

# Fructose sugar makes maturing human fat cells fatter, less insulin-sensitive

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Fructose, the sugar widely used as high-fructose corn syrup in soft drinks and processed foods, often gets some of the blame for the widespread rise in obesity. Now a laboratory study has found that when fructose is present as children's fat cells mature, it makes more of these cells mature into fat cells in belly fat and less able to respond to insulin in both belly fat and fat located below the skin.

The results will be presented Sunday at The Endocrine Society's 92nd Annual Meeting in San Diego by lead author Georgina Coade, a PhD student at the University of Bristol in the U.K.

"Our results suggest that high levels of fructose, which may result from eating a diet high in fructose, throughout childhood may lead to an increase in visceral [abdominal] obesity, which is associated with increased cardiometabolic risk," Coade said.

Defined by a large waistline, abdominal obesity raises the risk of heart disease and [Type 2 diabetes](#). The [abdominal cavity](#) contains one of two major types of fat in the body: visceral fat. The other type, subcutaneous fat, is found below the surface of the skin.

Although researchers have shown the negative effects of fructose on the fat distribution of rodents, the effects of this sugar on human adipocytes, or fat cells, are not clear, according to Coade. Therefore, she and her fellow researchers studied biopsy specimens of both subcutaneous and visceral fat from 32 healthy-weight children who had not yet gone

through puberty.

From the biopsy samples, the investigators obtained preadipocytes—the precursors to fat cells that have the potential to differentiate, or mature, into fat-containing adipocytes. They then allowed the [precursor cells](#) to mature for 14 days in culture media containing normal glucose (the main sugar found in the bloodstream and the principal source of energy in the body), [high glucose](#) or high fructose. The researchers assessed [cell differentiation](#) by measuring activity of an enzyme (GPDH) and the abundance of the adipocyte fatty acid binding protein, which are both present only in mature fat cells.

Fructose, the research team found, had different effects to that of glucose and caused the fat cells to differentiate more—that is, to form more mature fat cells—but only in visceral fat.

For both types of fat cells, maturation in fructose decreased the cells' insulin sensitivity, which is the ability to successfully take up glucose from the bloodstream into fat and muscles. Decreased insulin sensitivity is a characteristic of Type 2 diabetes.

Although prolonged exposure to fructose had a negative effect on insulin sensitivity, when Coade and her co-workers exposed mature fat cells, rather than preadipocytes, to fructose for 48 hours, the cells' insulin sensitivity increased. The reason why is unknown. However, she said, "Fructose alters the behavior of human fat cells if it is present as the [fat cells](#) mature. We can maybe compare this [timing] to periods in children when they are making their fat."

Provided by The Endocrine Society

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