Relation between Body Mass Index and Lung Cancer Risk in Men and Women Never and Former Smokers

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The authors assessed body mass index (BMI), measured as Quetelet's index (weight in kilograms divided by the square of height in meters), in relation to lung cancer risk in never and former smokers by using data from a population-based, individually matched, case-control study conducted in New York State from 1982 to 1985. To be included in the study, subjects must never have smoked more than 100 cigarettes in their lifetime (never smokers) or not have smoked more than 100 cigarettes during the last 10 years (former smokers). Data on height and weight were complete for 412 of 439 case-control pairs. A positive relation was found between BMI and lung cancer risk for both never smokers (188 case-control pairs) and former smokers (224 pairs). When subjects were combined, those in the eighth (highest) octile (BMI > 30.84) had more than twice the odds of being cases compared with those in the lowest octile (BMI ≤ 21.26, 95 percent confidence interval: 1.2, 4.4). These study results are consistent with those from studies of BMI and other cancer sites but differ from lung cancer results usually found in predominantly smoking populations. Am J Epidemiol 2000;152:506–13.

body mass index; case-control studies; lung neoplasms; obesity; smoking; tobacco

Quetelet's index, a measure of body mass index (BMI), is defined as a person's weight in kilograms divided by the square of height in meters. BMI is often used in epidemiologic studies as a proxy measure of obesity and has been shown to be positively associated with risk of a variety of cancers, including postmenopausal breast cancer (1–8), endometrial cancer (2, 9–12), and colon adenomas and adenocarcinomas (13–18). The data are suggestive for prostate cancer as well (19). The major exceptions are premenopausal breast cancer (1–4, 20–24) and lung cancer (25–31), for which an inverse association with BMI is usually seen.

The issue of whether obesity is protective for lung cancer remains unresolved. A major reason is that most studies have been conducted in populations that smoke, where the strong impact of smoking on both BMI and lung cancer risk may obscure the true relation between BMI and lung cancer risk. Although these studies attempt to adjust statistically for the effect of smoking, it is not clear that such adjustment can fully compensate for the true effect of smoking on this relation.

Given the problem of confounding by tobacco, the cleanest method of studying the relation between BMI and lung cancer risk is to study a population of persons minimally exposed to tobacco. However, such a population is difficult to assemble, because the vast majority of lung cancer patients have a history of smoking. Two studies have found a protective effect of obesity on lung cancer risk in never smokers, one a cohort study with 10 cases who never smoked (25) and the other a hospital-based case-control study that found this relation for female but not male never smokers (26). We performed such a study in a large population of lung cancer cases and controls who were never and former smokers; subjects were individually matched by smoking history either as never smokers or as former smokers who had been nonsmokers for at least the previous 10 years.

MATERIALS AND METHODS

Data collection methods for this study have been described previously (32–34). Briefly, a population-based, individually matched, case-control study was conducted in New York State from 1982 to 1985. Twenty-three counties, representing seven Standard Metropolitan Statistical Areas in upstate New York, were chosen for inclusion. This area contained about 10 million people, with about 125 diagnostic facilities from which cases could be ascertained.

Selection of cases

A special system of rapid case ascertainment was set up so that the time from diagnosis to inclusion in the study...
would be minimized. These diagnostic facilities as well as the New York State Tumor Registry were checked frequently for all new lung cancer cases, whether diagnosed clinically, histologically, or both. The mean time from diagnosis to reporting was 45 days, and the mean time from reporting to interview was 102 days.

To be included as a case in this study, the patient had to reside in the 23-county area, be aged 20–80 years, and have been given a diagnosis of primary lung cancer between July 1, 1982, and December 31, 1984. In addition, the patient was required to not have smoked more than 100 cigarettes in his or her lifetime (classified as a never smoker) or more than 100 cigarettes during the last 10 years (classified as a former smoker). Smoking information was determined initially by using the patient’s medical records and then was confirmed by telephone contact and again at the time of interview. To confirm the lung cancer and history classification, an outside pathologist reexamined pathologic specimens and clinical records. If the pathologist’s determination of histology type differed from the initial determination, a third pathologist read the pathology slides. When reading the slides, pathologists always were blinded to smoking status. Interviews were completed successfully for 76 percent of the potentially eligible cases.

