

Metformin combats adipose tissue expansion via AMPK

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(HealthDay)—AMPK activation by metformin is associated with

inhibition of interstitial fibrosis and suppression of transforming growth factor β -1 (TGF- β 1), according to a study published online May 13 in *Diabetes*.

Ting Luo, from the Boston University School of Medicine, and colleagues explored the initiation of aberrant extracellular matrix (ECM) remodeling of [white adipose tissue](#) (WAT) during obesity development.

The researchers found that metformin treatment inhibited excessive ECM deposition in WAT of ob/ob mice and diet-induced obese mice, with reduction in collagen deposition surrounding adipocytes and expression of fibrotic genes, including the regulator of collagen cross-linking, LOX. Metformin-induced inhibition of interstitial fibrosis may be due to activation of AMPK and suppression of TGF- β 1/Smad3 signaling, which leads to enhanced systemic insulin sensitivity. In primary cells from the stromal vascular fraction, the dominant negative AMPK abolished the ability of metformin to suppress TGF- β 1-induced fibrogenesis. AMPK agonists and the constitutively active AMPK suppressed TGF- β 1-induced insulin resistance in 3T3L1 adipocytes. There was also a correlation for interstitial fibrosis with AMPK inactivation, TGF- β 1/Smad3 induction, aberrant ECM production, myofibroblast activation, and adipocyte apoptosis in omental fat depots of obese humans.

"Collectively, integrated AMPK activation and TGF- β 1/Smad3 inhibition may provide a potential therapeutic approach to maintain ECM flexibility and combat chronically uncontrolled [adipose tissue](#) expansion in obesity," the authors write.

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