

# Strong Association of Smoking with Lumbar Degenerative Spine Disease

Manoj Kumar Sharma\* and Elena Petrukhina

*Nerve Diseases, Medical Genetics and Neurosurgery, Tver State Medical Academy, Tver, 170100, Russia*

**Abstract:** *Study Design:* A prospective case control study.

*Purpose of Research:* To investigate the role of cigarette smoking in the development of early lumbar degenerative spine disease.

*Summary of Background Data:* Smoking is a growing and serious social as well as health problem all over the world. Cigarette smoking has many unfavourable impacts on human spinal column. The increased predisposition to low back pain in individuals who smoke has been well established. Exposure to nicotine has impact on disc metabolism and is believed to reduce the pain threshold of central nervous system.

*Materials and Methods:* The 300 patients included in the study were with a mean age of  $40.6 \pm 6.0$  years who were diagnosed with lumbar degenerative spine disease confirmed by MRI. Among them 162 (54%) patients were females. Patients were interviewed to assess their smoking status/history.

*Results of Research:* In the study it was found that 76.7% patients were smokers. The patient's who smoked; mean duration of smoking was  $17.8 \pm 7.3$  years and mean for cigarettes per day was  $16.3 \pm 7.4$ /day. Among the smokers 60% were between the ages of 30-40 years. The mean of pack year consumed by the patients was  $15.2 \pm 10.2$ .

*Conclusion:* From this study it is evident that cigarette smoking is a leading risk factor for lumbar degenerative spine disease and its association with early onset of lumbar degenerative spine disease. Further research is necessary to establish a temporal relationship between smoking and degenerative spine disease.

**Keywords:** Degenerative spine disease (DSD), smoking, nicotine.

## INTRODUCTION AND BACKGROUND

Disc degeneration is influenced by various factors including age, mechanical, genetic, shear and toxins [1, 2]. Nutrition, metabolic disorders; low grade infection, autoimmune therapy and toxic factors such as nicotine to tobacco use have also been found to be responsible for degenerative changes to the spine [2]. Nicotine impacts disc metabolism by causing vasoconstriction and reduced blood supply to the vertebrae [3]. In vitro studies in cultured bovine cells have established that nicotine inhibits disc cell proliferation and synthesis of the extracellular matrix [4]. Animal studies in rats have shown passive smoking causes down regulation of collagen genes which is a precursor to disc degeneration [5]. It is believed exposure to nicotine reduces the pain threshold of the central nervous system [6]. Studies have suggested that the elevated CO-haemoglobin content in the blood of smokers causes an imbalance of oxidative metabolism and the resultant chronic cough may cause mechanical damage to the disc by increasing intradiscal pressure [7]. Smoking is implicated in speeding up aortic atherosclerosis and stenosis of arteries innervating the spinal column [8, 9].

The objective of this study is to investigate the role of cigarette smoking in the development of lumbar degenerative spine disease.

## RESEARCH METHODOLOGY

### Study Population

The study investigated 300 patients between the ages of 30 and 50 years of age of Tver State Regional Hospital, Russia, who have clinical signs and symptoms of lumbar degenerative spine disease and were diagnosed with lumbar degenerative spine disease confirmed by MRI.

We recruited the patients in consecutive order from the outpatient department.

90% of patients were diagnosed with L4-L5 and L5-S1 disc disease and 10% were with L3-L4 disc disease.

### Inclusion Criteria

Patients diagnosed with lumbar degenerative spine disease for the first time. The patients had the signs and symptoms of degenerative spine disease for less than one year.

\*Address correspondence to this author at the Flat No: 21, House No: 111, Hostel No: 4, Street: St. Peterburg, Tver, 170036, Russia; Tel/Cell: +7 904 026 19 31; E-mail: [saurovjyoti@gmail.com](mailto:saurovjyoti@gmail.com)

All the patients had sciatic pain (back and leg pain). A few patients (25 patients) experienced numbness and altered sensation over the affected dermatome without any motor deficit.

