Cancer Research

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Tea Plant
(Thea Sinensis)

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Call for Abstracts for 1999 AACR Annual Meeting
Abstract Deadline: Nov. 6, 1998

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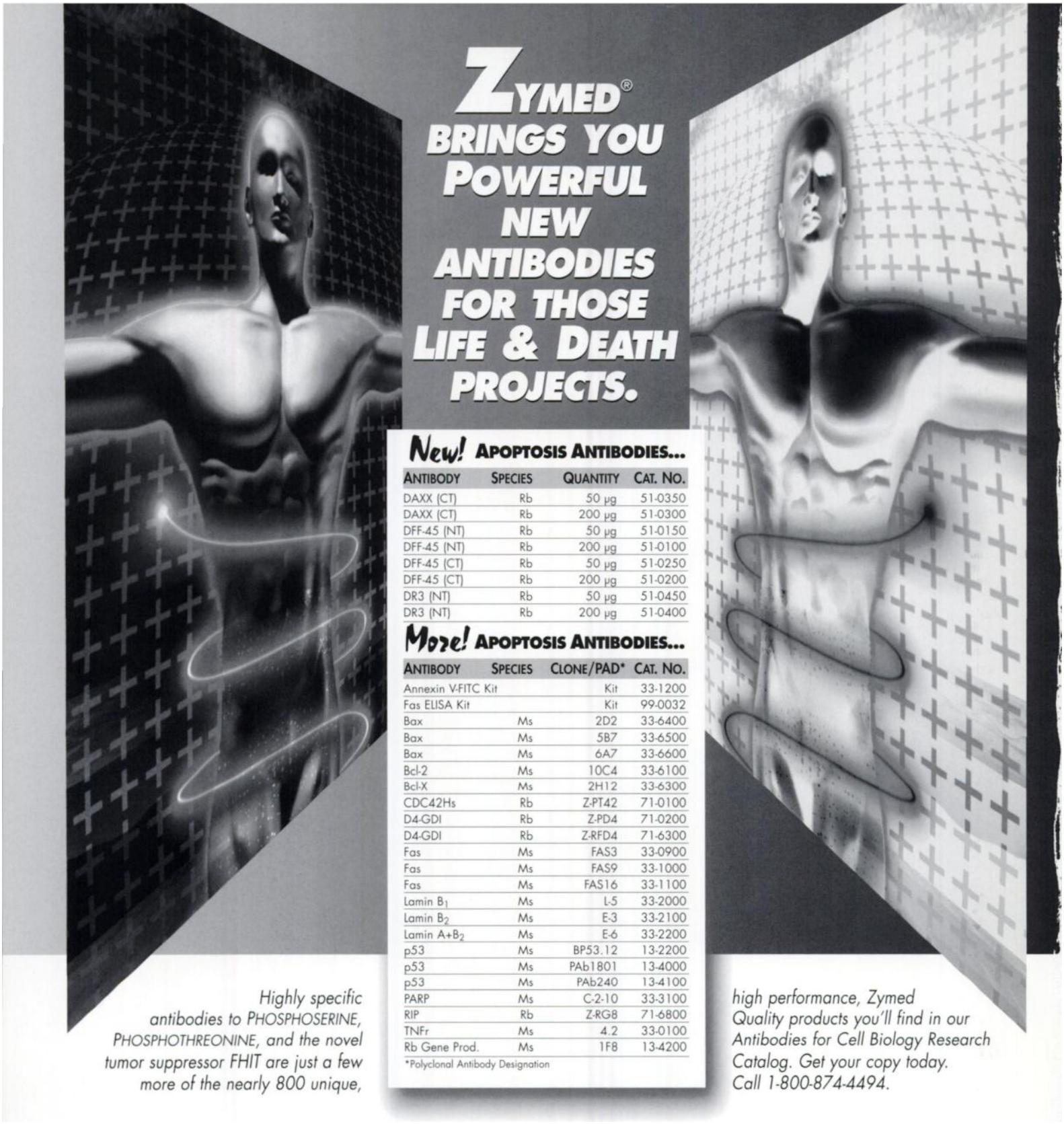

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1999 AACR-Pezcoller International Award for Cancer Research

Fondazione PEZCOLLER Trento - Italy

The AACR-Pezcoller International Award for Cancer Research is given annually to a scientist who has made a major scientific discovery in the field of cancer, who continues to be active in the field, and whose ongoing work holds promise for future contributions to cancer research. The Pezcoller Foundation was established in 1982 by Professor Alessio Pezcoller, a dedicated Italian surgeon who has made important contributions to medicine during his career and who, through his foresight, vision, and generous gift in support of the formation of the Foundation, stimulated others to make significant advances in cancer research. Over the past decade the Pezcoller Foundation has given a major award for outstanding contributions to cancer and cancer-related biomedical science.

