Glucose-dependent insulinotropic polypeptide (GIP) receptor deletion leads to reduced bone strength and quality.

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Résumé en anglais	Bone is permanently remodeled by a complex network of local, hormonal and neuronal factors that affect osteoclast and osteoblast biology. In this context, a role for gastro-intestinal hormones has been proposed based on evidence that bone resorption dramatically falls after a meal. Glucose-dependent insulinotropic polypeptide (GIP) is one of the candidate hormones as its receptor, glucose-dependent insulinotropic polypeptide receptor (GIPR), is expressed in bone. In the present study we investigated bone strength and quality by three-point bending, quantitative x-ray microradiography, microCT, qBEI and FTIR in a GIPR knockout (GIPR KO) mouse model and compared with control wild-type (WT) animals. Animals with a deletion of the GIPR presented with a significant reduction in ultimate load (--11%), stiffness (-16%), total absorbed (-28%) and post-yield energies (-27%) as compared with WT animals. Furthermore, despite no change in bone outer diameter, the bone marrow diameter was significantly increased and as a result cortical thickness was significantly decreased by 20% in GIPR deficient animals. Bone resorption at the endosteal surface was significantly increased whilst bone formation was unchanged in GIPR deficient animals. Deficient animals also presented with a pronounced reduction in the degree of mineralization of bone matrix. Furthermore, the amount of mature cross-links of collagen matrix was significantly reduced in GIPR deficient animals and was associated with lowered intrinsic material properties. Taken together, these data support a positive effect of the GIPR on bone strength and quality.

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