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PASSIVE AND ACTIVE SMOKING AND BREAST CANCER: THE ACCUMULATING EVIDENCE. *K C Johnson (Public Health Agency of Canada, Ottawa, ONT, Canada) and *A J Wells

The aim of the study was to examine the risk of breast cancer associated with passive and active smoking and to explore risk heterogeneity between studies. Seventeen of 18 located published studies of passive smoking and breast cancer risk among women met basic quality criteria. Pooled relative risk estimates for breast cancer were calculated for: 1) life-long nonsmokers with regular passive exposure to tobacco smoke; and 2) women who smoked. They were compared to women who were never regularly exposed to tobacco smoke. The pooled breast cancer relative risk estimate associated with passive smoking among life-long nonsmokers was 1.40 (95% confidence interval (CI), 1.17–1.68). In the subset of 5 studies (all case-control studies) with more complete exposure assessment (quantitative long-term information on the three major sources of passive smoke exposure: childhood, adult residential and occupational), the pooled risk estimate for exposed non-smokers was 1.89 (95%CI, 1.52–2.36). For the studies with less complete passive exposure measures the risk was 1.09 (95%CI, 0.97–1.23) overall, 1.03 for cohort, and 1.24 for case-control studies. Asian, but not North American cohort studies suggested increased breast cancer risk for higher exposure. The overall premenopausal breast cancer risk associated with passive smoking among lifelong non smokers was 1.86 (95%CI 1.49 – 2.66), and 2.20 (95% CI 1.70–2.85) for those studies with more complete exposure assessment. For women who had smoked the breast cancer risk estimate was 1.53 (95%CI 1.22- 1.91) when compared to women with neither active nor regular passive smoke exposure; 2.08 (95% CI 1.44-3.01) for more complete and 1.15 (95% CI 0.98–1.35) for less complete passive exposure assessment. Studies with thorough passive smoking exposure assessment implicate passive and active smoking as risk factors for premenopausal breast cancer.

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LEUKEMIA MORTALITY AFTER FRACTIONATED MODERATE-DOSE-RATE IONIZING RADIATION IN THE CANADIAN FLUOROSCOPY COHORT. *L B Zablotska, G R Howe (Columbia University, New York, NY 10032)

Risks of leukemia associated with exposures to high doses of ionizing radiation at high-dose-rates have been firmly established in the atomic bombings and radiation treatment studies. Canadian fluoroscopy cohort was used to analyze leukemia mortality due to exposure to fractionated moderate-dose-rate ionizing radiation between 1950 and 1987. A substantial fraction (32%) of subjects received multiple chest x-ray fluoroscopies during the course of pneumothorax for treatment of tuberculosis. Individual bone marrow doses were estimated from the combination of the number of fluoroscopies, interviews with physicians, and experimental human phantom study of organ doses produced by contemporary fluoroscopes. Cumulative person-year experience was cross-classified by sex, 5-year categories of age at risk and calendar time, province (Nova Scotia vs non-Nova Scotia), and lagged cumulative dose (2-year lag). Poisson regression analyses were used to estimate excess relative and absolute rates per gray and associated measures of uncertainty. There were 149 deaths from leukemia among 63,589 subjects (20 chronic lymphocytic leukemia (CLL)). Although single fluoroscopy carries exposure on the order of 0.01 gray (Gy), over the course of treatment exposed subjects accumulated substantial exposures (mean bone marrow dose=0.17 Gy and 0.25 Gy among those with non-zero doses). Categorical analyses showed a pattern of increasing risks with increasing dose. Linear dose-response analyses showed increased risks for all leukemia (ERR=0.56, 95%CI: <0.25, 2.36) and leukemia excluding CLL (ERR=0.85, 95% CI: <0.25, 2.98). In summary, the Canadian fluoroscopy cohort provides an opportunity for directly studying the long-term effects of low to moderate doses of highly fractionated ionizing radiation on the subsequent risk of leukemia mortality in a large cohort of North American population. Risks estimated in this cohort do not differ from those estimated in other ionizing radiation studies and their confidence intervals overlap. Since by the end of 1987, only 39.1 percent of the cohort has died, continued follow-up of this cohort will increase the number of cases of leukemia available for analysis as well as increase the power of statistical analyses.

