BRIEF REPORT

Colorectal cancer and solar radiation

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It has been suggested that sunlight might have a role in the prevention of colorectal cancer via a mechanism involving vitamin D. We used data from nine population-based cancer registries in the United States to analyze incidence rates for colon and rectal cancer during 1973-84 as a function of regional variation in the levels of available solar radiation. Data were restricted to include only those persons born and diagnosed in the same state. Incidence rates of colon and rectal cancer among men tended to increase with decreasing levels of solar radiation. Compared to rates in New Mexico and Utah, for example, rates in the Detroit area (MI), Connecticut, and western Washington were 50 percent to 80 percent higher. Among women, colon cancer rates showed a similar trend, though of smaller magnitude; rates of rectal cancer among women did not vary in relation to levels of available solar radiation.

Key words: Colorectal neoplasms, incidence, sunlight, United States.

Introduction

Geographic variation in the occurrence of colorectal cancer has been well-documented, and possible reasons for this variation have been suggested. In one such study, Garland and Garland, observed that for states in the United States there was an inverse association between age-adjusted colon-cancer mortality rates and average levels of solar radiation. Based on these findings and on the known relationship between sunlight and vitamin D production in the body, they proposed that vitamin D might have a protective role in colon carcinogenesis. Further support for a sunlight-vitamin D hypothesis was provided by a Canadian study which examined colon cancer mortality in relation to acid-haze air pollution. Colon cancer mortality rates from 18 Canadian cities were found to be associated positively with measurements of sulfur dioxide and haze, both of which are known to block transmission of the ultraviolet light necessary for vitamin D production.

Prospective studies have strengthened the epidemiologic evidence that vitamin D has a role in reducing the risk of colon cancer. Higher serum 25-hydroxyvitamin D levels were found to be associated with a reduced risk of developing colon cancer in a nested

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case-control study, and dietary vitamin D was found to be inversely correlated with colorectal cancer risk in a 19-year prospective study.

The present analysis further examines the sunlight-vitamin D hypothesis by extending the analysis to incidence rates for colon and rectal cancer as a function of regional variation in solar radiation.

Methods

Data on cases from nine population-based cancer registries in the US were obtained from the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute. All malignant, histologically confirmed cancers of the colon or rectum diagnosed between 1 January 1973 and 31 December 1984 among residents of the states of Iowa, New Mexico, and Utah, and the metropolitan areas of Atlanta (GA, from 1 January 1975), Detroit (MI), and San Francisco/Oakland (CA), and 13 counties of western Washington (from 1 January 1974) were identified. Those cases with an adenocarcinoma of the colon or rectum as determined using the first four digits of the morphology codes of the International Classification of Diseases for Oncology (ICD-O) were selected for study. Analyses were restricted to Whites only and to persons between the ages of 25 and 84 years at the time of diagnosis.

Birthplace data for the cancer cases were obtained by the SEER registries, which routinely abstract this information from the medical records of all persons for whom a diagnosis of cancer is reported. Codes are assigned indicating state of birth (for those persons born in the US) or country of birth (for those persons not born in the US). For the nine SEER registries combined, the percentage of colon cancer cases with missing birthplace data was 24 percent; the corresponding figure for rectal cancer cases was 23 percent.

As a means of limiting the potential for bias due to migration, cases were restricted to include only those persons born in the same state as the one in which they were diagnosed. The populations at risk were similarly restricted. It seemed likely that, on average, such persons would have had a longer duration of residence in the given state than someone not born there, and thus would have been exposed to the corresponding local level of solar radiation for a greater portion of their lives. Cases in a given population for whom birthplace was unknown were assigned the same age and sex-specific birthplace distribution as the cases in that same population for whom birthplace was known. This assumed that, for each age-sex stratum, there was an underlying probability of a missing birthplace that was independent of the actual birthplace (i.e., that information on birthplace was missing at random).

The US Bureau of the Census provided denominator data which classified the 1980 populations of Whites for each of the SEER areas by sex, age, and state of birth (unpublished). These data were estimated by the Census Bureau from a 19 percent sample of each SEER area. In the calculation of incidence rates for the period of study, the total population at risk was approximated by multiplying the 1980 sex, age, and birthplace-specific population of each SEER area by the number of years that the particular SEER registry had been in operation between 1973 and 1984.

Solar radiation data consisting of long-term annual averages for daily global radiation (megajoules per square meter per day, MJ/m²/day) were obtained from the National Technical Information Service and represented data collected by National Weather Service Stations over the period 1952-75. For the SEER sites Atlanta, Detroit, San Francisco/Oakland, and western Washington, the actual value reported for the particular city or area was used. For the SEER sites Connecticut, Hawaii, Iowa, New Mexico, and Utah, a simple average of values from all the stations in the state was used.

Age-adjusted incidence rates for each sex were calculated by direct standardization to the age-distribution of the 1980 US population. Rate estimates and variance estimates were calculated using a likelihood-based method, which accounted for the presence of cases with missing birthplace data. Upper and lower confidence limits were estimated for the ratio of two incidence rates using the standard error of the log of the adjusted rate ratio. To test for a trend in adjusted rates over the range of solar radiation data, weighted least squares regression was used to account for the differing variances of the adjusted rates.

Results

Age-adjusted incidence rates for colon cancer and rectal cancer in Whites for the period 1973 to 1984 are presented in Table 1. As noted above, these results apply to persons who were born in the same state as that in which they were diagnosed. The SEER sites are ordered according to their average annual solar radiation, which ranged from 11.9 MJ/m²/day in western Washington to 20.1 MJ/m²/day in New Mexico. Using the incidence rates for New Mexico as a baseline, rate ratios and 95 percent confidence limits (CI) were computed comparing the incidence rate of each of the SEER areas to this reference rate; these estimates are included in the table. Examination of the directly