

## Invited Speaker Abstract

ISI8

### **Cortical bone: forgotten frontier**

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Fragility fractures are an important public health problem. Fuller Albright signalled the occurrence of vertebral fractures in postmenopausal women and since that time, some 65 years ago, research aimed at understanding the pathogenesis of bone fragility has concentrated on the trabecular bone loss and vertebral fractures, because vertebrae are largely trabecular. In this historical context, vertebral fractures and trabecular bone loss remain the flagships of osteoporosis and are almost synonymous with bone fragility.

Cortical bone loss and non-vertebral fractures (apart from hip fractures) have been largely neglected despite cortical bone comprising 80% of the skeleton. Moreover, 80% of all fractures and 80% of all the disability due to fractures in the community are the result of non-vertebral fractures.

Recent studies from our department challenge these prevailing notions and suggest that age-related bone loss is largely cortical, not trabecular, and that this bone loss arises from the intracortical envelope of bone, not the endocortical or trabecular envelopes. This loss of bone occurs by increasing intracortical porosity which in turn increases the available surface for remodelling so that ~70% of the decrease in apparent volumetric density and compressive strength is the result of intracortical bone loss. Similarly, the process of age-related cortical thinning appears to be largely due to intracortical porosity. Thus, emphasis on trabecular bone and vertebral fractures is an incomplete presentation of the pathogenesis of fragility and epidemiological burden of fractures.