
ORIGINAL RESEARCH

Cigarette smoking and chronic low back pain in the adult population

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Abstract

Purpose: Chronic low back pain (LBP) is one of the main causes of disability in the community. Although there have been studies suggesting an association between smoking and LBP, these studies were limited by the small numbers of patients, and they did not control for confounders. The objective of this study was to determine whether cigarette smoking is associated with an increased risk of chronic LBP among adults.

Methods: Using Canadian Community Health Survey (cycle 3.1) data, 73,507 Canadians aged 20 to 59 yr were identified. Self-reported chronic LBP status, smoking habits, sex, age, height, weight, level of activity and level of education were identified as well. Back pain secondary to fibromyalgia was excluded. Multivariate logistic regression analysis was used to detect effect modification and to adjust for covariates. Design effects associated with complex survey design were taken into consideration.

Results: The prevalence of chronic LBP was 23.3% in daily smokers and only 15.7% in non-smokers. Age and sex were found to be effect modifiers ($P < 0.0001$), and the relationship between smoking and chronic LBP risk was dependent on sex and age. The association between daily smoking and the risk of chronic LBP was stronger among

younger individuals. Occasional smoking slightly increased the odds of having chronic LBP.

Conclusion: Daily smoking increases the risk of LBP among young adults, and this effect seems to be dose-dependent. Back pain treatment programs may benefit from integrating smoking habit modification. Further research is required to develop effective prevention strategies.

Low back pain (LBP) is a very common problem among adults. Up to two-thirds of adults suffer from LBP at some time.¹⁻³ It is estimated that, every year, between 2% to 5% of the population seeks medical attention because of LBP.^{4,5} Many adults with LBP will eventually develop Chronic LBP, which is one of the main causes of disability in the community and has a huge economic impact.⁶⁻⁸ Back pain may originate from many spinal structures, including facet joints, ligaments, intervertebral discs, nerve roots, vertebral bodies and paravertebral muscles. The onset of back pain most often occurs between the ages of 30 and 50 years.^{1,4,9}

Studies have reported different risk factors for chronic LBP; these include age, sex, genetics, education level, activities, socioeconomic status, lifestyle and smoking.^{3,5,10,11} Unfortunately, most of these factors are not easily changed by medical intervention. Nevertheless, lifestyle factors such as smoking could be changed with some effective interventions.^{10,12}

Animal models and biological studies support the association between smoking and the health of intervertebral discs.¹³⁻¹⁵ In humans, some studies have reported a possible link between cigarette smoking and chronic LBP,¹⁶⁻¹⁸ while others did not.^{3,19} Goldberg et al.²⁰ reviewed 38 studies that looked at the association between cigarette smoking and the development of chronic LBP, but the results were inconsistent. This might be because a large percentage of the studies did not account for possible confounders, whereas the studies that accounted for confounders were heterogeneous in their covariates. These authors also found that the most consistent confounders were age, sex, body mass index, level of activity and level of education.

The prevalence of smoking among Canadians was recently reported to be around 23%²¹ and, according to the latest reports from Health Canada, about five million Canadians were smokers at the time of the survey. The association between chronic LBP and smoking in the Canadian community receives limited attention in the current literature.^{1,22,23}

We hypothesised that there is an association between smoking and the risk of chronic LBP among adults aged 20 - 59 yr. In examining this association, we took into consideration the most likely covariates (age, sex, BMI, activity and education). We used a large sample size, which provides an opportunity to examine this association with good precision. This is the first study to assess the relationship between chronic LBP and smoking exposure among adults, based on a large sample size.

Materials and Methods

Study population

This study used data from the Canadian Community Health Survey (CCHS) cycle 3.1.²⁴ Conducted by Statistics Canada in 2005, the CCHS 3.1 was a cross-sectional survey using multi-staged, stratified random sampling procedures and targeting persons aged ≥ 12 yr who were living in privately occupied dwellings spanning 122 health regions in the ten provinces and three territories of Canada. Individuals living on Indian reserves, Crown Land or institutional residence, full-time members of the Canadian armed forces and residents of certain remote regions were excluded from the sampling frame.

