ASPO

Ninth Annual Meeting of the

American Society of Preventive Oncology

April 10–12, 1985

Royal York Hotel

Toronto, Ontario, Canada

MASTER AGENDA

Wednesday , April 10

MORNING	AFTERNOON	EVENING
	Registration 12:00 - 5:00 pm	**Free evening**
	Workshop - Quantitation of carcinogens in the environment 2:00 - 5:00 pm QUEBEC ROOM	

Thursday, April 11

MORNING	AFTERNOON	EVENING
Registration 8:00 am - 4:30 pm	**Videotape** Cigarette advertising and the health risks of smoking 12:00 - 12:30 pm and 1:00 - 1:30 pm	**Reception** 6:30 - 7:30 pm TERRITORIES ROOM
Welcoming remarks 8:30 - 8:45 am ALBERTA ROOM	**Distinguished achievement award** 1:30 - 2:30 pm	**Banquet** Goals of the NCI for cancer prevention - the year 2000 program 7:30 - 10:30 pm TERRITORIES ROOM
Keynote address The role of toxicology in strategies for cancer prevention 8:45 - 9:30 am	**Selected papers** 2:30 - 4:45 pm	
Symposium I Aids: Etiology and prevention 10:00 am - 12:00 pm		

Friday, April 12

MORNING	AFTERNOON	EVENING
Presidential address Screening for cancer: Issues and future directions 8:30 - 9:15 am ALBERTA ROOM	**Selected papers** 1:30 - 2:30 pm	
Selected papers 9:15 - 9:55 am	**Symposium III -** Prevention of primary hepatocellular carcinoma 2:30 - 4:30 pm	
Symposium II - Public policy issues related to cancer prevention 10:10 am - 12:10 pm	**Adjourn**	

PROGRAM AND SELECTED PAPERS

Welcome to Toronto!

Program chairman: W. Thomas London, MD

sponsored by:

American Society of Preventive Oncology

American Cancer Society

Conference information

This ninth annual meeting of the American Society of Preventive Oncology focuses on some of the current major issues in cancer prevention: quantitation of environmental carcinogens, AIDS, public policy in smoking and asbestos control, and the prevention of primary hepatocellular carcinoma. The program chairman, Dr. W. Thomas London, and the Executive Committee of ASPO have brought together what promises to be an extraordinary series of presentations by leaders in their fields of scientific inquiry. This meeting is supported by ASPO and by a generous grant from the American Cancer Society.

ASPO

In its ninth year ASPO is striving to

- promote the exchange and dissemination of information and ideas relating to cancer prevention and control;
- identify and stimulate research areas in cancer prevention and control;
- foster the implementation of programs in cancer prevention and control.

A growing and active membership facilitates achievement of these goals. The Executive Committee and council members listed below are very interested in hearing from prospective or current members.

Anthony B. Miller, M.B., F.R.C.P.
NCIC Epidemiology Unit
McMurrich Building
University of Toronto
Toronto, Ontario
Canada M5S 1A8
(416)978-5097

Nicholas Petrakis, M.D.
University of California
Dept. of Epidemiology &
International Health
1699 HSW
San Francisco, CA 94143
(415)666-2001

David Schottenfeld, M.D.
Memorial Sloan-Kettering Cancer Center
Chief, Epidemiology & Preventive Medicine Service
1275 York Avenue
New York, N.Y. 10021
(212)794-6580

Nathaniel I. Berlin, M.D.
Northwestern University
303 E. Chicago Avenue
Chicago, IL 60611
(312)266-5250

Joseph W. Cullen, Ph.D.
National Cancer Institute
Bldg. 31, Rm. 4A32
900 Rockville Pike
Bethesda, MD 20205
(301)496-9569

Virginia L. Ernster, Ph.D.
University of California
Dept. of Epidemiology &
International Health
San Francisco, CA 94143
(415)666-1424

Robert S. Fontana, M.D.
200 First St., S.W.
Rochester, MN 55901
(507)284-2494

Richard R. Love, M.D.
Cancer Prevention Program
1300 University Ave. - 7C
Madison, WI 53706
(608)263-7066

W. Thomas London, M.D.
Institute for Cancer Research
7701 Burholme Ave.
Philadelphia, PA 19111
(215)728-2203

Joseph F. Fraumeni, Jr., M.D.
Environmental Epidemiology Branch
National Cancer Institute
Landow Bldg.
7910 Woodmont Ave.
Bethesda, MD 20205
(301)496-1611

