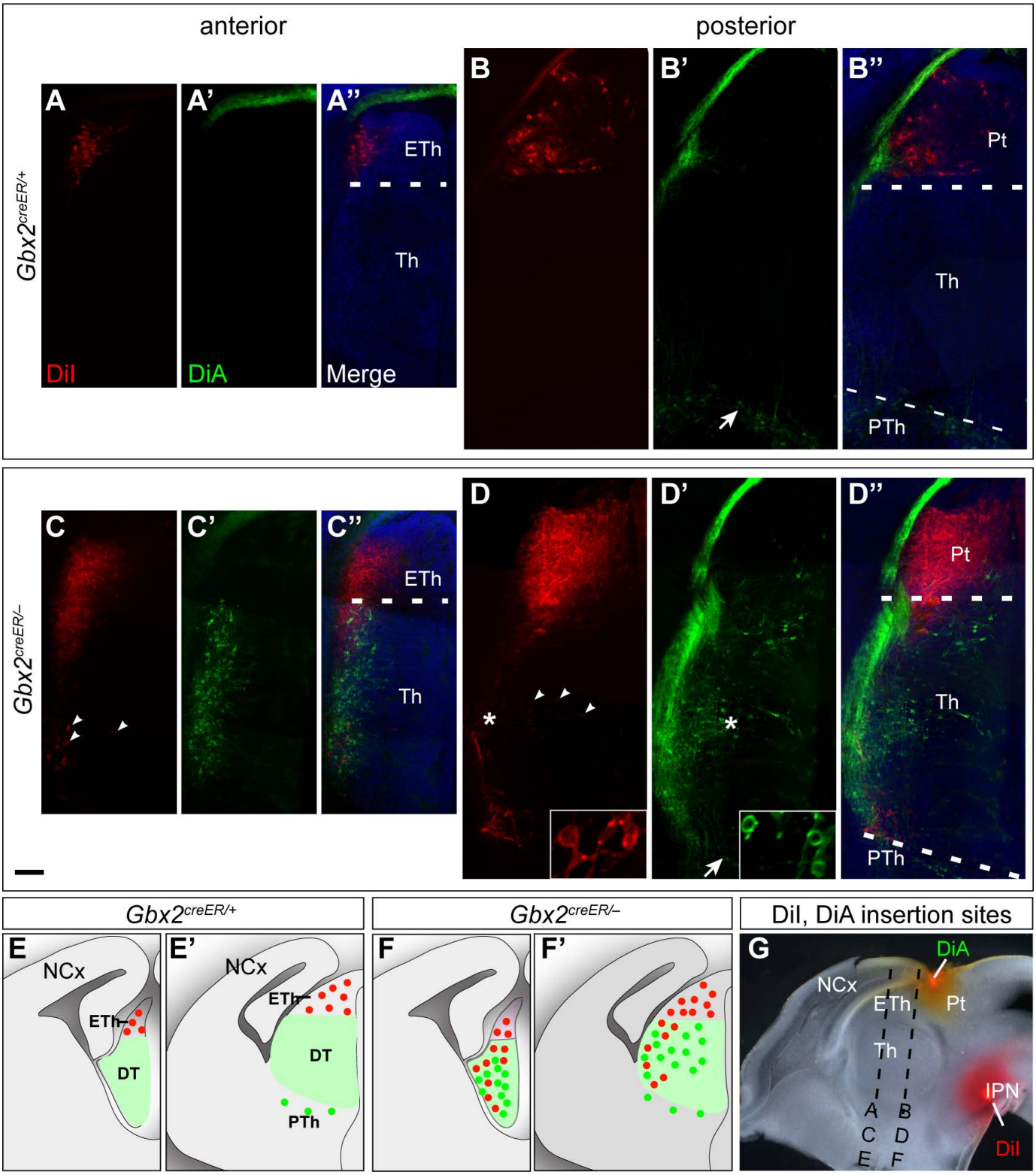

Fig. S1. Analyses of trajectory defects of thalamic axons lacking *Gbx2* using lipophilic dye labeling. (**A-D″**) Confocal images of coronal sections of E14.5 control (A-B″) and *Gbx2*-mutant (C-D″) brains at the anterior and posterior levels. Broken lines indicate the rostral and caudal limits of the thalamus; arrows indicate back-labeled DiA^+ cells in the thalamic reticular nucleus; arrowheads indicate ectopic DiI^+ and DiA^+ neurons, respectively, in the thalamus lacking *Gbx2*; asterisks demarcate cells that are enlarged in insets (D,D′). (**E-F′**) Schematic presentation of the distribution of the back-labeled neurons by DiI (red) and DiA (green). (**G**) Bisected E14.5 brain to show that DiI and DiA crystals are placed in the ventral mesencephalon and dorsal midline of the diencephalon, respectively. Broken lines indicate the plane of sections. Scale bar: 200 μm.

