

Taste buds may play role in fostering obesity in offspring

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Cornell food scientists show in animal studies that a mother's high-fat diet may lead to more sweet-taste receptors and a greater attraction to unhealthy food in their offspring—resulting in poor feeding behavior,

obesity in adulthood.

The researchers' findings were published July 31 in *Scientific Reports*.

Maternal exposure to a high-fat diet during the perinatal period—before the animal gets pregnant—appears to induce physical, detectable changes in the [taste buds](#) for offspring, said senior author Robin Dando, associate professor of food science in the College of Agriculture and Life Sciences.

"We see this is something actually happening in the taste buds themselves," Dando said. "Adult progeny, fed such a diet, have more sweet-taste receptors inside their taste buds than in the control group, whose mothers ate a steady, healthy diet."

Five weeks before mating, [female mice](#) were fed high-caloric, high-fat meals; other mice were also fed the high-fat diet from their pregnancy through lactation.

The progeny, weaned after the lactation period, ate healthy, high-quality laboratory chow. When the offspring became adults, the mice received their first taste of the high-fat diet.

"Up until then, the animals showed no difference between themselves and the [control group](#)," Dando said. "But as soon as the offspring of the moms who consumed the unhealthy diet had access to it, they loved it and they over-consumed it."

The offspring only encountered a [high-fat diet](#) by way of the maternal environment.

"If a mother has an unhealthy diet where she consumes a lot of calories through high-fat and sugary products," Dando said, "the offspring are

going to have a predisposition for liking the unhealthy diet. The origin of this is not only the changes the brain, but there are other [physical changes](#) happening within the taste buds."

As Dando stressed, these results are in mice, but obesity in humans combined with an environmental component, the heritability is between 40% to 70%. "Obesity in the offspring is strongly predicted by the metabolic state of the parents," he said.

While the specific mechanism remains unclear, Dando said, the results introduce the concept of "taste" to the list of metabolic alterations arising from fetal programming.

"Our research adds to the evidence that the taste bud plays a role in the etiology of obesity," he said. "From a public health standpoint, improving our knowledge of prenatal and early postnatal factors that program obesity in [offspring](#) may provide insight into therapeutic targets to combat the obesity epidemic—a disease easier to prevent than to cure."

More information: Ezen Choo et al. Offspring of obese mice display enhanced intake and sensitivity for palatable stimuli, with altered expression of taste signaling elements, *Scientific Reports* (2020). [DOI: 10.1038/s41598-020-68216-7](https://doi.org/10.1038/s41598-020-68216-7)

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