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Sclerostin antibody reduces long bone fractures in the oim/oim model of osteogenesis imperfecta

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Résumé en anglais	Osteogenesis imperfecta type III (OI) is a serious genetic condition with poor bone quality and a high fracture rate in children. In a previous study, it was shown that a monoclonal antibody neutralizing sclerostin (Scl-Ab) increases strength and vertebral bone mass while reducing the number of axial fractures in oim/oim, a mouse model of OI type III. Here, we analyze the impact of Scl-Ab on long bones in OI mice. After 9 weeks of treatment, Scl-Ab significantly reduced long bone fractures (3.6 ± 0.3 versus 2.1 ± 0.8 per mouse, $p < 0.001$). In addition, the cortical thickness of the tibial midshaft was increased (+42%, $p < 0.001$), as well as BMD (+28%, $p < 0.001$), ultimate load (+86%, $p < 0.05$), plastic energy (+184%; $p < 0.05$) and stiffness (+172%; $p < 0.01$) in OI Scl-Ab mice compared to OI vehicle controls. Similar effects of Scl-Ab were observed in Wild type (Wt) mice. The plastic energy, which reflects the fragility of the tissue, was lower in the OI than in the Wt and significantly improved with the Scl-Ab treatment. At the tissue level by nanoindentation, Scl-Ab slightly increased the elastic modulus in bones of both OI and Wt, while moderately increasing tissue hardness (+13% compared to the vehicle; $p < 0.05$) in Wt bones, but not in OI bones. Although it did not change the properties of the OI bone matrix material, Scl-Ab reduced the fracture rate of the long bones by improving its bone mass, density, geometry, and biomechanical strength. These results suggest that Scl-Ab can reduce long-bone fractures in patients with OI.
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