Curtis J. Mettlin, Ph.D.
Roswell Park Memorial Institute
666 Elm Street
Buffalo, N.Y. 14263
(716)845-4406

Daniel G. Miller, M.D.
PMI-Strang Clinic
55 E. 34 Street
New York, N.Y. 10016
(212)683-1000

Guy R. Newell, M.D.
University of Texas System Cancer
System
Texas Medical Center
Houston, TX 77030
(713)792-3020

John H. Weisburger, Ph.D.
American Health Foundation
Valhalla, N.Y. 10595
(914)592-2600, Ext. 302

Charles Key, M.D.
University of New Mexico
School of Medicine
Albuquerque, New Mexico 87131
(505)277-5541

The National Office for ASPO is at the University of Wisconsin, where the executive secretary, Ms. Joan Wiegel, may be contacted for any information or assistance: 1300 University Ave. - 7C, Madison, WI 53706, (608)263-2118.

CME credit

No arrangements for continuing medical education credit for this meeting have been made.

Messages

Please see Ms. Joan Wiegel at the registration desk if you wish to leave or expect a message.

Banquet

Please let Ms. Joan Wiegel know immediately of your plans to attend the banquet on Thursday evening.

Dining and discovering Toronto

The booklet "Metropolitan Toronto Discovery Guide 1985" is available at the registration desk.

Special acknowledgement

The Executive Committee of ASPO wishes to offer special thanks to Dr. W. Thomas London, the program chairman, for his tireless efforts in arranging this meeting.

Wednesday, April 10

12:00 pm - 5:00 pm	REGISTRATION
2:00 pm - 5:00 pm	**WORKSHOP** **Quantitation of carcinogens in the environment**
QUEBEC ROOM	Moderator: John Weisburger, PhD
	Dose-response studies
	David Gaylor, PhD
	Risk assessment in animals and humans
	Daniel Krewski, PhD
	Classification of carcinogens and mechanisms of carcinogenesis variation by species
	Gary Williams, MD

Thursday, April 11

8:00 am - 4:30 pm	REGISTRATION
8:30 am - 8:45 pm	WELCOMING REMARKS
ALBERTA ROOM	Anthony Miller, MB, FRCP(C) President, ASPO
8:45 am - 9:30 am	KEYNOTE ADDRESS **The role of toxicology in strategies for cancer prevention** Ian C. Munro, MD
9:30 am - 10:00 am	REFRESHMENT BREAK
10:00 am - 12:00 pm	SYMPOSIUM I **AIDS: Etiology and prevention** Moderator: David Schottenfeld, MD **Viral etiology** Jay Levy, MD **Discussant** Robert J. Biggar, MD **Risk factors and prevention** Michael Marmor, MD **Discussant** Colin Soskolne, PhD
12:00 pm - 1:30 pm	LUNCH
12:00 pm - 12:30 pm	VIDEOTAPE PRESENTATION **Mixed messages: Cigarette advertising and the health risks of smoking** Virginia Ernster, PhD
1:00 pm - 1:30 pm	VIDEOTAPE PRESENTATION **Mixed messages: Cigarette advertising and the health risks of smoking** Virginia Ernster, PhD

1:30 pm - 2:30 pm		ASPO DISTINGUISHED ACHIEVEMENT AWARD PRESENTATION **1985 Award recipient:** Sam Shapiro **Presentation of award:** Anthony B. Miller, MB, FRCP(C) **Awardee address: Cancer control and the health care system**
2:30 pm - 3:30 pm		SELECTED PAPERS Moderator: Anthony B. Miller, MB, FRCP(C)
2:30 pm - 2:50 pm	1*	**Neuroblastoma and paternal occupation** Margaret R. Spitz, MD
2:50 pm - 3:10 pm	2	**Factors that increase the risk of a second tumor in children with a primary soft-tissue sarcoma** Wick R. Williams, PhD
3:10 pm - 3:30 pm	3	**Cholecystectomy and colon cancer: Lack of association** Guy R. Newell, MD
3:30 pm - 3:45 pm		REFRESHMENT BREAK
3:45 pm - 4:45 pm		SELECTED PAPERS Moderator: Nicholas Petrakis, MD
3:45 pm - 4:05 pm	4	**Elevated levels of bioassayed serum prolactin (PRL) in women at risk for familial breast cancer** Richard R. Love, MD, MS
4:05 pm - 4:25 pm	5	**Glutathione transferase as a possible marker of risk for lung cancer** Ronald W. Pero, PhD
4:25 pm - 4:45 pm	6	**Appearance of nevi, palpable arm nevi, and the clinical diagnosis of non-familial dysplastic nevus** George C. Roush, MD
4:45 pm - 5:30 pm		BUSINESS MEETING
6:30 pm - 7:30 pm		RECEPTION
TERRITORIES ROOM		
7:30 pm - 10:30 pm		BANQUET
TERRITORIES ROOM		**Goals of the National Cancer Institute for cancer prevention - the Year 2000 program** Peter Greenwald, MD

