Environmental Tobacco Smoke and the Risk of Pancreatic Cancer

Findings from a Canadian Population-based Case-control Study

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ABSTRACT

Background: Despite the fact that tobacco is a well-recognized risk factor for pancreatic cancer, no study has yet reported on the association between environmental tobacco smoke (ETS) and this malignancy. We investigated the relationship between pancreatic cancer and childhood and adult exposure to ETS using a case-control study design.

Methods: Our study population consisted of 583 pancreatic cancer cases and 4,813 population-based controls that were identified within 8 Canadian provinces between 1994 and 1997. Mail-out questionnaires were used to collect risk factor information and a lifetime residential and occupational history of exposure to ETS.

Results: Among never smokers, those who were exposed to ETS both as a child and as an adult had an odds ratio of 1.21 (95% CI=0.60-2.44) relative to those with no exposure. For active smoking, when the referent group consisted of never smokers who had not been regularly exposed to ETS, the risk increases were more pronounced with an increased number of years of smoking, cigarette pack-years, years since quit smoking, and average number of cigarettes smoked daily.

Conclusions: Overall, our results are suggestive of a weak association between pancreatic cancer and ETS. Perhaps more importantly, they suggest that ETS smoking exposures may confound the risk of pancreatic cancer associated with active smoking measures commonly used in epidemiologic studies.

La traduction du résumé se trouve à la fin de l'article.

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Pancreatic cancer is currently the fourth leading cause of cancer mortality in Canada.[1] The prognosis of patients diagnosed with this malignancy is quite grim as less than 5% of such individuals survive five years.[2] For this reason, primary prevention through the identification of modifiable risk factors represents an important means to reduce the incidence of this disease. Although physical activity, obesity and diet have recently been identified as possibly playing a role in the etiology of this disease,[3,4] apart from smoking, there are no other established modifiable risk factors. It has been estimated that between 27-33% of all pancreatic cancer cases are attributable to the effects of cigarette smoking.[5,6] Both active and passive components of tobacco smoke contain numerous carcinogenic constituents that may increase the risk of cancer. In particular, environmental tobacco smoke contains 43 known carcinogens.[7] Although environmental tobacco smoke contains many of the same toxic constituents as mainstream tobacco smoke, more of these constituents of ETS appear in the vapour phase (rather than particulate phase), and therefore, ETS particle sizes are smaller. Furthermore, the uptake and deposition of these particles may differ between active and passive smokers.[8-10] For these reasons, it remains unclear which cancer sites may be most affected by exposure to ETS.

The objective of this study was to explore the relationship between ETS active smoking and the incidence of pancreatic cancer. To the best of our knowledge, the association between ETS and the incidence of pancreatic cancer has not been examined. Therefore, this study provides an important contribution to the existing literature of etiologic risk factors for pancreatic cancer. Furthermore, because past studies have not taken into account ETS when deriving risk estimates for cigarette smoking, such work may understate the overall impact that smoking has on the incidence of this disease, as the referent group would include individuals potentially exposed to ETS either at work or home. The research objectives were addressed by using data collected from a Canadian population-based case-control study that was conducted in 8 provinces between 1994 and 1997.

METHODS

Study population

The National Enhanced Cancer Surveillance System (NECSS) was a large-scale Canadian research initiative undertaken a decade ago to investigate environmental causes of cancer. The etiologic roles of several risk factors have been examined within the case-control component of the NECSS. This component consists of data that were collected from a population-based sample of 20,730 Canadians diagnosed with one of 18 different types of cancer and from 5,039 controls. On April 1, 1994, the NECSS began to collect data from participants in 7 of the 10 Canadian provinces (Alberta, British Columbia, Manitoba, Prince Edward Island, Nova Scotia, Newfoundland, and Saskatchewan) and in May 1995, data collection commenced in an eighth province (Ontario). By July 31, 1997, data collection was complete in all these provinces.

We examined the same study subjects who formed the basis of a previous risk assessment to evaluate the associations between pancreatic cancer and alcohol, coffee consumption and measures of tobacco use among smokers.[11] The participating provincial cancer registries tried to identify pancreatic cancer cases as early as possible in the registration process to minimize the loss of subjects due to severe illness or death. Pancreatic cancer cases were defined according to International Classification of Diseases, rubric 157.[12] Physicians were contacted to obtain consent to recruit the patients and this was given for approximately 85% of the cases. With the exception of the Ontario cancer registry, questionnaires were not mailed out to next of kin of pancreatic cases known to be deceased.