### Exclusion Criteria

Patients identified with: previously diagnosed degenerative disc or spine disease by MRI/CT scan or X-ray examination, previous surgery of lumbar spine, Congenital anomalies of lumbar spine, history of major trauma of lumbar spine, malignancy in the spinal region or any other malignancy, history of infection in the spinal region, serious internal disease such as, hypertension, diabetes mellitus, ischaemic disease of heart and lower limbs, bronchial asthma, BMI over 40, severe liver, renal and haematological disease. Patients with history of exposure to other risk factors like obesity, heavy physical activity (moving or pushing heavy furniture (75 lbs or more), carrying household items weighing 25 lbs or more up a flight or stairs, or shovelling coal into a stove. Standing, walking or walking down a flight of stairs while carrying objects weighing 50 lbs or more).

### Data Collection

Patients diagnosed with lumbar degenerative spine disease, by MRI were interviewed to assess their smoking status/history. The grading of disc degeneration was done as per non modified Pfirman's grading on T2-weighted Mid-sagittal fast spin-echo images. Distribution of patients as per Pfirman's grading with grade 1- 0, grade 2 -105, grade 3 - 135, grade 4- 40, grade 5- 20. To reduce the chances of observer and subjective bias we have taken the patients with duration of pain < 1 year and patients were assessed by independent observers (a neurologist and a radiologist). The whole spine MRI screening was done and degeneration was predominantly confined to lumbar region. No gross changes of other region were noticed in MRI.

### Statistical Analyses

All analyses were conducted using the statistical software package SPSS (version 10.0; SPSS, Inc., Chicago, IL, USA). Statistically significant differences were assumed when  $p < 0.05$ . The regression modelling analysis was also conducted. Chi square test was performed to see the variant significance. Analysis of smoking history was done in cigarettes per day, duration of smoking in years and pack year consumption. The analysis was done in relation to age and gender distribution also.

### Ethical Issues

The ethics committee of the academy approved all the studies and the patients gave their informed consent for participation.

### Results

The 300 patients included in the study were between the ages of 30 and 50 years with a mean age of  $40.6 \pm 6.0$  years, among which 138 patients (46%) were males (mean age of  $39.9 \pm 5.9$  years) and 162 patients (54%) were females with mean age of  $41.2 \pm 6.0$  years. Among these patients 230 (76.7%) were smokers (Table 1).

As per Table 1 the mean (average) number of cigarettes smoke /day stands 16.3 cig/day and average duration of smoking in years was 17.8 yrs. with mean pack year consumption of 15.2 pack years.

Among the smokers, highly significant 138 patients (48.7%) were between the age of 30- 40 years (Table 2).

In the study population out of 300 patients, only 70 (23.3%) patients were non-smokers and among these 62 (88.6%) patients were above the age of 40 years. Only 8 (5.5%) patients were less than 40 years old (Table 2). We have taken the non-smoker group as control group. Our limitation was that we could not get a large control group because of non-availability of patients.

It is found that 20% smoked more than 20 cigarettes per day, of which 63.0% were between the age of 30-40 years of age; 55.7% smoked 10-20 cigarettes per day of which 54.6% were less than 40 years and 24.3% smoked less than 10 cigarettes per day, of which 69.6% were less than 40 years of age (Table 3).

The patients who smoked, 74 patients (32.2%) were smokers for more than 20 years; 106 patients (46.08%) were found to be smoking for 10-20 years, while 50 patients (21.8%) smoked for less than 10 years. In Table 4a the smoking related to age was illustrated and it shows patients aged between 30-40 years smoked an average of 15.6 cig/day for 14.1 years. The patients with age of 41-50years the average stand at 17.3 cig/day for 23.3 years. It was a very significant amount of smoking as shown in Table 6b. The t test and chi-square correlation (Table 4b) was performed for these values it was found to be significant.

