Cigarette smoking: an independent risk factor for impotence?

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Cigarette Smoking: An Independent Risk Factor for Impotence?

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The authors sought to determine whether current cigarette smoking was associated with impotence among middle-aged men. This is a secondary analysis of a cross-sectional survey of 4,462 US Army Vietnam-era veterans aged 31–49 years who took part in the Vietnam Experience Study in 1985–1986. The main outcome measurement was the odds ratio for reported impotence, which was calculated by comparing current smokers with nonsmokers while controlling for multiple confounders. The study sample consisted of 1,162 never smokers, 1,292 former smokers, and 2,008 current smokers. The prevalence of impotence was 2.2% among never smokers, 2.0% among former smokers, and 3.7% among current smokers (p = 0.005). The unadjusted odds ratio (OR) of the association between smoking and reported impotence was 1.8 (95% confidence interval (CI) 1.2–2.6). The association held even after adjustments were made for confounders, including vascular disease, psychiatric disease, hormonal factors, substance abuse, marital status, race, and age (OR = 1.5, 95% CI 1.0–2.2). Neither years smoked nor cigarettes smoked daily were significant predictors of impotence in current smokers. The authors concluded that, among the men in this study, a higher percentage of cigarette smokers reported impotence than did nonsmokers. This observation could not be totally explained by comorbidity factors related to smoking. Am J Epidemiol 1994;140:1003–8.

Although its epidemiology has not been well studied, impotence is a common problem among men. Some researchers think it may affect as many as 10 million men in the United States (1). Thirty years ago, 90 percent of impotence cases were thought to have a psychogenic cause, but today over 50 percent of such cases are thought to have an organic cause (2). Many factors, including hypertension, medications, diabetes mellitus, aging, and ethanol use, are associated with impotence (3, 4). Although several studies (5–7) have evaluated the risk of impotence among cigarette smokers, most involved few men and did not control for confounders such as alcohol intake, hypertension, and depression.

In this study, our objective was to examine whether smoking was an independent risk factor for impotence among 4,462 Vietnam-era veterans aged 31–49 years.

MATERIALS AND METHODS

Complete medical history and physical and psychological examination data were obtained in 1985 and 1986 from 4,462 ran-
domly selected Vietnam-era US Army veterans as part of the Vietnam Experience Study (8–10). As described elsewhere (8, 11), the men examined were randomly selected from more than 14,000 veterans who participated in a large telephone interview study.

All subjects completed medical questionnaires that were administered by trained physician’s assistants (12). We defined impotence as a positive response to this question: “Have you experienced persistent difficulty in getting a satisfactory erection for sexual purposes within the past year?” Subjects were asked to report any prior physician-diagnosed hypertension or diabetes mellitus and to list all their current medications. We identified subjects being treated with antihypertensives, diuretics, sedatives, anxiolytics, antidepressants, or neuroleptics, which are all associated with impotence (2, 3, 13, 14).

A physical examination was also completed on each subject (12). We considered subjects with a mean diastolic blood pressure of greater than 95 mmHg, a brachial/ankle blood pressure ratio of less than one, or an absent posterior tibial pulse to have evidence of vascular disease.

Psychological health among members of the study group was assessed by using the Diagnostic Interview Schedule (15). We considered subjects to have a condition of interest (depression, generalized anxiety, alcohol abuse or dependence, or drug abuse or dependence) if, during the previous year, they reported a pattern of symptoms that met psychiatric criteria for that condition (15).

Blood was drawn for multiple assays, including testosterone and luteinizing hormone levels, in the morning (16). We classified subjects with testosterone levels of less than 12.1 nmol/liter (350 μg/liter) as having low testosterone levels and subjects with luteinizing hormone levels of less than 7 IU/liter as having low luteinizing hormone levels (17).

A complete smoking history was obtained on each subject (12). For some analyses, we grouped the former smokers and never smokers together and called them “nonsmokers.”

We considered subjects to have vascular factors associated with impotence if they reported hypertension, diabetes mellitus, or use of diuretics or antihypertensives, or if their physical examination results showed evidence of vascular disease. We considered subjects who had generalized anxiety or depression or who reported using sedatives, anxiolytics, antidepressants, or neuroleptics to have psychological factors associated with impotence. We considered subjects who had low testosterone levels or low luteinizing hormone levels to have hormonal factors associated with impotence. We considered subjects who reported abusing alcohol or drugs in the past year, or who reported consuming six or more alcoholic drinks on the days they were drinking to have substance abuse factors associated with impotence. We also classified each subject by marital status (married or not married) and race (white or not white).

We used logistic regression and chi-square tests for association from the SAS library (SAS Institute, Cary, North Carolina). We used logistic regression to model impotence as the outcome variable and to model smoking (categorized as nonsmoking or current smoking) as the exposure of primary interest. As potential confounders and effect modifiers, we included vascular factors, psychological factors, hormonal factors, substance abuse factors, marital status, race, and age (17). We assessed the goodness-of-fit of the logistic models using a summary measure of the errors (18).