In Ontario, 132 of 271 returned questionnaires were completed by proxy respondents while data were received from only 8 proxies for cases in the other provinces. Our analyses are based on cases and controls, 30 years of age and older, who completed questionnaires between 1994 and 1997. Overall response rates for female and male cases were 56% and 55%, respectively. Response rates of 71% and 65% were obtained from the respective female and male control populations. Our findings are based on analyses that were conducted using information collected from a total of 583 cases and 4,813 controls.

TABLE I

Selected Characteristics of the Pancreatic Cases and Controls, NECSS Study, Canada, 1994-1997

Characteristic	Cases	(%)	Control	(%)
Sex				
Males	322	55.2	2452	50.9
Females	261	44.8	2361	49.1
Age Group				
30-39	18	3.1	464	9.6
40-49	52	8.9	791	16.4
50-59	140	24.0	933	19.4
60-69	245	42.0	1643	34.1
≥70	128	22.0	982	20.4
Income Adequacy				
Low	86	14.8	784	16.3
Low middle	91	15.6	831	17.3
Upper middle	158	27.1	1225	25.5
High	103	17.7	785	16.3
Unknown	145	24.9	1188	24.7
Smoking Status				
Never	180	30.9	1819	37.8
Ever	403	69.1	2994	62.2
Proxy Response				
Yes	140	24.0	0	0.0
No	443	76.0	4813	100.0
Province				
British Columbia	94	16.1	814	16.9
Alberta	94	16.1	593	19.3
Saskatchewan	30	5.1	255	5.3
Manitoba	41	7.0	293	6.1
Ontario	271	46.5	1894	39.4
New Brunswick	41	7.0	293	6.1
Prince Edward Island	3	0.5	255	5.3
Newfoundland	9	1.5	593	12.3
Total	583	100.0	4813	100.0

The NECSS used frequency matching to select population-based controls in order to obtain a similar age and sex distribution to that of all cancer cases. Strategies for control selection varied by province depending on data accessibility. In Prince Edward Island, Nova Scotia, Manitoba, Saskatchewan, and British Columbia, provincial health insurance plans were used to obtain a random sample of the provincial population stratified by age-group and sex. In each of these provinces, more than 95% of residents are covered by these public plans. Current military personnel and their families and indigenous peoples were excluded from study because they are covered by other plans. In Ontario, Ministry of Finance data were used to obtain a stratified random sample while Newfoundland and Alberta used random digit telephone dialling to obtain a population sample.

Data collection

Mailed questionnaires, with telephone follow-up when necessary for clarification, were used to obtain information on subjects' residential and occupational histories and on other risk factors for cancer. Included were questions on ethnicity, education, income, smoking, height, weight, exposure to specific occupational carcinogens, physical activity, diet two years before interview (60-item food frequency instrument), and general changes in the diet compared with twenty years ago. The dietary questionnaire was modelled after two instruments that have been extensively validated: the reduced Block questionnaire[13] and the instrument used in the Nurses Health Study cohort.[14] Minor modifications to the questionnaire items were made to take into account differences between U.S. and Canadian diets.

Exposure to environmental tobacco smoke (ETS)

We applied previous developed methods that were used to evaluate the relationship between ETS and other type cancers within the NECSS.[15,16] The questionnaire used in the NECSS asked subjects to describe their lifetime history of potential exposure to passive smoking, both at home and at work. For each Canadian residence that was occupied for at least one year, subjects were asked to provide the address, first and last year of residence, and to indicate the number of regular smokers who usually lived in the home. Similarly, for each job

TABLE II

Childhood and Adult Exposure to Residential and Occupational Environmental Tobacco Smoke and the Risk of Pancreatic Cancer Among Never Smokers, NECSS Study, Canada, 1994-1997

