 0.0001$), respectively. Aortic fatty streak areas were reduced in the vitamin E diet group compared to the control, –36% ($p < 0.04$) and low tea –45% ($p < 0.01$) diets. Lag phase of conjugated diene production was greater in the vitamin E diet compared to the control, low tea, and high tea diets, 41% ($p < 0.0004$), 40% ($p < 0.0004$), and 39% ($p < 0.0008$), respectively. Rate of conjugated diene production was reduced in the vitamin E diet compared to the control, low tea, and high tea diets, –63% ($p < 0.002$), –57% ($p < 0.005$), and –59% ($p < 0.02$), respectively[CS005]. Infusion of black tea leaves was taken by 31 men (ages 47 ± 14) and 34 females (ages 35 ± 13) in a 4-week study. Six mugs of tea were taken daily vs placebo (water, caffeine, milk, and sugar) and blood lipids, bowel habit, and blood

pressure measured during a run-in period and at the end weeks 2, 3, and 4 of the test period. Compliance was established by adding a known amount of *p*-aminobenzoic acid to selected tea bags and then measure it excretion in the urine. Mean serum cholesterol values during run-in, placebo and on tea drinking were 5.67 ± 1.05, 5.76 ± 1.11, and 5.69 ± 1.09 mmol/L ($p = 0.16$). There were also no significant changes in diet, LDL-cholesterol, high-density lipoprotein (HDL) cholesterol, triacylglycerols, and blood pressure in the tea intervention period compared with placebo. Stool consistency was softened with tea compared with the placebo, and no other differences were observed in bowel habit. The results were unchanged within 15 "noncompliers" whose *p*-aminobenzoic acid excretion indicated that fewer than six tea bags had been used, were excluded from the analysis, and when differenced between run-in and tea periods were considered separately for those who were given tea first or second[CS167].

Anti-inflammatory effect. Epigallocatechin-3-gallate was shown to mimic its anti-inflammatory effects in modulating the IL-I β-induced activation of mitogen activated protein kinase in human chondrocytes. It inhibited the IL-I β-induced phosphorylation of c-Jun N-terminal kinase (JNK) isoforms, accumulation of phospho-c-Jun and DNA-binding activity of AP-1 in osteoarthritis chondrocytes, IL-I β but not epigallocatechin-3-gallate, and induced the expression of JNK p46 without modulating the expression of JNK p54 in osteoarthritis chondrocytes. In immune complex kinase assays, epigallocatechin-3-gallate completely blocked the substrate phosphorylating activity of JNK but not p38-mitogen activated protein kinase (MAPK). Epigallocatechin-3-gallate had no inhibitory effect on the activation of extracellular signal-regulated kinase p44/p42 (ERKp44/p42) or p38-MAPK in chondrocytes. Epigallocatechin-3-gallate did not alter the total nonphosphorylated levels of either p38-MAPK or ERKp44/p42 in osteoarthritis chondrocytes[CS033]. Epigallocatechin-3-gallate administered to primary human osteoarthritis chondrocytes at a concentration of 100 μM in cell culture, inhibited the IL-I β-induced production of nitric oxide by interfering with the activation of nuclear factor (NF)κB[CS042]. Tea, in culture with bovine nasal and metacarpophalangeal cartilage and human nondiseased osteoarthritis and rheumatoid cartilage with and without reagents known to accelerate cartilage matrix breakdown, produced chondroprotective effect that may be beneficial for the arthritis patient by reducing inflammation and the slowing of cartilage breakdown. Individual catechins were added to the cultures and the amount of released proteoglycan and type II collagen were measured by metachromatic assay and inhibition enzyme-linked immunosorbent assay (ELISA), respectively. Possible nonspecific or toxic effects of the catechins were assessed by lactate output and proteoglycan synthesis. Catechins, particularly those containing a gallate ester, were effective at micromolar concentrations at inhibiting proteoglycan and type II collagen breakdown[CS043].

