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CHANGING PATTERNS IN LUNG CANCER INCIDENCE BY HISTOLOGIC TYPE IN CANADA, 1970-1996.

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This study was to examine the incidence patterns of lung cancer by histologic type in Canada since 1970. Incidence data of lung cancer were obtained from the Canadian Cancer Registry. Secular trends in the overall age-adjusted rates and the age-specific rates were examined by histologic type. The changes between 1970-72 and 1994-96 were compared. The age-adjusted incidence rates of lung cancer increased slightly in males, from 48.7 per 100,000 in 1970-72 to 51.3 per 100,000 in 1994-96, while they reached a peak of 61.6 per 100,000 in 1982-84. For females, the age-adjusted incidence rates increased substantially, by 3-fold from 9.5 per 100,000 in 1970-72 to 29.6 per 100,000 in 1994-96. The incidence patterns varied appreciably by histologic type and age group. While squamous cell carcinoma decreased in recent years, adenocarcinoma has increased and become the most common type among men. Recent increases among women mainly came from the adenocarcinomas and the elderly, while squamous and small cell carcinomas stabilized or appeared to decline. Other unspecified carcinomas of the lung decreased among men, but still constitute one-third of lung cancer cases. The observed patterns for the three major histologic types are consistent with the changes in smoking prevalence although advances in histopathologic diagnosis of lung cancer may have an impact on the trends. It is anticipated that the rates of squamous and small cell carcinomas will continue to decline among males but stabilize among females, while the increasing adenocarcinoma among older women will slow down in the near future.

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COMPARATIVE ANALYSIS OF BREAST CANCER RISK FACTORS AMONG AFRICAN-AMERICAN WOMEN AND WHITE WOMEN.

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To compare the similarity of breast cancer risk factors for black women and white women by age or menopausal status, we investigated several biologic and lifestyle factors in a 1993-1996 case-control study of black women (350 cases and 353 controls) and white women (522 cases and 417 controls). Multivariate logistic regression analyses indicated that associations with breast cancer for menopausal status, lactation history, family history of breast cancer, BMI, waist-to-hip ratio, education, history of benign breast disease, and history of biopsy were similar overall for blacks and whites in magnitude and pattern. In contrast, the increased risk noted only for early age at menarche (<12 years) among white women was evident for average age at menarche (12-13 years) as well among black women. Similarly, the increased risks seen with nulliparity and with age at first full-term pregnancy older than 29 years among white women were not evident among black women. Additionally, stratification by age (< 50 yrs vs. ≥ 50 yrs) revealed that a history of breastfeeding was not protective among younger white women and a family history of breast cancer was a much stronger risk factor for younger black women. Stratification by menopausal status resulted in notable changes in magnitude of the odds ratios associated with parity, oral contraceptive use, and education among black women.

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CYP1A1, GSTM1, AND GSTT1 POLYMORPHISMS AND BREAST CANCER: A CASE-CONTROL STUDY IN RIO DE JANEIRO, BRAZIL.

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CYP1A1 polymorphism has been shown to be associated with breast cancer risk in African-Americans, but not in Caucasians. GSTM1 and GSTT1 have also been reported to be polymorphic and recently are being related to an earlier onset of the disease. The relation between CYP1A1, GSTM1 and GSTT1 polymorphisms and breast cancer according to skin color was examined in a case-control study carried out in Rio de Janeiro City. 128 cases of invasive breast cancer admitted to the main hospital of Instituto Nacional de Câncer and 256 controls recruited among out-patients from Hospital Universitário Pedro Ernesto were included. Information obtained by a standardized questionnaire was collected from all participants. Polymorphism characterization was performed by PCR procedures. Odds ratios (OR) and 95% confidence were adjusted for age, age at menarche, menopause status, parity, family history of breast cancer, and tobacco smoking. No risk of breast cancer associated with any of the genotypes, alone or in combination, was observed considering all women studied. Among Non-Whites, a reduction in risk associated with m1 homozygous and heterozygous genotypes appeared after adjusting for potential risk factors (OR=0.33, 95% CI 0.13-0.84) while the presence of combined null GSTM1 and GSTT1 genotypes tended to increase risk . Additional analysis and further studies are necessary to clarify the role of these enzymes in the etiology of breast cancer among Brazilian population.

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EFFECT OF METABOLIC POLYMORPHISMS ON BENZIDINE-INDUCED BLADDER CANCER IN CHINESE WORKERS: A NESTED CASE-CONTROL STUDY.

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Slow acetylation activity and its associated *NAT2* genotype are generally associated with increased risk for bladder cancer in general populations and in workers exposed to arylamine mixtures. For workers exposed to benzidine, a borderline protective association has been reported. The purpose of this study was to evaluate the impact that metabolic polymorphisms have on bladder cancer in Chinese workers exposed only to benzidine. This nested case-control study included 30 cases with transitional cell carcinoma of the urinary bladder, diagnosed after 1991. Sixty seven controls, frequency-matched by decade of birth, were selected. NAT2 and CYP1A2 enzymatic activities were characterized by measuring urinary caffeine metabolite ratios as phenotypic function markers. Polymerase chain reaction-based methods were used to identify genotypes for the following genes: *NAT2*, *NAT1*, *GSTM1*, *GSTT1*, *GSTP1*, and *UGT1A1*. Crude analysis of bladder cancer risk shows an odds ratio of 0.1 (95% confidence interval: 0.01 – 0.92) for workers with the *NAT2* slow genotype. Adjustment for lifetime smoking, age, and *NAT1* genotype did not alter the findings. A protective but not significant association was found for slow acetylation phenotype. *NAT1*, *GSTM1*, and *GSTT1* polymorphisms showed no association with bladder cancer risk; frequencies of the rare alleles for *GSTP1* and *UGT1A1* were too low for analysis. These findings suggest that the risk of bladder cancer from benzidine exposure is due to activation by *N*-acetylation.

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