Period of Passive Exposure	Cases	Controls	Age and Sex Adjusted Odds Ratio (95% CI)		Multivariate Odds Ratio (95% CI)†	
All Subjects						
Never exposed	16	191	1.0	–	1.0†	–
Child only§	7	80	1.52	(0.59-3.92)	1.37	(0.46-4.07)
Adult only‖	17	169	1.16	(0.56-2.38)	1.01	(0.41-2.50)
Child and adult	65	705	1.22	(0.69-2.18)	1.21	(0.60-2.44)
Male Subjects						
Never exposed	6	56	1.0	–	1.0‡	–
Child only§	3	14	2.79	(0.59-13.24)	4.54	(0.67-30.70)
Adult only‖	8	57	1.25	(0.41-3.87)	1.38	(0.28-6.89)
Child and adult	20	221	0.92	(0.35-2.43)	1.06	(0.27-4.21)
Female Subjects						
Never exposed	10	135	1.0	–	1.0‡	–
Child only§	4	66	1.21	(0.36-4.11)	0.85	(0.20-3.60)
Adult only‖	9	112	1.07	(0.42-2.76)	0.85	(0.27-2.68)
Child and adult	45	484	1.42	(0.69-2.92)	1.18	(0.51-2.73)
Total Never Smoking Patients	105	1145				

* Restricted to participants for whom exposure to environmental tobacco smoke could be ascertained for at least 75% of the etiologic period.
§ Exposed to either residential or occupational environmental tobacco smoke up to age 19.
‖ Exposed to either residential or occupational environmental tobacco smoke from age 20 onwards.
† Odds ratios were adjusted for: age, sex, body mass index, income adequacy and province of residence.
‡ Odds ratios were adjusted for: age, body mass index, income adequacy and province of residence.

TABLE III

Lifelong Exposure to Residential and Occupational Environmental Tobacco Smoke* and the Risk of Pancreatic Cancer Among Never Smokers, NECSS Study, Canada, 1994-1997

Lifetime Exposure to Environmental Tobacco Smoke	Cases	Controls	Age and Sex Adjusted Odds Ratio (95% CI)		Multivariate Odds Ratio (95% CI)†	
Residential Years						
No residential/occupational exposure	16	191	1.0		1.0	
Occupational exposure only	17	166	1.25	(0.60-2.59)	1.43	(0.61-3.36)
1-20 years	18	261	1.13	(0.56-2.29)	0.97	(0.42-2.26)
21-34 years	19	251	1.06	(0.53-2.15)	0.90	(0.39-2.11)
≥35 years	35	276	1.39	(0.74-2.61)	1.43	(0.66-3.11)
Occupational Years						
Never exposed	16	191	1.0		1.0‡	
Residential exposure only	26	327	1.01	(0.52-1.94)	0.97	(0.43-2.18)
1-9 years	19	209	1.51	(0.74-3.08)	1.55	(0.68-3.56)
10-21 years	19	199	1.45	(0.71-2.95)	1.16	(0.49-2.74)
≥22 years	25	219	1.22	(0.62-2.38)	1.20	(0.54-2.67)
Residential and Occupational Combined						
Never exposed	16	191	1.0		1.0‡	
1-35 years	28	311	1.38	(0.72-2.66)	1.33	(0.60-2.93)
36-56 years	25	325	1.08	(0.56-2.10)	1.09	(0.49-2.41)
≥57 years	36	318	1.24	(0.67-2.31)	1.15	(0.54-2.47)
Total Never Smoking Patients	105	1145				

* Restricted to participants for whom exposure to environmental tobacco smoke could be ascertained for at least 75% of the etiologic period.
† Odds ratios were adjusted for: age, sex, body mass index, income adequacy and province of residence.
‡ Odds ratios were adjusted for: age, body mass index, income adequacy and province of residence.

held for at least one year, occupational data were collected which included the specific years employed, and the number of regular smokers in the subject's immediate work environment. To reduce the potential for ETS exposure misclassification, we restricted our analysis to those individuals for whom residential ETS exposure could be ascertained for at least 75% of the individual's lifetime.

Statistical analysis

Two series of analyses were undertaken to determine whether ETS was associated with pancreatic cancer. First, we modelled the relationship between pancreatic cancer and ETS exposure among never smokers. ETS exposures were characterized according to the period of exposure (child or adult) as well as the nature of the exposure (work or home). Risk assessment was performed for males, females and both sexes combined. The second series of analyses examined whether the risks associated with selected active smoking variables were more pronounced when the referent category of never active cigarette smokers excluded those who reported exposure to ETS. The smoking variables that were used in this analysis were: the average number of cigarettes smoked daily, the number of smoking years, the age when first started smoking, the number of cigarette pack-years, and the number of years since cessation.

