Figure 2 Supplement

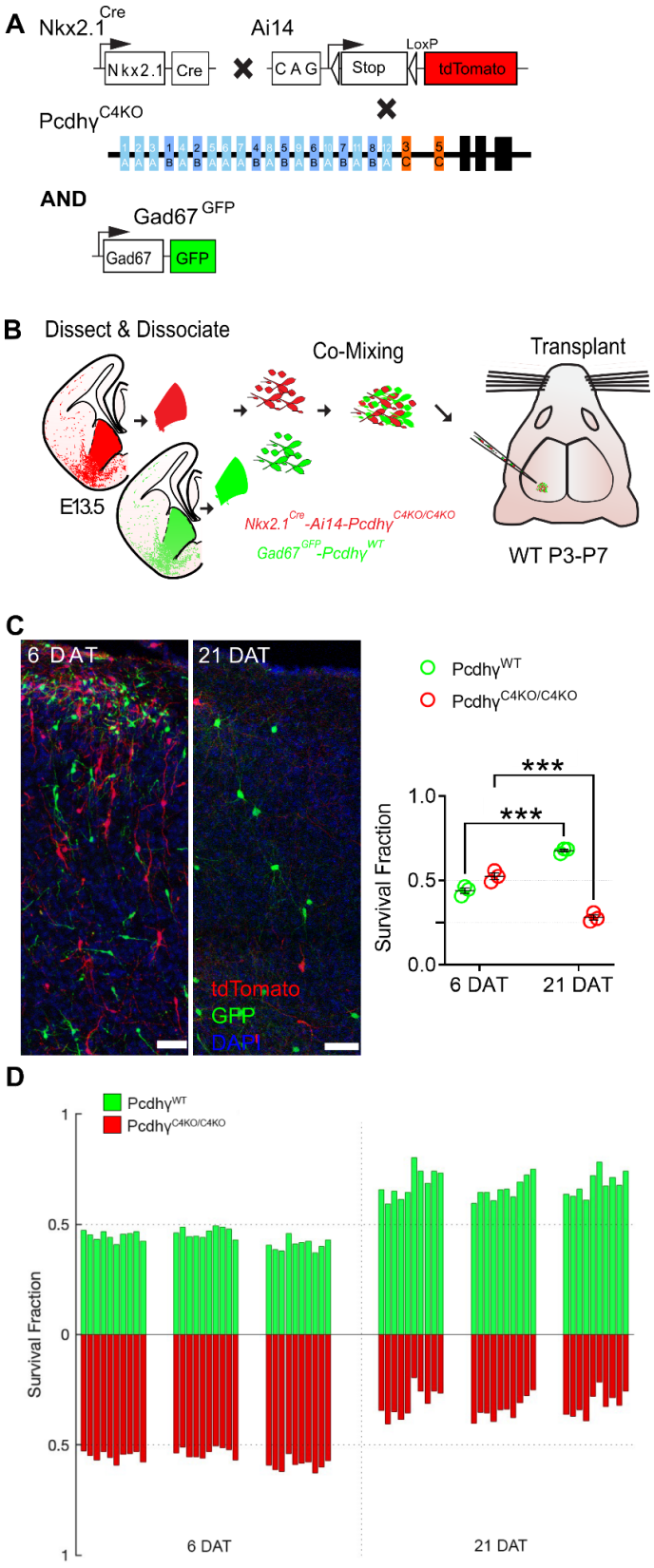

Figure 2 Supplement - Genetic deletion of Pcdhγc4 increased cell death in MGE-derived cINs.

A. Diagram of genetic crosses. Pcdhγ^{C4KO} homozygous MGE cells were labeled via the MGE-specific genetic reporter *Nkx2.1^{Cre}* mice that also carry the conditional Ai14 allele. Control Pcdhγ^{WT} MGE cells were labeled with GFP via the Gad67-GFP report mice.

B. Schematics of transplantation protocol. The MGEs of Pcdhγ^{C4KO} homozygous mutant or control E13.5 embryos were dissected, dissociated, and mixed in similar proportions. The mixture of GFP+ (Pcdhγ-WT) and tdTomato+ (Pcdhγ$^{C4KO/C4KO}$) cells were grafted into the cortex of WT neonate mice.

C. Left - Confocal images from the cortex of 6 and 21 DAT mice. The transplanted cells were labeled with GFP (Pcdhγ^{WT}) or tdTomato (Pcdhγ$^{C4KO/C4KO}$). Right - Quantifications (shown as survival fraction) of surviving GFP or tdTomato labeled MGE-derived cINs at 6 and 21 DAT. Both the GFP and tdTomato labeled cells undergo programmed cell death between 6 and 21DAT, but Pcdhγ$^{C4KO/C4KO}$ cells are eliminated at higher rates.

D. Survival fraction quantification from (C) shown by the brain section (each bar) and separated by animals at 6 and 21 DAT.

Scale bar = 50 um, Nested-ANOVA, ***p = 0.0002 , n = 3 mice per time point and 10 brain sections quantified per mouse.