Selection of controls

Individually matched controls were selected by screening the New York State Department of Motor Vehicle records and selecting a random sample of persons from this source. This source of controls was considered appropriate because it was population-based and provided much of the information needed to match cases to controls. Virtually all cases had driver’s licenses as well, suggesting that the Department of Motor Vehicles was a good population-based source for controls. Initially, roughly six potential controls were individually matched to their potential case on age, sex, and district of residence. On average, for each case, two potential controls had to be contacted before one was identified who matched the case on smoking status and also agreed to serve as a control for that case. In most instances, the controls were then further matched to cases on interview type (self vs. surrogate) in an attempt to collect information on each member of the case-control pair in a similar manner. This step was performed to minimize recall bias that might occur if a surrogate tended to over- or underreport certain exposures.

Data collection

The original purpose of data collection was to study the relation between passive smoking and lung cancer risk. Data were collected on 439 case-control pairs. A pretested, precoded, structured questionnaire was used to interview cases and controls face-to-face in their homes. Basic demographic information on such variables as age, sex, income, education, religion, and ethnicity was collected, as was information on job history, health history, diet history, family history of cancer, exposures to chemical and physical factors, and other variables. The questionnaire took about 1 hour to administer.

The interview included one question each on self-reported height and weight prior to illness for cases and 1 year ago for controls. Answers were later converted into body mass index for analysis. No other attempts were made to elicit information on other anthropometric variables or to validate the self-reports of height and weight. Of the 439 individually matched case-control pairs accrued, both the case and the control in 412 pairs (188 never smokers and 224 former smokers) provided height and weight information and thus were included in our analyses.

Statistical analysis

Given the matched design of our study, conditional logistic regression analysis was used to account for the effect of matching on the relation under investigation. This modification of logistic regression has been described by Holiford et al. (35). Data management was performed with SAS (36) software, and conditional and unconditional logistic regression modeling were done by using both EGRET (37) and STATA (38) statistical software packages.

RESULTS

Anthropometric variables

Demographic characteristics of this population are shown in table 1. The mean height of the study population was 1.68 m, and cases were slightly shorter than controls (1.68 vs. 1.69 m, respectively). Cases also were heavier than controls (mean weight: 75.1 vs. 73.0 kg, respectively). These data translated into an average BMI of 26.6 (range: 14.3–54.9) for cases versus 25.5 (range: 17.0–38.4) for controls. There tended to be a higher proportion of cases in lower strata of income, education, and consumption of servings of raw fruits and vegetables and in higher strata of intensity and duration of smoking for former smokers.

By using BMI octile cutpoints determined by the study population, we observed an increasing mean BMI across increasing octiles of BMI that was driven by an increase in mean weight, while height remained stable (data not shown). Overall, the ratio of cases to controls reversed with increasing octiles of BMI, from about 1.5 controls per case in octile 1 (the lowest) to almost 2 cases per control in octile 8 (the highest) (table 2). A similar pattern appeared throughout all strata of the matching variables. Of note, of the 16 persons with the highest BMI in this data set, 15 were cases. Of these 15 cases, 11 were female, 7 were never smokers, and 5 had surrogate interviews.

Main effect of BMI on lung cancer

The unadjusted odds ratio for lung cancer associated with a five-unit linear increase in BMI was 1.33 (95 percent confidence interval: 1.13, 1.57) in matched analysis. The BMI-lung cancer odds ratio was mildly larger for women versus men (odds ratio (OR) = 1.35 vs. OR = 1.22, respectively)
and was virtually identical for former smokers and never smokers (OR = 1.28 vs. OR = 1.31, respectively). This odds ratio was larger for younger subjects (aged less than 70 years) compared with older subjects (OR = 1.42 vs. OR = 1.13, respectively). No considerable modification of the BMI-lung cancer odds ratio was found after stratification by income, education, or diet (servings per month of raw fruits and vegetables).

TABLE 1. Demographic characteristics (selected variables) of men and women never and former smokers, New York lung cancer study, 1982–1985*

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<th>Variable</th>
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* Includes only the 412 case-control pairs with complete data on height and weight.
† Some percentages do not total 100% because of rounding.
‡ OR, odds ratio.
§ Refers to matched strata.
Confounding of the BMI-lung cancer association

Several variables previously have been shown to be associated with lung cancer risk in this data set (33, 34, 39, 40). To check for confounding of the BMI-lung cancer association, each data set variable associated with lung cancer in univariable analysis was included in a bivariable analysis of the effect of BMI on lung cancer risk (data not shown). Adjustment for education and income, both separately and together, reduced the magnitude of the BMI association by 10–30 percent, depending on how the BMI variable was categorized. No smoking-related variables were shown to confound the BMI-lung cancer association when tested individually or together (age at which smoking started, age at which smoking stopped, number of years of smoking, number of years since quitting, total pack-years, number of cigarettes smoked per day, and lifetime passive smoke exposure).

