

#### **Tobacco and Bone Health**

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Cigarette smoking is commonly identified by orthopedic trauma surgeons as one of the factors to cause delayed union and nonunion.

- Tobacco has been implicated as a major factor for the development of Osteoporosis.
- Smoking has been found to decrease oxygen levels in the cutaneous and subcutaneous tissues leading to poor wound healing.
- Nicotine has been found to decrease vascularization at fracture sites, increasing the chances for the development of osteomyelitis.

#### Introduction

- Bone mineral density,
- Lumbar disc disease,
- The rate of hip fracture,
- The dynamics of bone and wound healing.

# **Smoking Adversely affects:**

A review of multiple studies into the adverse effects of tobacco use on fracture repair revealed that there are several hypotheses as to the mode of action:

- A reduced blood supply,
- High levels of reactive oxygen intermediates,
- Low concentrations of antioxidant vitamins
- Effects of nicotine on arteriole endothelial receptors bringing vasoconstriction

## . Nicotine in high doses is directly toxic to proliferating osteoblasts.

• Nicotine seems to affect the early revascularization of the fractured bone, probably through down-regulated gene transcription of fibroblast growth factor, vascular endothelial growth factor, and bone morphogenetic protein cytokines known to be important to angiogenesis and osteoblast function.

## Tobacco has negative effects on fracture healing in diaphyseal fractures

- Smoking is believed to affect bone healing in diaphyseal fractures of Femur, Tibia and Humerus.
- Other diaphyseal fractures may be affected too but this has not been formally demonstrated yet, except for the negative effect of tobacco in healing of scaphoid nonunion and of lumbar arthrodesis.

# Bones affected by smoking

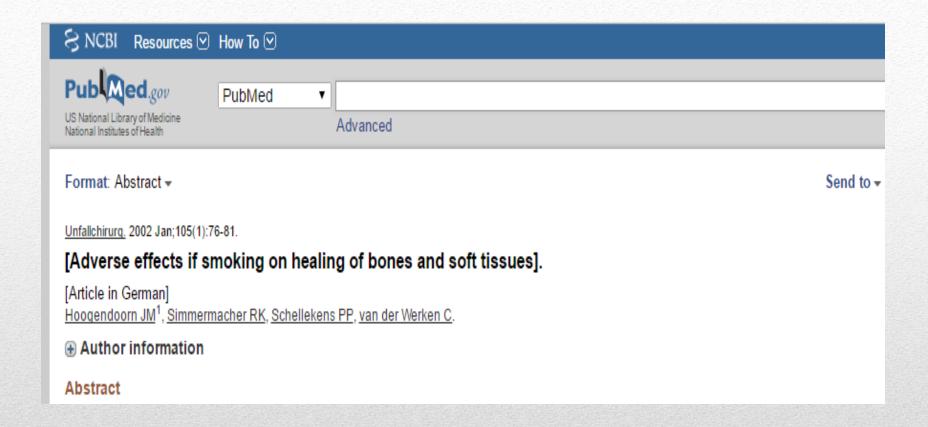
- Delayed acute fracture healing.
- Failure of nonunion treatment.
- Failure of bone healing associated with spinal fusion
- Failure of bone healing associated with osteotomies.

Reference (1-5)

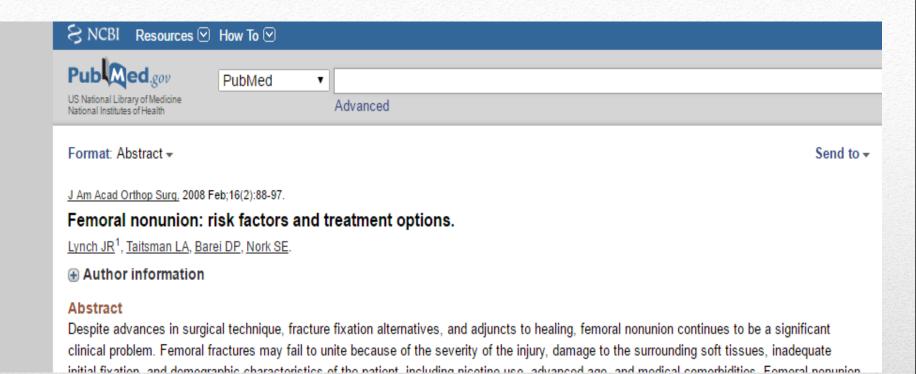
# Evidence shows that smoking is linked with:



# Even a history of prior smoking and exposure to second-hand smoke has been shown to delay bone healing: Castillo RC et al



Not only does smoking affect bone healing, it also increases the risk of other complications such as acute infection and osteomyelitis: Hoogendorn JM



