



**Figure 2 – figure supplement 1. Model**

(Left) Loss of *Maf* and *Mafb* in the MGE leads to overproduction of HINs (*Sst*<sup>+</sup> and/or *Pnoc*<sup>+</sup>). (Right) DEX analysis revealed that at P0, *Maf* cDKOs have upregulated expression of *Sst*, *Npy*, *Nrp1* and *Pnoc*; downregulated expression of *Maf*, *Mafb*, *Mef2c*, *Cxcr4*, *Snap25*, *Nxph1* and *Nxph2*. Our data suggested that prenatally, *Maf* and *Mafb* promote *Mef2c* expression to drive PV IN production; postnatally, *Maf* and *Mafb* regulate *Mef2c* and *Snap25* expression to regulate IN neurite morphogenesis.