Controlling bone formation to prevent osteoporosis

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Aging disrupts the balance between bone formation and bone destruction, resulting in osteoporosis, which is characterized by reduced bone mass and increased risk of fracture. Recent data have suggested that this imbalance is a result of a decrease in formation of bone forming osteoblast cells from mesenchymal cells upon aging. Instead, these cells form more fat cells.

Insight into this age-related switch in cell type generation has now been provided by a team of researchers, led by Hiroshi Takayanagi, at Tokyo Medical and Dental University, Japan, working in mice. The data generated might provide new avenues of research for those developing approaches to treat age-related osteoporosis.

In the study, the gene regulatory protein Maf was found to promote mesenchymal cell generation of osteoblasts and suppress their generation of fat cells. Consistent with this, mice lacking Maf showed delayed bone formation. Furthermore, Maf levels were found to decrease in mouse mesenchymal cells upon aging and to be reduced by increased oxidative stress, something that occurs upon aging. Both the authors and, in an accompanying commentary, Laurie McCauley, at University of Michigan, Ann Arbor, believe these data could lead to new approaches to treat age-related osteoporosis.

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