RESULTS

The study sample consisted of 4,462 Vietnam-era Army veterans (table 1), of whom 45 percent were current smokers. Their mean age was 38 years, with a range of 31–49 years (table 2).

Current smokers, former smokers, and never smokers had similar prevalences of vascular risk factors associated with impotence (table 3). Current smokers were more likely to be non-white and unmarried and
had higher prevalences of psychological risk factors and substance abuse risk factors and a lower prevalence of hormonal risk factors. Impotence was reported by 2.2 percent of never smokers, 2.0 percent of former smokers, and 3.7 percent of current smokers ($p = 0.005$); the unadjusted odds ratio (OR) comparing impotence among smokers with that among nonsmokers was 1.8 (95 percent confidence interval (CI) 1.2–2.6).

Impotence was more commonly reported among most subgroups with selected risk factors (table 4). We found no evidence of effect modification. After adjusting for vascular factors, psychological factors, hormonal factors, substance abuse factors, marital status, race, and age, current smoking was associated with impotence (OR = 1.5, 95 percent CI 1.0–2.2, goodness-of-fit statistic = 3.97 with 8 degrees of freedom, $p = 0.86$). After adjusting for only vascular risk factors, psychiatric risk factors, race, and marital status, current smoking was associated with impotence (OR = 1.5, 95 percent CI 1.0–2.1, goodness-of-fit statistic = 7.39 with 8 degrees of freedom, $p = 0.50$).

The results of the analyses restricted to current smokers showed that neither the number of cigarettes smoked daily nor the number of years smoked was a predictor of impotence.

**DISCUSSION**

After controlling for other factors associated with impotence, we found an independent association between current smoking and impotence in this population of Vietnam era veterans.
Population estimates of the prevalence of impotence are difficult to obtain, in part because of problems with the definition of impotence (19, 20). In our study, the overall prevalence of "persistent difficulty in getting a satisfactory erection for sexual purposes" in the previous 12 months was 3 percent, which is consistent with other studies of men in this age range (20, 21).

Vascular risk factors, including atherosclerosis, hypertension, antihypertensive medications, and diabetes mellitus, and psychological risk factors, including depression and medication use, are believed to have a major effect on impotence (2–4, 22–25). Subjects with either one or more vascular risk factors or one or more psychological risk factors reported a higher prevalence of impotence than subjects without these factors in all smoking categories.

Alcohol consumption has similarly been associated with impotence, but its mechanism of action is unclear (26, 27). In our study, current smokers and never smokers with substance abuse had a higher prevalence of impotence but not former smokers.

Hormonal factors are thought to be important in the association between smoking and impotence (4, 27), although smokers are reported to have higher testosterone levels (28). Hormonal risk factors were more prevalent among nonsmokers than current smokers in this study, and were associated with an increased prevalence of impotence only among nonsmokers (table 4).

The overall lack of association between age and impotence in this study is likely related to the larger effects of the vascular and psychological factors and the narrow age range of our study group.

Cigarette smoking is clearly associated with vascular pathology that could affect erectile function (7). Pharmacologic factors may also be important (29, 30), as some studies suggest that cigarette smoking might exert an effect on the autonomic nervous system similar to the pharmacologic effects of the many medications associated with impotence.

Results of epidemiologic studies of impotent men show that cigarette smoking may be an important risk factor (31–33). The results of our comparison of current

<table>
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<th>Former smokers</th>
<th>Current smokers</th>
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<td></td>
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<td>%</td>
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smokers with nonsmokers are consistent with these findings. We did not, however, detect a dose-response relation between smoking and reported impotence. This may be because the study population is relatively young; we would expect many more of the men to develop vascular pathology as they age, especially the smokers (34). In addition to controlling for known risk factors for impotence, we also controlled for race and marital status, which we found to be associated with reported impotence in our univariate analysis of the data.

Several potential biases may affect our ability to generalize from the results of this study. Study participants were all Vietnam-era Army veterans and may not be representative of the US male population. Standardized questionnaires such as those we used might elicit fewer reports of impotence than would clinical interviews and examinations. We did not document impotence in this study using tumescence or vascular evaluations, and we did not have data on whether our subjects had attempted intercourse.

Despite these limitations, we suspect that underreporting would have tended to occur to a similar extent among current smokers and nonsmokers; if this is true, our results would underestimate the association between smoking and impotence. An alternative explanation for our findings, however, is that smokers report symptoms more frequently than nonsmokers (35).

Results of our study suggest that tobacco smoking is associated with an increased prevalence of self-reported impotence in a population of Vietnam-era veterans. After we adjusted for other risk factors, current smokers had 50 percent more reported impotence than nonsmokers. Since smoking may be a risk factor in impotence, a smoking history should be obtained on all men visiting sexual dysfunction clinics. This association between smoking and impotence should provide clinicians with yet another opportunity to encourage patients, especially men with impotence, to stop smoking.

REFERENCES

19. Masters WH, Johnson VE. Principles of the new