



Review Article

## Adverse impact of smoking on the spine and spinal surgery

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### ABSTRACT

**Background:** Tobacco smokers and companies are well aware that smoking increases the risks for cancers, vascular morbidity, and early mortality. This is a review of the plethora of adverse effects chronic smoking has on spinal tissues and spinal surgery.

**Methods:** Medline (PubMed) and Google Scholar databases were searched for pertinent literature through keywords related to smoking, spondylosis, and spinal surgery.

**Results:** Smoking accelerates spondylosis by impairing spinal tissue vascular supply through atherosclerosis and thrombosis, while inducing local hypoxia, inflammation, proteolysis, and cell loss. It, thus, compromises disc, cartilage, synovium, bone, and blood vessels. It can lead to early surgery, delayed wound healing, increased surgical site infection, failed fusion, more re-operations, and chronic spinal pain.

**Conclusion:** There is ample evidence to support surgeons' declining to operate on chronic smokers. The need for immediate and permanent smoking cessation and its potential benefits should be emphasized for the patient considering or who has undergone spinal surgery.

**Keywords:** Pseudarthrosis, Smoking, Spinal fusion, Spondylosis, Surgery, Tobacco

### INTRODUCTION

Mainstream cigarette smoke drawn into a smoker's mouth consists of 8% tar and 92% gaseous components and contains thousands of toxic chemical compounds, about  $10^{15}$  free radicals per puff, and the addictive substance, nicotine.<sup>[1,7,13]</sup> Anti-smoking media campaigns usually emphasize the adverse health effects of cigarette smoking as including emphysema and chronic bronchitis, lung and other cancers, and cardiovascular diseases.<sup>[21]</sup> Here, the substantial negative impact of smoking on the spine and spinal surgery is emphasized, as cigarette smoke toxins compromise spinal blood flow and nutritional supply,<sup>[1,9]</sup> accelerate spondylosis,<sup>[12]</sup> and increase other surgical complications such as skin incision necrosis and dehiscence, delayed wound healing, and infection [Table 1].<sup>[15,20,26]</sup>

### METHODS

The literature was reviewed using keyword searches on Medline (PubMed) and Google Scholar search engines. Keywords included smoking, complications of spinal surgery, disc arthroplasty,

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**Table 1:** Adverse effects of smoking on spinal surgery.

Author (yr)	Study design	Characteristics	Statistical method (s)	Morbidity of smoking	Conclusion
Andersen <i>et al.</i> (2001) <sup>[3]</sup>	PS+RS	n=426; lumbar spinal fusion; 1993–1997; avg 2-yr f/u	Chi-square; Mann-Whitney	Nonunion risk increased with history of smoking (OR 2.01; P<0.16); nonunion risk increased in smokers undergoing multi-lvl fusion (OR 3.03; P<0.001)	Smoking doubles risk of nonunion and increases risk of patient dissatisfaction with surgery
Bydon <i>et al.</i> (2014) <sup>[5]</sup>	RS	n=281; 1–2 lvl PLF; 1990–2011	Student’s t; Chi-square; univariate+multivariate	Higher pseudoarthrosis rates in smokers (29.17%) versus nonsmokers (10.92%) undergoing 2-level PLF (P=0.019)	Smoking almost triples rate of pseudoarthrosis in 2-lvl PLF patients
Bydon <i>et al.</i> (2015) <sup>[6]</sup>	RS	n=500; 1–3 lvl lumbar laminectomies; 1990–2012; avg 4-yr f/u	Multiple logistic regression	Increased re-operation rates in single and multi-lvl laminectomy patients (OR 2.06; P=0.04)	Smokers have worse clinical outcomes after lumbar laminectomy versus nonsmokers
Glassman <i>et al.</i> (2000) <sup>[10]</sup>	RS	n=357; 1–2 lvl PILF; 1992–1996; min. 2-yr f/u	Student’s t; Chi-square; ANOVA	Nonunion rate 14.2% in nonsmokers versus 26.5% in smokers (P<0.05). Patients who quit smoking for>6 months after surgery returned to work at 74.6% versus 53.4% nonquitters (P<0.005). Smokers reported less satisfaction with outcome versus nonsmokers (P<0.05)	Smoking>doubles rate of nonfusion in lumbar spine. Smokers who quit for>6 months more likely to return to work. Smokers less satisfied with surgical outcome
Grisdela Jr <i>et al.</i> (2017) <sup>[11]</sup>	RS	Cervical disc degeneration with (n=26,636) or without (n=324,288) myelopathy; 2007–2013	Chi-square	Peak age group for cervical spinal surgery 60–79 yrs in nonsmokers versus 40–59 yrs in smokers (P<0.001). Smokers twice as likely to undergo cervical spinal surgery (P<0.001)	Smoking increases risk of requiring cervical spinal surgery and at an earlier age
Hilibrand <i>et al.</i> (2001) <sup>[13]</sup>	RS	n=190; 2–3 lvl ACDF (n=131) or 1–4 lvl corpectomy with strut graft (n=59); 1973–1992; min. 2-yr f/u	Chi-square; rank-sum	Overall rate of solid union 81% in nonsmokers versus 62% smokers (P<0.02). Fusion in 76% of multi-lvl ACDF nonsmokers versus 50% smokers (P<0.02). Fusion in 93% of corpectomy nonsmokers and smokers. Better outcomes reported in nonsmokers versus smokers (P<0.03)	Lower fusion rates in multi-lvl ACDF smokers. Poorer outcome reporting in smokers.
Kong <i>et al.</i> (2017) <sup>[15]</sup>	MA	26 spinal surgery studies; 1999–2016 (n=67,405)	15 cohort+11 case-control	10.37% increased risk of SSI; range 1.82–20.14%	Smoking increases SSI risk

