From Adipose Tissue Insulin Resistance to Hepatic Inflammation

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Adipose tissue dysfunction, particularly visceral fat accumulation, is presented as a major driver of metabolic dysfunction-associated steatohepatitis (MASH) and type 2 diabetes through mechanisms of lipotoxicity and glucotoxicity.

KEY POINTS

- Visceral adipose tissue releases free fatty acids directly into the portal vein, which the liver takes up to synthesize triglycerides; dysfunctional visceral fat also releases pro-inflammatory adipokines including leptin, TNF-alpha, and resistin while decreasing adiponectin.
- The speaker reported that individuals with MASH and normal BMI (around 21) had almost double the visceral fat of controls without liver disease, demonstrating that visceral fat accumulation can occur even without obesity.
- The speaker's group found a strong correlation between lipolysis and hepatic glucose production, and reported that individuals with MASH without diabetes showed increased gluconeogenesis especially at fibrosis stages F2-4, which was further elevated in those with diabetes.
- Adipose tissue insulin resistance, measured as the product of free fatty acid and insulin concentration, was shown to increase stepwise with fibrosis severity from F0-1 to F2 to F3-4 in biopsy-proven NASH patients.
- Glucagon is described as having protective hepatic effects by increasing fatty acid oxidation and ketogenesis while decreasing de novo lipogenesis, hepatic triglyceride accumulation, and inflammatory cytokine production.
- The speaker stated that all drugs decreasing liver fat show strong correlation with decreased visceral fat, and those gaining visceral fat during trials show corresponding increases in liver fat.

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