



Complex Interactions Between Estrogen, Strain, and Exercise-Induced Periosteal Bone Growth

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Complex interactions between estrogen, strain, and exercise-induced periosteal bone growth

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Abstract:

Understanding the relationship between bone strain and bone growth is critical for interpreting variations in skeletal robusticity. Recently we presented a model for interactions between estrogen, strain, and periosteal bone growth, in which high estrogen (E₂) increases, and low (E₂) decreases, osteogenic responses to strain. We compared cortical growth in exercised and sedentary sheep (*Ovis aries*) with higher vs. lower estrogen levels, and showed that exercised animals with high E₂ added substantially more bone than those with lower E₂. However, without normal controls, it was unclear whether exercise-induced cortical growth was stimulated by high E₂, suppressed by lower E₂, or both. Here we present a broader test of interactions between E₂ levels (normal, low, high) and loading (exercised and sedentary). Low E₂ animals were vaccinated against GnRH to suppress estrogen, while high E₂ animals received estrogen implants. After 45 days, periosteal bone growth was measured at hindlimb midshafts. The results support the hypothesis that estrogen upregulates strain-induced cortical bone growth: exercised, high-E₂ animals grew 6-27% more bone than exercised animals with lower E₂ levels, or sedentary animals ($p < .05$). The effects of the anti-GnRH vaccine on bone growth are more complex. Assays showed that vaccinated animals had normal, not decreased, E₂ levels, but grew 34-39% less bone in response to exercise than normal controls ($p < .05$). This suggests the vaccine affected strain-induced bone growth without changing circulating E₂, an unexpected finding. These results demonstrate that variation in E₂ levels may produce differential growth response to similar mechanical loading through complex mechanisms.