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Progesterone for the prevention and treatment of osteoporosis in women

J C Prior [1] [2]

Affiliations

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Abstract

Estradiol (E2) is women's dominant 'bone hormone' since it is essential for development of adolescent peak bone mineral density (BMD) and physiological levels prevent the rapid (3-week) bone resorption that causes most adult BMD loss. However, deceasing E2 levels trigger bone resorption/loss. Progesterone (P4) is E2's physiological partner, collaborating with E2 in every cell/tissue; its bone 'job' is to increase P4-receptor-mediated, slow (3-4 months) osteoblastic new bone formation. When menstrual cycles are normal length and normally ovulatory, E2 and P4 are balanced and BMD is stable. However, clinically normal cycles commonly have ovulatory disturbances (anovulation, short luteal phases) and low P4 levels; these are more frequent in teen and perimenopausal women and increased by everyday stressors: energy insufficiency, emotional/social/economic threats and illness. Meta-analysis shows that almost 1%/year spinal BMD loss occurs in those with greater than median (~31%) of ovulatory disturbed cycles. Prevention of osteoporosis and fragility fractures requires the reversal of stressors, detection and treatment of teen-to-perimenopausal recurrent cycle/ovulatory disturbances with cyclic oral micronized progesterone. Low 'Peak Perimenopausal BMD' is likely the primary risk for fragility fractures in later life. Progesterone plus estradiol or other antiresorptive therapies adds 0.68%/year and may be a highly effective osteoporosis treatment. Randomized controlled trials are still needed to confirm progesterone's important role in women's bone formation.

Keywords: Progesterone; bone formation; bone resorption; estradiol; life cycle; menstrual cycle; osteoporosis; ovulatory disturbances; women.

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