

When it comes to obesity, the problem isn't an excess of fat but its loss of function, researchers argue

February 3 2022



The hallmarks of adipose tissue dysfunction. Credit: Sakers et al./Cell



Obesity is known to cause cardiometabolic diseases like hypertension and diabetes but attributing these diseases to merely an overabundance of fat is a simplification. On a basic level, fat acts as a receptacle to store energy, but upon a closer look it is an essential actor in vital bodily processes like the immune response, the regulation of insulin sensitivity, and maintenance of body temperature. In a review published in the journal *Cell* on February 3rd, researchers argue that the negative health effects of obesity stem not simply from an excess of fat but from the decline in its ability to respond to changes, or in other words, its plasticity.

The makeup and functioning of this tissue changes in response to weight fluctuations and aging. As fat declines in plasticity due to aging and obesity, it loses its ability to respond to bodily cues. In the current model of this phenomenon, the rapid growth of adipose tissue outpaces its blood supply, depriving the fat cells of oxygen and causing the accumulation of cells that no longer divide. This leads to insulin resistance, inflammation, and <u>cell death</u> accompanied by the uncontrolled spill of lipids from these cells.

"The central role of adipose tissue dysfunction in disease and the incredible plasticity of fat tissue supports the promise of modulating fat tissue phenotypes for therapeutic purposes," write the authors, led by Claudio J. Villanueva from the College of Life Sciences/David Geffen School of Medicine and Patrick Seale from Perelman School of Medicine at the University of Pennsylvania. "Many questions and opportunities for future discovery remain, which will yield new insights into adipose tissue biology and hopefully lead to improved therapies for human disease."

More information: *Cell*, Sakers et al. "Adipose tissue plasticity in health and disease" <u>www.cell.com/cell/fulltext/S0092-8674(21)01454-9</u>, <u>DOI: 10.1016/j.cell.2021.12.016</u>



Provided by Cell Press

Citation: When it comes to obesity, the problem isn't an excess of fat but its loss of function, researchers argue (2022, February 3) retrieved 28 June 2024 from https://medicalxpress.com/news/2022-02-obesity-problem-isnt-excess-fat.html

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