

July 15, 1992

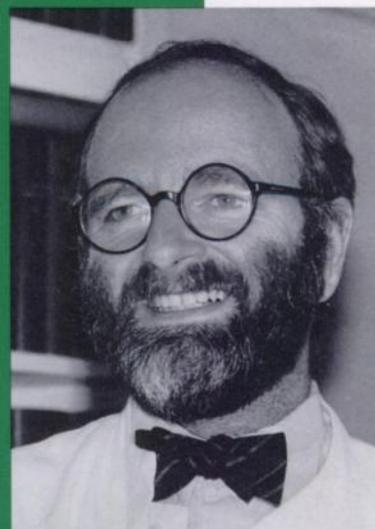
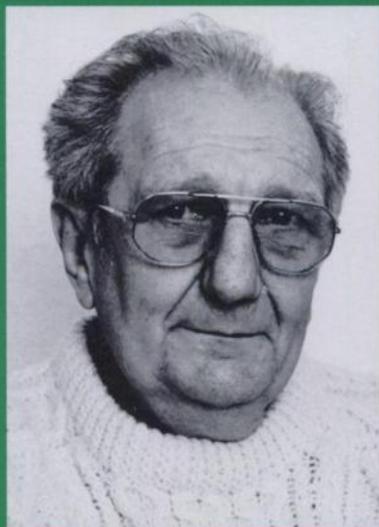
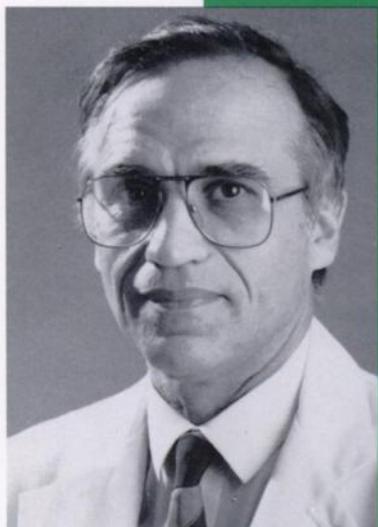


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AACR SPECIAL CONFERENCE IN CANCER RESEARCH

Genetics of Cancer

November 4-8, 1992

Marriott Hilton Head Resort, Hilton Head, South Carolina



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Information and Application Forms

American Association for Cancer Research
Public Ledger Building
620 Chestnut Street, Suite 816
Philadelphia, PA 19106-3483

215-440-9300 215-440-9313 (FAX)

Application Deadline: August 10, 1992

AACR SPECIAL CONFERENCE IN CANCER RESEARCH

Molecular and Biochemical Methods in Cancer Epidemiology and Prevention - The Path Between the Laboratory and the Population

September 23-26, 1992
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Keynote Addresses

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Assessment of Exposure to Genotoxic Agents

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Dietary Biomarkers in Preventive Intervention Studies

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John D. Potter / Minneapolis, MN
Gladys Block / Bethesda, MD
David P. Rose / Valhalla, NY

Measurement of Endogenous Sex Steroid Hormones in Breast and Prostate Neoplasia

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David Schottenfeld / Ann Arbor, MI

Disorders of Immune Function in Human Carcinogenesis

Charles Rabkin / Bethesda, MD
Ian T. Magrath / Bethesda, MD
David T. Purtle / Omaha, NE

Evaluation of the Applications of Biochemical and Molecular Markers in Epidemiological Studies

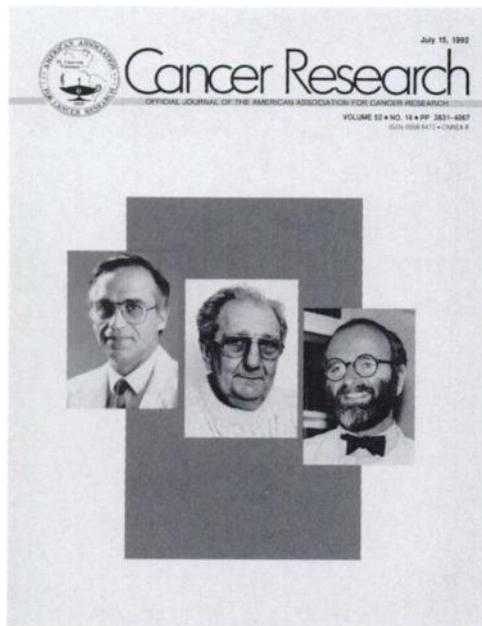
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COVER LEGEND



Alcoholic beverages such as wine and beer have been consumed since antiquity. Distilled spirits were produced as early as the 4th century B.C., but they became available broadly only in the last few centuries as part of the Industrial Revolution. [Alcohol consumption is the subject of a detailed review in International Agency for Research on Cancer (IARC), Monograph 44, 1988.]

