

# Medical school identifies placental protein as possible birthweight regulator

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New findings from the University of Minnesota Medical School are helping uncover why some people are more likely to be overweight and develop Type 2 diabetes—and it starts in the womb.

Previous association studies have shown that low birthweight among infants is a strong determinant for eventual obesity and Type 2 [diabetes](#).

The placenta of infants with a low birthweight have reduced levels of mTOR (mechanistic target of rapamycin), and the placenta of bigger infants have increased levels of mTOR. Building off of that research, a U of M Medical School study, published in *JCI Insight*, is the first to directly implicate mTOR, a nutrient-sensor protein in the placenta, as a possible regulator of an infant's birthweight.

"It is clear from human and preclinical studies that Type 2 diabetes has fetal origins, but we do not yet know the mechanisms of how this programming of metabolic dysfunction or Type 2 diabetes occurs," said senior author, Emilyn Alejandro, Ph.D., an associate professor in the Department of Integrative Biology and Physiology. "Our study is the first to show a direct role of a placental protein, like mTOR."

They found that in preclinical studies:

- After eliminating mTOR in the placenta, [female offspring](#) had lower birthweights and had an [increased risk](#) for obesity and insulin resistance in adulthood.
- In contrast, after increasing mTOR signaling in the placenta, female adult [offspring](#) were protected from high-fat diet induced obesity.

"A [causal relationship](#) between placental mTOR and the metabolic health of the offspring has not been tested before, and our study suggests that manipulating mTOR in the [placenta](#) is sufficient to cause permanent and lasting impact on the health trajectory of the offspring," said Brian Akhaphong, first author and a post-baccalaureate trainee in the Alejandro Lab. "Our hope is that we can identify proteins that we may target therapeutically through maternal health to reduce the prevalence of Type 2 diabetes."

The research team will continue their study, probing which metabolic

tissues in the offspring are permanently impacted by placental mTOR signaling. Megan Beetch, Ph.D., a postdoctoral fellow, will look at the epigenetics, or heritable changes in gene expression, that do not involve changes to the underlying DNA sequence.

**More information:** Brian Akhaphong et al, Placental mTOR-Complex1 regulates fetal programming of obesity and insulin resistance in mice, *JCI Insight* (2021). [DOI: 10.1172/jci.insight.149271](https://doi.org/10.1172/jci.insight.149271)

Provided by University of Minnesota Medical School

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