Antimutagenic activity. The anticarcinogenic activity of tea phenols has been demonstrated in rats and mice, transplantable tumors, carcinogen-induced tumors in digestive organs, mammary glands, hepatocarcinomas, lung cancers, skin tumors, leukemia, tumor promotion, and metastasis. The mechanisms of this effect indicated that the inhibition of tumors maybe the result of both extracellular and intracellular mechanisms indicting the modulation of metabolism, blocking or suppression, modulation of DNA replication and repair effects, promotion, inhibition of invasion and

Metastasis, and induction of novel mechanisms[CS002]. Green and black teas, administered orally to human adults, were effective. Between 60 and 180 minutes after the teas were administered, the antimutagenic active compounds were recovered from the jejunal compartment by means of dialysis. The dialysate appeared to inhibit the mutagenicity of the food mutagen 2-amino-3,8-dimethylimidazo[4,5-f]quinoxaline on *Salmonella typhimurium*. The maximum inhibition was measured at 2 hours after administration and was comparable for black and green teas. The maximum inhibition observed with black tea was reduced by 22, 42, and 78% in the presence of whole milk, semi-skimmed milk, and skimmed milk, respectively. Whole milk and skimmed milk abolished the antimutagenic activity of green tea by more than 90% and semi-skimmed milk by more than 60%. When a homogenized breakfast was taken with black tea, the antimutagenic activity was eliminated. When tea and mutagen 2-amino-3,8-dimethylimidazo[4,5-f]quinoxaline were added to the system, 2-amino-3,8-dimethylimidazo[4,5-f]quinoxaline mutagenicity was efficiently inhibited, with green tea showing a slightly stronger antimutagenic activity than black tea. The addition of milk had only a small inhibiting effect on the antimutagenicity. The antimutagenic activity corresponded with reduction in antioxidant capacity and with a decrease of concentration of catechin, epigallocatechin gallate, and epigallocatechin[CS014]. Chinese white tea, tested on rat liver S9 in assay for methoxyresorufin O-demethylase, inhibited methoxyresorufin O-demethylase activity and attenuated the mutagenic activity of 3-methylimidazo[4,5-f]quinoline (IQ) in absence of S9. Nine of the major constituents found in green and white teas were mixed to produce artificial teas according to their relative levels in white and green teas. The complete tea exhibited higher antimutagenic potency compared with the corresponding artificial tea[CS019]. Green and black tea polyphenols, applied to the surfaces of ground beef before cooking, inhibited the formation of the mutagens in a dose-related fashion[CS025]. Green or black tea polyphenols sharply decreased the mutagenicity of a number of aryl- and heterocyclic amines, of aflatoxin B_1, benzo[a]pyrene, 1,2-dibromoethane, and more selectively of 2-nitropropane, all involving an induced rat liver S9 fraction. Good inhibition was found with two nitrosamines that required a hamster S9 fraction for biochemical activation. No effect was found with 1-nitropyrene and with the direct-acting (no S9) 2-chloro-4-methyl-thiobutanoic acid[CS027]. Hot water extract on the leaf was evaluated in cell cultures on various systems vs decaffeinated and caffeinated teas. On mouse mammary gland vs decaffeinated and caffeinated teas, IC_{s50} were 10 mg/mL and 10 μg/mL on CA-A427, IC_{50} 27 mg/mL and 31 μg/mL, and on epithelial cells, IC_{50} 0.01 ng/mL and 0.3 ng/mL[CS169]. Hot water extract of the leaf, on agar plate at a concentration of 1 mg/plate, was active on Salmonella typhimurium TA98 vs 2-amino-3-methylimidazo [4,5-f]quinoline-induced mutagenesis and produced weak activity vs benzo[a]pyrene-induced mutagenesis[CS168]. Infusion of the leaf, on agar plate at a concentration of 0.7 mg/plate, was active on *Salmonella typhimurium* TA98 and TA100 vs 2-amino-3-methylimidazo[4,5-f]quinoline-; 3-amino-1,4-dimethyl-5H-pyrid[4,3-b]indole(Trp-1); aflatoxin B1-; 2-amino-6-methyl-dipyrido[1,2-A:3,2-d]imidazole-, and benzo[a]pyrene-induced carcinogenesis[CS170]. Infusion of the leaf, on agar plate at a concentration of 50 mg/plate, was active on *Salmonella typhimurium* TA98 vs 2-amino-3-methylimidazo[4,5-f]quinoline-; 2-amino-3,4-dimethyl-imidazo[4,5-f]quinoline-;2-amino-3,8-dimethylimidazo[4,5-f]quinoxaline-;

2-amino-1-methyl-6-phenylimidazo[4,5-b]-pyridine-; 2-amino-3,7,8-trimethylimidazo[4,5-f]quinoxaline-; 2-amino-3,4,7,8-tetramethyl-3H-imidazo-[4,5-f]quinoxaline-inoxaline-; 3-amino-1,4-dimethyl-5H-pyrid[4,3-b]indole (Trp-P-I)- and 3-amino-1-methyl-5H-pyrido [4,3-b]indole-induced mutagenesis. Metabolic activation was required for positive results[CS171].

