Accelerated immune aging may contribute to obesity-linked metabolic disease

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Obese individuals are at an elevated risk of developing comorbid cardiovascular and metabolic diseases, such as type 2 diabetes. Some research suggests that these comorbid diseases develop in response to chronic inflammation that occurs in visceral adipose tissue, the fat deposits that accumulate around organs. Although obesity is linked to changes in immune cells, it is not clear how these changes influence pathological inflammation in specific fatty tissues.

Motoaki Sano and colleagues at the Keio University School of Medicine examined how changes in immune cell populations contribute to chronic visceral fat inflammation in a new study published this week in the *JCI*.

They found that large amounts of a T cell subtype with the characteristics of aging cells accumulate in the visceral fat of obese mice.

Transplanting this aged T cell population from obese mice into normal mice caused the normal mice to develop obesity-like inflammation in their visceral fat stores.

The normal mice also developed insulin resistance similar to that observed in obese mice.

Identifying this immune cell population as a driving force in obesity-linked inflammation points to a potential therapeutic target for preventing comorbid metabolic and cardiovascular disorders in obese individuals.

More information: Kohsuke Shirakawa et al, Obesity accelerates T cell senescence in murine visceral adipose tissue, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI88606

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