(Contd...)

**Table 1:** (Continued)

Author (yr)	Study design	Characteristics	Statistical method (s)	Morbidity of smoking	Conclusion
Lau <i>et al.</i> (2014) <sup>[16]</sup>	RS	n=160; 1–3 lvl corpectomy; 2006–2011; 1-yr f/u	Multivariate logistic regression; Chi-square; ANOVA	Current smokers had significantly higher odds of complications (OR 2.87, 95% CI 1.15–3.04; P=0.012), with a larger proportion of infections (P=0.013) versus nonsmokers. Current smokers had higher odds of pseudarthrosis (OR 1.72, 95% CI 1.13–2.63) + longer hospital stays (9.5 days vs. 4.8 days) than nonsmokers	Smoking increases risk of infection, longer hospital stay and pseudarthrosis
Lee <i>et al.</i> (2015) <sup>[17]</sup>	RS	n=1,038; 1–4 lvl ACDF; 1999–2010; avg 4.2-yr f/u	Kaplan-Meier; Cox proportional hazards	Smoking increased risk of re-operation for adjacent segment disease by 1.9 times (95% CI 1.2–3.0; P=0.008)	Smoking doubles risk of surgery for adjacent segment disease
Olsson <i>et al.</i> (2015) <sup>[22]</sup>	RS+PS	n=100; 1–3 lvl ACDF; 2008–2012; avg 2.75-yr f/u	Student’s t; Chi-square; Pearson correlation; questionnaire	Smokers had higher preoperative pain scores versus nonsmokers (6.4 versus 5.0; P=0.02) + reported more severe dysphagia scores versus nonsmokers (1.17 versus 0.54; P=0.02)	Smokers report more severe dysphagia following ACDF
Saeedinia <i>et al.</i> (2015) <sup>[25]</sup>	PS	n=987 spinal surgery patients; various spinal diagnoses/conditions; 2010–2013; 1 month f/u	Multivariate+logistic regression analyses; Chi-square; ANOVA	Current smokers had a surgical site infection rate of 8.3% compared with 1.9% in nonsmokers (P<0.0001)	Smoking is an independent risk factor for SSI. Cease smoking before elective surgeries

ACDF: Anterior cervical discectomy and fusion, ANOVA: Analysis of variance, avg: Average, f/u: Follow-up, lvl: Level, MA: Meta-analysis, min: Minimum, n: Number of patients, PILF: Posterior instrumented lumbar fusions, PLF: Posterolateral fusion, PS: Prospective, RS: Retrospective, SSI: Surgical site infection, yr: Year

disc degeneration, facet joint arthropathy, infection, pseudarthrosis, spinal fusion, spondylosis, vertebral bone, and wound healing.

## RESULTS

### Disc degeneration

The nutritional supply of intervertebral discs, which are nonvascular tissues, depends on diffusion from adjacent vertebral bodies.<sup>[8,9,27]</sup> That supply is compromised by smoking-induced vascular atherosclerosis in both local arteries and arterioles, thus promoting generalized tissue ischemia.<sup>[9]</sup> Smoking also induces a hypoxic and low pH

state in intervertebral discs, leading to a reduction in normal cellular activity that impairs collagen and proteoglycan production, while enhancing enzymatic proteolysis.<sup>[8,9,12]</sup> Smoking’s disruption of cells, extracellular matrix, and the ordered histological architecture in spinal discs contributes to fissures/tears and fibrotic macroscopic hardening of the nucleus pulposus.<sup>[27]</sup> Together, these adverse factors advance the loss of disc height/hydration (desiccation) and promote the onset of earlier and more severe symptomatic lumbar spondylosis. In studies of identical twins where one smoked and the other did not, the smoker was found to have higher disc degeneration and lower bone mineral density (BMD) scores.<sup>[8-10,12,27]</sup>