## Smoking is a risk factor for femoral nonunions: Lynch J B et. al.



# Smoking decreases efficiency even in the bone generation machine: Ilizarov distraction osteogenesis: McKee MD



# Smoking delays radiological bone healing in all bones, foot bones, spinal fusions and also at surgical osteotomy sites: Kranitz KW et al

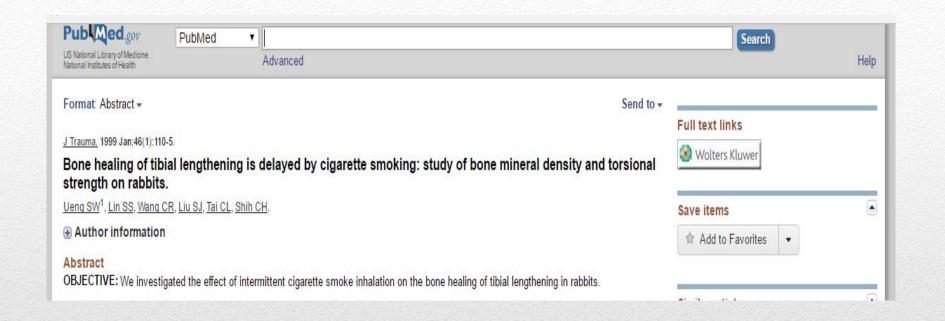
- Vasoconstrictive properties of nicotine inhibit tissue differentiation and the normal angiogenic responses in the early stages of fracture healing.
- Nicotine directly interferes with osteoblast function.

• Referrences: 5-8

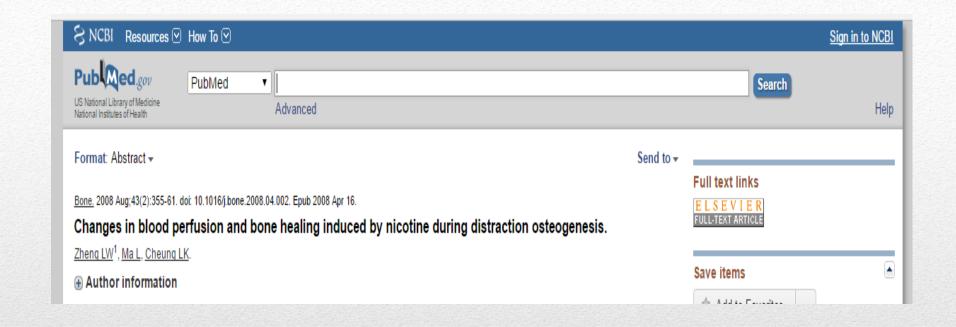
#### **Animal studies**



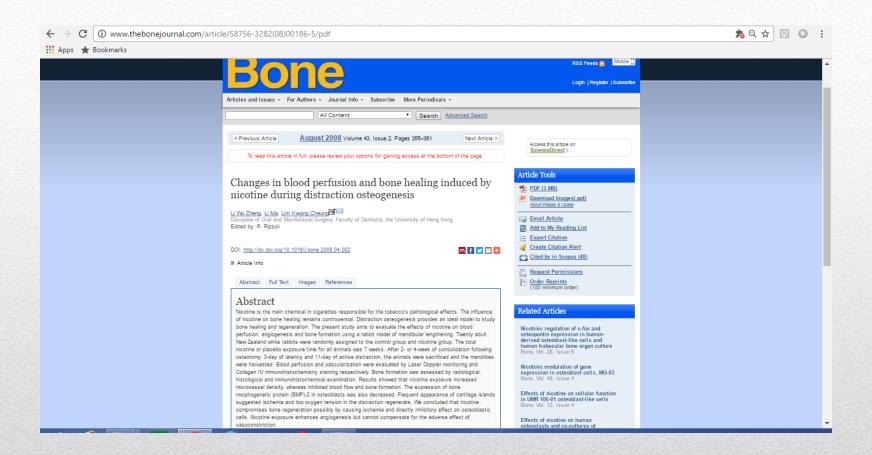
## Revascularization of the bone graft in a Rabbit model was found to be delayed by nicotine: Daftari T K et al



## Tibial lengthening in a Rabbit model was delayed by cigarette smoking: Ueng SW



Nicotine compromises bone regeneration possibly by causing ischemia and direct inhibitory effect on osteoblastic cells: Zheng L W



Nicotine exposure enhances angiogenesis but cannot compensate for the adverse effects of vasoconstriction: Zheng L W

