# The Relationship Between The Lumbar Disc Degeneration And Smoking

*Lomber Disk Dejenerasyonu ile Sigara Kullanımı Arasındaki İlişki*

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**Abstract**
The purpose of this study was to determine whether smoking was related to lumbar disc degeneration. We included 200 patients at the age of between 15 and 70 years, who presented at the neurosurgery polyclinic for lumbago and had lumbar magnetic resonance images, and who had been diagnosed with herniated lumbar disc; the patients' histories, complaints, and whether they smoked were recorded. We excluded those with a systemic disease and did not meet the age criteria. Smoking was found to have no effects on degeneration when compared the groups. We concluded that smoking was not a risk factor associated with lumbar disc degeneration.
**Key words:** Intervertebral disc degeneration, lumbar vertebrae, risk factors, smoking.

**Özet**
Bu çalışmanın amacı sigara kullanımının bel ağrısı ile ilişkisi olup olmadığını belirlemekti.Çalışmamıza, beyin ve sinir cerrahi polikliniğine bel ağrısı nedeniyle başvurmuş olup ve lomber disk hernisi ön tanısı ile Lomber MRG yapılan olgularda, Lomber MRG değerleri pozitif olan, 15-70 yaş arası 200 hasta dahil edildi; olguların öyküleri ,yakınmaları ve sigara içip içmedikleri kayıt altına alındı. Sistemik hastalığı olanlar ve yaş aralığı dışındakiler değerlendirmeye alınmadı.Gruplar karşılaştırıldığında sigara kulanımının dejenerasyon üzerine etkisi olmadığı tespit edildi. Sigara kullanımının lomebr disk dejenerasyonu ile ilişkili bir risk faktörü olmadığı kanısına varıldı.
**Anahtar kelimeler:** İntervertebral disk dejenerasyonu, lomber vertebra, risk faktörleri, sigara içmek.

## Introduction

The lumbago, also known as low back pain, is a clinical disorder that restricts daily activities and affects the life of quality of people. 65% to 80% of world population complains about lower back pain some time in their life [1]. Intervertebral disc degeneration is one of the causes of lumbago. Intervertebral discs begin to degenerate at the moment of human's standing up and walking [2,3]. Disc degeneration usually presented with mechanically changed intervertebral disc cells under excessive stress. However, many extrinsic, intrinsic and genetic factors are known to play a role in disc degeneration in addition to mechanical causes. Systemic diseases, trauma, and genetic factors may lead to degeneration of the disc [4,5].

Recent studies have indicated that exposure to nicotine, a component of cigarette, may result in disc degeneration. However, the mechanism between the cigarette and degeneration is not completely cleared in those studies [6-9]. Some other recent studies have reported the otherwise [10-12].

The present study was intended to investigate that the cigarette could be a risk factor for intervertebral disk degeneration.

## Materials and Methods

We included 200 patients at the age of between 15 and 70 years, who presented at the neuro-

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surgery polyclinic for lumbago and had lumbar magnetic resonans images, and who had been diagnosed with herniated lumbar disc; the patients' histories, complaints, and whether they smoked were recorded. We excluded those with a systemic disease and did not meet the age criteria.

Schneiderman Disc Degeneration classification was used for neuroradiologic evaluation of the patients [6,13]. The degeneration levels of patients at Lumbar 4-5 discs level were examined. Chi Square analysis was performed after reviewing descriptive statistics of variables. SSPS 19.0 program was used to conduct the analyses, and the margin of error was considered to be 5%.

## Results

The study involved 200 patients. The mean age of the patients was 40,34±10.54, and there were 90 males (44,8%) and 110 females (55,1%) (Table 1).

**Table 1.** Distribution table for ages and gender of the patients.

| | | n | % |
|---|---|---|---|
| | Females | 110 | 55.1 |
| Gender | Males | 90 | 44.8 |
| | Total | 200 | 100.0 |
| Age | Min. | Max. | Mean±S.D. |
| | 18 | 62 | 40.34±10.54 |

The age of the patients in the study was categorized into three groups: ranging 18-30 (36 patients, 17,9%), 31-50 (124 patients, 62,1%), and 50 and over (40 patients, 19,9%) (Table 2). As to smoking, the non-smoking group had 114 patients (%57,1), the group smoked less than 10 years had 15 patients (%7,5), and the group smoked for 10 years and over had 71 patients (%35,3) (Table 2).