Figure 3 supplement

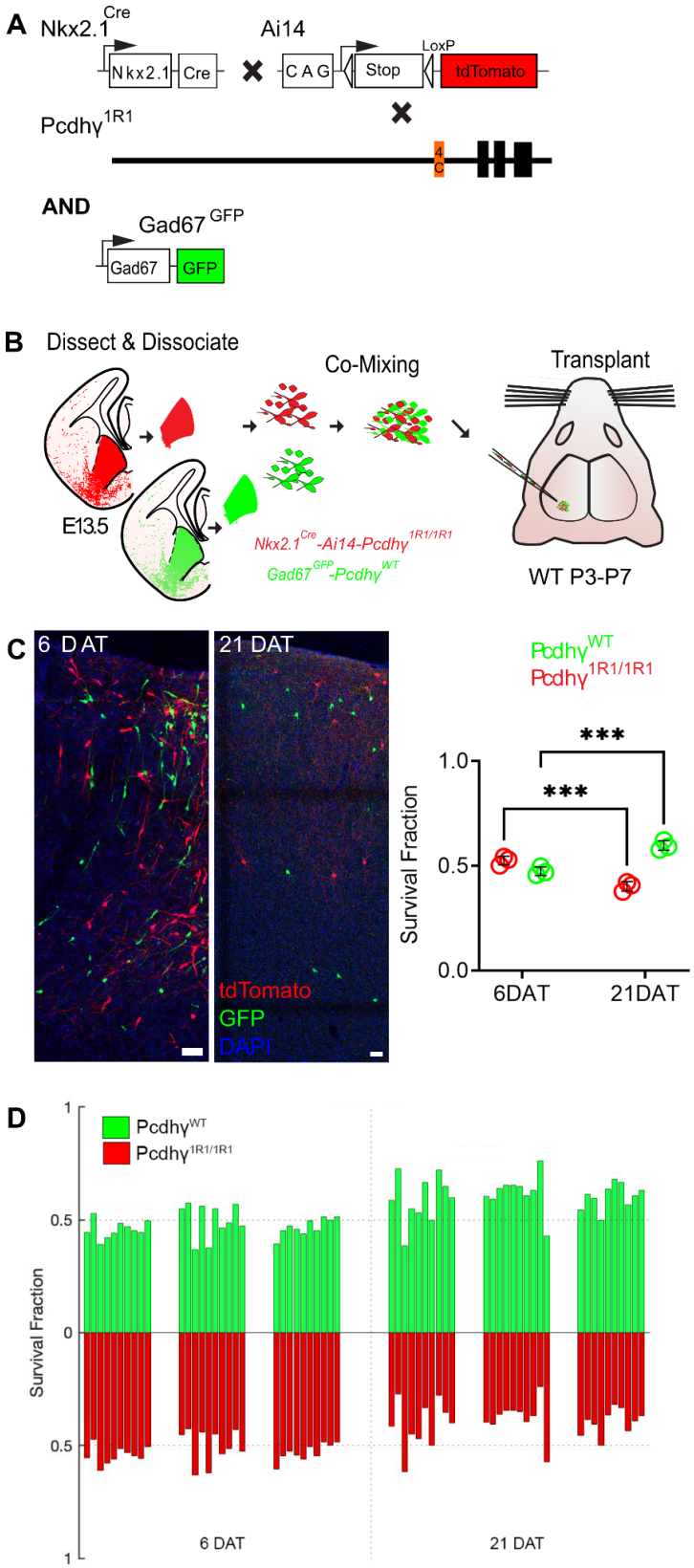

Figure 3 - supplement - Expression of Pcdhγc4 is sufficient for the survival of most MGE-derived cINs.

A. Diagram of genetic crosses between MGE/POA-specific reporter *Nkx2.1*^{Cre};Ai14 and Pcdhγ^{1R1} mice. Control cells were obtained from *Gad67-GFP* embryos.

B. Schematics of transplantation protocol. The MGEs from E13.5 Pcdhγ^{1R1} homozygous or control embryos were dissected, dissociated, and mixed in similar proportions. The mixture of GFP+ (Pcdhγ^{WT}) and tdTomato+ (Pcdhγ$^{1R1/1R1}$) cells was grafted into the cortex of WT neonate mice.

C. Left - Confocal images from the cortex of 6 and 21 DAT mice. The transplanted cells are labeled with GFP (Pcdhγ^{WT}) or tdTomato (Pcdhγ$^{1R1/1R1}$). Right - Quantifications (shown as survival fraction) of surviving MGE-derived cINs at 6 and 21 DAT. Both the transplanted GFP and tdTomato-labeled cells undergo programmed cell death between 6 and 21 DAT, but the Pcdhγ$^{1R1/1R1}$ cells are eliminated at higher rates.

D. Survival fraction quantification from (C) shown by the brain section (each bar) and separated by animals at 6 and 21 DAT.

