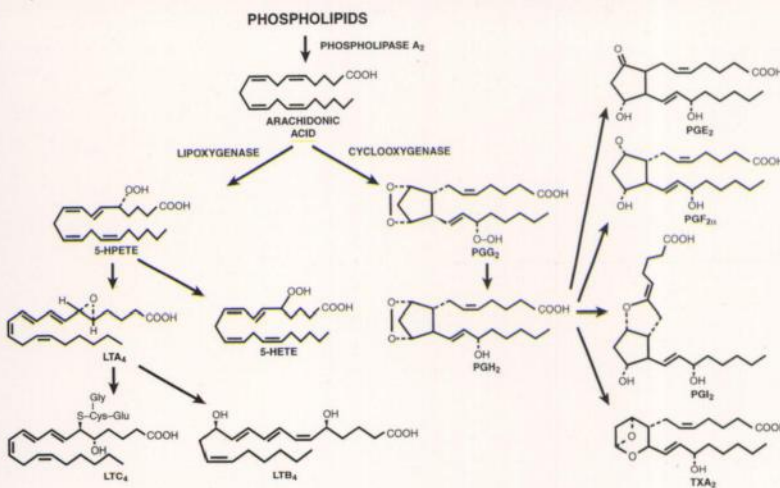




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DECEMBER 12-16, 1997

DNA Methylation, Imprinting, and the Epigenetics of Cancer

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Bestor, New York, NY
El Conquistador Resort and Country Club, Las
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JANUARY 9-13, 1998

Molecular Mechanisms of Apoptosis Regulation

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Dixit, Ann Arbor, MI
Renaissance Esmeralda Resort, Indian Wells (Palm
Springs), CA

JANUARY 24-28, 1998

Angiogenesis and Cancer

Chairpersons: Judah Folkman, Boston, MA; Michael
Klagsbrun, Boston, MA
Hyatt Orlando, Orlando, FL

FEBRUARY 16-21, 1998

Innovative Molecular Biology Approaches to the Prevention, Diagnosis, and Therapy of Cancer

Joint Meeting with the Japanese Cancer Association
Chairpersons: Edward Bresnick, Worcester, MA;
Kaoru Abe, Tokyo, Japan
Maui Marriott Resort, Maui, HI

MARCH 28-APRIL 1, 1998

89th Annual Meeting

Chairperson: Frank J. Rauscher III, Philadelphia, PA
Morial Convention Center, New Orleans, LA
Abstract Deadline: October 28, 1997

JUNE 14-18, 1998

Proteases and Protease Inhibitors in Cancer

Co-Sponsored by the Danish Society for Pathology
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Nyborg Strand Conference Center, Nyborg, Denmark

SEPTEMBER 24-28, 1998

Viral Targets and Cellular Growth Control (tentative title)

Chairperson: Thea D. Tlsty, San Francisco, CA
(additional chairpersons to be announced)
Marriott's Laguna Cliffs Resort, Dana Point, CA

OCTOBER 14-18, 1998

Gene Regulation and Cancer (10th Anniversary of the AACR Special Conferences)

Chairpersons: Phillip A. Sharp, Cambridge, MA, and
Steven L. McKnight, Dallas, TX
The Homestead, Hot Springs, VA

NOVEMBER 11-15, 1998

Endogenous Sources of Mutations

Chairpersons: Lawrence J. Marnett, Nashville, TN;
James A. Swenberg, Chapel Hill, NC; Tomas A.
Lindahl, Herts, England
Sanibel Harbour Resort and Spa, Ft. Myers, FL

DECEMBER 2-6, 1998

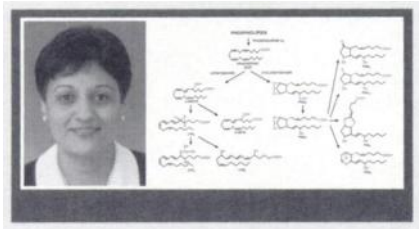
Basic and Clinical Aspects of Prostate Cancer

Chairpersons to be announced
Hyatt Grand Champions Resort, Indian Wells (Palm
Springs), CA

AACR members will receive brochures on the above conferences as soon as they are available. Nonmembers should call or write:



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Numerous studies in nutritional carcinogenesis had supported the view that polyunsaturated fatty acids were required to promote carcinogenesis in several animal tumor models (*Adv. Cancer Res.*, *1*: 451, 1953; *32*: 237, 1980; *Cancer Res.*, *27*: 1737, 1967; *35*: 3231, 1975). By 1981, it was widely accepted that all types of polyunsaturated fatty acids [n-3 present in fish oil (eicosapentaenoic, EPA, C20:5, n-3 and docosahexaenoic, DHA, C22:6, n-3) and n-6 present in vegetable oils (linoleic, C18:2, n-6)] had tumor-promoting activity in experimental models (*J. Natl. Cancer Inst.*, *66*: 517, 1981).