Anti-neoplastic effect. Green tea, administered orally at a dose of 6 g per day in six doses to 42 patients who were asymptomatic and had manifested, progressive prostate specific antigen elevation with hormone therapy, produced limited anti-neoplastic activity. Continued use of luteinizing hormone-releasing hormone agonist was permitted. However, patients were ineligible if they had received other treatments for their disease in the preceding 4 weeks or if they had received a long-acting antiandrogen therapy in the preceding 6 weeks. The patients were monitored monthly for response and toxicity. Tumor response, defined as a decline of 50% or greater in the baseline prostate-specific antigen (PSA) value, occurred in a single patient, or 2% of the cohort (95% confidence interval [CI], 1–14%). This one response was not sustained beyond 2 months. At the end of the first month, the median change in the PSA value from baseline for the cohort increased by 43%[CS031]. Infusion of the leaf, administered in the drinking of female mice at a concentration of 1.25%, was active vs UV radiation-induced papillomas and tumors[CS172]. Leaves in the drinking water of female mice at a dose of 0.6% reduced lung tumor multiplicity and volume in 4-(methylnitrosamine)-1-(3-pyridyl)-1-butanone (NNK) treated mice[CS173].

Antioxidative effect. Tea, administered orally to rats, decreased the thiobarbituric acid reactive substances (TBARS) contents in urine and lowered the esterified and total cholesterol contents in plasma as compared with a control group. TBARS contents in liver, plasma, and cholesterol levels in the liver were not affected. The lower plasma cholesterol concentration could not be explained by increased fecal excretion of cholesterol or bile acids. On the other hand, a relationship between decreased plasma cholesterol and significantly higher acetate concentrations in the cecum, colon, and portal blood of rats was assumed. Copper absorption was significantly increased while iron absorption was not affected[CS007]. Epigallocatechin gallate, tea polyphenols, and tea extract were added to human plasma and lipid peroxidation induced by the water-soluble radical generator 2,2'-azobis (2-amidinopropane) dihydrochloride. Following a lag phase, lipid peroxidation was initiated and it occurred at a rate that was lower in a dose that was lowered in a dose-dependent manner by the polyphenols. Similarly, epigallocatechin gallate and the extract added to plasma strongly inhibited 2,2'-azobis(2-amidinopropane) dihydrochloride-induced lipid peroxidation. The lag phase preceding detectable lipid peroxidation was the result of the antioxidant activity of endogenous ascorbate, which was more effective at inhibiting lipid peroxidation than the tea polyphenols and was not spared by these compounds. When eight volunteers consumed the equivalent of six cups of tea, the resistance of their plasma to lipid peroxidation did not increase over a period of 3 hours[CS009]. Black tea leaves, administered to human red blood cells, was effective against damage by oxidative stress induced by inducers such as phenylhydrazine, Cu^{2+}-ascorbic acid, and xanthine/xanthine oxidase systems. Lipid peroxidation of pure erythrocyte membrane and of whole red blood cell was completely prevented by black tea extract. Similarly, the tea provided total protection against

degradation of membrane proteins. Membrane fluidity studies as monitored by the fluorescent probe 1,6-diphenyl-hexa-1,3,5-triene showed considerable disorganization of its architecture that could be restored back to normal on addition of black tea or free catechins. The tea extract in comparison to free catechin seemed to be a better protecting agent against various types of oxidative stress[CS013]. Ethanol/water (7:3) extract of green tea, tested on 2,2-azino-di-3-ethylbenzthiazoline sulphonate, produced antioxidant activity compared with that of ascorbic acid (10 mmol/L)[CS018]. The Nonpolyphenolic fraction of residual green tea (after hot water extraction) produced a significant suppression against hydroperoxide generation from oxidized linoleic acid in a dose-dependent manner. Using silica gel TLC plate, chlorophylls a and b, pheophytins a and b, β-carotene, and lutein were isolated. All of these constituents exhibited significant antioxidant activites, the ranks of suppressive activity against hydroperoxide generation were chlorophyll a > lutein > pheophytin a > chlorophyll b > b-carotene > pheophytin b[CS047].

Antiproliferative activity. Green tea fractions, tested on human stomach cancer (MK-1) cells, indicated six active flavan-3-ols, epicatechin, epigallocatechin, epigallocatechin gallate, gallocatechin, epicatechin gallate, and gallocatechin gallate. Among the six active flavan-3-ols, epigallocatechin gallate and gallocatechin gallate produced the highest activity. Epigallocatechin, gallocatechin, and epicatechin gallate followed next, and the activity of epicatechin was lowest. This suggests that the presence of the three adjacent hydroxyl groups (pyrogallol or galloyl group) in the molecule would be a key factor for enhancing the activity[CS032].

