Inflammation in body fat is not only pernicious

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It has been a common opinion that inflammation in adipose tissue may cause insulin resistance, and thereby type 2 diabetes. However, recent research from the Swedish medical university Karolinska Institutet, published in the *New England Journal of Medicine*, question the theory that inflammation in the body fat is only pernicious. Instead the findings suggest that a certain form of body fat inflammation is necessary for fat cell turnover in the lean, healthy state.

In 2008 the same group of researchers showed that adult humans constantly produce new fat cells to replace equally rapid break down of the already existing fat cells due to cell death, and that overweight people generate and replace more fat cells than do lean. It is also well established that obesity is a very strong risk factor for the most common form of diabetes (type 2). The mechanisms linking obesity to diabetes are not clear, but it is believed that disturbances in the function of adipose tissue which are induced by obesity impair the action of insulin on carbohydrate and lipid metabolism (insulin resistance) thereby causing type 2 diabetes.

One of the most investigated pathogenic factors behind the link between insulin resistance and obesity is inflammation of adipose tissue (i.e. where the body fat is stored). Adipose inflammation is pronounced among obese and even more so when obesity is accompanied by type 2 diabetes.

"The hypothesis has been that inflammation in adipose tissue indirectly causes insulin resistance and thereby type 2 diabetes", says Professor Peter Arner, who led the study. "However, when we investigated adipose tissue from healthy and lean young women, we found that inflammation also is necessary for fat cell turnover in the lean, healthy state."

The researchers determined the expression of a number of inflammatory factors produced by the adipose tissue. The most important of these factors in adipose tissue is the signal protein TNF-alpha. This so called cytokine influences the generation, growth and death of fat cells in the body (adipocyte turnover) and also inhibits insulin action thereby causing insulin resistance. The researchers also observed a very strong relationship between, on the one hand, the ability of adipose tissue to produce and secrete TNF-alpha and, on the other hand, the size and number of fat cells in the adipose tissue among the healthy, lean women.

"This observation was only made for TNF-alpha, since there was no relationship between other inflammatory factors and adipocytes size or number", says Professor Arner. "Furthermore, the relationship was not found in obese women who were investigated at the same time as the lean ones."

Professor Arner and his group will now continue their search for how TNF-alpha and other factors regulate the regeneration and break down of fat cells, with the goal to find new targets in adipose tissue for medical treatment of type 2 diabetes.

More information: 'Tumor Necrosis Factor Alpha and Regulation of Adipose Tissue', Erik Arner, Mikael Rydén, Peter Arner, New England Journal of Medicine, 25 March 2010.

Provided by Karolinska Institutet

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