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Summary

Unraveling Steatotic Liver Diseases: Insights from Amino Acid and TCA Cycle Metabolomics

Masaaki Mino

UEG Week Berlin 2025 | Presentation

Hepatobiliary

October 6, 2025

The speaker presented clinical and mouse model data showing that MASLD is associated with altered amino acid metabolism, and that the investigational compound MA-5 improved mitochondrial function, ATP production, and liver fibrosis in a mouse model of MASH.

KEY POINTS

- In a Japanese biobank study of approximately 23,000 subjects, the speaker reported that plasma levels of branched-chain amino acids and glutamic acid were elevated in patients meeting cardiometabolic criteria for MASLD compared to normal controls.
- In a 20-week high-fat high-carbohydrate diet mouse model mimicking human MASH, the speaker found that liver amino acids, TCA cycle intermediates, and ATP levels were significantly reduced, with electron microscopy showing mitochondrial abnormalities including rounding and decreased cristae.
- Treatment with MA-5, an investigational synthetic indole compound, significantly reduced liver transaminases and improved pericellular fibrosis in the mouse model, as stated by the speaker.
- The speaker reported that MA-5 restored mitochondrial morphology and cristae structure, improved liver ATP levels, increased glucogenic amino acids, and upregulated oxidative phosphorylation genes in treated mice.
- A phase two clinical trial of MA-5 is currently underway in Japan for mitochondrial diseases, with the speaker noting in discussion that few adverse events such as nausea or diarrhea have been reported and that the compound may have potential in MASLD and other conditions with mitochondrial dysfunction.

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