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Microfibril-associated glycoprotein-1 (MAGP1) is an extracellular matrix protein that interacts with fibrillin and is involved in regulating the bioavailability of signaling molecules such as TGFβ. Mice with MAGP1 deficiency (*Mfap2*$^{-/-}$) progressively develop increased adiposity, insulin resistance, and reduced cancellous bone mass. In this study, MAGP1-deficient mice were used to study the relationship between peripheral adiposity and marrow adipose tissue (MAT), a fat depot located within the bone marrow space that has been shown to respond to metabolic disease. By two months, *Mfap2*$^{-/-}$ mice had reduced cancellous bone and were hyperglycemic. At 10 months, *Mfap2*$^{-/-}$ mice became insulin resistant and showed a five-fold increase in MAT relative to the WT group. As the MAT expansion was coincident with the development of insulin resistance rather than the progressive cancellous bone loss, a Prx-Cre model was used to conditionally delete MAGP1 from the limbs to further explore this relationship. Mice with a deletion of MAGP1 in the limbs (Prx1-Cre; *Mfap2*$^{-/-}$) did not show any changes in peripheral adiposity, insulin response, or MAT volume. However, by 24 weeks, Prx1-Cre; *Mfap2*$^{-/-}$ had cancellous bone loss. This suggests that changes in MAT are not regulated by the bone microenvironment expression of MAGP1, but are rather tied to global metabolic functioning.