

From fat to chronic inflammation

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Researchers may have found a key ingredient in the recipe that leads from obesity to chronic low-grade inflammation, according to a report in the September issue of *Cell Metabolism*.

Chronic inflammation within fat tissue is now recognized as a contributor to the many ill health consequences that come with [obesity](#), from diabetes to cardiovascular disease, explains Yuichi Oike of Kumamoto University in Japan. The new discovery may therefore point to a targeted therapy designed to limit the health impact of the obesity epidemic, the researchers say.

The new culprit Oike's team identifies is a fat-derived protein called angiopoietin-like protein 2 (Angptl2). In mice, Angptl2 levels are elevated in many organs, but especially in fat tissue, they show. Those levels increase further under the oxygen-deprived conditions typically found within obese fat tissue. In humans, too, they find higher Angptl2 levels in the blood of people with higher [body mass index](#) and insulin levels.

Obese mice lacking Angptl2 show less inflammation in their fat tissue and are less insulin resistant, they report. Likewise, otherwise healthy mice made to have higher than normal Angptl2 levels in their [fat tissue](#) develop inflammation and insulin resistance.

They also showed additional details of what Angptl2 does. The protein starts an inflammatory cascade, causing blood vessels to remodel and attracting immune cells called macrophages.

The researchers conclude that Angptl2 is a key adipocyte-derived inflammatory mediator linking obesity to systemic [insulin resistance](#) and identify it as a new molecular target that could be used to improve the diagnosis and treatment of obesity and related metabolic diseases.

Oike says he thinks drugs that would act on Angptl2 not only have considerable promise, but are also likely to come with limited side effects.

"In healthy animals and people, the precise role of Angptl2 has not been clarified," he said. "However, mice in which Angptl2 was deleted genetically were born normally and showed normal growth compared to genetically normal [mice](#). Therefore, we speculate that the possibility of the occurrence of a serious unfavorable side effect due to treatments that decrease Angptl2 expression in animals or people is low."

Source: Cell Press ([news](#) : [web](#))

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