In 1984, Rashida A. Karmali (*cover*), then at the Memorial Sloan-Kettering Cancer Center, first reported a difference in the activities of n-3 and n-6 polyunsaturated fatty acids on the growth of a transplantable mammary tumor (*J. Natl. Cancer Inst.*, *73*: 147, 1984). Dr. Karmali demonstrated that instead of having tumor-promoting activity in experimental tumor models, n-3 fatty acids actually had protective effects. She proposed that both the marine and plant (α -linolenic) n-3 fatty acids could abrogate the tumor-promoting activity of n-6 polyunsaturated fatty acids by competitively inhibiting the metabolism of arachidonic acid.

Dr. Karmali received a B.Sc. in 1971 at Makerere University in Kampala (Uganda) and a M.Sc. in 1973 at Aberdeen University (Scotland). She earned a Ph.D. at the University of Newcastle Upon Tyne (England) on the role of prolactin in the immune system and on experimental tumors of lung and muscle in mice (*Eur. J. Cancer*, *13*: 685, 1977). Subsequently, she moved to the Clinical Research Institute of Montreal (Canada), where her research interests turned to the mechanism of action of prostaglandins and related substances in different disease conditions. Dr. Karmali moved to East Carolina University and carried out a series of studies on the role of polyunsaturated fatty acids in cancer and in autoimmune diseases (*Prog. Lipid Res.*, *20*: 655, 1981). She continued to develop these studies when she joined Robert A. Good at the Memorial Sloan-Kettering Cancer Center in 1980, and later, in 1984, at Rutgers University. A major area of research evolved, namely, biochemical and pathophysiological actions of arachidonic acid products, specifically, prosta-

glandins (PG), thromboxanes (TX), leukotrienes, and hydroxy fatty acids and hydroperoxy fatty acids [collectively referred to as eicosanoids (*see cover illustration*)], in a variety of paraneoplastic syndromes, including tumor initiation and promotion, cell proliferation, tissue invasiveness, metastatic spread, and subversion of immune surveillance. Dr. Karmali demonstrated that cyclooxygenase (cox) enzyme activity was significantly increased in cancerous tissues compared to their normal counterparts. Elevated levels of cox2 products PGE₂ and TXB₂ were associated with tumor growth and metastasis in breast cancer (*Eur. J. Cancer Clin. Oncol.*, *19*: 817, 1983; *Br. J. Cancer*, *48*: 689, 1983). Dr. Karmali demonstrated that intervention with pharmacological agents that inhibit eicosanoid synthesis (non-steroidal anti-inflammatory agents) resulted in inhibition of tumorigenesis (*Prostaglandins Leukot. Med.*, *20*: 283, 1985). Similarly, n-3 fatty acids, and specifically eicosapentaenoic acid (EPA), a competitive inhibitor of arachidonic acid (at the level of lipoxygenase and cyclooxygenase enzymes), also inhibited tumor development, growth, and metastasis in some models (*Prev. Med.*, *16*: 493, 1987; *J. Intern. Med.*, *2225* (Suppl. 1): 197, 1989; *Br. J. Cancer*, *61*: 290, 1990; *Nutrition*, *12*: S2-S4, 1996). In turn, EPA is converted to trienoic prostanoids and thromboxane, and to pentane leukotrienes, all having attenuated activity in comparison with corresponding arachidonic acid products. Both the n-6 and n-3 fatty acids are essential and as such must be provided in the diet. Linoleic acid (n-6) is found in vegetable seeds and oils such as safflower, sunflower, soybean, and corn. Linolenic acid (n-3) is found in dark green leafy plants and in some vegetable oils such as linseed, rapeseed, walnut, and blackcurrant. Deep cold water fatty fish are rich sources of marine n-3 fatty acids (EPA and DHA). The marine food chain is based on n-3 fatty acids which are present in plankton and algae on which fish feed (*Nutr. Today*, *10*: March 1988).

The intriguing question of how a marine diet has protected Greenland Eskimos and the Japanese from coronary heart disease and some types of cancers resulted in a rapidly expanding interest in the protective role of n-3 fatty acids in the prevention and/or treatment of human cancers. Eskimos eat fish and meat from marine mammals rich in n-3 fatty acids. Japanese fishermen have the highest consumption of fish per capita, and clinical investigations with fish and fish oil supplements have demonstrated antithrombotic effects in some patients (*Lancet*, *1*: 441, 1980; *N. Engl. J. Med.*, *318*: 549, 1988). Dr. Karmali performed pioneering research on the protective effects of n-3 fatty acids in cancer models at a time when the widely held notion was that n-3 polyunsaturated fatty acids, like the n-6 fatty acids, had tumor-promoting activity.

In addition to her other degrees, Dr. Karmali also earned a J.D. degree at Rutgers University School of Law in New Jersey, and she is now an attorney specializing in intellectual property and technology transactions at Stroock & Stroock & Lavan LLP in New York. She continues to publish articles on the chemopreventive and protective role of n-3 fatty acids in cancer and has published approximately 100 papers, reviews, and book chapters in all. She is an active member of the American Association for Cancer Research and of Women in Cancer Research. In addition, she was named Outstanding Young Woman of America, 1982, and was honored with the Outstanding Achievement Award, Aga Khan Health Board, 1987.

Sidney Weinhouse