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Summary

Is ageing a metabolic disease?

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The speaker proposed that adipose tissue senescence represents a key link between ageing and metabolic diseases, with senescent adipocytes creating a systemic pro-inflammatory environment that drives obesity, insulin resistance, and liver fibrosis.

KEY POINTS

- The speaker stated that adipose tissue is the largest reservoir of senescent cells that accumulate with ageing and serve as a major source of pro-inflammatory cytokines (SASP).
- Senescent adipocytes were described as impairing lipid storage, releasing free fatty acids and cytokines that promote ectopic fat deposition in liver, pancreas, and muscle, leading to insulin resistance and metabolic dysfunction.
- The speaker reported that inflamed adipocytes exhibit a memory effect that resists weight loss maintenance, associated with epigenetic changes and CD4 T-cell infiltration according to recent articles in Nature and EMBO Reports.
- Mitochondrial dysfunction was identified as highly associated with ageing, driving reactive oxygen species production, decreased energy expenditure, and ectopic fat accumulation.
- The speaker noted that semaglutide has been approved for MASH with fibrosis and suggested GLP-1 therapy may improve outcomes by reducing adipose tissue, though no published data on GLP-1 and senescent ageing currently exist.
- In the Q&A, the speaker stated that new GLP-1/glucagon combination therapies are being developed for obesity and MASH treatment, noting glucagon's role in stimulating fatty acid oxidation in postprandial states.

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