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Invited Plenary Abstract
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Pathogenesis of bone fragility

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At the microscopic level, osteoporotic bone is normally mineralized but reduced in volume. There is a generalized thinning of trabecular elements with the total loss of some trabeculae. The combination of less bone and disrupted micro-architecture greatly reduces the strength of osteoporotic bone. Loss of bone is sometimes pathological, but more commonly occurs as a normal part of the ageing process. With age, the efficiency with which osteoblasts refill resorption cavities is reduced, and there is also an increase in bone resorption, probably resulting from hypogonadism. There is an increase in fracture risk with age which is partly independent of BMD.

Whether or not an individual develops osteoporosis depends on their peak bone mass, their rate of loss of bone in later life, and their longevity. Probably the strongest influence on peak bone density is genetic, though the major genetic contributors remain to be determined. Genetic mechanisms probably underlie the significant racial differences in fracture incidence, and might involve differences in bone architecture as well as BMD. Body weight is an important influence on bone density throughout life, heavier people having greater bone mass. This is probably mediated via hormonal mechanisms and by direct skeletal load. The role of calcium intake in determining bone density is limited, and vitamin D deficiency, which is common in older individuals may also contribute to reduced bone mass. Physical activity contributes only moderately to the differences in bone density that exist in postmenopausal women.