* = Presenter; S = The work was completed while the presenter was a student

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THE BURDEN OF CANCER IN ONTARIO FIRST NATIONS PEOPLE, 1968–2001. *L Marrett, D Nishri, B Green, C Jones (Cancer Care Ontario, Toronto, ONT Canada)

Traditionally, cancer incidence has been lower in First Nations/Native American peoples. However, previous work (covering the period 1968–1991) by the investigators had indicated increasing incidence rates for common cancers, especially those of the lung and colorectum, in the Ontario First Nations population (FN). The primary purpose of the current work was to extend this earlier work to 2001. Computerized probabilistic linkage between FN registered with Indian and Northern Affairs Canada between 1968 and 1991; new diagnoses in the Ontario Cancer Registry and among Ontario residents in the Manitoba Cancer Registry in 1968–2001; and the Ontario mortality database for 1968–2001. Incidence and mortality rates were calculated using the person-years-at-risk, and age-standardized to the 1991 Canadian population for both the FN and the Ontario population (ON). Trends were examined across 4 time periods. Survival experiences for cancer patients in the two populations were compared for major types of cancer using Cox proportional hazard models. Incidence rates for all cancers combined continue to be much lower in FN compared to ON, although the gap is closing. While breast and prostate cancer incidence rates continue to be lower in FN, the incidence of lung and colorectal cancers in FN have risen over time and are currently very similar in FN and ON. Increases were also noted among FN for a number of other cancers strongly related to use of tobacco or unhealthy diet/obesity/physical inactivity. Almost all of the increase in cancer incidence in FN results from these cancers. While cancer mortality has declined for many cancers in ON, it continues to rise in FN. Survival is significantly worse in FN for those major cancers where prognosis is moderate to good (colorectum, breast, prostate) but no different when prognosis is very poor (lung). Results suggest that priority areas for primary prevention include reduction of cigarette use and improved diet, weight control and physical activity. Improvements will require implementation of both policies and health promotion strategies in concert.

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HISTORY OF DIABETES AND SUBSEQUENT PROSTATE CANCER RISK IN THE NIH-AARP DIET AND HEALTH STUDY. *M F Leitzmann, B Calton, S-C Chang, ME Wright, A Subar, F Thompson, V Kipnis, D Midthune, T Mouw, P Hurwitz, D Campbell, A Hollenbeck, A Schatzkin (National Cancer Institute, Bethesda, MD 20892)

Inverse associations between history of diabetes and prostate cancer risk have been reported in several studies, but available evidence is inconsistent. Some previous investigations have been limited by small sample sizes and diagnostic bias. We prospectively examined the association between diabetes diagnosis (mostly adult-onset) and subsequent prostate cancer risk in a cohort of 313,233 U.S. men aged 50 to 69 without a cancer diagnosis in 1995 who were followed for 5 years. A prior history of diabetes was assessed using a self-administered questionnaire at baseline. Incident cases of prostate cancer were ascertained by matching cohort participants to state cancer registries and the National Death Index. Multivariate relative risks (RR) and 95% confidence intervals (CI) of prostate cancer associated with diabetes were estimated using Cox regression. During 1,420,897 person-years of follow-up, 10,890 prostate cancer cases were documented. A prior history of diabetes was associated with decreased prostate cancer risk. The age-adjusted RR of prostate cancer among men with diabetes was 0.70 (95% CI=0.65–0.75) compared to men without diabetes. After additional adjustment for 15 other purported risk factors for prostate cancer the RR was 0.73 (95% CI=0.68–0.78). Restricting the analysis to men who had PSA tests (RR=0.71; 95% CI=0.65–0.78) and excluding the first year of follow-up (RR=0.72; 95% CI=0.67–0.79) did not materially change the results. Analyses of advanced prostate cancer (RR=0.77; 95% CI=0.64–0.92) and fatal prostate cancer (RR=0.88; 95% CI=0.57–1.36) showed weaker inverse relations. The results from this large prospective study suggest that diabetes is associated with a reduced risk of prostate cancer.

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