## Genes discovery linked to osteoporosis and fracture risk

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A large number of genetic variants have for the first time been linked to the risk of osteoporosis and bone fracture, according to a major new international study.

Osteoporosis is a silent but devastating age-related disease that kills half of those who fracture their hip after the age of 80 within 12 months. Women aged over 65 are at greater risk of death after hip fracture than from breast cancer.

Researchers around the world, including from The University of Western Australia, found that variants in 56 regions of the genome influenced bone mineral density, while 14 of these variants increased the risk of bone fracture.

Bone mineral density is the most widely used measurement to diagnose osteoporosis and assess the risk of fracture, with higher density associated with lower risk of fracture.



In the largest genetic study of osteoporosis to date, investigators from more than 50 studies across Europe, North America, East Asia and Australia studied more than 80,000 individuals.

The study, led by researchers from Holland's Erasmus University Medical Centre in Rotterdam, was published yesterday in the leading international journal *Nature Genetics*.

Co-author Professor Richard Prince, from UWA's Bone and Vascular Research Group, said osteoporosis was strongly related to gene variation.

"We have found new genes strongly related to bone structure. This latest research has helped pinpoint many factors in critical molecular pathways that may lead to therapeutic treatments.

"This research also leads to better understanding of the biology of skeletal health and fracture susceptibility."

Researchers also found that women with an excess of bone mineral density-decreasing genetic variants had up to 56 per cent higher risk osteoporosis and a 60 per cent higher risk of all types of fractures.

<sup>\*</sup> Estrada K, et al. Genome-wide meta-analysis identifies 56 bone mineral density loci and reveals 14 loci associated with risk of fracture. Nat Genet. 2012 Apr 15;44(5):491-501.