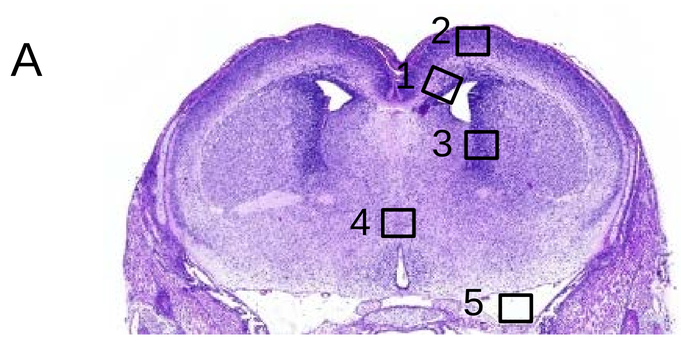

1) Neocortex (deep layers)
2) Neocortex (upper layers)
3) SVZ
4) Thalamic region
5) NR-like structure

		Neocortex (deep layers)	Neocortex (upper layers)	SVZ	Thalamic region	NR-like structure	
Brg1$^{fl/fl}$	Deletion E7.5	B	C	D	E	*Structure is not present.*	BRG1
Sox2-creER^{T2}*::Brg1*$^{fl/fl}$	Deletion E7.5	F	G	H	I	J	
	Deletion E9.5	K	L	M	N	*Structure is not present.*	
Ini1$^{fl/fl}$	Deletion E9.5	O	P	Q	R	*Structure is not present.*	INI1
Sox2-creER^{T2}*::Ini1*$^{fl/fl}$	Deletion E7.5	S	T	U	V	*Structure is not present.*	
	Deletion E9.5	W	X	Y	Z	*Structure is not present.*	

Fig. S1: Overview of *Brg1* and *Ini1* deficient cells in *Sox2-creER*^{T2}*::Brg1*$^{fl/fl}$ and *Sox2-creER*^{T2}*::Ini1*$^{fl/fl}$ embryos. A schematic overview of all regions with morphological alterations and/or BRG1/INI1 negative cells in mutants is shown in A. In control brains, all cells stain positive for BRG1 (B-E). After *Brg1* deletion at E7.5, only few BRG1 negative cells are found in the neocortex (F, G) and the SVZ (H), whereas about 50 % of cells in the cell accumulation close to the basal part of the cerebrum (J) have lost its expression. In mutants in which *Brg1* loss was introduced at E9.5, the number of BRG1 negative cells in the neocortex is more pronounced, but the majority retained its expression (K, L). The knockout in the SVZ is similar to the E7.5 mutants (M). In both mutant groups, there are BRG1 negative cells in the thalamic region (I, N). The INI1 staining in *Sox2-creER*^{T2}*::Ini1*$^{fl/fl}$ embryos and controls reveals that in the latter, all cells in the brain are INI1 positive (O-R). In mutants, INI1 negative cells are predominantly found in the thalamic region and in case of INI1 deprivation at E9.5 also in the neocortex and seldom in the SVZ (S-Z). All panels show representative images of E18.5 frontal brain sections in a 200x magnification. Yellow and blue arrows highlight BRG1 and INI1 negative cells, respectively. Scale bar in B is representative for all panels and corresponds to 20 µm. n≥3.