Out of 230 smokers, 53.9% were males, and 46.08% were females. For male smoking population mean value for cigarettes/day was 17.7 cig/day and for duration of smoking 18.9 years and for female these were 14.7cig/day and 16.5

**Table 1. Descriptive Statistics of smokers among the total patients**

	Number of Patients (Frequency)	Minimum	Maximum	Mean	Std. Deviation
Age	300	30	50	40.6	6.0
cigar/ day	230	4	35	16.3	7.4
Duration of smoking in years	230	5	35	17.8	7.3
Consumption of cigarettes in pack years	230	1.2	52.5	15.2	10.2

**Table 2. Cross Tabulation of Smoker and Non-smoker with Reference to Age Distribution**

			Smoker	Non-smoker	Total
Age	30-40 yrs.	Frequency	138	8	146
		% Age	94.5%	5.5%	100.0%
		% of group	60.0%	11.4%	48.7%
	41-50 yrs	Frequency	92	62	154
		% Age	59.7%	40.3%	100.0%
		% of group	40.0%	88.6%	51.3%
Total	Frequency		230	70	300
	% Age		76.7%	23.3%	100.0%
	% of group		100.0%	100.0%	100.0%

**Table 3. Distribution of Cigarettes Consumed /Per Day by Smoking Population**

		Frequency	Percentage of Total no. of Patients	Percentage of Smoking Population
Smoker	Upto 10 cig/day	56	18.7	24.3
	11-20 cig/day	128	42.7	55.7
	More than 20 cig/ day	46	15.3	20.0
	Total	230	76.7	100.0
Non-smoker	Total	70	23.3	
Total		300	100.0	

years respectively. 'T' test was done for both frequency of cigarettes consumption per day and duration of smoking with gender distribution it was found a significant number of patients of both the genders smoke a significant amount of cigarettes per day for a long duration (Table 5).

Out of 230 smokers, 53.9% were males, and 46.08% were females. For male smoking population mean value for cigarettes/day was 17.7 cig/day and for duration of smoking 18.95 years and for female these were 14.72 cig/day and 16.53 years respectively. 'T' test was done for both frequency of cigarettes consumption per day and duration of smoking with gender distribution it was found a significant number of patients of both the genders smoke a significant amount of cigarettes per day for a long duration (Table 5).

The consumption of cigarettes was considered in pack year and the mean stands at  $15.2 \pm 10.2$  pack years (Table 1) and 66.1% of smokers smoked  $\frac{1}{2}$ -1 pack year (Table 6a). This is very significant. In Table 6b chi square test was performed and value was significant.

## DISCUSSION

Limited research has been conducted on the relationship between smoking and lumbar degenerative spine disease. Majority of the research focuses on the association between cigarette smoking and development of low back pain. Cigarette smoking has several significant unfavourable effects on the human spinal column. Heavy smokers are

more susceptible to bony degradation resulting in degenerative disease of the spinal column as well increased incidence of vertebral injury. Nicotine has an adverse effect on osteoblastic cells. It not only reduces bone mineral density and blood supply to the bone, but also adversely affects cellular proliferation of osteoblasts, collagen synthesis and cellular metabolism of osteoblasts. Moreover, spinal fusion procedures are often less successful, clinically and radiographically in smokers compared to non-smokers [10]. Although smoking has been associated with low back pain and intervertebral disc degeneration, its significance as a causative factor remains to be elucidated [11, 12]. Russia leads the world in cigarette smoking.

It has been reported that there are 44 million adult smokers in Russia. The prevalence of smoking among males is reported to be 60%, while the figure stands at 20% among females [13].

From the results it is evident that in the sample of patients studied, smoking is strongly associated with degenerative spine disease. It is also found that the early degeneration of disc and spine is quiet rare or uncommon in younger age group than that of smokers. In our study it was found 60% of smokers were of age between 30-40years. It is a serious concern. It was also evident from the result that most of the patients smoked a large number of cigarettes per day for long time (mean pack year  $15.20 \pm 10.25$ ).