Scale bar = 50 um, Nested-ANOVA , ***p = 0.009 , n = 3 mice per time point and 10 brain sections per mouse

Figure 4 supplement 2

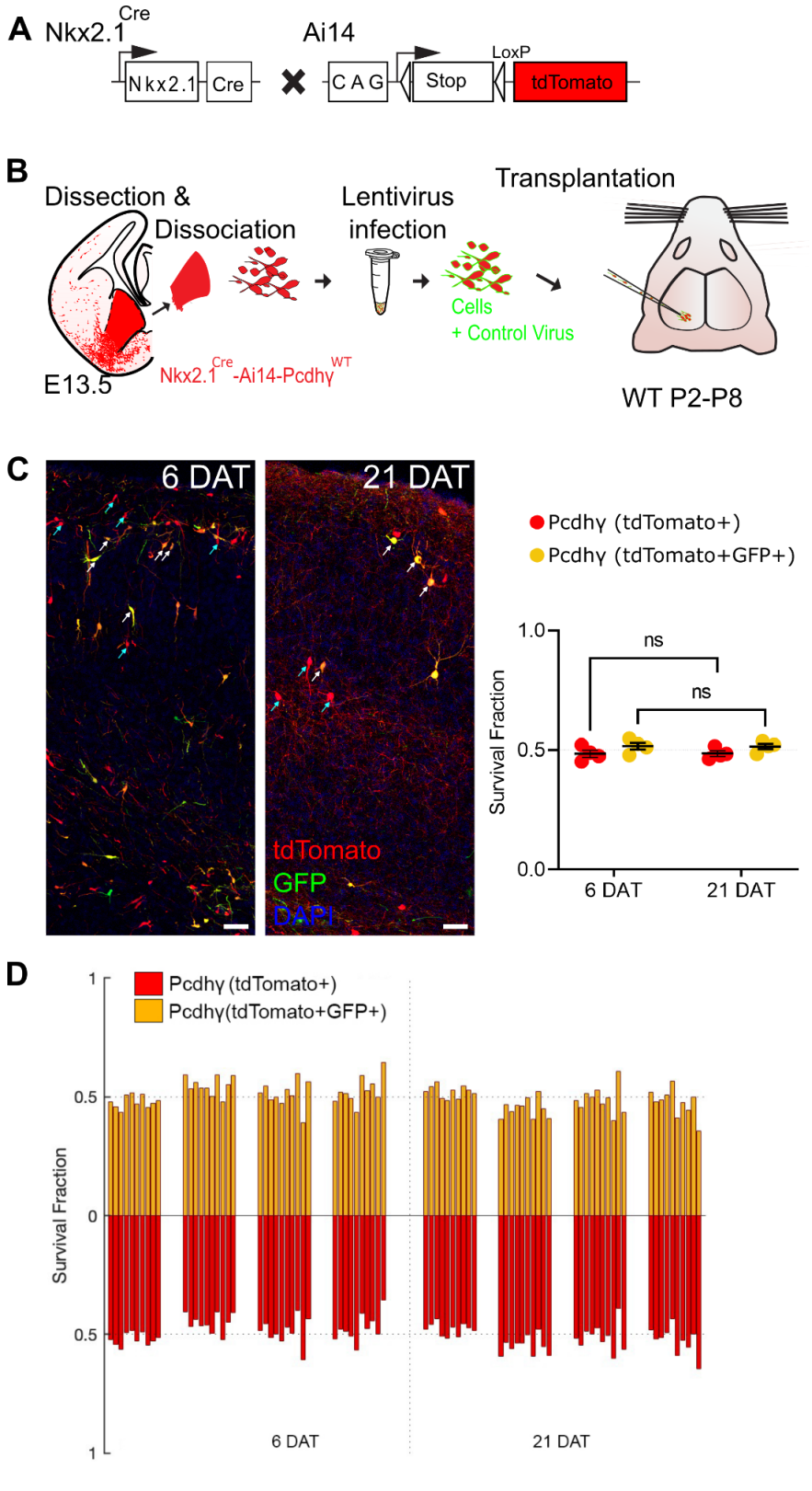

Figure 4 supplement - Infection with lentivirus and expression of GFP does not affect the survival of cINs

A Diagram of mouse crosses to obtain Pcdhγ^{WT} MGE cells labeled with MGE-specific genetic reporter.

B. Schematic of lentiviral infection and transplantation of MGE cIN precursors. The MGEs of *Nkx2.1*^{Cre}-Ai14 embryos that carry Pcdhγ^{WT} were dissected, dissociated, and infected in suspension with lentivirus expressing GFP. The infected cells were grafted into the cortex of WT recipient mice P0-P8.

C. Confocal acquired images of the transplanted cINs in the cortex at 6 and 21 DAT. Notice that all transplanted cells are labeled with tdTomato (red), while cells expressing virally-driven GFP are labeled in green. Quantifications of the tdTomato+ only (teal arrows) or tdTomato+GFP+ (yellow cells, white arrows) cells are shown as the fraction of cells from the total tdTomato+ cells at 6 and 21 DAT.

D. Survival fraction quantification from (C) shown by the brain section (each bar) and separated by animals at 6 and 21 DAT.

Scale bar = 50 um, Nested-ANOVA , ns = not significant, n = 4 mice per time point and 10 brain sections per mouse from one transplant cohort.