Bisphosphonates suppress periosteal osteoblast activity independently of resorption in rat femur and tibia

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Abstract:

Recent studies demonstrate that bisphosphonates suppress bone resorption by leading to apoptosis of the osteoclast and inhibiting the differentiation to mature osteoclasts. The influence of bisphosphonates on bone formation is unknown, although it has been hypothesized that bisphosphonates inhibit osteoblast apoptosis and stimulate osteoblast proliferation and differentiation in vitro, leading to increased bone formation. The purpose of this study was to investigate the effect of bisphosphonates on bone formation. We administered risedronate at 0.05, 0.5 or 5.0 μg/kg/day or alendronate at 0.1, 1.0 or 10 μg/kg/day subcutaneously for 17 days to 6-month-old female Sprague–Dawley rats. Control rats were given a daily subcutaneous injection of saline. Following sacrifice, the femoral and tibial mid-diaphyses were harvested and mineralizing surface (MS/BS), mineral apposition rate (MAR) and bone formation rate (BFR/BS) were measured on periosteal and endocortical surfaces. In the femur, periosteal MAR was significantly lower in all treatment groups (22–29% for risedronate, 26–36% for alendronate) than in control. In the tibia, periosteal MAR and BFR of all treatment groups were significantly lower (41–50% for risedronate, 43–52% for alendronate) than in the control group. Because the periosteal surfaces of these bones are only undergoing bone formation in modeling mode, our results show that bisphosphonates suppress bone formation independently of bone resorption. Because this effect is seen on

periosteal MAR rather than on periosteal MS/BS, we hypothesize that bisphosphonates affect the activity of individual osteoblasts at the cell level. This may help to explain the reason that the anabolic effects of teriparatide are blunted when administered concurrently with or following a course of bisphosphonates in humans.

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