Clin Orthop Relat Res (2017) 475:903–905 / DOI 10.1007/s11999-016-5217-1

Clinical Orthopaedics and Related Research®
A Publication of The Association of Bone and Joint Surgeons®

Published online: 3 January 2017

CORR Insights

CORR Insights®: Exposure to Secondhand Smoke Impairs Fracture Healing in Rats

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Where Are We Now?

We do not need double-blind crossover randomized clinical trials to understand that smoking causes cancer. Though we are not "lung doctors or cancer doctors" (as I tell my patients), we understand that when the preponderance of clinical results—along with experimental and mechanistic data—strongly indicate a detrimental affect, we scientifically accept those conclusions and use them to guide and treat our patients.

Following that paradigm, but focusing on musculoskeletal tissues, multiple systematic reviews have shown that smoking has negative consequences in the healing of ligament, cartilage [8], tendon, labrum [13], and specifically relevant to the paper by Santiago and colleagues, bone [11, 14]. Mechanistically, we have come to understand the osteobiologic underpinnings of some of the effects of cigarette smoke, including its modulation of the osteoprogenitor population [1], preosteogenic chondrogenesis [3], and osteoblastic gene expression [12]. The conclusion is clear: Smoking is bad for the bone.

While surgeons strive to decrease smoking in their patients with fractures in an attempt to avoid complications and improve healing, we must also consider the issue of secondhand smoke, which is even less under our control. Among the many undesired compounds in secondhand smoke are polycyclic aromatic hydrocarbons, which have been implicated in specific-receptor mediated loss of bone mass [7], and likely impair callus generation in fractures [9].

Santiago and colleagues conclude that if you smoke cigarettes, or are close enough to it to inhale it, you are at risk. The consistency in the results only enhances their conclusion—callus maturity, density, and mechanical resistance were all negatively affected by secondhand smoke. A more clinically relevant finding in their study showed that exposure to secondhand smoke before the fracture was less detrimental to the patient than exposure before and after the fracture. This is a compelling scientific rationale to strongly encourage cessation at the time of injury, which is typically the orthopaedic surgeon's first encounter with a patient.

In other words, being a nonsmoker is best, but stopping after the fracture still is helpful.

Where Do We Need To Go?

Several topics pertaining to tobacco smoke and fractures call for further

This CORR Insights® *is a commentary on the article* "Exposure to Secondhand Smoke Impairs Fracture Healing in Rats" *by Santiago and colleagues available at:* DOI: 10.1007/s11999-016-5184-6.

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This *CORR* Insights® comment refers to the article available at DOI: 10.1007/s11999-016-5184-6.

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CORR Insights

elucidation. First, while the results from Santiago and colleagues provide a rational association between secondhand smoke and negative effects on fracture healing, they do so in a controlled experimental setting. We do not yet know whether secondhand smoke, as inhaled by people in real life, is in fact linked to poor fracture healing.

Second, we do not clearly know the relative negative contributions of the components of tobacco smoke (first or secondhand) such as polycyclic aromatic hydrocarbons or the active stimulant ingredient of nicotine. This may become increasingly important as marketing efforts for, and the popularity of, nicotine-containing smokeless products increase; it also presents a dilemma for cessation efforts as we do not clearly know how nicotine from a gum or a patch, without the rest of tobacco smoke, may affect fracture healing.

Third, our data (including those from the current study) do not provide a robust dose-response relationship. Does some reduction help or is there a threshold that must be crossed? Is that threshold essentially zero, including secondhand smoke? Answers to these questions will improve our ability to properly counsel and treat our patients.

That said, this information would be much less necessary, if we could get all of our patients to quit smoking (except perhaps the understanding of inhaled nicotine, should this become a popular independent "habit" rather than a proxy for cigarette smoking). That brings me to another important place for us to go: Leading our patients with fractures to smoking cessation.

How Do We Get There?

We have amassed enough good scientific evidence to conclude that eliminating tobacco smoke from our patients with fractures will help them heal, and will save medical-economic resources. But it is beyond the scope of this commentary to delineate the scientific experiments and clinical studies to resolve the three issues mentioned in the section above. Rather, I will consider the larger problem at hand: Getting our patients with fractures to stop smoking. Here, we not only have a challenge, but an opportunity.

Consider that for some of our patients, hospital admission is part of the care of the fracture; this generally provides temporary disruption of smoking, which has been associated with cessation [6]. Further, the acute event of fracture and surgeon intervention may provide a "teachable moment," which has been used successfully to improve healthy-heart practices after myocardial infarction [4, 5], and to promote smoking cessation within other hospital settings [2]. Consistent with the Health Belief Model for healthcare change, McBride and colleagues [10] have formalized a model to study such interventions. For example, an acute fracture can become a teachable moment for smoking cessation because it: (1) Increases expectation of personal risk, (2) prompts a strong emotional response, and (3) redefines self-concept.

Our real challenge is to use the lessons learned from others regarding healthcare behavior change, combine them with insights gleaned as orthopaedic surgeons to further understand the critical issues, and help our fracture patients quit smoking.

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