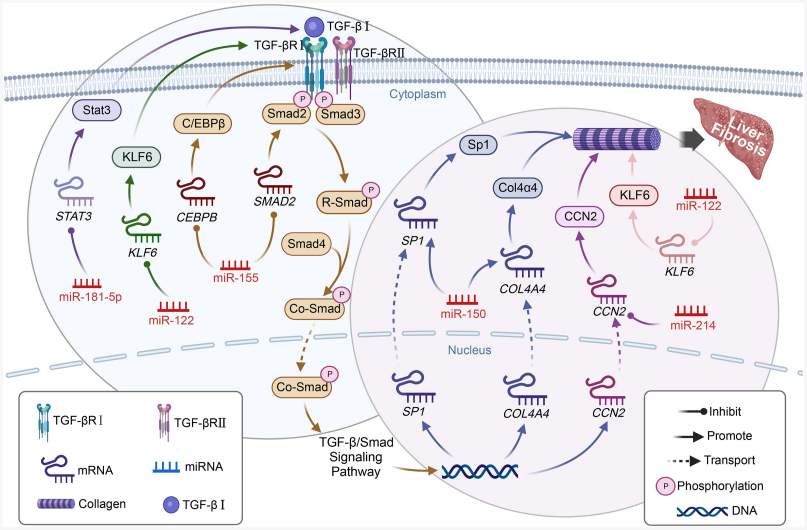
MicroRNAs Offer New Hope in the Battle Against Adipose Tissue Fibrosis

SHANNON, CLARE, IRELAND, April 20, 2025 /EINPresswire.com/ --
A new review published in Genes & Diseases highlights the transformative role of microRNAs (miRNAs) in regulating and potentially reversing adipose tissue fibrosis, a condition closely linked to obesity, diabetes, and cardiovascular disease. Fibrosis, driven by abnormal extracellular matrix (ECM) accumulation, disrupts normal adipose tissue function and contributes to broader organ dysfunction. The review explores how miRNAs act as potent molecular regulators, capable of fine-tuning signaling pathways and gene expression patterns that influence fibrotic progression.



TGF-β/Smad signaling pathway and miRNA regulation in liver fibrosis. Tissue fibrosis intricately links with the TGF-β/Smad signaling pathway.

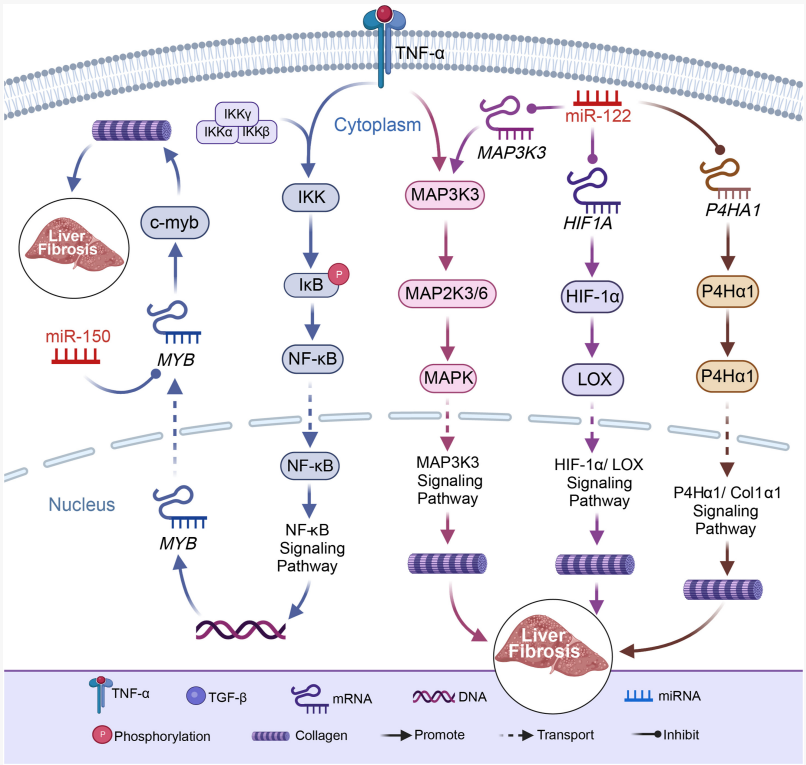
miRNAs, a class of small non-coding RNAs, can suppress or promote the translation of target genes involved in fibrogenic processes. Within adipose tissue, their regulation of pathways such as TGF-β/Smad, PI3K/AKT, and PPAR-γ plays a pivotal role in determining the balance between healthy tissue maintenance and pathological fibrosis. Specific miRNAs such as miR-122, miR-140, miR-150, miR-30b, and miR-155 demonstrate diverse functions, from blocking collagen synthesis to preventing the conversion of adipogenic cells into fibrogenic ones.

