Updates on Smoking and Low Back Pain

This review article highlights current knowledge on the association between low back pain and smoking, with an emphasis on the role of nicotine.
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Volume 14, Issue #10

An estimated 1 in 5 adults in the United States uses tobacco products every day or some days. This equates to 50 million people and 21.3% of the overall population.1 Smoking is more common among men (26.2%) than women (15.4%) and is most common among adults aged 25 to 44 years (25.2%).1 Tobacco use remains the leading cause of preventable death in the United States, accounting for more than 480,000 deaths each year.2 In addition, more than 16 million Americans suffer from a smoking-related disease, including cancer, and respiratory and vascular diseases.2

Interestingly, the prevalence of tobacco use is nearly 2-fold higher among patients with chronic pain.3-5 The reasons for this increased prevalence are believed to be related to many factors, encompassing both behavioral and biological mechanisms. This review will cover current knowledge on the biological mechanisms and pathophysiology behind the relationship between smoking and chronic pain. The behavioral mechanisms are discussed in a Q&A with Joseph W. Ditre, PhD [3].
Cigarettes contain many compounds that produce physiologic effects, but this review focuses on nicotine, which is the most widely studied of these compounds and is believed to play a role in pain modulation. Nicotine has analgesic properties that, at first, can help relieve acute pain. However, over time, nicotine can alter pain processing and contribute to the development of chronic pain and greater pain intensity. This paradox is an important aspect of both acute and chronic pain management for patients who use tobacco products.

**Mechanism of Nicotine Action**

A variety of factors are believed to contribute to the analgesic effects of short-term exposure to nicotine.

First, nicotine is an agonist of nicotinic acetylcholine receptors (nAChRs), which are found throughout the peripheral and central nervous system, and notably in brain regions associated with pain transmission, such as the dorsal horn, locus ceruleus, and thalamus. Specifically, nicotine acts on the α3β4 ganglion type in the autonomic ganglia and adrenal medulla, and the α4β2 nicotinic receptors in the central nervous system. The increased binding of nAChRs produces central antinociceptive effects that activate the spinal cord descending pain-inhibitory pathways, resulting in discharge of epinephrine from the adrenal medulla and catecholamines from sympathetic nerve endings.

Activation of nAChRs also potentiates the release of other neurotransmitters, such as dopamine, that play a role in nicotine-mediated analgesia. This increased stimulation of dopamine also activates the mesolimbic dopamine reward system, which increases the importance of incentive cues associated with nicotine use. Through this reward system, painful stimuli can become a conditioned cue for smoking. Interestingly, activation of nAChRs is similar to activation of opioid receptors in that both stimulate the release of dopamine in the nucleus accumbens, which mediates the rewarding effects of nicotine and plays a role in pain perception.

Second, the antinociceptive effects of nicotine also may be mediated by activation of endogenous opioid systems. Smoking stimulates the release of beta-endorphins, which are endogenous opioid polypeptide compounds that are similar to opioids in their analgesic effects. The more cigarettes smoked per day, the higher the plasma concentration of beta-endorphins.

Third, smoking causes changes in the neuroendocrine system that could modulate pain perception. In general, the stress response (sympathetic and hypothalamic-pituitary-adrenal [HPA] activation) causes a decrease in pain perception. However, this stress response is blunted in chronic smokers, possibly because of attenuated modulation of endogenous opioids on the HPA.

Fourth, pressor actions on the cardiovascular system have been hypothesized to play a role in the analgesic effect of nicotine. Some studies suggest that passive smoking and smoking cigarettes increases blood pressure, which in turn has been linked to reduced pain sensitivity.

Other proposed mechanisms for the analgesic effects of nicotine include attentional narrowing, release of norepinephrine and serotonin, γ-aminobutyric acid (GABA) receptor activity, regulation of inflammatory responses, and suppression of pain-related evoked potentials.

While all of these analgesic effects of nicotine occur with brief exposure to nicotine, chronic nicotine exposure is linked to nAChR desensitization and tolerance and changes in the endogenous opioid system that may alter pain processing.