### BONE MORPHOGENETIC PROTEIN MRNA EXPRESSION IN HUMAN PERIOSTEUM OF FRACTURED BONES

C. Chassanidis, Z. Dailiana, P. Kollia, T. Koromila, K. Malizos, S. Samara, S. Varitimidis Published 20 September 2010

Article

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#### Current Issue

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#### Abstract

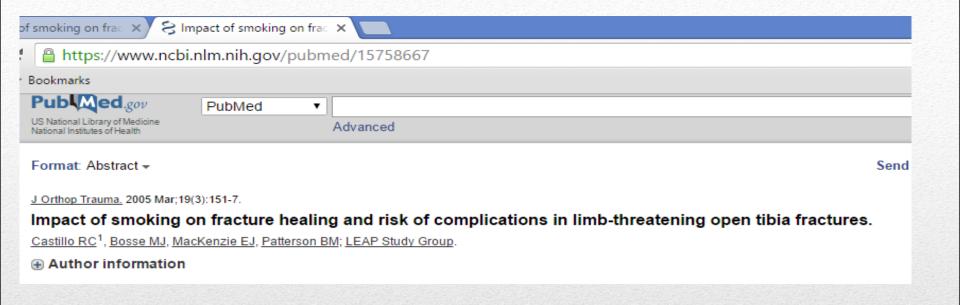
Introduction: Periosteum is a tissue with pluripotential mesenchymal cells (MSCs).

During fracture repair several growth factors are released from periosteum, including bone morphogenetic proteins (BMPs), which induce the differentiation of bone marrow stromal cells towards the osteoblastic lineage, therefore increasing the pool of mature bone forming cells and enhance the differentiated function of osteoblasts.

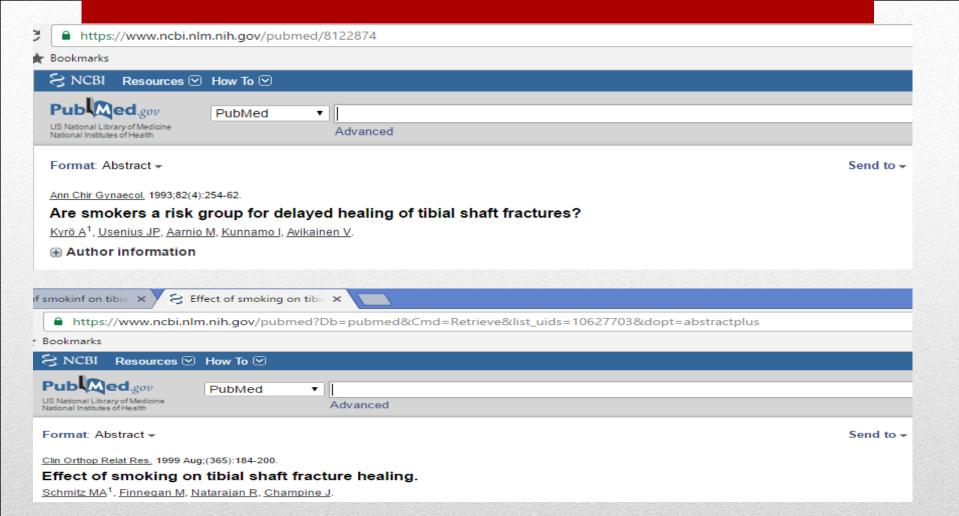


Recent human data, where samples of fractured and non-fractured bones from smokers and nonsmokers were assayed for BMP-2, -6, -4, and -7 using polymerase chain reaction, indicate that smoking reduces periosteal bone morphogenetic protein (BMP) gene expression

#### **Human Evidence**



• Smokers with open tibia fractures treated with intramedullary (IM) nails were found, in a prospective study by Castillo et al., <sup>49</sup> to be 37% less likely to achieve union and 3.7 times more likely to develop osteomyelitis than nonsmokers.



Smoking was found to delay healing in a dosedependent manner after closed management of tibial shaft fractures.



• In the setting of Ilizarov limb reconstruction, McKee et al. demonstrated that smoking was associated with multiple complications. The overall complication rate was over three times higher rates of persistent infection, nonunion, and amputation.

- Despite being one of the few risk factors that is potentially modifiable, smoking cessation in the face of the stresses associated with acute fracture is exceedingly difficult.
- Despite these challenges, it is prudent to advocate and support smoking cessation in all patients with fractures at risk for nonunion and in those facing nonunion repair.
- Given the direct adverse effects of nicotine on bone healing, nicotine supplementation (e.g., nicotine patch) as part of a smoking cessation program should be avoided.

#### Recommendation



Animal data supports the fact that transdermal nicotine patch leads to nonunion and decreased mechanical strength of healing fractures: Donigen J A



Approximately 50% of smokers return to their habit. It is best for healing of bone and soft tissue if they can abstain while being treated for their injury.