Odds ratios and their 95% confidence intervals were calculated using unconditional logistic regression. In order to allocate an optimal number of controls across categories of continuous variables (e.g., number of smoking years, average number of cigarettes smoked daily) in smokers, categories were created based on tertiles obtained from the frequency distribution observed in the control population. Multivariate models were adjusted for the effects of other known or suspected risk factors of pancreatic cancer. These factors included: age, sex, body mass index, physical activity, income adequacy, province of residence and dietary fat and caloric intake. Participants' levels of dietary fat and caloric intake were estimated by assigning published nutrient values to the dietary items in the NECSS questionnaire.

RESULTS

Descriptive data for the study population are presented in Table I.

Among never smokers, there were only 16 pancreatic cancer cases who reported no exposure to either occupational or residential ETS (Table II). Relative to never smokers with no reported ETS exposure, non-significant increased risks of pancreatic cancer were observed among never smokers who reported exposure to ETS in early childhood (OR=1.37; 95% CI=0.46-4.07). Individuals who reported ETS exposure both as a child and as an adult had an odds ratio of 1.21 (95% CI=0.60-2.44). When analyses were stratified by sex, there

were no statistically significant differences in the risk of pancreatic cancer according to exposure to ETS.

No statistically significant associations between occupational or residential passive smoking history and risk of pancreatic cancer were observed (Table III). The odds ratios among individuals in the highest tertile of years of occupational and residential (combined) passive smoking was 1.15 (95% CI=0.54-2.47).

Statistically significant associations were noted between case status and the total number of smoking years, average number of cigarettes smoked daily, total number of pack-years and years since cessation of smoking (Tables IV, V). The number of smoking years was a more important determinant of risk than the average number of cigarettes smoked daily. In particular, the addition of this variable attenuated the risk estimate for the number of cigarettes smoked daily. The risks for the active smoking variables presented in Table IV were more pronounced when the referent group was restricted to never smokers with no reported exposure to ETS (Table V). For example, the odds ratio for those with greater than 30 pack-years of smoking relative to a never smoker was 1.59 (95% CI=1.12-2.24), while it was 1.79 (95% CI=0.91-3.52) relative to a never smoker with no reported ETS exposure. Similarly, the relative risk among those who had smoked for at least 34 years was 1.48 (95% CI=0.93-2.35) relative to a never smoker, while it was 1.67 (95% CI=0.80-3.50) relative to a never smoker with no reported ETS exposure.

Our results did not change appreciably with further adjustment for dietary fat, energy intake, physical activity, and parity.

TABLE IV

The Risk of Pancreatic Cancer* According to Selected Smoking Variables Using a Referent Group that does not Take into Account Exposure to ETS, NECSS, Canada, 1994-1997

Smoking Characteristics	Cases	Controls	Age and Sex Adjusted Odds Ratio (95% CI)		Multivariate Odds Ratio (95% CI)	
Smoking status - never smoker§	105	1145	1.0		1.0	
Ever smoker	250	1961	1.31	1.02-1.68	1.23	0.92-1.69
Age when first started to smoke‖						
Never smoker	105	735	1.0		1.0	
≤15	84	737	1.25	0.91-1.72	0.65	0.39-1.10
16-18	101	489	1.37	1.02-1.85	0.64	0.30-1.04
>18	65	1145	1.28	0.92-1.79	0.68	0.42-1.11
Average cigarettes a day‖						
Never smoker	105	1145	1.0		1.0	
1-10	61	643	1.00	0.72-1.40	0.61	0.38-0.98
>10-20	118	846	1.47	1.10-1.96	0.73	0.44-1.21
>20	71	472	1.51	1.08-2.12	0.77	0.44-1.35
Total smoking years¶						
Never smoker	47	1145	1.0		1.0	
>0-19	72	628	0.89	0.62-1.28	0.71	0.45-1.12
>19-34	131	672	1.16	0.84-1.60	0.98	0.62-1.56
≥34	105	661	1.81	1.35-2.42	1.48	0.93-2.35
Smoking pack-years**						
Never smoker	105	1145	1.0		1.0	
>0-11.8	46	620	0.86	0.60-1.24	0.81	0.54-1.22
>11.8-30	88	690	1.38	1.01-1.87	1.31	0.92-1.86
>30	116	651	1.68	1.24-2.26	1.59	1.12-2.24
Years since quit smoking¶						
Never smoker	27	1145	1.0		1.0	
≥25	74	399	0.55	0.35-0.86	0.49	0.28-0.84
10-24	60	553	1.33	0.96-1.83	0.98	0.57-1.69
<10	89	327	1.98	1.39-2.81	1.70	0.86-3.35
Current smokers	105	682	1.49	1.10-2.03	1.22	0.60-2.48