Of particular interest is the therapeutic application of adipose-derived stem cells (ADSCs) transfected with targeted miRNAs. These engineered cells produce a secretome—a vesicle-rich fluid carrying anti-fibrotic miRNAs—that can be delivered to affected tissues without triggering immune rejection. This approach enables precise molecular intervention, targeting key proteins like Smad3, PDGFR-β, Runx1, and PPAR-γ, which are central to fibrosis development.

The review also draws attention to miRNAs' systemic impact, noting how alterations in adipose tissue can influence fibrosis in distant organs, including the liver, heart, and kidneys. For

example, miR-410-5p, elevated in high-fat diet-induced obesity, enhances fibrosis by downregulating protective factors like Smad7 in cardiac tissue. Conversely, restoring miR-140 or delivering miR-30b can mitigate these fibrotic responses.

Ultimately, the findings underscore the potential of miRNA-based therapies as a non-invasive, targeted strategy to combat fibrosis in both adipose tissue and other organs.



Decoding miRNA-mediated regulation of fibrotic pathways: Insights into liver fibrosis inhibition. Hypoxia-inducible factor 1-alpha (HIF-1α) enhances collagen and elastin expression by targeting LOX (lysyl oxidase), promoting liver fibrosis.

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Genes & Diseases publishes rigorously peer-reviewed and high quality original articles and authoritative reviews that focus on the molecular bases of human diseases. Emphasis is placed on hypothesis-driven, mechanistic studies relevant to pathogenesis and/or experimental therapeutics of human diseases. The journal has worldwide authorship, and a broad scope in basic and translational biomedical research of molecular biology, molecular genetics, and cell biology, including but not limited to cell proliferation and apoptosis, signal transduction, stem cell biology, developmental biology, gene regulation and epigenetics, cancer biology, immunity and infection, neuroscience, disease-specific animal models, gene and cell-based therapies, and regenerative medicine.
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Reference
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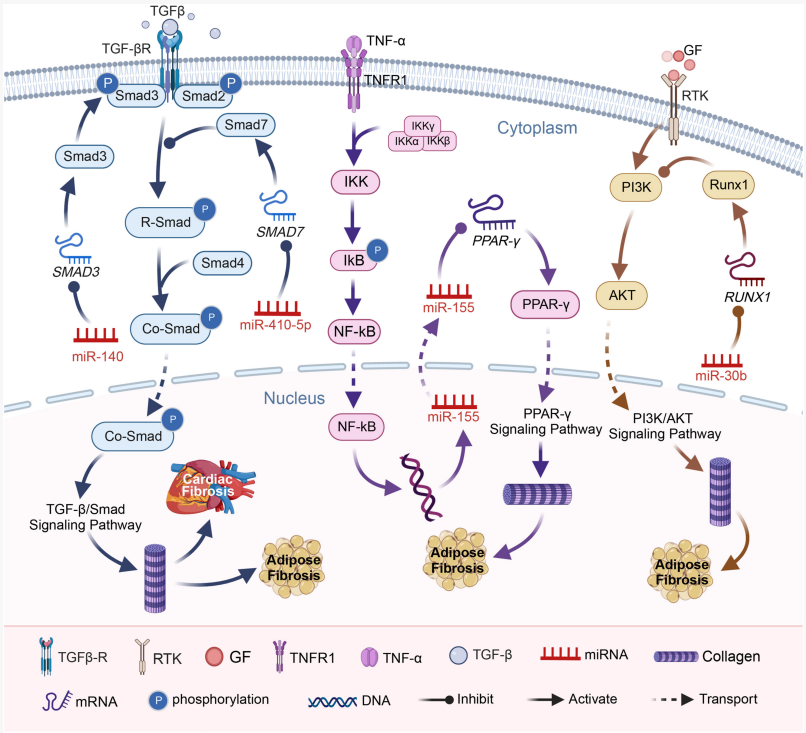

The role and therapeutic potential of miRNAs in adipose tissue fibrosis induced by high-fat diet. Transforming growth factor β (TGF-β) activates Smad3 upon binding to its receptor, and miR-140 interferes with phosphorylated SMAD3, inhibiting Smad3 express

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