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SCIENCE DIRECT®
Preventive Medicine 42 (2006) 200–205
Preventive Medicine
www.elsevier.com/locate/ypmed

Active cigarette smoking, household passive smoke exposure, and the risk of developing pancreatic cancer

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Available online 3 February 2006

Abstract

Objective. The objective of this study was to examine the association between active cigarette smoking, household passive smoke exposure, and pancreatic cancer risk using a prospective cohort design.

Methods. Two cohorts were established in Washington County, Maryland in 1963 (n = 45,749) and 1975 (n = 48,172). The Washington County Cancer Registry was used to ascertain the occurrence of pancreatic cancer in the 1963 cohort from 1963–1978 and in the 1975 cohort from 1975–1994. Poisson regression was used to analyze the associations between active smoking and household passive smoke exposure and pancreatic cancer risk.

Results. Current active smoking was associated with a two-fold increased risk of pancreatic cancer in both cohorts. Among never-smokers in each cohort, exposure to household passive smoke was not associated with an increased risk of pancreatic cancer, although the confidence limits were wide due to a small number of cases.

Conclusions. This study further documents the approximate doubling of pancreatic cancer risk in current active smokers. Our results also indicate that household passive smoke exposure is not associated with pancreatic cancer risk, although our risk estimates lacked precision.
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Keywords: Pancreatic cancer; Smoking; Tobacco smoke pollution; Cohort studies

Introduction

Pancreatic cancer causes 30,000 deaths per year in the United States, making it the fifth most common cause of cancer death (American Cancer Society, 2005). Although few risk factors for pancreatic cancer have been identified, epidemiological studies have consistently shown that cigarette smoking increases an individual's risk for pancreatic cancer (Ghadirian et al., 2003; U.S. Department of Health and Human Services (USDHHS), 2004; International Agency for Research on Cancer (IARC), 2004). In all, we identified nine prospective cohort studies examining the relationship between cigarette smoking and pancreatic cancer incidence using the Surgeon General's 2004 report (U.S. Department of Health and Human Services (USDHHS), 2004) and a MEDLINE search of the terms 'smoking' and 'cancer' (Engeland et al., 1996; Fuchs et al., 1996; Harnack et al., 1997; Nilsen and Vatten, 2000; Stolzenberg-Solomon et al., 2001; Isaksson et al., 2002; Lin et al., 2002; Jee et al., 2004; Larsson et al., 2005); the results of most of these studies indicate that cigarette smokers have a two- to three-fold increase in the risk of developing pancreatic cancer compared to non-smokers and that the risk of pancreatic cancer increases incrementally with the number of cigarettes smoked and the number of years smoked. Further, it has been estimated that smoking accounts for approximately one-fourth of pancreatic cancer incidence (Fryzek et al., 1997).

Cigarette smoking has consistently been reported to be a strong risk factor for pancreatic cancer, but additional data from

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0091-7435/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.ypmed.2005.12.014

large cohort studies are of value in enabling the association to be more thoroughly characterized and better understood. Furthermore, a paucity of literature exists examining the relationship between passive or secondhand smoke exposure and the risk of pancreatic cancer. Secondhand smoke contains many of the same toxic constituents as mainstream tobacco smoke, including 43 known carcinogens (Villeneuve et al., 2004; Brownson et al., 2002). Thus, it is biologically plausible that passive smoke exposure may be associated with the risk of developing pancreatic cancer. In a recently published case–control study, secondhand smoke exposure was not significantly associated with the risk of pancreatic cancer among 105 non-smoking pancreatic cancer cases and 1145 non-smoking population-based controls identified within 8 Canadian provinces (Villeneuve et al., 2004). No cohort studies addressing the association between passive smoke exposure and pancreatic cancer have been published. With this background in mind, we therefore conducted a community-based, prospective cohort study to investigate active and passive smoking in relation to the risk of developing pancreatic cancer.

Methods

Study population

This study was carried out with approval from the Institutional Review Board of the Johns Hopkins University Bloomberg School of Public Health. The study is based on two cohorts established when records were collected during two private censuses of the residents of Washington County, Maryland. The first census was conducted in 1963 and the second census was conducted in 1975. Details of the data collection methods, which were carried out at the Johns Hopkins Comstock Center for Public Health Research and Prevention (formerly the Training Center for Public Health Research), are described elsewhere (Trimble et al., 2005). Approximately 98% of households in Washington County participated in the 1963 census, and 90% of the households participated in the 1975 census.

