Public Abstract
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Gestational diabetes mellitus (GDM) is the most common pregnancy disorder. Women with GDM give birth to offspring that are more likely to develop obesity and diabetes as adults. As offspring experience these adverse effects during their reproductive years, GDM has the potential to propagate disease for many generations.

Obesity drives GDM in a majority of women, but a third of women with GDM are lean. We developed a mouse model with hyperglycemia during pregnancy, but not obesity to study the effects of GDM in offspring of lean mothers. Offspring of lean hyperglycemic mothers had normal glucose tolerance, but were obese.

Leptin, a hormone that promotes satiety in non-pregnant animals but not during pregnancy, was elevated in the lean hyperglycemic mothers, so we used two different high maternal leptin (hyperleptinemia) models to determine whether maternal leptin played a role in protecting the glucose tolerance of the offspring. Maternal hyperleptinemia promoted offspring insulin sensitivity, and improved glucose intolerance.

This work demonstrates that compared to offspring of obese GDM mothers, offspring of lean GDM mothers may become obese while remaining metabolically healthy, and without an increased risk for diabetes. This effect is potentially as a result of maternal hyperleptinemia acting to improve offspring insulin sensitivity. These studies point to leptin as a potential target for improving offspring outcomes in pregnancies complicated by GDM.