**Table-4a. T-Test Group Statistics of Cigarettes Per Day and Duration of Smoking with Age Distribution**

	Age	Frequency	Mean	Std. Deviation	t	Sig. (2-tailed)
cigar/ day	30-40 yrs	138	15.6	7.5	1.6	.093
	41-50 yrs	92	17.3	7.1		
duration of smoking in years	30-40 yrs	138	14.1	5.3	11.5	.000
	41-50 yrs	92	23.3	6.6		

**Table 4b. Correlations with Chi Square of Statistics of Cigarettes Per Day and Duration of Smoking with Age Distribution**

		cigar/ day	duration of smoking in years
Cigar/day	Pearson Correlation	1	.186(**)
	Sig. (2-tailed)	.	.005
	Frequency (smoker)	230	230
duration of smoking in years	Pearson Correlation	.186(**)	1
	Sig. (2-tailed)	.005	.
	Frequency (smoker)	230	230

\*\* Correlation is significant at the 0.01 level (2-tailed).

**Table-5. T-Test Group Statistics of Cigarettes Per Day and Duration of Smoking with Gender Distribution**

	Gender	Frequency	Mean	S. D	t	Sig. (2-tailed)
Cigar/ day	Male	124	17.7	8.1	3.1	.002
	Female	106	14.7	6.2		
duration of smoking in years	Male	124	18.9	7.6	2.5	.002
	Female	106	16.5	6.9		

Keeping in mind of quality of collecting and reporting of data, the patients were not selected from the previously diagnosed group. All the patients were recruited freshly. As the patients were not previously selected, the investigator had control over the quality of collection or reporting of the data.

Studies have demonstrated a significant association between smoking and low back pain after taking into account confounding factors [14, 15], as well as a dose –response relationship between present smoking status and incidence of back pain [16] and likelihood of prolonged back pain [17]. A recent meta-analysis [18] on the association between smoking and low back pain demonstrated that occurrence of low back pain differed with study population, year of publication, thereby indicating that aetiology of low back pain varied with region and ethnic populations.

Battie *et al* (1991) in a study of 20 pairs of identical twins established that disc degeneration was more common (18%) among the individuals who smoked compared to their twins who did not. The study also revealed that the incidence disc degeneration was greater in the upper lumbar vertebrae compared to the lower lumbar spine [19]. Making use of

magnetic resonance imaging of the lumbar spine, it was observed that disc degeneration was elevated in the smokers, although smoking history had no relationship with the incidence of pain. As the degenerative changes were observed across the lumbar spine it was hypothesized that smoking had a systemic effect. Similarly, histological evidence of disc degeneration in rabbits exposed to nicotine levels equivalent to those of heavy smokers also found [20].

It is important to keep in mind the multifactorial aetiology of low back pain while interpreting the results as, it causal factors have been found to have a weak effect in multifactorial diseases [21]. A 14 year prospective study was carried out on middle-aged farmers and established that smokers were 10 times as likely to develop sciatica compared to their non-smoking counterparts [22]. Furthermore, people suffering from respiratory diseases have been found to exhibit an increased association between smoking and low back pain [7, 23] this is attributed to chronic cough and expiratory obstruction that results in mechanical stress on parts of the spinal column, similar to stress due to heavy physical work.

**Table 6a. Cross Tabulation of Cigarette Consumption in Pack Year with Cigarettes/Day**

		Cigarettes/ day			Total	
			Up to 10	11-20	More than 20	
Pack year	< 1/2	Frequency	24	6	0	30
		% within Pack year	80.0%	20.0%	.0%	100.0%
		% within cig/day	42.9%	4.7%	.0%	13.0%
	1/2 - 1.	Frequency	30	100	22	152
		% within Pack year	19.7%	65.8%	14.5%	100.0%
		% within cig/day	53.6%	78.1%	47.8%	66.1%
	> 1	Frequency	2	22	24	48
		% within Pack year	4.2%	45.8%	50.0%	100.0%
		% within Cig/day	3.6%	17.2%	52.2%	20.9%
Total (smoker)		Frequency	56	128	46	230
		% within Pack year	24.3%	55.7%	20.0%	100.0%
		% within Cig/day	100.0%	100.0%	100.0%	100.0%

**Table 6b. Chi-Square Tests of Pack Year with Cigarettes /Day**

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	87.9(a)	4	.000
Likelihood Ratio	79.8	4	.000
Linear-by-Linear Association	64.4	1	.000
No. of Cases (smoker)	230		

(a)- 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.00

Moreover, smoking is a known risk factor for osteoporosis [24, 25], which causes low back pain. Studies have demonstrated an increase in the level of circulating pro-inflammatory cytokines [26, 27], which results in an elevated sensitivity to pain [27]. Smoking may cause degenerative changes to spinal structures by alteration in genetic expression in the intervertebral discs; it down-regulates collagen genes and up-regulates aggrecan and the tissue inhibitor of metalloproteinase-1 genes [5].

