This Paper Will Change Your Practice – It Changed Mine. New Information That You Need to Know: Basic Science

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The burden of musculoskeletal disease has surpassed cardiovascular disease as the major health burden in the world, and bone fractures contribute substantially to the overall burden of musculoskeletal disease. In the US, there are over 600,000 fractures per year with a substantial number of these fractures exhibiting delayed healing or non-union. The gold standard to stimulate bone union been autologous bone grafting, which although generally good, remains problematic due to limited graft supply, donor site morbidity, and potential complications. Therefore, understanding mechanisms of normal fracture healing to develop effective therapies to treat patients is imperative.

Generally, fracture repair occurs through two processes: direct bone (intramembranous ossification) and the formation of bone through a cartilage intermediate (endochondral ossification). With the exception of an initial inflammatory process, adult healing is similar to that observed during bone development. Previous work suggested that in adult repair, stem cells in the periosteum and endosteum give rise to chondrocytes that form the soft callus during endochondral ossification, and subsequently, during vascular invasion of the cartilage callus, osteoprogenitor cells are delivered to the fracture site to form new bone.

Recent findings, however, challenge this assumption. This presentation will summarize an article (Hinton et al., 2017) that reviews data suggesting that suggests that a significant number of bone cells are derived directly from the transformation of chondrocytes. They discuss other recent findings that show demonstrate this concept (see other references below).

These finding are potentially paradigm-shifting. Traditionally, strategies for stimulating bone repair seek to stimulate the process of direct bone formation. However, given that the majority of long bone fractures heal with some degree of callus formation (with the exception of those treated with absolute stability) and bone is formed directly through the transformation of chondrocytes, successful fracture repair therapies might target the endochondral rather than intramembranous ossification process. These findings have the potential to affect the way fracture healing, bone incorporation, and bone tissue engineering strategies are developed and employed.

Reference:

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