

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 G₁/S and G₂/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 \(subscription or payment may be required\)](#)

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