*Number refers to the abstract printed in the selected paper section of this program.

Friday, April 12

8:30 am - 9:15 am		**PRESIDENTIAL ADDRESS** **Screening for cancer: Issues and future directions** Anthony B. Miller, MB, FRCP(C)
9:15 am - 9:55 am		**SELECTED PAPERS** Moderator: Richard R. Love, MD, MS
9:15 am - 9:35 am	7	**The impact of a community breast self-examination program** James E. Enstrom, PhD
9:35 am - 9:55 am	8	**Attitudes toward cancer and having had cancer-related tests** Zili Amsel, ScD.
9:55 am - 10:10 am		**REFRESHMENT BREAK**
10:10 am - 12:10 pm		**SYMPOSIUM II** **Public policy issues related to cancer prevention** Moderator: Virginia Ernster, PhD **Smoking** John Pinney **Discussant** Mary Jane Ashley, MD **Asbestos** J. Fraser Mustard, PhD **Discussant** Richard Lemen
12:10 pm - 1:30 pm		**LUNCH** Executive Committee Lunch PRINCE EDWARD ISLAND ROOM
1:30 pm - 2:30 pm		**SELECTED PAPERS** Moderator: David Schottenfeld, MD
1:30 pm - 1:50 pm	9	**Mutagenic cervical mucus in women smokers** Elizabeth A. Holly, PhD, MPH

1:50 pm - 2:10 pm	10	**The sex ratio of the offspring of patients with cancer of the prostate** Gerry B. Hill, MD
2:10 pm - 2:30 pm	11	**Prevention of chronic liver disease and liver cancer: Hepatitis B virus replication and tuberculin reactivity** Katherine McGlynn, PhD
2:30 pm - 4:30 pm		**SYMPOSIUM III** **Prevention of primary hepatocellular carcinoma** Moderator: W. Thomas London, MD **Epidemiology** R. Palmer Beasley, MD, DPh **Discussant** Paul M. Newberne, DVM, PhD **Prevention** James F. Maynard, MD, PhD **Discussant** Brian McMahon, MD
4:30 pm		**ADJOURNMENT**

INVITED SPEAKERS

Mary Jane Ashley, MD
University of Toronto
Toronto, Canada

Zili Amsel, ScD
Fox Chase Cancer Center
Philadelphia, Pennsylvania

R. Palmer Beasley, MD, DPh
Director, University of Washington
Medical Research Unit
Taipei, Taiwan

Robert J. Biggar, MD
Environmental Epidemiology Branch
National Cancer Institute
Bethesda, Maryland

James E. Enstrom, PhD
University of California
School of Public Health
Los Angeles, California

Virginia Ernster, PhD
University of California, San Francisco
Medical School
Department of Epidemiology and International Health
San Francisco, California

David Gaylor, PhD
National Center for Toxicological Research
Jefferson, Arkansas

Peter Greenwald, MD
Director, Division of Cancer Prevention and Control
National Cancer Institute
Bethesda, Maryland

Gerry B. Hill, MB
Alberta Cancer Board
Edmonton, Alberta Canada

Elizabeth A. Holly, PhD, MPH
Northern California Cancer Program
Palo Alto, California

Daniel Krewski, PhD
Health Protection Branch
Health and Welfare
Ottawa, Ontario Canada

Richard Lemen
Director, Division of Standards Development and Technology Transfer
National Institute of Occupational Safety and Health
Cincinnati, Ohio

Jay Levy, MD
Cancer Research Institute
Department of Medicine
University of California School of Medicine
San Francisco, California

