Acute continuous moderate-intensity exercise, but not low-volume high-intensity interval exercise, attenuates postprandial suppression of circulating osteocalcin in young overweight and obese adults

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Background. Osteoblasts are bone forming cells which are primarily involved in the synthesis, deposition, and mineralization of type 1 collagen to form bone during growth. Osteoblasts also synthesize and secrete paracrine proteins including osteocalcin (tOC), and the undercarboxylated form (ucOC), which participate not only in bone mineralization but also glucose homeostasis**.** Serum tOC and ucOC are suppressed during conditions of hyperglycemia, such as after the consumption of a meal or an oral glucose tolerance test. This suppression may contribute to increased fracture risk in populations who are insulin resistant. In contrast, acute exercise transiently increases tOC and ucOC, and is proposed to enhance glucose regulation. However, the effects of acute exercise and exercise-intensity on postprandial levels of tOC and ucOC are unknown.

Methods. Twenty-seven adults that were overweight or obese (age: 30±1 years; BMI: 30±1 $kg \cdot m^{-2}$; mean±SEM) were randomly allocated to perform a single session of low-volume high-intensity interval-exercise (LV-HIIE; 9 females, 5 males) or continuous moderate-intensity exercise (CMIE; 8 females, 5 males) 1 hour after consumption of a standard breakfast. Serum tOC, ucOC, and ucOC/tOC were measured at baseline, 1 hour, and 3 hours after breakfast consumption on a rest day (no exercise) and the exercise day (exercise 1 hour after breakfast).

Results. Compared to baseline, serum tOC and ucOC were suppressed 3 hours after breakfast on the rest day (-10±1% and -6±2%, respectively; $P<0.05$), whereas ucOC/tOC was elevated (2.5±1%; $P=0.08$). Compared to the rest day, CMIE attenuated the postprandial-induced suppression of tOC (rest day: -10±2% *versus* CMIE: -5±2%, $P<0.05$) and ucOC (rest day: -6±4% *versus* CMIE: 11±2%, $P<0.05$), and increased postprandial ucOC/tOC (rest day: 3±2% *versus* CMIE: 15±1%, $P<0.05$). In contrast, LV-HIIE did not alter postprandial tOC, ucOC or ucOC/tOC (all $p>0.1$).

Conclusions. Acute CMIE, but not LV-HIIE, attenuates the postprandial-induced suppression of tOC and ucOC. CMIE may be an effective tool to control the circulating levels of tOC and ucOC following meal consumption in overweight/obese adults.