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Smoking and Dose Dependent Early Effects of Nicotine on Bone Mechanical Properties and Histology

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SMOKING AND DOSE DEPENDENT EARLY EFFECTS OF NICOTINE ON BONE MECHANICAL PROPERTIES AND HISTOLOGY

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INTRODUCTION

In the United States, about 50 million adults are smokers; smoking has been associated with debilitating bone disease. In a study done by Daniell (1), most people who showed symptoms of osteoporosis before the age of 65 were smokers. Lappe (2) and Friedl (3) found that female army recruits who developed stress fractures were more likely to report current smoking or past smoking habits. Nicotine has been implicated as the agent responsible for bone loss and reduced healing rates in spine fusion (4). However, a recent study done by France et al (5,6) showed that nicotine either helped or did not reduce spine fusion rates. An additional study done on rats to see if nicotine had any effect on bone that had not undergone surgery showed that nicotine did not weaken the strength of the bone (7). Few studies have been done to measure the effect of smoking on bone's mechanical properties. In this study, the effect of smoking on bone's ability to resist crack growth (i.e. fracture toughness) was examined.

METHODS

Twenty-six skeletally mature adult (24- 29 month) New Zealand white rabbits (male retired breeders) were obtained and randomly divided into four groups. This study was done in conjunction with another study to determine the effect of nicotine on spine fusion. Each group received a single level posterolateral, intertransverse process fusion with autologous iliac crest bone, and sacrificed 5 weeks later. The groups were control (n=5), nicotine patch (10.5 mg)(n=9), and two smoking groups (n=12). The first smoking group only received cigarette smoke for four weeks starting one week after the surgery. The second smoking group received the cigarette smoke for all five weeks starting immediatey after the surgery. The smoke was delivered via a smoking chamber. A mode I compact tension specimen was made from each rabbit tibia (Figures 1). The control, 10.5 mg patch and the two smoking groups were compared using ANOVA. JMP statistical software was utilized. Significance was set at $p < 0.05$.

RESULTS

The fracture toughness values were significanty lower for the five week smoking group compared to the control group and the four week smoking group. There was also a close to significant difference (p=.062) in the nicotine group compared to the five week smoking group (Figure 2). There was a close but non-significant (p = .01115) difference between the porosity of the control group and the five week smoking group (Figure 3). The power analysis indicated that the number of rabbits needed in each group to determine significant difference was 16. The average pore radius size was significantly (p< .05) different between the control and four week smoking groups compared to the five week smoking group (Figure 4).

DISCUSSION

This study suggested that the nicotine had no effect on weakening bones' fracture toughness; however, cigarette smoke does. A cigarette contains over 4,000 chemicals; therefore, some agent other than nicotine may be responsible for the effects of smoking on bone. For example, a study done on ovariectomized rats with estrogen replacement, using Polycyclic Aromatic Hydrocarbons (PAH), showed a decrease in BMD and in both vertebral compression strength and three point bending test failure load (8). It still remains to be shown that nicotine is the agent in cigarettes that is responsible for debilitating bone disease found in smokers.

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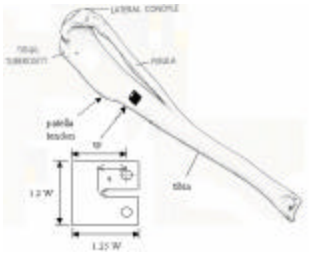

Figure 1: Location of the fracture toughness specimen on the tibia with adaptation with CT specimen (9)

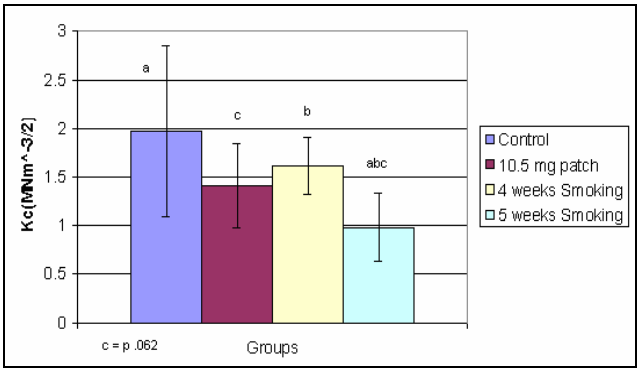

Figure 2: Fracture Toughness

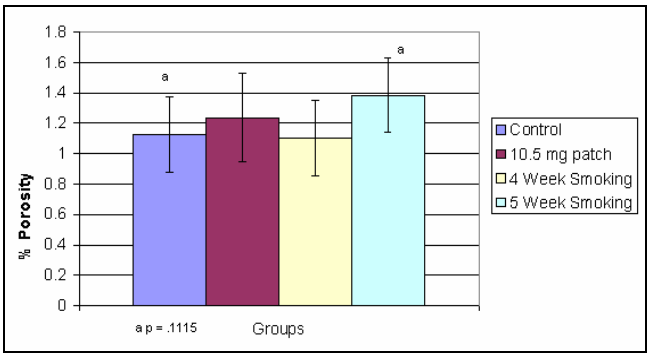

Figure 3: Porosity

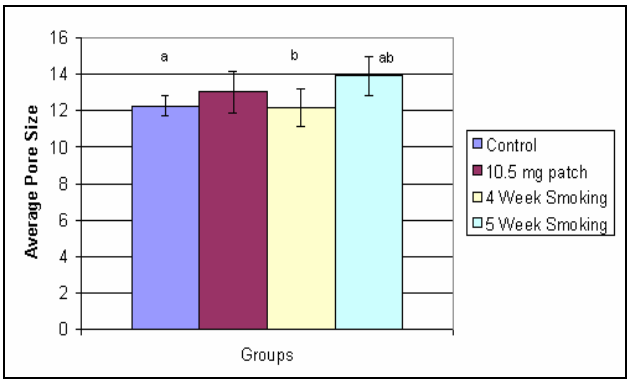

Figure 4: Average pore radius