

Adiponectin is a metabolic link between obesity and bone mineral density

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Researchers at the University of Toronto, Faculty of Medicine, Toronto, Canada, have discovered that adiponectin, a protein secreted from adipocytes, is a metabolic link that can explain, in part, the known positive relationship between obesity and both bone mineral density and reduced susceptibility to fractures. This study appears in the December issue of *Experimental Biology and Medicine*.

Circulating adiponectin levels are significantly lower in obese humans and rodent models than in lean controls. It is known that excess body weight and elevated body mass index are strongly correlated with high bone mineral density, and that weight loss is associated with loss of bone mineral density and increased risk of fractures. However, the mechanism for this relationship is unclear.

The research team, Dr. Michael C. Archer, Earle W. McHenry Professor and Chair, Dr. Wendy E. Ward, Associate Professor, Dr. Kafi Ealey, Postdoctoral Fellow and predoctoral student Jovana Kaludjerovic, in the Department of Nutritional Sciences, investigated whether adiponectin modulates bone development using transgenic mice that overexpress this protein. These mice were initially developed by Dr. P. Scherer's research group at Albert Einstein College of Medicine, N.Y. Bone mineral density and biomechanical strength properties, surrogate measures of fracture risk at multiple skeletal sites, were the outcomes used to assess bone development.

Female mice overexpressing adiponectin had weaker vertebra at 8 weeks

of age than control mice and this delay in bone development persisted through to the end of the study period, representing early adulthood. The weaker vertebra model compression fractures of the lumbar spine in humans, among the most common type of fragility fracture associated with low bone mass and osteoporosis.

The strength of the femur neck, representing the hip, was also weaker in both females and males overexpressing adiponectin. Serum adiponectin levels were inversely correlated with femur bone mineral content, further emphasizing that a high level of adiponectin impedes bone development at not only the lumbar spine but also the hip. Whether or not the delay in bone development resolves in later life or is sustained and leads to an increased risk of fragility fracture, particularly during aging when bone loss rapidly occurs due to declining levels of sex steroids, requires further investigation.

In summary, elevated circulating adiponectin was associated with lower bone mass and weaker bones in growing mice compared to control animals. Furthermore, these effects of adiponectin were observed in the absence of differences in body weight between the two groups of mice. Dr. Archer commented, "A unique and important feature of the adiponectin transgenic mice is that they exhibit significantly elevated circulating adiponectin but have similar body weights as control animals, thus eliminating obesity from confounding the study findings – mechanical load resulting from an obese state can modulate bone metabolism". Moreover, Dr. Archer said, "female mice exhibited a stronger response than males, and this is likely due to the sexual dimorphism in these mice whereby females have significantly higher circulating levels than males".

Dr. Steven R. Goodman, Editor-in-Chief of *Experimental Biology and Medicine* said " These intriguing results form the University of Toronto team supplies significant insight into adiponectin secretion, from

adipocytes, and loss of bone mineral density and resulting increased fractures. I consider this a major advance for this field of research.

Source: Society for Experimental Biology and Medicine

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