Antiprotozoan activity. Ethanol (50%) extract of the entire plant, in broth culture at a concentration of 125 μg/mL, was inactive on *Entamoeba histolytica*[CS161].

Antispasmodic activity. Hot water extract and tannin fraction of the dried entire plant were active on the rabbit and rat intestines vs pilocarpine-induced spasms and barium-induced contractions[CS160].

Antiviral activity. Epigallocatechin-3-gallate, administered to Hep2 cells in culture, produced a therapeutic index of 22 and an IC_{50} of 25 μM. The agent was the most effective when added to the cells during the transition from the early to the late phase of viral infection suggesting that the polyphenol inhibits one or more late steps in virus infection[CS016].

Ethanol (50%) extract of the entire plant, in broth culture at a concentration of 50 μg/mL, was inactive on Raniket and Vaccinia viruses[CS161]. Hot water extract of the leaf in cell culture was active on Coxsackie A9, B1, B2, B3, B4, and B6 viruses, Echo type 9 virus, herpes simplex virus, poliovirus III, vaccinia virus, and REO type 1 virus[CS163].

Anti-yeast activity. Ethanol (50%) extract of the entire plant, in broth culture at a concentration of 1 mg/mL, was inactive on *Candida albicans*, *Cryptococcus neoformans*, and *Sporotrichum schenckii*[CS161]. Ethanol extract of the leaf on agar plate produced MIC 9.3 mg/mL on *Candida albicans* [CS164].

Coronary heart disease prevention. Tea, taken by men and women age 30 to 70 years at a dose of 480.0 mL per day, produced a positive dose–response effect[CS008].

Cytochrome P50 expression. Fresh leaves of green, black, and decaffeinated black tea enhanced lauric acid hydroxylation. The decaffeinated black tea produced no significant effect. Green tea and black tea but not decaffeinated black tea, stimulated the O-dealkylations of methoxy-, ethoxy-, and pentoxy-resorufin indicating upregulation of cytochrome P50 (CYP)1A and CYP2B. Immunoblot analysis revealed that green and black tea, but not decaffeinated black tea, elevated the hepatic CYP1A2 apoprotein levels. Hepatic microsomes from green

and black tea-treated rats, but not those from the decaffeinated black tea-treated rats, were more effective than controls in converting IQ into mutagenic species in the Ames test[CS001].

Dental enamel erosion. Herbal tea and conventional black tea, tested on teeth, resulted in erosion of dental enamel. After exposure to tea, sequential profilometric tracings of the specimens were taken, superimposed, and the degree of enamel loss calculated as the area of disparity between the tracings before and after exposure. Tooth surface loss resulted from herbal tea (mean 0.05 mm^2) was significantly greater than that which resulted from exposure to conventional black tea (0.01 mm2), and water (0.00 mm^2)[CS022]. Tannin, catechin, caffeine, and tocopherol, tested in vitro on tooth enamel, demonstrated that these components possess the property of increasing the acid resistance of tooth enamel. The effects increased dramatically when the components were used in combination with fluoride. A mixture of tannic acid and fluoride showed the highest inhibitory effect (98%) on calcium release to an acid solution. Tannin in combination with fluoride inhibited the formation of artificial enamel lesions in comparison with acidulated phosphate fluoride (APF) as determined by electron probe microanalysis, polarized-light microscopy, and Vickers microhardness measurement[CS024].

DNA effect. Green tea extract, in cell culture at a dose of 10 mg/L corresponding to 15 mmol/L EGCg for 24 hours, did not protect Jurkat cells against H_2O_2-induced DNA damage. The DNA damage, evaluated by the Comet assay, was dose-dependent. However, it reached plateau at 75 mmol/L of H_2O_2 without any protective effect exerted by the extract. The DNA repair process, completed within 2 hours, was unaffected by supplementation[CS021].

Fluoride retention. Tea, used as a mouth rinse, demonstrated strong avidity of enamel for tea and salivary pellicle components. Thirty-four percent of the fluoride was retained in the oral cavity. Differences in retention at the tooth surface in the presence and absence of an acquired pellicle were not statistically significant at incisor or molar sites. Fluoride from tea showed strong binding to enamel particles, which was only partially dissociated by solutions of ionic strength considerably greater than that of saliva[CS012].