The American Association for Cancer Research (AACR) was founded in 1907 by eleven physicians and scientists dedicated to the conquest of cancer and now has nearly 14,000 members in more than 60 countries who are experts in basic, clinical, and translational cancer research. The mission of the AACR is to foster cancer research; this is accomplished in part through outstanding scientific publications, meetings, and training and educational programs. Because of the commitment of the Foundation and the AACR to scientific excellence in cancer research, these two organizations are collaborating annually on the presentation of the AACR-Pezcoller Award. This jointly sponsored award will strengthen international collaborations and will be a catalyst for advancements in cancer research internationally. The awardee will be selected by an international committee of AACR members appointed by the AACR President along with the agreement of the Council of the Pezcoller Foundation. While normally the Award will be presented to a single investigator, in exceptional cases two individuals may be selected to share the award when their investigations have resulted in related prizeworthy work. The committee will make its selection solely on the basis of the awardee's scientific accomplishments without regard to race, gender, nationality, or religious or political views. The candidate will give an award lecture during the AACR Annual Meeting in Philadelphia, USA (April 10-14, 1999) and will receive the award in an official ceremony at the Foundation's headquarters in Trento, Italy, after the annual meeting. The award consists of an honorarium of US$75,000 and a commemorative plaque.

The Pezcoller Foundation and the AACR are now soliciting nominations for the 1999 Award. Nominations can be made by any scientist who is now or has been affiliated with an institution engaged in cancer research. Institutions or organizations are not eligible for this award, and candidates may not nominate themselves.

There is no official application form for this award. The nomination package should consist of the following:

- the candidate's curriculum vitae and full list of published works
- an indication of the candidates's most important publications
- a letter of recommendation in English (500 words, maximum) explaining why the candidate is deserving of this prestigious Award. This letter should summarize the candidate's major scientific achievements, indicate which of the candidate's publications best describe these achievements, and explain the impact of these achievements on progress in cancer research.

Nominators are asked to maintain the confidentiality of the nomination process and to refrain from informing the candidate about the nomination.

The deadline for receipt of nominations for the 1999 Award is **October 1, 1998.** Questions about the nomination process should be directed to the AACR via FAX at (215) 440-9322 or E-mail at aacr@aacr.org. Nominations should be submitted to the AACR. Please forward the original nomination letter plus 15 copies of the letter and any accompanying materials to:

Peter K. Vogt, Ph.D., Chairperson, Selection Committee
AACR-Pezcoller International Award for Cancer Research
c/o American Association for Cancer Research
Public Ledger Building, Suite 826
150 S. Independence Mall West
Philadelphia, PA 19106-3483
USA

AACR SPECIAL CONFERENCE IN CANCER RESEARCH

Cancer Biology and the Mutant Mouse: New Methods, New Models, New Insights

January 31-February 5, 1999
Keystone Resort & Conference Center
Keystone, Colorado

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Tak W. Mak / Toronto, Ontario, Canada
Neal G. Copeland / Frederick, MD
Stanley J. Korsmeyer / St. Louis, MO
Pier Paolo Pandolfi / New York, NY

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Terry A. Van Dyke / Chapel Hill, NC
Erwin Wagner / Vienna, Austria
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Model Systems II
Raju Kucherlapati / Bronx, NY
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Timothy C. Thompson / Houston, TX

The conference will also feature a methods workshop session and a late-breaking research session.

Registration Deadline: December 4, 1998

Information and Registration Forms:

American Association for Cancer Research
Public Ledger Building, Suite 826
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215-440-9300 215-440-9313 (FAX)
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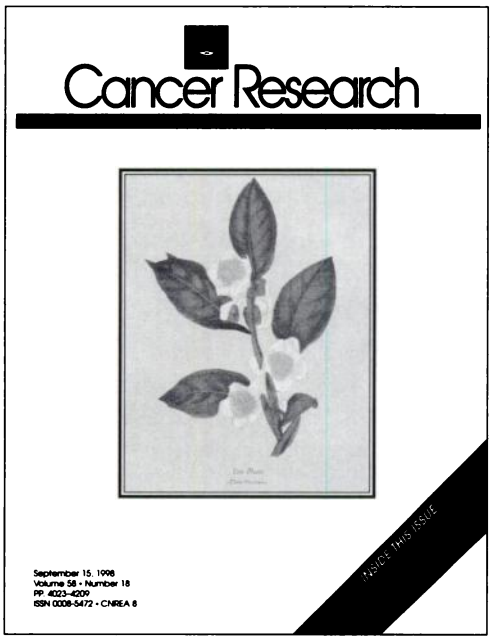