The known association of alcoholic beverages with cancer is quite recent. Regular heavy consumers of any type of alcoholic beverage run a risk of liver damage, alcoholic cirrhosis, and liver cancer. Dr. Charles S. Lieber has studied the mechanisms whereby the regular consumption of alcoholic beverages leads to the sequence of events resulting in liver cancer and liver diseases in general (Proc. Natl. Acad. Sci. USA, 72: 437, 1975; Cancer Res., 39: 2863, 1979; Hepatology, 6: 1005, 1986; Annu. Rev. Pharmacol. Toxicol., 30: 219, 1990). Alcohol is metabolized by alcohol dehydrogenase to acetaldehyde and then oxidized to acetate. The collaboration between Drs. Lieber and Helmut Karl Seitz led to the discovery that chronic or high intake of alcohol induces microsomal enzymes, the cytochrome P-450 system and, particularly, cytochrome P450IIE1. Their function is to oxidize alcohol and specific types of chemical carcinogens and also various drugs, retinoids, and vitamins, thus accounting for the interactions observed between alcohol intake and the effect of the other agents [Annu. Rev. Nutr., 8: 99, 1988; Annu. Rev. Pharmacol. Toxicol., 30: 219, 1990; Eur. J. Clin. Invest., 11: 33, 1981; Carcinogenesis (Lond.), 3: 1457, 1982; Cancer Res., 47: 3123 and 4305, 1987; J. Am. Coll. Nutr., 10: 602, 1991].

Dr. Ernst L. Wynder (Cancer Res., 35: Cover 2, 1975)

described the occurrence of cancer in the oral cavity, larynx, and esophagus in individuals who smoke and consume alcohol. This interaction has been the subject of detailed studies in Europe by Albert Tuyns (Bull. Cancer, 64: 45, 1977) who demonstrated that in certain regions of France there was a greater incidence of cancer in the esophagus even in nonsmokers (Int. J. Cancer, 32: 443, 1983; 41: 483, 1988; Nutr. Cancer, 9: 81, 1987; Br. J. Cancer, 64: 415, 1991). Alcohol modifies the metabolism of carcinogens in the esophagus and increases DNA synthesis and cell replication [Cancer Res., 41: 2849, 1981; Gastroenterology, 93: 362, 1987; Carcinogenesis (Lond.), 10: 303, 1989]. Thus, there is a synergistic interaction between alcohol and tobacco.

Cancer of the large bowel is generally associated with a high fat, low fiber nutritional regimen. However, cancer of the rectum also occurs more frequently in regular drinkers. Alcohol use may account for rectal, but not colon, cancer in populations, like that of Japan, on a low fat diet. Dr. Seitz (*Alcoholism: A Molecular Perspective*. New York: Plenum Publishing Corp., 1991) has contributed much to the understanding of the relevant mechanism. In laboratory rodents, alcohol increases the rate of cell replication in the rectum (Gut, 27: 278, 1986). Acetaldehyde, a reactive metabolite of alcohol, was also present in the rectum (Gastroenterology, 98: 1 and 406, 1990). This particular mechanism may also apply to the esophagus. Thus, the effect of alcohol and acetaldehyde involves several mechanisms, including the induction of enzymes and nutritional deficiencies, especially of vitamin A, and also cytotoxicity, promotion, and increased cell replication. In the presence of carcinogens, such as those from tobacco, alcohol can act as a cocarcinogen.

The research featured has been recognized by a number of awards to the individuals described.

Charles S. Lieber, M.D. (*upper left*), is Professor of Medicine and Pathology, Mount Sinai School of Medicine, and Director of the Alcohol Research and Treatment Center and the G.I.-Liver Training Program at the Bronx VA Medical Center, New York, New York. Dr. Lieber has authored over 700 scientific publications and five books dealing with the roles of nutrition and toxic factors in the pathogenesis of alcoholic liver disease. He is the recipient of the Hugh R. Butt Award for Clinical Research in Liver and Nutrition, given by the American Gastroenterological Association.

Helmut Karl Seitz, M.D. (*lower right*), is Professor of Medicine at the University of Heidelberg and Director of the Department of Internal Medicine, Salem Medical Center, Heidelberg, Germany. He has published over 150 papers and edited two books.

Albert Tuyns, M.D. (*middle*), officially retired in 1982, but he maintains his affiliation with the IARC, Lyon, France. Among his accomplishments of continuing value are the establishment of Cancer Registries in Europe, especially in Latin countries, and the development of World Health Organization and IARC monographs and teaching manuals.

John H. Weisburger