The survey was conducted from January to December 2005 and had a 78.9% national response rate, with 132,947 individuals responding. The survey included questions related to health status, health care use and health determinants. Interviews were conducted equally in person and over the phone. Interviewers were trained, and computer-assisted interviewing was employed.

Participants aged 20 - 59 yr in the CCHS 3.1 database were identified. Their exposure status and outcomes were also identified, as well as information about possible confounders.

Exposure and Outcomes

The exposure of interest was smoking. Smoking status was determined based on the following questions:

- *In your lifetime, have you smoked a total of 100 or more cigarettes (about 4 packs)?*
- *Have you ever smoked a whole cigarette?*
- *At the present time, do you smoke cigarettes daily, occasionally or not at all?*
- *Have you ever smoked cigarettes daily?*

Based on the answers to these questions, respondents were classified as daily smokers (present or

TABLE 1. Smoking status classification

Smoking Status	Answers
Daily smoker (present or former)	Q3 (Daily), Q3 (Occasionally) and Q4 (Yes), Q3 (Not at all) and Q4 (Yes).
Occasional smoker (present or former)	Q3 (Occasionally) and Q4 (No), Q3 (Occasionally) and Q4 (No) and Q1 (Yes) or Q2 (Yes).
Non-smoker	Q3 (Not at all) and Q1, Q2, Q4 (No).

former), occasional smokers (present or former) or non-smokers (Table 1).

The health outcome was chronic LBP, excluding LBP secondary to fibromyalgia. The LBP should be diagnosed by a health care professional and have a duration of at least six months. A dichotomous outcome variable was created to classify individuals as either having chronic LBP or not, based on their responses.

Participants' age, sex, BMI, level of activity and education level were identified and analysed. Subjects were grouped into four age groups (20-29, 30-39, 40-49 and 50-59 yr). BMI was calculated as weight (kg)/height (m). Obesity status was then determined using the World Health Organisation (WHO) classification whereby participants with a BMI of ≥ 30 considered obese and those with a BMI < 30 are not. Participants were classified as active if they participated in daily physical activity for at least 15 min. Based on participants' education level, two groups were identified: a higher education group (participants who had been admitted to college or university and those with a post-secondary school certificate or diploma) and a lower education group (those who did not proceed beyond secondary education).

Analytical methods and strategy

Since the CCHS 3.1 involved a complex survey design, in which selection probabilities had to be accounted for in all point estimate- and variance-related calculations, we had to calculate the adjusted weight. We achieve this by taking into account the national average design effect and relative sampling weights.²⁵

The point prevalences of chronic LBP according to smoking status, age, sex, BMI, physical activity and level of education were calculated first. To examine

the relationship between smoking status and chronic LBP, univariate and multivariate logistic regression analysis were used before and after adjustment for covariates.

Using logistic regression analysis, odds ratios and their respective 95% confidence intervals were calculated and used to express the relationship between smoking status and chronic LBP; an odds ratio greater than one signifies increased risk of chronic LBP compared with the referent group.

Since the relationship between smoking status and the risk of chronic LBP may depend on participants' age, sex or both, these covariates were assessed for effect modification by including multiplicative interaction terms in each model.

Covariates were also assessed for potential confounding. Confounders were identified if they resulted in a 10% change in the odds ratio for the association. Effect modifiers, confounders and significant predictors of back pain were included in all models. Model parameters were estimated by using the method of maximum likelihood ratio and were tested if they were found to be statistically significant using the Wald statistical test.