### Cartilage degradation and facet arthropathy

Smoking alters joint tissue gene expression toward promotion of autoimmunity and chronic inflammation.<sup>[23]</sup> *In vitro* studies show that cigarette smoke extract induces (in a dose- and time-dependent manner) impaired function and viability of chondrocytes found in articular cartilage.<sup>[7]</sup> The rheumatological and orthopedic literature reports increased articular cartilage loss and peripheral joint pain in smokers versus nonsmokers.<sup>[2]</sup> Further, smokers with osteoarthritis also have relatively decreased polymer molecular size and concentration of hyaluronic acid, critical for synovial joint lubrication.<sup>[7]</sup>

### Bone compromise

Osteoblast function, cellularity, bone mineral content, tensile strength, and vertebral body blood supply are all compromised by smoking/tobacco use.<sup>[12]</sup> Smoking increases osteoporosis, avascular osteonecrosis, fractures, and impaired bone formation (e.g., both natural healing and fusion).<sup>[12,24]</sup> Meta-analyses have found that smokers have greater dose-dependent deficiencies in bone mass, with more adverse effects noted in men, and the elderly.<sup>[28]</sup> Further, women smoking one packet of cigarettes per day through adulthood have relatively reduced oestrogen levels and an approximate 5–10% lower BMD by the time they become menopausal, which also typically occurs earlier.<sup>[12,28]</sup>

### Vascular compromise

The gas and tar components of cigarette smoke contribute to oxidative stress attributed to reactive oxygen species that, in turn, contribute to atherosclerosis, increased inflammation, platelet aggregation, and thrombosis.<sup>[1]</sup> Circulating inflammatory pro-oxidative cytokines such as interleukin-6 and tumor necrosis factor-alpha are found to be present at higher levels in smokers versus nonsmokers.<sup>[19]</sup> In the microcirculation, this leads to increased endothelial cell damage with impaired vasorelaxation (e.g., from nitric oxide and prostacyclin signaling compromise), activation of circulating white cells, arteriolar thickening, and increased platelet adhesion.<sup>[18]</sup> Further, smoking directly or indirectly contributes to spinal tissue ischemia and degradation of bone, endplates, the synovia, and discs.<sup>[7-9,12,27]</sup>

### Infection and impaired wound healing

Meta-analyses have shown that smokers have significantly increased rates of spinal surgical site complications including more skin incision necrosis, delayed wound healing, more wound dehiscence, and increased susceptibility to infection [Table 1].<sup>[15,16,20,25,26]</sup> The higher risk of infection is typically attributed to smoking impairing the normal phagocytic

activity of neutrophils and macrophages against, for example, *Staphylococcus aureus* and *Escherichia coli* pathogens.<sup>[26]</sup>

### Earlier operation and increased reoperation rate

Tobacco smokers are more likely to develop symptomatic cervical spondylotic myelopathy, typically warranting spinal surgery approximately two decades earlier than their nonsmoking counterparts.<sup>[11]</sup> Cessation of smoking for those undergoing cervical spine surgery is critical to avoid the two-fold greater incidence for developing “surgical” adjacent segment disease versus nonsmokers.<sup>[4,14,17]</sup> Further, smoking serves as an independent predictor for re-operation following single or multi-level lumbar laminectomies, where re-operations include surgery for other nondegenerative complications (e.g., adjacent segment disease, wound infection, dehiscence, and pseudarthrosis) [Table 1].<sup>[6,14]</sup>

### Smoking increases rates of fusion failure

Smoking is known to impede all stages of bone healing and fusion.<sup>[4,12,14]</sup> Smoking initially inhibits the normal inflammatory response (first stage) and decreases fibroblast and osteoblast proliferation and function (first and second stages).<sup>[4,12]</sup> It then disrupts the normal vascular supply and neovascularization (second stage), while promoting bone’s net resorption instead of its net formation (third stage).<sup>[4,14]</sup> Notably, smokers have a two-fold greater rate of pseudarthrosis following lumbar or cervical fusion surgery.<sup>[3,4,10,13,16]</sup> In addition, lower rates of fusion are encountered in smokers undergoing multi-level posterolateral fusions versus single-level arthrodesis [Table 1].<sup>[5]</sup>

## CONCLUSION

Through a multitude of pathophysiological mechanisms, smoking negatively impacts the structure of the spine and the results of spinal surgery.<sup>[3,6,10,11,13,14,22]</sup> Patients anticipating undergoing spinal procedures should therefore quit smoking immediately and permanently.<sup>[3,4,10,14,25]</sup>

### Declaration of patient consent

Patient consent not required as patient identity not disclosed or compromised.

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### Conflicts of interest

There are no conflicts of interest.

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