Understanding increased insulin secretion in pancreas in healthy pregnant women

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Description

Pregnancy involves several physiological adjustments that are essential to prevent refusal of conception and to ensure successful fetal development. In healthy pregnant women, the insulin secretory response increases gradually, but peripheral insulin sensitivity declines only in the third trimester of pregnancy. Increased insulin secretion appears to be independent of insulin sensitivity, suggesting that the pancreas undergoes unique adaptations during pregnancy.

Whether functional enhancement, decreased apoptosis, increased proliferation, or neo-genesis of these cells results in increased insulin secretion is still debated, but metabolic alterations of prolactin, human placental lactogen, are still being debated. It is clearly associated with increased serum levels and progesterone. In healthy women, decreased serum PRL levels during early pregnancy were independent predictors of decreased cellular function, whole body insulin sensitivity, and glucose tolerance during pregnancy and postpartum. The low potency of PRL can be explained by the low expression of the receptor in human cells. This is also associated with a limited proliferative response during pregnancy. In contrast to humans, the detection of pancreatic-specific knockout mice clearly demonstrates how important PRL is for increased cell mass and glucose-dependent insulin secretion.

Patients with T1D improve glycemic control during pregnancy, but it is not clear whether this is simply the result of increased motivation and frequent metabolic control advice from medical professionals, or whether resuming insulin production can also contribute. Studies measuring fasting or induced C-peptide, a surrogate marker of insulin production, in pregnant women are conflicting. Prospective studies examining changes in pregnancy within the peripheral immune system in T1D are also lacking. We therefore wanted to investigate the long-term effects of pregnancy on endogenous insulin production in women with L-T1D used an immunoassay. Function Insulin secretion was shown to recover during pregnancy but slowly decline after delivery. Analyzed plasma protein levels were dynamic in pregnant women with L-T1D, although a limited number of analytes may be associated with improved cell function.

Several mechanisms within the pancreas have been proposed that lead to increased insulin secretion in healthy pregnant women. Cells are known to be a heterogeneous population containing proliferative subsets and quiescent cells with a mature phenotype. Furthermore, it was recently found that L-T1D patients retain a small number of cells. Although our study does not provide evidence or mechanisms for reduced cell apoptosis, proliferation, or neogenesis in pregnant women with T1D, an intriguing link between increased C-peptide secretion and circulating proteins indicating improved cellular function Higher serum PRL levels in parallel with improved glucose-induced insulin secretion during pregnancy suggest that PRL modulates secretory pathways in residual cells.

The decline in CCL11 levels between the first and third trimesters of pregnancy is consistent with findings in healthy women who report that CCL11 and other pro-inflammatory chemokines decrease during pregnancy and rise again after delivery increase. Including NP women with L-T1D allowed us to compare metabolic and protein variables with controls. These features make it difficult to draw firm conclusions about the origin and functional consequences of different plasma proteins and thus their impact on pancreatic islets. It is also important to question the relevance of imputed protein annotations, as many bio-informatic data are based on genetic analyses.

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Conflict of Interest

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