Data for the exposure, the outcome or any of the covariates were found to be missing in only 2.8% of participants. Because the percentage of participants with missing data was small, they were excluded from the analysis. All statistical analyses were conducted using the statistical software package SAS, version 9.1.²⁶

Results

A total of 73,507 individuals were identified and analysed (Table 2). Among these, the overall prevalence of chronic LBP was 19.6%. About one-third of our

TABLE 2. Characteristics of the study population

Characteristic	No.	%*
Chronic Low back pain	15,372	19.6
Smoking Status		
Daily smoker (present or former)	37,905	46.7
Occasional smoker (present or former)	13,160	18.9
Non-smoker	22,442	34.4
Sex		
Male	35,242	51.0
Female	38,265	49.0
Age (Yr)		
20-29	15,582	23.6
30-39	18,812	23.6
40-49	19,221	29.5
50-59	19,892	23.33
BMI		
Not obese	59,817	84.0
Obese	13,690	16.0
Education		
Secondary education or less	21,359	16.0
Post-secondary education	52,148	84.0
Activity		
Not active	46,525	64.2
Active	26,982	35.8

* Weighted to the Canadian population

study population consisted of non-smokers, while 46.8% of participants were current or former daily smokers; 51% of the participants were men, the majority had some post-secondary education, only 16% were obese and about one-third classified themselves as active persons.

All the included covariates had an association with the prevalence of chronic LBP (Table 3). Obesity was associated with increased prevalence of chronic LBP, and this increase in the prevalence was almost consistent regardless of smoking status. As shown in Table 3, age was associated with a consistent increase in the prevalence of chronic LBP. Individuals who had some postsecondary education in general had less chronic LBP. Physical activity was associated with a decreased prevalence of chronic LBP; this effect was minor.

The analysis based on individuals' smoking status (Table 3) showed that the prevalence of chronic LBP is different between daily smokers, occasional smok-

TABLE 3. Prevalence of chronic back pain associated with smoking, age, sex, BMI, educational level and activity status

	Daily smokers (present or former)			Occasional smokers (present or former)			Non-smokers		
	No.	Cases	%*	No.	Cases	%*	No.	Cases	%*
Total	37,905	9,199	23.3	13,160	2,392	17.2	22,442	3,760	15.7
Sex									
Male	19,108	4,653	23.8	6,511	1,144	16.2	9,634	1,507	14.7
Female	18,797	4,546	22.7	6,649	1,248	18.2	12,808	2,253	16.7
BMI									
Not obese	30,442	7,128	22.5	10,936	1,872	16.1	18,470	2,902	15.0
Obese	7,463	2,071	27.1	2,224	520	23.6	3,972	858	20.2
Age (yr)									
20-29	6,826	1,296	18.1	3,087	342	11.8	5,673	624	10.4
30-39	8,323	1,766	20.9	3,789	612	15.9	6,697	1,046	15.2
40-49	10,838	2,793	25.3	3,240	698	19.7	5,145	991	18.0
50-59	11,918	3,344	26.4	3,044	740	22.5	4,927	1,099	21.2
Education									
Secondary education or less	13,479	3,439	24.8	2,861	568	18.0	5,004	940	18.2
Post-secondary education	24,426	5,760	22.6	10,299	1,824	17.0	17,348	2,820	15.1
Activity									
Not active	37,905	6,120	23.8	8,108	1,506	17.5	13,901	2,410	16.3
Active	13,382	3,079	22.5	5,052	886	16.6	8,541	1,350	14.8

* Weighted to the Canadian population

TABLE 4. Unadjusted and adjusted (sex and age) ORs and 95% CIs for chronic back pain in relation to smoking

	Daily smokers (present or former)				Occasional smokers (present or former)			
	Unadjusted		Adjusted*		Unadjusted		Adjusted*	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Men								
Age (yr)								
20-29	1.86	1.59-2.18	1.87	1.62-2.17	1.01	0.8-1.24	1.07	0.88-1.3
30-39	1.5	1.34-1.7	1.46	1.29-1.66	0.98	0.83-1.14	0.91	0.77-1.07
40-49	1.58	1.4-1.8	1.69	1.52-1.89	1.13	0.97-1.32	1.17	1.01-1.36
50-59	1.53	1.3-1.75	1.57	1.38-1.79	1.39	1.13-1.59	1.29	1.07-1.54
Women								
Age (yr)								
20-29	1.89	1.66-2.15	1.84	1.6-2.11	1.0	0.85-1.2	1.26	1.06-1.51
30-39	1.43	1.28-1.6	1.36	1.2-1.54	1.09	0.94-1.26	1.24	1.06-1.47
40-49	1.36	1.22-1.52	1.36	1.23-1.51	1.19	1.02-1.38	1.08	0.93-1.26
50-59	1.26	1.15-1.4	1.17	1.05-1.31	1.01	0.89-1.16	0.97	0.82-1.14

