A longitudinal study of the effect of subcutaneous estrogen replacement on bone in young women with Turner's syndrome.


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It is desirable that young women with primary ovarian failure achieve normal peak bone mass to reduce the subsequent risk of osteoporosis, and that there are management strategies to replace bone that is already lost. While estrogen (E2) is generally considered to prevent bone loss by suppressing bone resorption, it is now recognized that estrogen also exerts an anabolic effect on the human skeleton. In this study, we tested whether estrogen could increase bone mass in women with primary ovarian failure. We studied the mechanism underlying this by analyzing biochemical markers of bone turnover and iliac crest biopsy specimens obtained before and 3 years after E2 replacement. Twenty-one women with Turner's syndrome, aged 20-40 years, were studied. The T scores of bone mineral density at lumbar spine and proximal femur at baseline were -1.4 and -1.1, respectively. Hormone replacement was given as subcutaneous E2 implants (50 mg every 6 months) with oral medroxyprogesterone. Serum E2 levels increased incrementally from 87.5 pM at baseline to 323, 506, 647, and 713 pM after 6 months and 1, 2, and 3 years of hormone replacement therapy (HRT), respectively. The bone mineral density at the lumbar spine and proximal femur increased after 3 years to T scores of -0.2 and -0.4, respectively. The cancellous bone volume increased significantly from 13.4% to 18.8%. There was a decrease in activation frequency, but the active formation period was increased by HRT. There was a significant increase in the wall thickness from 33.4 microm at baseline to 40.9 microm after 3 years of HRT, reflecting an increase in bone formed at individual remodeling units. Although there was an early increase in
biochemical markers of bone formation, these declined thereafter. Our results show that estrogen is capable of exerting an anabolic effect in the skeleton of young women with Turner's syndrome and low bone mass.

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