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Effects of Risedronate in Runx2 Overexpressing Mice, an Animal Model for Evaluation of Treatment Effects on Bone Quality and Fractures

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Résumé en anglais	Young mice overexpressing Runx2 specifically in cells of the osteoblastic lineage failed to gain bone mass and exhibited a dramatic increase in bone resorption, leading to severe osteopenia and spontaneous vertebral fractures. The objective of the current study was to determine whether treatment with a bisphosphonate (risedronate, Ris), which reduces fractures in postmenopausal as well as in juvenile osteoporosis, was able to improve bone quality and reduce vertebral fractures in mice overexpressing Runx2. Four-week-old female Runx2 mice received Ris at 2 and 10 µg/kg subcutaneously twice a week for 12 weeks. Runx2 and wild-type mice received vehicle (Veh) as control. We measured the number of new fractures by X-ray and bone mineral density (BMD) by DEXA. We evaluated bone quality by histomorphometry, micro-CT, and Fourier transform infrared imaging (FTIRI). Ris at 20 µg/kg weekly significantly reduced the average number of new vertebral fractures compared to controls. This was accompanied by significantly increased BMD, increased trabecular bone volume, and reduced bone remodeling (seen in indices of bone resorption and formation) in the vertebrae and femoral metaphysis compared to Runx2 Veh. At the femur, Ris also increased cortical thickness. Changes in collagen cross-linking seen on FTIRI confirmed that Runx2 mice have accelerated bone turnover and showed that Ris affects the collagen cross-link ratio at both forming and resorbing sites. In conclusion, young mice overexpressing Runx2 have high bone turnover-induced osteopenia and spontaneous fractures. Ris at 20 µg/kg weekly induced an increase in bone mass, changes in bone microarchitecture, and decreased vertebral fractures.
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