* Adjusted for BMI, education and activity status.

ers and non-smokers. Whereas 23.3% of daily smokers had chronic LBP, only 17.2% of occasional smokers and 15.7% of non-smokers had back pain. Daily smokers showed an approximately 50% increase in the prevalence of chronic LBP when compared to non-smokers, whereas occasional smokers had only a slightly increased prevalence of chronic LBP.

Among daily smokers (Table 3), men had a slightly higher prevalence of chronic LBP; the opposite was observed among occasional smokers and non-smokers. We also noticed that daily smoking (Table 3) was associated with an increased risk of chronic LBP. This association was dependent on individuals' age: daily smoking was associated with an approximately 80% increase in the risk of chronic LBP among participants aged 20 to 29 years when compared to non-smokers in the same age range, but the risk was only 24% higher for daily smokers aged 50 - 59 yr compared with their non-smoking age peers.

Univariate regression analyses demonstrated that smoking is a significant predictor of lower back pain ($P < 0.0001$). In the multivariate analysis, sex and age were found to be effect modifiers, BMI and education were found to be confounders ($P < 0.0001$) and physi-

cal activity was found to be a marginally significant risk factor ($P = 0.054$).

Multivariate analysis, which took into account the effect modifiers and adjusted for risk factors (Table 4), demonstrated that, among men who are daily smokers, the increase in the odds of having chronic LBP was dependent on age: those who were aged 20 - 29 yr had an 87% (OR 1.87 with 95% CI 1.62-2.17) increase in their odds of having chronic LBP compared with non-smokers in the same age range, whereas the odds of daily smokers aged 30 - 39 yr increased by only 46% (OR 1.46 with 95% CI 1.29-1.66). For older men who were daily smokers, the increase in the odds of having chronic LBP ranged between 57% to 69% when compared with non-smoking men in the same age range.

A similar pattern was found among women who were daily smokers (Table 4): those aged 20 - 29 yr had an 84% (OR 1.84 with 95% CI 1.60-2.11) increase in their odds of having chronic LBP, whereas for women aged 30 - 49 yr the odds increased by 36% (OR 1.36 with 95% CI 1.20-1.54) and for those aged 50 - 59 yr the increase was only 17% (OR 1.17 with 95% CI 1.05-1.31), when compared with non-smoking women in the same age range.

Occasional smokers (Table 4) did not differ much from non-smokers, and their risk of chronic LBP was still dependent on sex and age. One interesting finding was that among men who were occasional smokers, the increase in the odds of having chronic LBP was statistically significant only among those aged 40 - 59 yr when compared with non-smokers within the same age range. However, for women who were occasional smokers, the increase in the odds was statistically significant only in the younger groups (20 - 39 yr) when compared with non-smoking women in the same age range.

Discussion

In this study of more than 75,000 Canadian adults, daily smoking was associated with increased odds of having chronic LBP; this is consistent with the results of similar studies that were conducted in the USA.^{10,20} The exact mechanism behind this finding remains unclear, but there are several theories that potentially explain it. Smoking reduces bone mineral content, which increases the risk of osteoporosis and microfractures of the trabeculae of the vertebral bodies, causing an increase in degenerative changes in the spine.^{4, 13} Another theory is that smoking increases coughing, leading to increased intradiscal and intra-abdominal pressure, predisposing patients to disc herniation.²⁷ Other theories focus on the fact that smoking causes a reduction in blood flow to the discs and vertebral bodies, which in turn affects the metabolic balance of the discs, leading to disc degeneration that accelerates spinal degenerative processes and makes the spine more susceptible to mechanical deformity and injury.²⁸

We found that the relationship between smoking and the risk of chronic LBP was dependent on the sex and age of the participants. This association is receiving limited attention in the current literature.²⁹ The association was examined using multivariate analysis in which sex and age were found to be effect modifiers.

