Beneficial effects of a N-terminally modified GIP agonist on tissue-level bone material properties.

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Résumé en anglais	Bone remodeling is under complex regulation from nervous, hormonal and local signals, including gut hormones. Among the gut hormones, a role for the glucose-dependent insulinotropic polypeptide (GIP) has been suggested. However, the rapid degradation of GIP in the bloodstream by the ubiquitous enzyme dipeptidyl peptidase-4 (DPP-4) precludes therapeutic use. To circumvent this problem, a series of N-terminally modified GIP agonists have been developed, with N-AcGIP being the most promising. The aims of the present study were to investigate the effects of N-AcGIP on bone at the micro-level using trabecular and cortical microstructural morphology, and at the tissue-level in rats. Copenhagen rats were randomly assigned into control or N-AcGIP-treated groups and received daily injection for 4 weeks. Bone microstructural morphology was assessed by microCT and dynamic histomorphometry and tissue-level properties by nanoindentation, qBEI and infra-red microscopy. Four week treatment with N-AcGIP did not alter trabecular or cortical microstructural morphology. In addition, no significant modifications of mechanical response and properties at the tissue-level were observed in trabecular bone. However, significant augmentations in maximum load (12%), hardness (14%), indentation modulus (13%) and dissipated energy (16%) were demonstrated in cortical bone. These beneficial modifications of mechanical properties at the tissue-level were associated with increased mineralization (22%) and collagen maturity (13%) of the bone matrix. Taken together, the results support a beneficial role of GIP, and particularly stable analogs such as N-AcGIP, on tissue material properties of bone.
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