BMI as a categorical variable

Next, BMI was categorized into quartiles and octiles based on the distribution of the study population as a whole to determine whether the BMI-lung cancer association was linear. In these unadjusted analyses, the association increased with increasing quartile and octile of BMI; most of the increase occurred in the uppermost octile of the BMI distribution. From these analyses it appeared that BMI octile 8 was driving the majority of this increase in lung cancer risk.

Multivariable modeling of main effects

The results of conditional logistic regression modeling of the BMI-lung cancer relation are shown in table 3. BMI was modeled as a three-category variable, with octile 1 (lowest 12.5 percent) as the referent (BMI ≤ 21.26), octiles 2–7 combined as the first comparison group (BMI > 21.26 and ≤ 30.84), and octile 8 (highest 12.5 percent) as the second comparison group (BMI > 30.84). Education was used instead of income to adjust for socioeconomic differences among cases and controls because of the large quantity of missing data on income. The BMI-lung cancer association was further adjusted for income, while missing income was assigned to a separate

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category; no increased adjustment for confounding was found when this model was compared with the smoking- and education-adjusted models (data not shown). Otherwise, all covariates were modeled continuously to achieve a parsimonious model.

The results adjusted for both smoking and smoking and education showed an increase in the odds of lung cancer when BMI octile 8 was compared with octile 1 and mostly a moderate increase when octiles 2–7 were compared with octile 1. This pattern generally was found for most strata of
smoking (never vs. former), sex, age (less than vs. more than age 70 years), and interview type (self vs. proxy).

DISCUSSION

A positive relation was found between BMI and lung cancer risk in this population-based case-control study of never and former smokers. Most of this risk seemed to be for subjects in BMI octile 8. Those in the highest octile of BMI (≥30.84) had more than twice the odds of being cases compared with those in the lowest octile of BMI (≤21.26). Because of the nature of case-control studies in general, we could not determine the baseline risk of lung cancer for either never smokers or former smokers. Regardless, it appears that the obesity-related odds ratio of lung cancer was similar for both former and never smokers.

Identifying the relation between BMI and lung cancer incidence presents many of the same problems found when assessing the association between BMI and mortality. Most of the known biases inherent in a study of BMI and lung cancer tend to favor an inverse BMI-lung cancer risk relation. For example, the time at which BMI is measured relative to disease progression is important (27–29, 31). If preclinical weight loss in lung cancer cases has begun to occur before BMI is assessed, then cases would tend to be thinner than controls. Thus, a bias toward an inverse BMI-lung cancer association would occur that would attenuate or eliminate any positive BMI-lung cancer association that might exist. In the present study, BMI was assessed as self-reported height and “usual” weight “prior to your illness” for cases and “last year at this time” for controls. Adjustment for preclinical weight loss in the present study, had it been possible, would have resulted in an even stronger positive association than the one observed.

The effects of smoking-related variables on the BMI-lung cancer association are complex and therefore may not be amenable to statistical adjustment. Adjustment for smoking has been shown to change the shape of the mortality distribution with respect to BMI (41). For the BMI-lung cancer association, smoking is so strongly related to both variables that adjustment for smoking may leave much room for residual confounding. Because smoking is associated with a lower BMI and a higher risk of lung cancer, residual confounding from smoking might either produce an inverse BMI-lung cancer association or disguise a true positive BMI-lung cancer relation. Studies in populations that smoke show that statistical adjustment for smoking does readjust the observed inverse association toward the null value, although a statistically significant inverse association often remains. In the present study, our attempt to minimize the impact of smoking exceeded such efforts by other studies in two major ways. It both matched on smoking status (never vs. former) and excluded anyone who had been a smoker during the previous 10 years. Consequently, to our knowledge it is the only matched study of its kind and included more cases who never smoked (n = 188) than any published BMI-lung cancer study has. The similarity in the association between never and former smokers adds credibility to the results regarding the never smokers in this study.

Kabat and Wynder’s (26) study that found an inverse BMI-lung cancer association for women never smokers is the only other such study with substantial numbers of never smokers; roughly 100 such cases were available. Their study was hospital-based, and use of hospital-based controls may have resulted in a control group that was more obese than the population from which the cases were drawn. It is possible that the difference in results between the Kabat and Wynder study and ours is due to different criteria used to select controls.

Methods of sensitivity analyses described by Greenland (42) were used to explore the effect of potential control selection bias and differential recall of cases and controls. If nonparticipating controls were more likely than participating controls to be obese, then a true lack of association could be observed as a positive obesity-lung cancer association. Sensitivity analyses of potential control selection bias revealed this explanation to be highly unlikely for the association observed (data not shown).