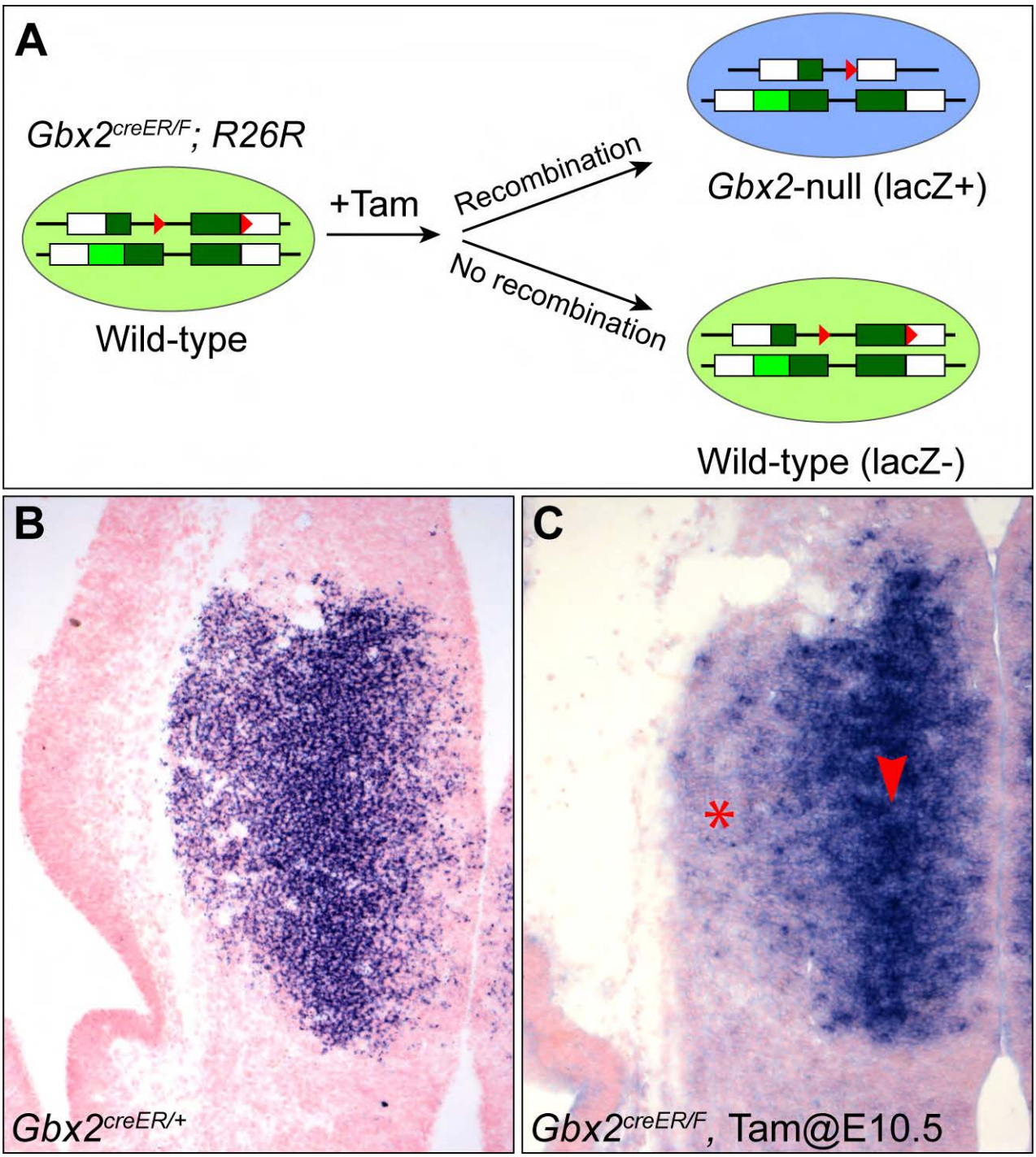

Fig. S2. Generation of *Gbx2* mosaic mutation. (**A**) Schematic representation of the generation of *Gbx2* mosaic mutant thalamus. (**B**,**C**) In situ hybridization using a *Gbx2* RNA probe that recognizes sequence that is deleted by Cre-mediated recombination on coronal section of E12.5 $Gbx2^{creER/+}$ (B) and $Gbx2^{creER/F}$ (C) embryos that received tamoxifen at E10.5. The *Gbx2* expression in the intermediate zone (arrowhead) is unaffected, but is mostly abolished in the mantle zone (asterisk) in $Gbx2^{creER/F}$ embryos.