**Smoking and Chronic Pain Studies**

It is important to differentiate the effects of nicotine in experimental versus epidemiologic studies. While
experimental studies suggest an analgesic and antinociceptive effect of tobacco use in acute pain, epidemiologic studies point to a causal association between smoking and chronic pain.

**Experimental Studies**

Experimental studies point to analgesic and antinociceptive effects in acute pain. For example, in early research by Tripathi et al, the researchers assessed the analgesic effect of nicotine by measuring tail-flick latency in rats and mice. Brain levels of nicotine reached a maximum at 10 minutes, whereas antinociception was maximal at 2 minutes. Tachyphylaxis to antinociception developed in 10 minutes and lasted up to 14 hours. In a 1998 study, Jamner et al found that nicotine increased pain threshold and tolerance ratings of men but had no effect on pain ratings of women. In 2004, Flood et al studied the effects of intranasal nicotine for postoperative pain treatment. They found that patients treated with nicotine reported lower pain scores during the first hour after surgery and less pain 24 hours after surgery, and used half the amount of morphine as the control group.

While short-term exposure to nicotine appears to have antinociceptive effects, chronic exposure to nicotine may change pain perception due to receptor desensitization. Indeed, studies suggest that nicotine withdrawal increases pain sensitivity and blunts stress response in chronic smokers in experimental pain tests, and that this effect drives excessive nicotine use via corticotropin-releasing factor signaling pathways.

**Epidemiologic Studies**

While an early landmark meta-analysis by Leboeuf-Yde did not show a consistent statistically significant positive association between smoking and low back pain (LBP) in studies published between 1974 and 1996, a resurgence of interest in this issue has produced more research in this field. Numerous recent studies have demonstrated an association between smoking and the prevalence of a variety of chronic pain conditions. In addition, chronic pain patients who smoke have greater pain intensity than those who do not smoke.

In a study involving 10,916 patients, Ekholm et al found that cigarette smoking was significantly increased in individuals suffering from chronic pain; in opioid users, smoking was further increased. In addition, in a well-organized survey involving 73,507 people (aged 20-59 years), Alkherayf et al found a higher prevalence of LBP in daily smokers compared with nonsmokers (23.3% vs 15.7%; P<0.0001). This association was statistically significant in all age groups and genders, and was strongest in younger age groups. The data suggested a positive correlation between the smoking dose and the risk for LBP.

Further evidence of the link between smoking and pain chronicity is found in a study of 6,092 women over the age of 18 years reported by Mitchell et al. Women who smoked daily had a 2-fold higher prevalence of chronic pain compared with women who never smoked. In addition, women who were “some-day” smokers had a 1.8-fold increased prevalence of chronic pain.

Smoking was linked to an increased risk for transitioning from subacute back pain to chronic back pain in a 2014 study by Petre et al. Brain imaging in the study participants suggested that the risk for development of chronic back pain was linked to corticostratal circuitry involved in addictive behavior and motivated learning in the nucleus accumbens and medial prefrontal cortex.

While the etiology of chronic pain is multifactorial, these data suggest that smoking is a risk factor for development of chronic pain.

**Nicotine-Induced Structural Changes Might Contribute to Chronic Pain Conditions**

In addition to altering pain processing, smoking can contribute to structural changes that may increase
the risk for chronic pain conditions. For example, smokers are at increased risk for osteoporosis, fracture, lumbar disc disease, impaired wound healing, muscular damage, and impaired healing from spinal fusion, possibly because of impaired oxygen delivery to tissues and accelerated degenerative processes.

**Conclusion**

Although clinicians barely have time to finish their medical records, this should not preclude them from talking to their pain patients about smoking cessation. We must dedicate time to this task, provide our patients with educational materials, and keep the door open to bring up the subject on subsequent follow-ups. In a well-designed cohort study, Kaye et al showed that persistence on the part of the physician can significantly reduce both the amount and prevalence of nicotine consumption among pain patients in a relatively short amount of time. Given the impact of smoking on pain chronicity, it is imperative that physicians implement strategies to help their patients stop smoking.

**References:**


View Sources [5]


First published on: December 1, 2014

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[5] #fieldset