Except for studies on alcohol, it is only recently that the question of beverage use has become a subject of interest. Tea ranks second only to water as a major component of fluid intake worldwide. It is a safe beverage, since it is made generally with boiling water, an important consideration in places where pure, uncontaminated water is not available. Yet, detailed research on the health effects of tea, particularly regarding its role in cancer, is quite recent.

Tea is derived from the leaf of the plant *Camellia sinensis* (*cover*),* which was discovered thousands of years ago in China (Prev. Med., *21:* 334, 1992; Food Rev. Int., *11:* 371, 1995; *Handbook of Antioxidants*, E. Cadenas and L. Packer (eds.), p. 469, 1996; Cancer Lett., *114:* 315, 1997). The leaf contains caffeine, but the caffeine content of a cup of tea is only one-third that of coffee. Antioxidant polyphenols comprise about one-third of the weight of the dried leaf. An enzyme, polyphenol oxidase, is a key component. Upon harvesting, the tea leaves are dried, rolled and crushed, and the enzyme is then liberated; however, it is inactivated when the dried leaves are immediately steamed or heated, yielding green tea. A delay in enzyme inactivation permits conversion of the indigenous tea polyphenols to other polyphenols. When the action of the enzyme is relatively short (about 30 minutes), prior to final heating and drying of the processed leaf, the product is oolong tea. When the enzyme activity proceeds for one to two hours, black tea results (Prev. Med., *21:* 334, 1992; J. Natl. Cancer Inst., *85:* 1038, 1993; Int. J. Oncol., *8:* 221, 1996). The main polyphenol in green tea is epigallocatechin gallate (EGCG). Polyphenol oxidase converts EGCG to theaflavin gallates (which are responsible for the orange-reddish color of black tea infusions). These and other potent polyphenols are believed to account for the beneficial attributes of tea. The tea leaf also contains small amounts of essential oils and terpenes, providing part of the flavor. Fluoride is present as well, having a protective effect on tooth enamel, and the bacteriostatic action of the tea polyphenols might also improve dental health [Chem. Pharm. Bull. (Tokyo), *38:* 717, 1990; Agric. Biol. Chem., *54:* 2925, 1990]. Green tea is consumed mainly in the Far East and North Africa, oolong tea in Taiwan and Southern China, and black tea in the rest of the world. In such places as Turkmenistan and Northern Iran, large amounts are traditionally consumed very hot, a habit that might account in part for the elevated incidence of cancer of the esophagus in those places (IARC Monograph, *51:* 207, 1991), due not to the tea, but to the high temperature at which it is consumed.

Consumption of green tea is associated with a lower risk of cancer of the stomach, esophagus, and lung in some but not all studies (J. Natl. Cancer Inst., *85:* 1038, 1993; Nutr. Rev., *54:* S67, 1996; Int. J. Oncol., *8:* 221, 1996; Carcinogenesis, *18:* 2361, 1997; Nutr. Cancer, *29:* 7, 1997). Tea drinkers have a reduced cardiovascular disease rate (Lancet, *342:* 1007, 1993; Br. J. Cancer, *310:* 693, 1995; J. Agric. Food Chem., *43:* 2798, 1995; Am. J. Clin. Nutr., *65:* 1489, 1997; *66:* 261, 1997). The role of tea in the Western types of cancer, such as cancer of the breast and colon, is still controversial (Eur. J. Cancer Prev., *5:* 425, 1996; Nutr. Cancer, *27:* 1, 1997). In postmenopausal women, tea intake was associated with a lower risk of cancer of the digestive and urinary tracts (Am. J. Epidemiol., *144:* 175, 1996). Intake of black tea together with vegetables and fruits seemed to decrease the incidence of breast and colon cancer by 30–40%, but this effect of tea was not apparent when correcting for vegetable and fruit intake. The conclusion was drawn that tea drinkers often have a healthier lifestyle in terms of avoiding tobacco use, maintaining wholesome nutritional habits, and exercising regularly (Prev. Med., *23:* 377, 1994; J. Natl. Cancer Inst., *88:* 93, 768, 1996).

