Exercise increases nonshivering thermogenesis but not bone mass during cold exposure in a mouse model of humans

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Recent studies suggest exercise can activate brown adipose tissue (BAT) in humans, producing heat. This finding is surprising since exercise itself generates heat, and BAT upregulation is more commonly associated with cold stress. To understand the effects of exercise and cold on uncoupling protein (UCP1) in BAT and on bone mass, we studied male C57BL/6J mice at 26C (thermoneutrality) and 16C (moderate cold) from 3-6 weeks of age. Half of each group had running wheels (exercise, EX) and half did not (control, CON). We hypothesized that exercise would increase UCP1 and cortical and trabecular bone volume at both temperatures. Results indicate that both exercise (+32-91%) and cold (+31-52%) increased UCP1 protein expression ($p<0.03$ for all) in BAT, such that it was highest in 16C EX and lowest in 26C CON. In the distal femur, EX mice had 27-28% lower trabecular bone volume fraction (BV/TV), 18-24% lower connectivity density (Conn.D), and 7-8% lower trabecular number (Tb.N) vs. controls at 16C and 26C ($p<0.05$ for all). Cold mice had 24% lower BV/TV, 8% lower Tb.N and 25-35% lower Conn.D independent of exercise ($p<0.05$ for all), such that trabecular bone properties were lowest in 16C EX and highest in 26C CON. There were no differences in midshaft femur cortical bone. These results show that exercise does increase UCP1 expression, but contrary to our hypothesis exercise and cold have inverse effects on UCP1 and trabecular bone microarchitecture. Low bone mass in cold-dwelling humans may reflect energetic tradeoffs among thermogenesis, activity, and skeletal acquisition.

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