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Role of galectin-3 in bone and fat cell differentiation: Implications for bone and adipose tissue development, metabolism and function

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Galectin-3 is expressed in bone, where it is considered a marker of chondrogenic and osteogenic cell lineages. Moreover, we recently reported that galectin-3 modulates osteogenic transdifferentiation of vascular smooth muscle cells and is involved in the process of atherosclerotic calcification. This lectin is also expressed in human adipocytes during cell differentiation and its circulating levels are positively associated with human and murine obesity. Therefore, we decided to investigate the *in vitro* differentiating capacity of bone cells and adipocyte precursors from glectin-3 knockout (Lgals3-/-) mice, and the structural and functional phenotype of bones and fat depots of these mice. Regarding the bone, osteoblasts and osteoclasts precursors derived from Lgals3-/-mice showed impaired terminal differentiation, reduced mineralization capacity and resorption activity, which translated into profound alterations of bone remodeling and structure throughout life, resulting in accelerated age-dependent bone loss and reduced bone strength. Regarding the adipose tissue, stromal precursor cells from Lgals3-/- mice showed defective adipocyte differentiation, as revealed by decreased expression of adipogenic markers and reduced ability to store lipids, which resulted in delayed maturation and function of adipose tissue *in vivo*. Adipose tissue dysfunction was associated with transient insulin resistance and inflammation at both the adipose tissue and systemic level, resulting in impaired glucose homeostasis. Therefore, these studies demonstrate that galectin-3 is a critical regulator of osteogenic and adipogenic differentiation, thus playing an important role in bone physiology and systemic energy metabolism. Overall, these findings identify galactin-3 as an attractive potential target to treat disorders of bone remodeling and energy metabolism.

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