W. Thomas London, MD
Fox Chase Cancer Center
Philadelphia, Pennsylvania

Richard R. Love, MD, MS
University of Wisconsin-Madison
Wisconsin Clinical Cancer Center
Madison, Wisconsin

Michael Marmor, MD
The Institute of Environmental Medicine
New York University Medical Center
New York, New York

James F. Maynard, MD, PhD
Director, World Health Organization
Collaborating Center for Reference and Research on Viral Hepatitis
Division of Viral Diseases
Center for Disease Control
Atlanta, Georgia

Katherine McGlynn, PhD
Institute for Cancer Research
Philadelphia, Pennsylvania

Brian McMahon, MD
Department of Medicine
Alaska Native Medical Center
Anchorage, Alaska

Anthony Miller, MB, FRCP(C)
President, ASPO
Director, NCIC Epidemiology Unit
University of Toronto
Toronto, Ontario Canada

SELECTED PAPERS

NEUROBLASTOMA AND PATERNAL OCCUPATION: A CASE-CONTROL ANALYSIS: Margaret R. Spitz, M.D., Christine Cole Johnson, M.P.H., Houston, Texas.

The peak incidence of neuroblastoma (NBL) during early infancy suggests that prezygotic or prenatal exposures to carcinogens could be implicated. Several recent epidemiologic studies have documented an association between parental exposure to hydrocarbons and the aircraft manufacturing industry and the development of nervous system cancer in the offspring. This is a case control epidemiologic analysis of the paternal occupation of 157 children under 15 years of age who died from NBL in Texas during the time period 1964 through 1978. As controls, 314 children were selected, by stratified random sampling, from all Texas live births with the same birth year distribution as the cases. Data on paternal occupation and industry and other demographic variables were abstracted from birth certificates. Neither hydrocarbon-related work exposures nor employment in the aircraft industry was associated with increased risk for NBL. However, children of fathers employed in occupations with electromagnetic field exposure were at significantly increased risk for NBL (odds ratio = 2.14). These occupations included electricians, electric and electronics workers, linemen, and utility employees and welders. The odds ratio was 11.3 for children of fathers who reported themselves to be electronics workers (6 cases, 1 control). Previous reports of exposure to electromagnetic fields and induction of cancer, as well as potential chemical exposures in these industries, are outlined.

FACTORS THAT INCREASE THE RISK OF A SECOND TUMOR IN CHILDREN WITH A PRIMARY SOFT-TISSUE SARCOMA. W.R. Williams, Ph.D., L.C. Strong, M.D., E. Lustbader, Ph.D., M. Stine, M.P.H. Fox Chase Cancer Center, Philadelphia, PA; M.D. Anderson Hosp. and Tumor Institute, Houston, TX.

A cohort of patients who had a soft tissue sarcoma diagnosed before 16 years of age and who had survived at least three years were identified from a list of patients seen at M.D. Anderson Hospital before 1975. Of the 161 patients, 9 have developed a second primary malignancy; less than one second malignancy was expected. Another 15 individuals have developed benign tumors.

To identify patient characteristics that increase the risk of a second tumor, survival analysis under the proportional hazards model was used to test the significance of epidemiologic (sex, age at diagnosis, race, histology), treatment (radiation and/or chemotherapy), and genetic variables. The genetic variable was determined from the results of segregation analysis involving the patient's 1st and 2nd degree relatives. This defined a subgroup of 11 families in which cancer appears to be transmitted as an autosomal dominant trait. The genetic variable we used reflects the likelihood that cancer in the patient's family is inherited vs. the likelihood that it occurred by chance.

An early age at diagnosis and having a rhabdomyosarcoma primary lesion were associated with increased risk of developing a second tumor. None of the cancer treatment variables were significant; however, the genetic variable was important ($p<.05$), and individuals whose family history of cancer was more likely to be due to an inherited mechanism were at increased risk to develop a second primary tumor. This finding suggests that family histories of patients should be obtained and close surveillance be maintained on those that have a strong family history of cancer. (Sup. by CA34097 and CA38929).

CHOLECYSTECTOMY AND COLON CANCER: LACK OF ASSOCIATION.

