



## **MitoRx Therapeutics presents new preclinical data on MTRX31, demonstrating restored mitochondrial density, at the 62<sup>nd</sup> EASD Annual Meeting**

- *MTRX31 shown to restore mitochondrial density in the liver of diet-induced obesity (DIO) mice to levels comparable to lean control mice*
- *MTRX31 also reduced lipid droplets and increased glycogen flexibility: evidence of repaired metabolic flexibility*

**Harwell Oxford, UK – 1 October 2026:** MitoRx Therapeutics (“MitoRx”), a biotech company targeting the restoration of healthy mitochondrial metabolism in high-risk obesity, announces new preclinical data for its lead asset, MTRX31, was presented at the 62<sup>nd</sup> Annual Meeting of the European Association for the Study of Diabetes (EASD), taking place in Milan, Italy, from 28 September - 2 October 2026.

The data was generated from a study using a diet-induced obesity (DIO) mouse model and was orally presented by Xavier Jacq, Chief Scientific Officer of MitoRx Therapeutics, on 30 September 2026.

MTRX31 is a novel small molecule that aims to eliminate ectopic fat from affected organs by resetting mitochondrial function rather than suppressing appetite, restoring healthy mitochondrial metabolism by resetting the balance between fat and carbohydrate oxidation.

In work carried out at the University of Oxford, Transmission Electron Microscopy (TEM) was used to examine lipid droplets, glycogen and mitochondrial number in liver tissue from diet-induced obesity (DIO) mice over 56 days. Obesity has been demonstrated in DIO mice to lead to a decrease in the number of mitochondria across various tissues, contributing significantly to a loss of metabolic flexibility.

Treatment with MTRX31 showed restoration of mitochondrial density per unit of tissue area to levels comparable to those observed in control mice. The TEM data also showed that MTRX31 reduced liver lipid droplets (steatosis) and restored glycogen flexibility in the DIO mice. Together, these findings indicate a cellular shift from fat to carbohydrate oxidation and a reduction in ectopic fat within the liver, restored mitochondrial function and metabolic flexibility.

This new data build on [previously presented results](#) in which MTRX31 was shown to drive durable, high-quality weight loss, preferential fat loss and preservation of lean mass in DIO mice, with greater weight loss than tirzepatide demonstrated as a monotherapy.

**Xavier Jacq, PhD, Chief Scientific Officer, MitoRx Therapeutics commented:** *“What makes these data so informative is that we can now see MTRX31’s effect directly within the tissue. The electron microscopy images show liver cells regaining their mitochondrial density, eliminating ectopic fat and restoring normal glycogen regulation. These are hallmarks of a metabolically flexible, healthy cell that can switch between carbohydrate and fat oxidation. Being able to characterize the mechanism at this resolution strengthens our conviction that MTRX31 acts on the root cause of high-risk obesity, rather than managing symptoms through curbing appetite.”*

**Jon Rees, PhD, CEO of MitoRx Therapeutics added:** *“The conversation in obesity is shifting from how much weight a therapy removes, to the quality of the weight lost. These new data presented at EASD 2026 build*

*on our existing evidence base and speak directly to that shift, with MTRX31 leading to high-quality weight loss and the restoration of metabolic flexibility, reducing the risk of progression into more serious diseases such as fatty liver disease, type 2 diabetes and atherosclerotic cardiovascular disease. Furthermore, our non-anorexic approach sidesteps the incretin-associated GI AEs that drive cessation, instead offering a durable therapy in patients for which that matters the most. We look forward to sharing further insights in the coming months as we continue to advance MTRX31 towards the clinic.”*

A webcast of the presentation will be made available for conference attendees on the [conference website](#).

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### **About MitoRx Therapeutics**

MitoRx is a biotech company targeting the restoration of healthy mitochondrial metabolism in high-risk obesity. This is critical for the 30-40% of patients living with metabolically unhealthy obesity – characterized by visceral adiposity and insulin resistance involving mitochondrial dysfunction – who are at disproportionate risk of metabolic-associated fatty liver disease, metabolic-associated steatohepatitis, type 2 diabetes and atherosclerotic cardiovascular disease.

Our first-in-class small molecule asset, MTRX31, targets the elimination of ectopic fat from affected organs by resetting mitochondrial function rather than suppressing appetite. This has the potential to deliver durable and higher quality weight loss, with implications for lower risk of cardiovascular complications and other serious disease, as well as benefits for body composition and improved quality of life. Preclinical studies showed high-dose MTRX31 driving significant weight loss that outperformed tirzepatide in magnitude, quality and durability after two months of treatment.

Find out more about our work on [LinkedIn](#) or visit our website at [mitorxtherapeutics.com](http://mitorxtherapeutics.com).

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