From among the total number of participants ascertained in the 1963 census (n = 91,908), the 1963 analytic cohort of the present study was limited to the 45,749 participants who were 25 years of age or older, had no prior cancer diagnosis, were not pipe or cigar smokers, and were not missing information on age, gender, or smoking status. Individuals were included if they had smoking status information but not data on the number of cigarettes smoked per day. After applying these eligibility criteria to the 1975 cohort (n = 94,881), 48,172 participants from the 1975 cohort were included in the present study. These two cohorts were then followed-up over time for first-time occurrences of pancreatic cancer by linking personal identifying information collected at baseline (participant first and last names and date of birth) with the Washington County cancer registry.

Exposure measurements

Measurement of cigarette smoking in 1963 consisted of assessing whether household members had ever or currently smoked cigarettes, the age of initiation of smoking, and the amount smoked per day (≤10, 11–20, and >20). The cigarette smoking history collected during the 1975 census was similar to that collected in 1963, except that the actual number of cigarettes per day was recorded and the age of initiation of cigarette smoking was not. Additionally, questions concerning pipe and cigar smoking measured ever use in 1963 and current use in 1975.

For this study, individuals who smoked only pipes or cigars were excluded. There were less than 2000 pipe and cigar smokers in each cohort (all male) and the majority of them were also cigarette smokers, making it difficult to examine cigar and pipe smoking alone in relation to the risk of pancreatic cancer. Active cigarette smoking was categorized as current, former, or never. Current number of cigarettes smoked per day was categorized as ≤10, 11–20, and >20. An individual was considered exposed to passive smoke if any household member other than that individual reported being an active cigarette smoker.

Outcome measurements

The occurrence of pancreatic cancer (ICD-9 157) among the participants in the two cohorts after the baseline data collection was ascertained by linkage to the Washington County Cancer Registry. This registry, established in 1948, ascertains cancer cases primarily via discharge records from the Washington County Hospital, the only general hospital in the county, and death certificates. Our cancer registrar collects these data and accepts the diagnosis and code from the sources (Washington County Hospital or death certificates). Washington County Hospital has an excellent cancer unit and tends to draw patients from the surrounding area. Even patients who go elsewhere are likely to be diagnosed in Washington County hospital and registered in the Washington County cancer registry. When compared with the Maryland Cancer Registry, the county registry has been at least as complete as the state registry (unpublished data). Within the two cohorts examined in these analyses, 56 cases of pancreatic cancer occurred between 1963 and 1978 in the 1963 cohort and 92 cases of pancreatic cancer occurred between 1975 and 1994 in the 1975 cohort.

Person-time of follow-up

Person-time of follow-up was estimated based on systematic samples of 5% of the households near the mid-point of the follow-up interval to identify characteristics associated with likelihood of remaining in the county. Specifically, a 5% random sample of households in each cohort was surveyed 8 (1963 cohort) or 10 (1975 cohort) years after the baseline data were collected to determine what factors (age, gender, marital status, education, and smoking status) were associated with the probability of remaining alive and in Washington County. The results of these analyses were used to assign the probability of remaining a resident in the county (Comstock and Tonascia, 1977). The probability factor calculated for each individual from this regression model was then multiplied by the maximum possible follow-up time for each cohort (15 years for the 1963 cohort and 19 years for the 1975 cohort) to estimate person-time correcting for the potential for emigration or death as determined by individual characteristics (age, marital status, education, smoking status). The maximum limit for follow-up was July 15, 1978 for the 1963 cohort and July 15, 1994 for the 1975 cohort. For a diagnosis of pancreatic cancer to contribute to the numerator of the incidence rate, the diagnosis had to occur within the participant's estimated follow-up time. This method was applied uniformly to both cohorts, regardless of whether an individual's specific survival information was actually known.

Statistical analyses

Because of the temporal changes in cigarette smoking habits that occurred during the time period in which the two cohorts were established (Burns et al., 1997), the 1963 and 1975 cohorts were analyzed separately. To examine the association between active cigarette smoking and the risk of developing pancreatic cancer, all participants in each cohort were included in the regression models. To elucidate the specific effect of passive smoke exposure on the risk of developing pancreatic cancer, only participants who were classified as never active-smokers were included in the passive smoking analyses. Because relative risks were similar for men and women for the associations between active and passive smoking and pancreatic cancer, the genders were combined for all analyses.

Poisson regression models (Breslow and Day, 1987) were employed to estimate the relative risks of developing pancreatic cancer among active smokers and participants exposed to passive smoke compared to never-active, never-passive smokers. Relative risks were calculated adjusting first for age and then for age, education, and marital status. All statistical analyses were performed using SAS (SAS Institute Inc, Cary, NC).

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