Mother's milk turns on the heat

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In newborn mice, at least, mother's milk appears to have some rather immediate and potentially far-reaching metabolic consequences. The milk intake kick-starts the liver to produce a molecule that then turns on heat-generating brown fat.

"A key phenomenon required after birth is to adapt the body to a lower environmental temperature with respect to that experienced when the fetus is inside the mother's womb," said Francesc Villarroya of the University of Barcelona. "We find that a key inducer of heat production in neonates is FGF21, released by the liver in response to the initiation of suckling."

FGF21 (short for fibroblast growth factor 21) has recently emerged as a novel regulator of metabolism, Villarroya explained. Scientists knew that FGF21 is produced primarily in the liver, where it is induced after fasting in adult rodents and humans. FGF21 can also correct metabolic disorders of obese and diabetic mice.

In the new study, the researchers wanted to know whether FGF21 also has a role in metabolic shifts as newborn animals transition to life in the world. It appears that it does.

Plasma FGF21 levels and FGF21 gene expression in the liver rise dramatically after birth in mice, the researchers report. That increase is initiated by suckling and depends on the intake of lipid-rich milk. When the researchers mimicked the FGF21 postnatal rise by injecting FGF21 into fasting neonates, they found that the treatment enhanced the expression of genes involved in heat generation, or thermogenesis, within brown fat, to increase body temperature. Brown fat cells treated with FGF21 showed increased expression of thermogenesis genes. The cells also expended more energy and burned more glucose.

Villarroya's team thinks what happens in those first hours of life may have consequences for the individual that carry over into adulthood, noting that FGF21 is a powerful antidiabetic agent.

"There are many evidences that alterations of dietary, genetic, environmental, or other origin in the metabolic performance during the fetal and early neonatal life can make an individual prone to develop diabetes and obesity in adulthood," he said. "The precise mechanisms by which this happens are not fully understood. We observe that a 'natural' event in the postnatal life is a burst in FGF21 levels in response to suckling. It will be important to know whether any disturbance in the intensity of this naturally occurring event may have negative consequences in adulthood."

Villarroya said that there has been something of a revolution in thinking about brown fat in recent years. That's because scientists have found active brown fat in adult humans and have reported evidence that greater activity within brown fat can lend an individual greater resistance to obesity.

He says he suspects the pathways observed in neonatal mice do play similar roles in newborn humans, and maybe in adults, too. "It remains to be demonstrated if FGF21 is also an activator of brown fat in adult humans, but this would be of utmost importance for studies on complex metabolic diseases in adult humans," he says.

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