The effect of smoking was more obvious among daily-smoking men than in women. This observation may be related to the fact that men who are daily smokers tend to smoke more heavily than women and the observed dose-response relationship between smoking and back pain. Deyo and Bass,³⁰ in their study of the influence of smoking on back pain, found that the prevalence of chronic LBP increased with increased pack-years of cigarette smoking. Another theory is that this finding may be related to hormonal differences; this theory should be studied in more detail.

Among daily smokers, the association between daily smoking and the risk of chronic LBP was stronger in younger individuals, a finding that is consistent with those of other studies.^{23, 30} This may reflect the multifactor aetiology of chronic LBP. Since in younger individuals there are few other risk factors for chronic LBP, daily smoking could be a major initiating event, whereas in older individuals many other aetiological factors may be operating (e.g., degenerative spine changes, spinal stenosis, cumulative trauma, tumours);⁵ thus, daily smoking alone would be a relatively less important risk factor in older individuals. The appearance of many other factors for chronic LBP with aging could explain the finding that, as age increases, the prevalence of chronic LBP increases even among non-smokers.

There was a tendency for occasional smokers to have a lower odds ratio of chronic LBP than daily smokers. It is not clear whether this constitutes a sign of causality or whether it means that occasional smokers have a reduced prevalence of chronic LBP for some other reason. This finding was also observed in other studies.^{22,29} Since daily smokers tend to smoke more cigarettes than occasional smokers, the dose-response relationship may actually explain this finding. Deyo and Bass³⁰ found that the prevalence of chronic LBP increased with increased pack-years of cigarette smoking.

We found that occasional smoking increased the odds of chronic LBP among women aged 20 - 39 yr but not in men in the same age range. This finding

was similar to what was observed in a prospective cohort study of adolescents.¹¹ We do not think that there is a difference in the biological effects of smoking between the two genders. One explanation is that women may report a higher prevalence of chronic LBP due to their monthly period. Another explanation could be that men misreport their smoking status.

Another interesting finding is that occasional smoking was associated with an increase in the odds of chronic LBP in men aged 40 - 59 yr but not in women of the same age. Such a finding has never been reported before. This finding could be related to the variation in smoking consumption between the two groups; it should be addressed in future studies before making a definitive conclusion.

Our study demonstrates that the overall point prevalence of back pain is 19.6% and this finding is consistent with the current literature.^{3,10,12} In our study, 46.7% of participants were classified as current or former smokers. This high percentage is not surprising, knowing that smoking prevalence was very high in previous decades. For example, the prevalence of smoking among Canadians was 35% in 1990.²¹

Our study showed that obesity is associated with an increased prevalence of chronic LBP, a finding similar to those previously reported.^{4,30} Obesity increases the load on the lumbar spine, which in turn increases the risk of degenerative changes to the spine, causing chronic LBP. We should keep in mind that this association could be confounded by other unmeasured lifestyle differences between obese and non-obese individuals.

There are few limitations to this study. Because it is a cross-sectional study, the data did not provide information on whether or not smoking preceded the development of chronic LBP. However, there is growing evidence supporting the hypothesis that smoking actually causes chronic back pain.^{11,16} It is possible that our results were affected by recall bias. Given that surveys were conducted by interview and also considering the size of our sample, recall bias was less likely to have a significant impact on our results. This study

is based on self-reported data, which makes it vulnerable to misclassification bias. In this case, smoking status misclassification would most likely result in shifting some non-smokers or occasional smokers to the daily smoker category, a misclassification that would shift the results towards the null hypothesis. Thus, the finding that daily smoking increases the odds of chronic LBP may actually underestimate the real effect of smoking.

In summary, this study demonstrates that smoking is associated with an increased risk of chronic LBP among adults; this risk was modified by age and sex. Also, this study suggests a positive relationship between smoking dose and risk of back pain. These findings suggest that smoking behaviour modification may contribute to reducing back pain in the adult population.

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