* based on the same series of cases and controls presented in Table V, namely, includes those subjects whose occupational and residential passive smoking history was complete for at least 75% of their lifetimes.
§ Adjusted for age, sex, BMI, province of residence, socioeconomic status and number of smoking years.
‖ Adjusted for age, sex, BMI, province of residence, socioeconomic status and average number of cigarettes smoked daily.
¶ Adjusted for age, sex, BMI, province of residence, and socioeconomic status.

DISCUSSION

We found no significant associations between indices of environmental tobacco smoke and the occurrence of pancreatic cancer. However, our findings of increased risks for several measures of active smoking after controlling for ETS exposure support the hypothesis that passive smoking may play a role in the etiology of this malignancy. Our results should be interpreted with caution due to wide confidence intervals, and the small number (n=16) of cases of pancreatic cancer that were observed among never smokers who had no exposure to ETS. However, our study offers several notable strengths that include the collection of a large series of risk factors for pancreatic cancer, a population-based design, and data that were, for the most part (76%), collected directly from the patients.

The measures of lifetime passive exposure we obtained from the questionnaire were basic, focussing on the number of smokers an individual subject lived with at each residence and the average number of people who smoked in the subjects' immediate work area for each job they held. These proxies for actual exposure obviously were unable to capture individual variations in actual levels of ETS exposure of those who lived or worked with smokers, and the actual amount of time they were exposed to other's cigarette smoke. Such misclassification of exposure, if non-differential between the case and control series, would attenuate our presented risk estimates.

Case-control studies may also be affected by recall bias whereby the case population recollects their exposure profile differently from persons who have not been diagnosed with disease. Although it is not possible to directly evaluate this bias, the results of the multivariate analysis suggest that this effect is likely small. Specifically, the addition of measures of socio-economic status (SES) into logistic regression models did not change the risk estimates for active smoking variables appreciably. These measures of SES are recognized correlates of ETS that are less likely to be influenced by recall bias than lifetime reports of ETS exposure. Moreover, we restricted our analysis, and potential for exposure misclassification, by using models that included only those subjects who had ETS exposures for at least 75% of their lifetimes.

Previous analyses of NECSS data have revealed positive associations between ETS exposure and cancers of the lung and breast. Specifically, among pre-menopausal women who were never active smokers, regular exposure to passive smoke was associated with an odds ratio of 2.3 (95% CI=1.2-4.6) relative to those with no expo-

TABLE V

The Risk of Pancreatic Cancer According to Selected Smoking Variables Using a Referent Group with no Active or Passive Smoking Exposure*, NECSS, Canada, 1994-1997

Smoking Characteristic	Cases	Controls	Age and Sex Adjusted Odds Ratio (95% CI)		Multivariate Odds Ratio (95% CI)	
No passive or active smoking	16	191	1.0	1.0		
Smoking status - never smoker§	89	954	1.18	0.68-2.06	1.16	0.59-2.27
Ever smoker	250	1961	1.50	0.88-2.57	1.39	0.73-2.66
Age when first started to smoke‖						
Never smoker	89	954	1.18	0.68-2.06	1.14	0.58-2.23
≤15	84	735	1.44	0.81-2.55	0.73	0.34-1.60
16-18	101	737	1.58	0.90-2.76	0.72	0.33-1.53
>18	65	489	1.48	0.83-2.63	0.76	0.36-1.63
Average cigarettes a day‖						
Never smoker	89	954	1.18	0.68-2.06	1.14	0.59-2.24
1-10	61	643	1.15	0.65-2.06	0.68	0.32-1.45
>10-20	118	846	1.69	0.97-2.94	0.82	0.38-1.78
>20	71	472	1.74	0.98-3.11	0.87	0.39-1.94
Total smoking years¶						
Never smoker	89	954	1.16	0.67-2.03	1.16	0.59-2.26
>0-19	47	628	1.10	0.56-1.84	0.80	0.38-1.68
>19-34	72	672	1.32	0.74-2.33	1.11	0.53-2.33
≥34	131	661	2.05	1.18-3.56	1.67	0.80-3.50
Smoking pack-years**						
Never smoker	89	954	1.17	0.67-2.05	1.15	0.59-2.25
>0-11.8	46	620	0.98	0.54-1.79	0.92	0.45-1.87
>11.8-30	88	690	1.57	0.90-2.76	1.48	0.75-2.91
>30	116	651	1.92	1.10-3.35	1.79	0.91-3.52
Years since quit smoking¶						
Never smoker	89	954	1.20	0.69-2.09	1.15	0.59-2.26
≥25	27	399	0.64	0.87-2.73	0.55	0.25-1.22
10-24	74	553	1.54	1.28-4.15	1.11	0.50-2.47
<10	60	327	2.30	1.28-4.15	1.93	0.78-4.74
Current smokers	89	682	1.73	0.99-3.05	1.38	0.55-3.47

