**From Adipocyte Plasticity to Systemic Energy Balance: How Hippo-YAP/TAZ Signaling Shapes Metabolic Health**

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Adipose tissue plays an essential role as an energy reservoir as well as an endocrine organ. Coordination of these critical functions is required to maintain metabolic health. For example, leptin production is tightly linked to fat mass, but the underlying molecular mechanism is yet to be identified. Here, we show that the transcriptional coregulators YAP and TAZ directly regulate leptin gene expression through interactions with the TEAD transcription factor, the canonical target of YAP/TAZ, which occupies TEAD-binding elements within the *Lep* gene enhancer. On the other hand, we show that Hippo-YAP/TAZ regulates adipose tissue storage capacity through repression of PPARG. Together, we demonstrate that YAP/TAZ activation in mature adipocytes functions on two distinct molecular axes: a YAP/TAZ-TEAD axis that increases systemic energy expenditure by increasing circulating leptin levels and a YAP/TAZ-PPARG axis that decreases adipose tissue mass by repressing PPARG target genes. Surprisingly, in mice with sustained activation of adipocyte YAP/TAZ results in an apparent uncoupling of leptin gene expression from adipose tissue mass. Despite being fatless, or lipoatrophic, these mice did not exhibit metabolic defects commonly associated with lipodystrophy. We show that the paradoxical increase in circulating leptin levels in these fatless mice compensates for their energy storage deficit by increasing fat oxidation and energy expenditure. These findings position adipocyte YAP/TAZ at the nexus of molecular crosstalk that links adipose storage capacity and systemic energy balance.