G. R. Newell, M. R. Spitz, V. F. Guinee, N. C. Russell.
University of Texas M. D. Anderson Hospital, Houston.

This study was conducted to test the null hypothesis of no association between right-sided colon cancer and previous cholecystectomy. Importance of the question rested with the prevalence of gallbladder removal in the U.S. A case-control, hospital chart review was conducted on 267 white women with right-colon cancer (RSC, cases) and 268 women with left-colon cancer (LSC, controls) to compare prevalence of prior removal of the gallbladder between the two groups. Thirty of 256 cases (11.7%) and 21 of 261 controls (8.0%) had a cholecystectomy 6 months or longer before diagnosis of colon cancer. The risk estimate for cases vs. controls was 1.5 (95% CI 0.8, 2.7). Previous studies exploring the association between cholecystectomy and colon cancer have yielded inconsistent findings. Results of this study, together with an independent one recently published, provide no evidence for a significant etiologic role of cholecystectomy in the development of right-sided colon cancer.

ELEVATED LEVELS OF BIOASSAYED SERUM PROLACTIN (PRL) IN WOMEN AT RISK FOR FAMILIAL BREAST CANCER.

Richard R. Love, M.D. and David P. Rose, Ph.D. Wisconsin Clinical Cancer Center, Madison, WI 53792 and American Health Foundation, Valhalla, NY 10595

In women at increased risk for breast cancer, specific endocrinologic abnormalities have been found inconsistently. Prolactin (PRL) has been of particular interest because of its demonstrated role in rodent mammary carcinogenesis. A new sensitive and specific bioassay for lactogenic hormones (prolactin and growth hormone, GH) has recently been described (J. Clin. Endocrinol. Metab. 51:1058, 1980). We studied 8 healthy, unrelated premenopausal women, each of whom had at least two first degree relatives with breast cancer and a history of familial breast cancer. Throughout one menstrual cycle, morning blood samples were taken and thyrotropin releasing hormone (TRH) stimulation tests were done in mid-follicular and mid-luteal phases in each woman. Compared with previously studied controls, basal and TRH stimulated serum PRL and GH levels determined by the standard radioimmunoassays were unremarkable, while basal levels of serum prolactin by bioassay were significantly elevated, and responses to TRH stimulation showed exaggerated bioactive to immunoreactive PRL ratios. We conclude that, as in the rat, different forms of PRL exist in humans, and that a bioassayable form of PRL is significantly elevated in a homogeneous subset of women at risk for familial breast cancer. Supported by K07-CA00721-05, NCI/NIH.

GLUTATHIONE TRANSFERASE AS A POSSIBLE MARKER OF RISK FOR LUNG CANCER.

Ronald W. Pero, Janeric Seidegard and Daniel G. Miller, PMI-Strang Clinic, New York City.

The glutathione transferases are a group of multifunctional proteins which play an important role in the biotransformation and detoxification of many different endogenous and exogenous compounds. Recently, we have determined the hereditary characteristics of a glutathione transferase (GT) in human mononuclear leukocytes having high activity towards trans-stilbene oxide. Our data are consistent with a dominant expression of a single gene located on an autosomal chromosome for this high GT activity. We have applied this GT assay to age-matched individuals with and without lung cancer who all had a strong previous history of cigarette smoking. The data in Table I show a significant under representation of high GT activity levels among individuals with lung cancer. Therefore, these data suggest that low GT activity may be a marker for increased risk to lung cancer.

TABLE I

Glutathione transferase activity (pmol/min/10^7 cells)

	Low Group (100)	High Group (600)
Lung Cancer Patients	n=35	n=20
Controls	n=32	n=46

Chi Square=5.72 p 0.02

APPEARANCE OF NEVI, PALPABLE ARM NEVI, AND THE CLINICAL DIAGNOSIS OF NON-FAMILIAL DYSPLASTIC NEVUS. Roush GC, Ernstoff M, Titus L, Kirkwood JM, Barnhill R, Duray PH, Stenn KS, Klaus SN, Lerner AB. Yale Melanoma Unit, New Haven, CT.

