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2007 Amgen/ANZBMS Outstanding Abstract Award Recipient

Winner: Ms Maria Chiu

Abstract:

The calcitonin receptor plays a physiological role in maintaining trabecular bone volume in young female and adult male mice

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To investigate the physiological role of the calcitonin receptor (CTR) in calcium homeostasis and bone turnover, we have generated a mouse model in which the CTR is deleted specifically in osteoclasts (OCL-CTR KO).

The phenotypes of OCL-CTR KO and control littermates were assessed at 6 and 12 weeks of age (n=8-10/group). Serum calcitonin, PTH and calcium levels were unchanged in female and male OCL-CTR KOs at 6 and 12 weeks of age. Osteoclast-specific deletion of the CTR in males resulted in a 37% decrease in trabecular bone (BV/TV) at 12 weeks of age ($P<0.01$). This was associated with a 23% decrease in trabecular thickness (TbTh) ($P<0.05$), a 14% decrease in mineralised surface ($P<0.05$), and a 24% decrease in bone formation rate ($P=0.06$). Although trabecular number was lower in OCL-CTR KOs, this was not significant ($P=0.08$). This bone loss may be attributed to an increase in bone turnover prior to 12 weeks of age, as evidenced by preliminary gene expression data demonstrating elevated RANK ($P<0.01$), TRAP, collagen 1a1 ($P<0.05$) and alkaline phosphatase ($P<0.05$) mRNA levels in OCL-CTR KO males at 6 weeks of age.

In contrast, OCL-CTR KO females had a 17% reduction in BV/TV ($P<0.05$) at 6 weeks of age due to an 8% decrease in TbTh ($P=0.05$).

We are further assessing the physiological role of the CTR in protecting against hypercalcemia in OCL-CTR KO and control mice.

In conclusion, we have demonstrated a physiological role of the CTR in trabecular bone maintenance in young female and adult male mice.