Differential recall bias of weight by cases versus controls is a real possibility. If cases underreported their weight relative to controls, then the positive association observed in this study would be an attenuation of the true association. In contrast, if cases differentially overestimated their weight before diagnosis, then the observed association would be larger than the true association. On the basis of sensitivity analyses, these results appear to be more sensitive to the potential for recall bias than for control selection bias.

There are four possible types of misclassification of obesity, defined in this study as being in BMI octile 8. Obese cases classified as nonobese (because of preclinical weight loss), or nonobese controls classified as obese, would result in an odds ratio smaller than the true value by producing a negative bias. Nonobese cases classified as obese, or obese controls classified as nonobese, would result in an odds ratio larger than the true value by producing a positive bias. In this study, the unmatched, unadjusted odds ratio was 1.87 for BMI octile 8 compared with octiles 1–7 combined. For this odds ratio to be entirely attributable to a positive bias from misclassification of obesity, at least 28 of the 374 controls classified as nonobese or 28 of the 66 cases classified as obese would have to have been misclassified. Any misclassification producing a negative bias would mean that a larger number of misclassifications that produce a positive bias would be required to nullify the observed odds ratio. The most likely source of recall bias, preclinical weight loss, would attenuate a true positive BMI-lung cancer association.

The result of a stronger BMI-lung cancer association in proxy-interviewed case-control pairs is difficult to interpret. For both cases and controls, roughly 28 percent of responses were by proxy. Attenuation of the association from misclassification related to proxy reporting of height and weight might be expected. If this misclassification were similar among proxy-interviewed cases and controls, the result would be an attenuation of the true association between BMI and lung cancer. Alternatively, cases who required a proxy interview may have been ill for a longer period of time and perhaps had more preclinical weight loss compared with individually inter-
viewed cases. Again, the expected result would be an attenuation of the association for proxy-interviewed case-control pairs. If disease in proxy-interviewed versus self-report cases generally had progressed to a later stage, then perhaps BMI-related risk is stronger for cases whose disease is more aggressive.

The lung is the only site, other than perhaps premenopausal cancer of the breast, for which an inverse relation has been consistently reported between BMI and cancer risk. It is also the site most likely to be affected by confounding from smoking, and the sparse data that exist on never smokers are ambiguous with regard to BMI and lung cancer risk.

The positive BMI-lung cancer association found in this matched case-control study of nonsmokers may have a biologically plausible explanation as well. Obese persons tend to have higher levels of estrogens, converted from androgens in fat tissue (43–45). In combination with lower levels of steroid hormone-binding globulin (44), there may be more circulating unbound estrogen, able to bind to its receptor in lung and other tissues. Estrogen may act as a promoter, with higher estrogen levels acting as a growth factor on cancer cells (45). Stratification of the data by sex and age, however, suggests that the adverse effects of obesity are not mediated via estrogen. That is, menopausal status and therefore estrogen levels are not responsible for the BMI-related lung cancer risk. In our data, the BMI-related risk was similar for women aged less than and more than 70 years (table 3) and less than and more than 50 years (data not shown). Obese persons are also known to have higher circulating levels of insulin, which also may promote cancer cells by acting as a growth factor (46, 47). The results of the present study are more consistent with the BMI-cancer associations found for other cancer sites, and similar biologic theories have been used to explain these associations.

The results shown in table 3 suggest that age may modify the effect of BMI on lung cancer risk in men but not women. This finding for men is consistent with the recent finding by Stevens et al. with regard to BMI and overall mortality (48).

The positive obesity-lung cancer relation found in this study of never smokers and of former smokers of more than 10 years is more similar to the data on BMI and other cancer sites than are the BMI-lung cancer associations that have been reported in predominantly smoking populations. Ideally, this relation would be reassessed by using a cohort design. However, the small number of never smokers who develop lung cancer would make such a study design prohibitively costly and time-consuming. Recent cohort studies of mortality have been reanalyzed to look at never smokers who subsequently died of lung cancer, but most included only a handful of such cases.

Ours appears to be the largest study of BMI and lung cancer risk in never smokers, with 188 case-control pairs matched on never-smoking status. Analysis of another 224 case-control pairs, matched on not having smoked for at least 10 years, produced a virtually identical association, adding credibility to the results for never smokers. These results disagree with the literature on BMI and lung cancer risk in general, perhaps because of the minimal effect of tobacco on these results.

ACKNOWLEDGMENTS

Data collection was supported by a grant from the National Cancer Institute (R01CA32088) and data analysis by a grant from the National Cancer Institute to the Yale Cancer Center (R25CA47883). Support for manuscript preparation came from a grant from the National Cancer Institute to the Department of Epidemiology at the University of North Carolina at Chapel Hill (CA09330-20).

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