Clinical recognition of the dysplastic nevus (DN) is deemed important in the prevention and follow up of melanoma. However, quantitative data are lacking. In this study, patients with newly diagnosed cutaneous melanoma (CM) without evidence for familial dysplastic nevus syndrome were examined and the most atypical nevus biopsied in every patient undergoing exam. Prior to biopsy, each mole was characterized independently by two examiners regarding presence or absence of features thought to characterize DN: diameter > 5 mm, a macular component, irregular border, ill-defined border, variegate color, and erythema. Histopathologic diagnosis of DN was positively and significantly correlated with presence of 4 or more clinical features (specificity for each examiner = 79%, sensitivity = 58% and 63%). Histopathologic diagnosis of DN was correlated with each of the 6 clinical features. Histopathologic diagnosis of DN was also strongly and significantly associated with total number of palpable nevi on the arms--a nevus count obtained because it has been strongly associated with CM in epidemiologic studies. Combinations of the 6 clinical features with total number of palpable arm nevi permitted adjustment of specificity (up to 93% with sensitivity maintained above 50%) or sensitivity (up to 83% with specificity above 70%). Although further refinement is vital, combinations of clinical and epidemiologic variables point to a diagnostic index applicable to research on etiology and prevention of CM and to research and clinical care of patients with CM.

THE IMPACT OF A COMMUNITY BREAST SELF-EXAMINATION PROGRAM.

James E. Enstrom, Ph.D., and Linda E. Kanim, M.A.
University of California, Los Angeles, CA 90024.

The goals of this study are to assess long-term breast self-examination (BSE) practice among a cohort of women trained to do BSE and to examine the impact of this practice on their subsequent incidence, staging, and mortality from breast cancer. Initially, 22,000 self-selected women were trained to do BSE during 1978-81 at five hospital-based breast examination training centers throughout the Los Angeles area as part of the Community Cancer Control/Los Angeles (CCC/LA) project. A random sample of 2500 of these women was telephoned in 1980-81 at 12 to 20 months after training to determine BSE frequency and technique. A second independent random sample of 500 women was telephoned in 1984 at 40-70 months after training and questioned about current BSE practice. A number of these women had their BSE technique examined in-person. This cohort of women, although self-selected, appears to be demographically similar to Los Angeles County adult women. For breast screening, the women rely primarily on BSE and physician examinations and not on mammography. Their overall level of breast screening is very high. About 40% of the women practiced BSE at the time of training and have continued to do so, 40% have taken up and continued BSE practice since training, and 20% have not practiced BSE at all. There has been a substantial increase in the portion of the cohort that practices BSE at least monthly--25% at time of initial training, 60% at 12-20 months follow-up, and 50% at 40-70 months. General population surveys indicate that about 35% of adult women practice BSE at least monthly. The cohort is currently being followed for breast cancer, incidence staging, and mortality. This is the first cohort study in the U.S. of BSE and may determine the impact of BSE on reducing breast cancer mortality.

ATTITUDES TOWARD CANCER AND HAVING HAD CANCER-RELATED TESTS

Zili Amsel, Sc.D. and Andrew Balshem, B.A., Fox Chase Cancer Center, Philadelphia, PA.

The purpose of this analysis is to examine the relationship of attitudes toward cancer and ever having had cancer-related tests. Residents of a high cancer mortality community were surveyed by mail to determine their levels of knowledge about cancer, their attitudes toward cancer and, their experiences and practices related to cancer. Responses were received from 1538 residents (23% of a random sample of 4320 households or approximately 6560 residents). Attitude statements were grouped according to the components of the Health Belief Model-perceived susceptibility of getting cancer, perceived severity of having cancer, perceived benefits of early detection and perceived social costs of detection. Respondents were asked to identify tests or procedures to detect cancer and which of these they had experienced (chest x-ray, examination of skin, digital rectal examination, proctoscopic examination, breast examination by a physician, pap test, regular oral self-examination, regular breast self-examination and regular skin-examination). Respondents were classified as to whether they had experienced each test or procedure. Preliminary analysis showed attitudes measuring perceived severity of having cancer being highly associated with have a chect x-ray, rectal examination and proctoscopic examination as well as regular performance of oral and skin self-examination. Measures of perceived benefits were important for rectal examination, and for women examination of breasts by a physician and performance of breast self-examination. Educational status was associated with performance of oral and skin self-examination in all respondents and, for women, examination of breasts by a physician and pap tests. The implications of these results for a community health education program are discussed.

This project is supported by NCI PHS grant # CA34856.