**Table 2.** Distribution table for age groups, smoking and degeneration of the patients.

| | | n | % |
|---|---|---|---|
| | 18-30 | 36 | 17.9 |
| Age Group | 31-50 | 124 | 62.1 |
| | over 50 | 40 | 19.9 |
| | none | 114 | 57.2 |
| Smoking | less than 10 years | 15 | 7.5 |
| | 10 years and over | 71 | 35.3 |
| | 0 | 25 | 12.8 |
| | 1 | 49 | 24.4 |
| Degeneration | 2 | 68 | 33.8 |
| | 3 | 58 | 27.9 |
| | Total | 200 | 100.0 |

The Modic degeneration scores of the patients are presented in the *Table 2*. Of those that did not smoke, 16,4% were in degeneration group, 24,3% were in degeneration group 2, 28,7% were in degeneration group 2, and 30,4% were in degeneration group 3. Those who smoked less than 10 years were approximately equally distributed into the degeneration groups with 33.3% being in the degeneration group 2, and 20% being in the degeneration group 0. For those who smoked for 10 years and over, the distribution was as follows: 5,8% were in degeneration group 0 and 43,5% were in degeneration group 2. The percentage of those in the Groups 1 and 3 was respectively 26,1% and 24,6% (Table 3). No statistically significant associations were found between smoking and degeneration groups (p>0,05).

**Table 3.** Analysis table between smoking and degeneration of the patients.

| | | | Degeneration | | | | |
|---|---|---|---|---|---|---|---|
| | | | 0 | 1 | 2 | 3 | Total |
| Smoking | none | n | 18 | 28 | 33 | 35 | 114 |
| | | % | 16.5% | 24.3% | 28.7% | 30.4% | |
| | less than 10 years | n | 3 | 3 | 5 | 4 | 15 |
| | | % | 20.0% | 20.0% | 33.3% | 26.7% | |
| | 10 years and over | n | 4 | 18 | 30 | 19 | 71 |
| | | % | 5.8% | 26.1% | 43.5% | 24.6% | |
| Total | | n | 25 | 49 | 68 | 58 | 200 |
| | | % | 13.1% | 24.6% | 34.2% | 28.1% | |

Chi-square= 7.876 p=0.247

## Discussion

Intervertebral disc begins to degenerate with the moment of human's standing up and walking. Many extrinsic, intrinsic and genetic factors are known to play a role in disc degeneration [2,3].

The literature, and the studies involving magnetic resonance imaging to find whether smoking could cause disc degeneration have reported that there is no relationship between smoking and disc degeneration [14,15]. Our study found no relationship either. However, the inci-

dence of degeneration was higher in those who smoked for over 10 years than those who smoked for less than 10 years**.**

The role of lumbago, which is a common complaint in the 60-80 of the society, is significant in loss of labor force. Surprisingly, it is reported that those who smoke are more likely to complain about lumbago on which large studies have been performed. The cause of lumbago is suggested to be smoking-related chronic cough and increased pressure on the disc due to coughing. There are publications supporting our theory that intervertebral disc is poorly supplied because of vascular and hematologic changes in those who have smoked for long years; that smoking results in changes in diffusion capacity; that the possibility of having lumbago following a diskectomy in patients with a history of smoking for over ten years is higher than those who have nor smoked, and that disc degeneration revealed by MRI is greater in smoking individuals [16,17].

The studies supporting that there is a relationship between smoking and lumbago suggest that smoking impairs oxygen transmission due to formation of carboxyhemoglobin [18], leads to vasoconstriction [12] and atherosclerosis of blood vessels, causes disturbance in fibrinolytic activity [19], and adversely affects supply of intervertebral disc because of decreased blood flow and increases the risk of occurrence of lumbago as a result of all these causes [10]. In addition, leucocyte and erythrocyte counts, hemoglobin concentration, and hematocrit value were found to be higher in patients who smoked [20]. It is argued that increased leucocyte count would result in obstruction of small veins which might lead to reduced blood flow and vascular endothelial diseases such as thrombosis [21]. As a result, decreased blood flow reduces the oxygen levels as well as synthesis of proteoglycan and collagen, and all these factors accelerate the degeneration of the discs [22]. It is suggested that nicotine contained in the cigarette produces adverse effects on the disc degeneration [23]. Various studies have showed that nicotine enters the capillary veins of intervertebral discs and goes into the nucleus pulposus through diffusion [24], affects vertebral chondrocytes [6], reduces mineral density of bones [25], inhibits synthesis of collagen [26,27], and reduces DNA synthesis and thus adversely affects cell proliferation [28,29]. Furthermore, it is reported that nicotine leads to intervertebral disc degeneration and results in decreased viscoelasticity of nucleus pulposus [30].

In conclusion; the present study investigated the relationship between smoking and the levels of lumbar vertebral degeneration. As a result, smoking was found to have no effects on degeneration when compared people with lumbar disc degeneration with those who did not have lumbar disc degeneration. However, studies with larger groups of patients as well as molecular studies are required.

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