The increased predisposition to low back pain among individuals who smoke has been well established [28]. Studies have found that smokers are nearly twice as likely (OR 1.3 – 2.5) to develop low back pain than their non-smoking counterparts [6, 7, 29-31]. The relationship between cigarette smoking and low back pain is found to be dose dependent [9]. It is believed that the presence of confounding factors such as genetic factors, lifestyle and occupational hazards also play a role in the development of degenerative spinal disease [28, 31, 32].

The elevated serum proteolytic activity accelerates degeneration of a previously degenerated neovascularised disc [33]. Moreover, the increased proteolytic activity may

be responsible for weakening of spinal ligaments leading to spinal instability, thereby accounting for the increased likelihood of low back pain among cigarette smokers.

The undesirable effect of cigarette smoking has been demonstrated for outcome of spinal surgery. Cigarette smoking is a risk factor for both non-union and poor clinical outcome in lumbar spine fusion surgery. In posterolateral spine fusion procedures, smoking is associated with reduced fusion rates due to an unfavourable healing environment [34].

For the assessment of lumbar disc degeneration grading system developed by Pffirman et al is extensively used. In this system of grading, Grade I: the structure of the disc is homogeneous, with bright hyperintense white signal intensity any normal disc height. Grade II: the structure of the disc is inhomogeneous, with the hyperintense white signal. The distinction between nucleus and annulus is clear, and the disc height is normal, with or without a horizontal gray bands. Grade III: the structure of the disc is inhomogeneous, with intermittent gray signal intensity. The distinction between nucleus and annulus is unclear, and the disc height is normal or slightly decreased. Grade IV: the structure of the

disc is inhomogeneous, with hypointense dark grey signal intensity. The distinction between the nucleus and annulus is lost; the disc height is normal or moderately decreased. Grade V: the structure of the disc is inhomogeneous, with hypointense black signal intensity. The distinction between nucleus and annulus is lost, and the disc space is collapsed. Grading is performed on T2-weighted Mid-sagittal fast spin-echo images [35].

The modified Pfirrmann grading comprises 8 grades of lumbar disc degeneration. The 8 grades represented progression from normal disc degeneration to severe disc degeneration. Grade 1 corresponds to no disc degeneration while grade 8 corresponds to end stage degeneration [36].

## CONCLUSION

In order to demonstrate a causal relationship between smoking and degenerative spine disease it is essential to establish a progressive or temporal relationship, in which cigarette smoking precedes onset of spinal degeneration. This cannot be demonstrated effectively in a cross sectional study due to ethical restraints. Further research is necessary to establish a temporal relationship between smoking and degenerative spine disease. From this study it is evident that cigarette smoking is a leading risk factor for degenerative spine disease as well as early onset of degenerative spine disease. It is looking obvious that the smoking initiates the early degenerative process by stimulating the degenerative gene in our body, which is responsible for early onset of degenerative spine disease and other degenerative diseases. But there is no concrete evidence of it till now. This topic needs more elaborate discussion as smoking is a major health hazard all over the world

Due to the cross sectional study design, a strong causal relationship between smoking and degenerative spine disease could not be established. A strong association between smoking and degenerative spine disease was found. Another limitation of the study was the absence of large number of literatures. Third limitation was that small control group due to non-availability of large number of non-smoking patients.

## ABBREVIATIONS

S.D	=	Standard deviation.
Cig/day	=	Cigarettes per day.
MRI-	=	Magnetic resonance imaging.
CT-	=	Computer tomography.

## CONFLICT OF INTEREST

The authors confirm that this article content has no conflicts of interest.

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