* Included only those subjects whose occupational and residential passive smoking history was complete for at least 75% of their lifetimes.
§ Adjusted for age, sex, BMI, province of residence, and socioeconomic status.
‖ Adjusted for age, sex, BMI, province of residence, socioeconomic status and number of smoking years.
¶ Adjusted for age, sex, BMI, province of residence, socioeconomic status and average number of cigarettes smoked daily.
** Adjusted for age, sex, BMI, province of residence, and socioeconomic status.

sure.[16] Among NECSS women diagnosed with lung cancer, the odds ratio for those with passive exposure as a child and as an adult was 1.63 (95% CI 0.8-3.5) and for those only exposed as an adult 1.20 (95% CI=0.5-3.0).[15] Given that ETS may be associated with an increased risk of several cancers, the continued implementation of policies that restrict such exposures in public and in the workplace are important public health initiatives. The value of restricting such exposures is further underscored given that a large portion of never smokers continue to be exposed to ETS. Within our study population, 81% (1,316/1,615) of never smokers reported some exposure to ETS.

In summary, our analyses suggest that ETS may be an important risk factor for pancreatic cancer and that the overall effects of active cigarette smoke in previous studies may have been confounded by the inability to exclude subjects with ETS exposures from the referent category. These previous studies have suggested that 27-33% of all pancreatic cancer cases may be attributable to cigarette smoking.[5,6] This figure may indeed be quite higher if the effects of passive smoking are considered. Further studies are needed to evaluate this association, particularly in light of the small number of pancreatic cancer cases among never smokers in this study population.

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RÉSUMÉ

Contexte : Bien que le tabac constitue un facteur de risque bien connu pour le cancer du pancréas, aucune étude n'a encore constaté d'association entre la fumée secondaire du tabac (FST) et ce type de tumeur maligne. À l'aide d'une étude cas-témoin, nous avons analysé la relation entre le cancer du pancréas et l'exposition à la FST durant l'enfance et à l'âge adulte.

Méthode : La population à l'étude comprenait 583 cas de cancer du pancréas et 4 813 témoins représentatifs identifiés dans huit provinces canadiennes entre 1994 et 1997. Nous leur avons posté des questionnaires pour recueillir de l'information sur leurs facteurs de risque et leurs antécédents d'exposition à la FST, à la maison et au travail, au cours de leur vie.

Résultats : Chez les personnes n'ayant jamais fumé, celles qui avaient été exposées à la FST durant l'enfance et à l'âge adulte avaient un rapport de cotes de 1,21 (IC de 95 % =0,60-2,44) comparativement aux personnes non exposées. Pour les fumeurs actifs (comparés à un groupe de référence composé de personnes n'ayant jamais fumé et n'ayant pas été régulièrement exposées à la FST), l'accroissement du risque était plus prononcé avec le nombre d'années de tabagisme, le nombre de paquets de cigarettes par année, le nombre d'années depuis le renoncement au tabac et le nombre moyen de cigarettes fumées quotidiennement.

Conclusions : Dans l'ensemble, nos résultats semblent indiquer une faible association entre le cancer du pancréas et la FST. Ils soulignent néanmoins un fait intéressant : l'exposition à la FST pourrait être un facteur de confusion dans les mesures couramment utilisées par les études épidémiologiques pour évaluer le risque de cancer du pancréas associé au tabagisme actif.

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