Tea polyphenols can block the formation of mutagens and carcinogens, such as nitroso compounds affecting the stomach or esophagus or heterocyclic amines that target the colon and breast (Mutagenesis, *11:* 597, 1996; Carcinogenesis, *17:* 2193, 1996). In animal models for cancer of the skin, lung, esophagus, mammary glands, and colon, intake of green or black tea as the sole drinking fluid lowered incidence, multiplicity, and volume of the induced tumors compared to animals on water (Cancer Res., *52:* 1943, 3875, 1992; *54:* 3428, 1994; J. Natl. Cancer Inst., *85:* 1038, 1993; Jpn. J. Cancer Res., *84:* 1007, 1993; Carcinogenesis, *16:* 2143, 1995; *17:* 1429, 1996; Fundam. Appl. Toxicol., *29:* 244, 1996; Cancer Lett., *104:* 205, 1996; *114:* 153, 287, 315, 327, 1997; Med. Res. Rev., *17:* 327, 1997). In many but not all systems, tea inhibited not only the initiation, but also the promotion or progression associated with active oxygen and hydrogen peroxide, blocked by tea antioxidants (Cancer Lett., *96:* 239, 1995; *116:* 47, 1997; Carcinogenesis, *18:* 497, 2163, 1997; Jpn. J. Cancer Res., *88:* 639, 1997). In fact, the growth of induced mouse skin papillomas was inhibited by oral green tea or by i.p. injected EGCG (Cancer Res., *52:* 6657, 1992; *57:* 4414, 1997).

Tea increases the levels of important metabolic enzymes, phase I enzymes of the cytochrome P450 type, and a phase II enzyme, glucuronyl transferase (Carcinogenesis, *15:* 2575, 1994; Xenobiotica, *24:* 119, 1994; Food Chem. Toxicol., *33:* 27, 1995; Int. J. Oncol., *8:* 221, 1996). This effect may be due in part to caffeine (Drug Metab. Dispos., *24:* 529, 1996). Genotoxic carcinogens form key DNA adducts and also generate active oxygen, yielding 8-OH-deoxyguanylate in DNA, an effect inhibited by the tea polyphenols [Cancer Res., *52:* 3875, 1992; Experientia (Basel), *52:* 922, 1996; Jpn. J. Cancer Res., *88:* 553, 1997]. The biochemical activation of carcinogens, measured as mutagenicity or induction of DNA repair, is strongly inhibited by tea polyphenols (Cancer Res., *41:* 67, 1981; Proc. Natl. Acad. Sci. USA, *79:* 5513, 1982; Carcinogenesis, *4:* 1631, 1983; Cancer Lett., *83:* 143, 1994; Int. J. Oncol., *8:* 221, 1996; Food Chem. Toxicol., *34:* 515, 1996; Mutation Res., *359:* 159, 1996; *371:* 57, 1996; *389:* 167, 1997; Environ. Mol. Mutagen., *30:* 468, 1997).

Thus, international research on the health effects of this important beverage consumed as a warm drink or, as is common in the United States, as a cold drink, suggests that perhaps tea should become a part of our dietary traditions to lower the incidence of major chronic diseases, including cancer.

Major contributors to knowledge on the health effects of tea include: J. Chen, F-Q. Lou, Y. Xu, and Y. Gao in China; J-K. Lin in Taiwan; Y. Hara, H. Fujiki, H. Hayatsu, T. Osawa, I. Tomita, M. Suganuma, K. Nakachi, I. Oguni, S. Kono, M. Nagao, T. Sugimura, T. Yamanishi, and M. Kosuge in Japan; M. Green in Israel; I. Stensvold and A. Tverdal in Norway; O. Korver, D. Kromhout, S. Goldbohm, and M. Hertog in the Netherlands; C. Ioannides in the United Kingdom; Z. Apostolides in South Africa; H. Stich in Canada; R. H. Dashwood, H. Graham, D. Balentine, V. Steele, H. Mukhtar, S. Katiyar, Z. Y. Wang, C. S. Yang, A. H. Conney, F-L. Chung, M-T. Huang, C-T. Ho, W. Chen, W. J. Blot, J. F. Fraumeni, Jr., and J. H. Weisburger in the United States.

*The illustration of the tea plant was kindly provided by J. Simrany, U.S. Tea Council.

John H. Weisburger