Gastrointestinal effect. Green tea, administered to rats fasted for 3 days, reverted to normal the mucosal and villous atrophy induced by fasting. Black tea ingestion had no effect. Ingestion of black tea, green tea, and vitamin E before fasting protected the intestinal mucosa against atrophy[CS003]. Characterization of melanin extracted from tea leaves proved similarity of the original compound to standard melanin. The Langmuir adsorption isotherms for gadolinium (Gd) binding were obtained using melanin. Melanin–Gd preparation demonstrated low acute toxicity. LD50 for the preparation was in a range of 1.25–1.50 g/kg in mice. Magnetic resonance imaging (MRI) properties of melanin itself and melanin-Gd complexes have been estimated. Gadolinium-free melanin fractions possess slighter relaxivity compared with its complexes. The relaxivity of lower molecular weight fraction was 2 times higher than relaxivity of Gd(DTPA) standard. Postcontrast images demonstrated that oral administration of melanin complexes in concentration of 0.1 mM provides essential enhancement to longitudinal relaxation times (T[1])-weighted spin echo image. The required contrast and delineation of the stomach wall demonstrated uniform enhancement of MRI with proposed melanin complex[CS049].

Hypocholesterolemic effect. Green tea, in human HepG2 cell culture, increased both LDL receptor-binding activity and protein. The ethyl acetate extract, containing 70% (w/w) catechins, also increased

LDL receptor-binding activity, protein, and mRNA, indicating that the effect was at the receptor level of gene transcription and that the catechins were the active constituents. The mechanism by which green tea upregulated the LDL receptor was investigated. Green tea decreased the cell cholesterol concentration (–30%) and increased the conversion of the sterol-regulated element binding protein (SREBP-1) from the inactive precursor form to the active transcription-factor form. Consistent with this, the mRNA of 3-hydroxy-3-methylglutaryl coenzyme-A reductase, the rate limiting enzyme in cholesterol synthesis, was also increased by green tea[CS050].

Immunomodulatory effect. To determine the effects of tea on transplant-related immune function in vitro lymphocyte proliferation tests using phytohemagglutinin, mixed lymphocytes culture assay, IL-2, and IL-10 production from mixed lymphocyte proliferation were performed. Tea had immunosuppressive effects and decreased alloresponsiveness in the culture. The immunosuppressive effect of tea was mediated through a decrease in IL-2 production[CS038]. Tea, assayed in cell culture, enhanced neopterin production in unstimulated peripheral mononuclear cells, whereas an effective reduction of neopterin formation in cells stimulated with concanavalin A, phytohemagglutinin or interferon (IFN)-γ was observed[CS041]. Theaflavins potently suppressed IL-2 secretion, IL-2 gene expression, and the activation of NF-κB in murine spleens enriched for CD4(+) T-cells. Theaflavins also inhibited the induction of IFN-γ mRNA. However, the expression of the T(H2) cytokines IL-4 and IL-5, which lack functional NF-κB sites within their promoters was unexpectedly suppressed by theaflavins as well[CS046].

Insulin-enhancing effect. Tea, as normally consumed, was shown to increase insulin activity more than 15-fold in vitro in an epididymal fat cell assay. The majority of the insulin-potentiating activity for green and oolong teas was owing to epigallocatechin gallate. For black tea, the activity was present in addition to epigallocatechin gallate, tannins, theaflavins, and other undefined compounds. Several known compounds found in tea were shown to enhance insulin with the greatest activity due to epigallocatechin gallate followed by epicatechin gallate, tannins, and theaflavins. Caffeine, catechin, and epicatechin displayed insignificant insulin-enhancing activities. Addition of lemon to the tea did not affect the insulin-potentiating activity. Addition of 5 g of 2% milk per cup decreased the insulin-potentiating activity one-third, and addition of 50 g of milk per cup decreased the insulin-potentiating activity approx 90%. Non-dairy creamers and soymilk also decreased the insulin-potentiating activity[CS034].

Iron absorption. Tea, administered by gastric intubation to rats, did not affect iron absorption when tea was consumed for 3 days but when delivered in tea the absorption was decreased. Rats maintained on a commercial diet were fasted overnight with free access to water and then gavaged with 1 mL of ^{59}Fe labeled FeCl3 (0.1 mM or 1 mM) and lactulose (0.5 M) in water or black tea. Iron absorption was estimated from Fe retention. Intestinal permeability was evaluated by lactulose excretion in the urine. Iron absorption was lower with given with tea at both iron concentrations but tea did not affect lactulose excretion[CS004].

Lipid peroxidation activity. Solubilized green tea, administered orally to rats for 5 weeks, reduced lipid peroxidation products. The treatment produced increased activity of glutathione (GSH) peroxidase and GSH reductase, increased content of reduced GSH, a marked decrease in lipid hydroperoxides and malondialdehyde in the liver, an increase in the concentration of vitamin A by about 40%. A minor change in the measured parameters was observed in the blood