Lithium and bone healing

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Researchers have described a novel molecular pathway that may have a critical role in bone healing and have suggested that lithium, which affects this pathway, has the potential to improve fracture healing.

The study, led by Benjamin Alman from the Hospital For Sick Children, Toronto, investigated the role of the beta-catenin signaling pathway, which activates T cell factor -dependent gene transcription, and which is known to have a key regulatory role in embryonic skeletal development.

By studying mice with fractures the researchers were able to show that beta-catenin-mediated gene transcription was activated in both bone and cartilage formation during fracture repair. In mice that lacked beta-catenin fracture healing was inhibited, whereas in mice expressing an activated form of beta-catenin bone healing was accelerated. Treating mice with lithium activated beta-catenin in the healing fracture, but healing was enhanced only when treatment was given after the fracture occurred, rather than before.

These results show that that beta-catenin functions differently at different stages of fracture repair. Although the relevance of this study to human fractures remains to be determined, activation of beta-catenin by lithium treatment has the potential to improve fracture healing, but probably only when given in later phases of fracture healing.

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