MUTAGENIC CERVICAL MUCUS IN WOMEN SMOKERS. EA Holly PhD, NL Petrakis MD, NL Friend MS, et al. University of California, San Francisco and Northern California Cancer Program, Palo Alto, California

This study was conducted to determine if mutagenic substances could be detected in the secretions of the uterine cervix of smokers more frequently than nonsmokers. Cervical fluids were obtained from 78 premenopausal women seen at the University of California dysplasia and diethylstilbestrol clinic, or in a private practice. The women were interviewed with regard to their smoking and reproductive histories. The cervical fluids were tested for mutagenicity using the Salmonella microsomal test. Fourteen of 36 women (39%) who smoked had positive tests as compared to 5 of 42 nonsmokers (12%). The age-adjusted estimate of the odds ratio (OR) was 7.8 with 95% confidence limits (CL) of 2.1 to 29.0. Among smokers, 13 of 14 women (93%) who were positive on the laboratory test had smoked within 6 hours prior to sample collection while 18 of 22 women (82%) with negative tests had smoked during this period (age-adjusted OR = 3.8 with 95% CL of .40 to 36.5). Women who smoked 3-10 and 11+ cigarettes per day were more likely to have positive laboratory test results when compared to nonsmokers (for 3-10 cigarettes, age-adjusted OR = 10.3 with 95% CL of 2.5 to 42.6; for 11+ cigarettes, OR = 3.3 with 95% CL of .90 to 11.9). Women with dysplasia or carcinoma in situ were more likely to be positive on the test for mutagenicity when compared with all other women (OR = 3.1 with 95% CL of .99 to 9.9). This relationship between smoking and mutagenic cervical fluids offers biochemical evidence for the association between cervical cancer and cigarette smoking that has been noted in previous epidemiologic studies.

THE SEX RATIO OF THE OFFSPRING OF PATIENTS WITH CANCER OF THE PROSTATE

G. B. Hill, M.B., S. Fincham, M.A.,
C. Wijayasinghe, M.P.V.M. and C. Haronga, B.A.

We have completed a population-based case-control study of prostatic cancer in Alberta. We focus here on the fertility of the 354 cases and 597 controls who had married at least once. Considering offspring of both sexes, the mean fertility of the cases was 3.17, not significantly different from the mean of 3.11 for the controls (X^2=0.22, p>0.10). However the sex-ratios were significantly different, 56.5 percent of the cases' offspring being sons, compared with 50.1 percent for the controls (X^2=11.47, p<0.001). The equality of total fertility, but increase in the sex ratio of the cases' offspring, persisted after stratification by age at first marriage, which was found to be a risk factor in this study. If the finding is confirmed by other studies then an abnormality of one of the sex chromosomes, or of the seminal fluid, may be implicated in prostatic cancer.

PREVENTION OF CHRONIC LIVER DISEASE AND LIVER CANCER: HEPATITIS B VIRUS REPLICATION AND TUBERCULIN REACTIVITY.

K. McGlynn, Ph.D., W.T. London, M.D., E. Lustbader Ph.D., B. McMahon, M.D., W. Heyward, M.D. Fox Chase Cancer Center Philadelphia, PA; Centers for Disease Control, Anchorage, AK; Indian Health Servce, Anchorage, AK.

Chronic carriers of hepatitis B virus (HBV) who are also positive for hepatitis B e antigen (HBeAg(+)) are actively replicating virus and are more likely to develop chronic hepatitis (CAH) than HBeAg(-) carriers. Carriers with CAH are at higher risk of developing cirrhosis and primary liver cancer than carriers without CAH.

In a prior study of southeast Asian refugees, a population with high prevalences of M.tuberculosis (TB) and HBV infections, we detected an inverse association between HBeAg status and tuberculin skin test (PPD) reactivity. Among 224 HBV carriers, PPD(+) persons were significantly less likely to be HBeAg(+) than PPD(-) persons. PPD positivity increased with age and HBeAg positivity decreased with age, but the HBeAg/PPD association remained constant across age groups. To test whether this relationship occurs in other populations, we studied carriers in the Alaska native American population.

We identified, from the Alaska hepatitis B registry, 613 carriers whose HBeAg status had been determined. We used records of the Alaska Native Medical Service and the Alaska Tuberculosis Control Program to ascertain the PPD reactivity of each subject. Analysis of this data confirmed the inverse relationship of HBeAg and PPD reactivity. The Mantel-Haenszel common odds ratios (across age groups) of the Alaskan and southeast Asian populations were identical; 2.13.

Since PPD reactors are more likely to be HBeAg(-), we are investigating whether bacille Calmette Guerin (BCG) vaccination has an effect similar to natural TB infection, i.e.; interferes with the replication of HBV in carriers.