

# Body's anticipation of a meal can be a diabetes risk factor

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Alterations in our response to the taste or smell of food may be another culprit responsible for Type 2 diabetes, according to scientists at Duke University Medical Center who have identified the specific mechanism in human specimens and in mice.

When we anticipate or smell a meal, the parasympathetic nervous system triggers salivation and increases [insulin production](#) in response to the expectation that glucose will be entering the blood stream.

"We think this parasympathetic response is potentially important in type 2 [diabetes](#)," said Vann Bennett, the James B. Duke professor in the departments of cell biology, biochemistry, and neurobiology and Howard Hughes Medical Institute investigator. "Our study showed there is a novel mutation in the gene encoding ankyrin-B, which increases the risk of type 2 diabetes. This happens through an impairment of the [insulin secretion](#) that is added by the parasympathetic nervous system."

The study was released online on Tuesday, March 16 in *Science Signaling*.

Bennett was the first scientist to delineate a molecule called ankyrin and for years has been studying its roles in the heart and brain, as well as other organ systems. Bennett and colleagues discovered the importance of ankyrin-B in the insulin response and the source of a mutation that could lead to diabetes.

In earlier experiments, the group found that [pancreatic beta cells](#) that are ankyrin-B deficient display impaired insulin secretion in mice. Ankyrin-B-deficient mice had high blood sugar after eating a source of glucose, but not if the glucose bypassed the mouse's mouth. These findings indicated that ankyrin-B deficiency impaired the parasympathetic chain of events that enhance insulin secretion and had a measurable impact on blood sugar levels.

The scientists then asked whether mutations involving ankyrin-B loss of function were associated with diabetes in humans. They used the American Diabetes Association's GENNID genetic specimen collection from families with type 2 diabetes to genotype 524 people with diabetes disorders and 498 non-diabetic controls. They were searching for three ankyrin-B mutations that had previously been shown in heart muscle cells to create severe loss of function.

They found that one of these mutations of ankyrin-B (R1788W) was associated with type 2 diabetes in about 1 percent of Caucasian and Hispanic individuals. "Genomewide studies have failed to identify more than a small fraction of the genetic heritability in diabetes as well as in other complex diseases," Bennett said. "There are estimates that only 6 percent of the heritability of type 2 diabetes has been detected, by multiple genomewide studies."

Bennett said this implies there is a large reservoir of genes yet to be identified, that are risk factors in type 2 diabetes. "We are excited by our findings of a specific mutation with a known mechanism because of the potential for personalized treatment of diabetes," he said. "This particular mutation is likely to play a role in 1 percent of adults with diabetes. We hope our finding will lead to strategies to specifically benefit these individuals."

Provided by Duke University Medical Center

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