Prolactin receptor signaling linked to expansion of beta-cells

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(HealthDay)—Prolactin receptor (PRLR) signaling plays a role in

expansion of maternal β-cells during pregnancy, according to a study published online May 23 in *Diabetes*.

Ronadip R. Banerjee, M.D., from the Stanford University School of Medicine in California, and colleagues generated a 'floxed' *Prlr* allele that allowed conditional loss of PRLR in β-cells in order to examine the in vivo requirement for PRLR signaling.

The researchers found that PRLR signaling loss in β-cells resulted in gestational diabetes mellitus (GDM), and in reduced proliferation of β-cells and failure to expand β-cell mass during pregnancy. Expression of the transcription factor *Foxm1*, both G_1/S and G_2/M cyclins, tryptophan hydroxylase 1, and islet serotonin production were impaired with targeted PRLR loss in maternal β-cells in vivo. PRLR signaling was also required for the transient gestational expression of the MafB transcription factor within a subset of β-cells in pregnancy. GDM was also produced with deletion of *MafB* in maternal β-cells; inadequate β-cell expansion was accompanied by failure to induce PRLR-dependent target genes, which regulate proliferation of β-cells.

"These results unveil molecular roles for PRLR signaling in orchestrating the physiologic expansion of maternal β-cells during pregnancy," the authors write.

More information: Full Text (subscription or payment may be required)