Estrogen and bone-muscle strength and mass relationships.

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The largest voluntary loads on bones come from muscles. To adapt bone strength and mass to them, special strain threshold ranges determine where modeling adds and strengthens bone, and where remodeling conserves or removes it, just as different thermostat settings control the heating and cooling systems in a house. If estrogen lowers the remodeling threshold, two things should occur. First, at puberty in girls, bone mass should begin to increase more than in boys with similar muscle strengths, owing to reduced remodeling-dependent bone losses, while gains from longitudinal bone growth and bone modeling continue normally. That increase in bone mass in girls should plateau when their muscle strength stops increasing, since their stronger bones could then reduce bone strains enough to turn modeling off, but could let remodeling keep conserving existing bone. Second, decreased estrogen secretion [or a related factor(s)], as during menopause, should raise the remodeling threshold and make remodeling begin removing that extra bone. That removal should also tend to plateau after the remaining and weaker bone lets bone strains rise to the higher threshold. Postmenopausal bone loss shows the second effects. Previously unremarked relationships in the data of a 1995 Argentine study showed the first effects. This supports the idea that estrogen can affect human bone strength and mass by lowering the remodeling threshold, and loss of estrogen would raise the threshold and help cause postmenopausal bone loss even if other factors help to do it. The Argentine study also suggested ways to study those things and the roles of muscle strength and other factors in controlling bone strength and mass in children and adult humans. Those factors included, in part, hormones, vitamins, calcium, diet, sex, race, age, medications, cytokines, genetic errors, gene expression patterns, and disease.

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