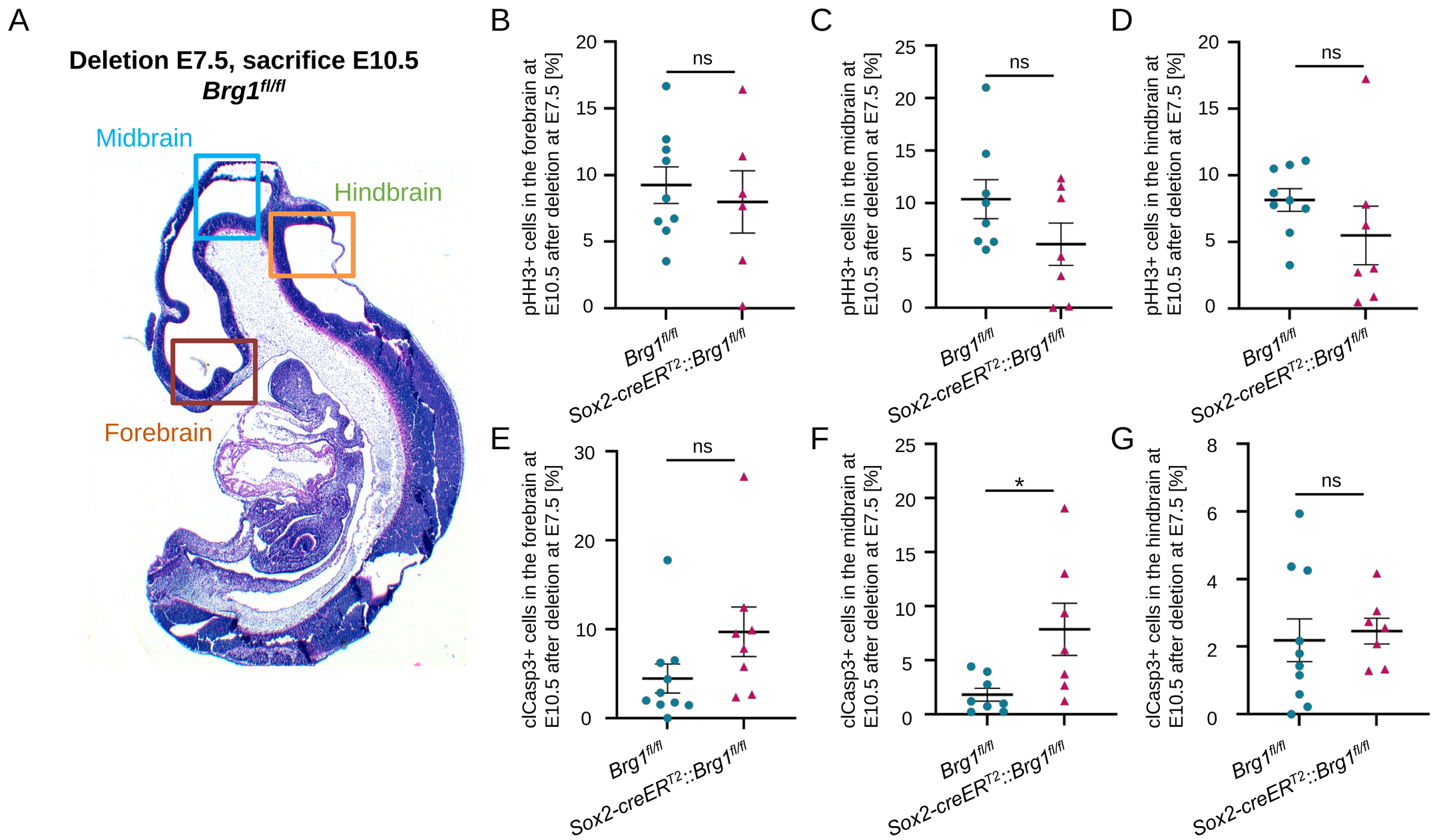

Fig. S2: Increased apoptosis after *Brg1* loss at E7.5 is predominantly found in the developing midbrain and still detectable at E12.5. A illustrates the regions forebrain, midbrain and hindbrain at E10.5 that were used for quantifications of clCasp3 and pHH3 stainings (10x magnification). Abundance of pHH3 positive cells in the forebrain (B), midbrain (C) and hindbrain (D) is compared in *Brg1*$^{fl/fl}$ and *Sox2-creER*^{T2}*::Brg1*$^{fl/fl}$ embryos at E10.5. Proportion of clCasp3 positive cells in the forebrain (E), midbrain (F) and hinbrain (G) is compared in *Brg1*$^{fl/fl}$ and *Sox2-creER*^{T2}*::Brg1*$^{fl/fl}$ embryos at E10.5. H and J show respective H&E stains of frontal E12.5 sections of *Brg1*$^{fl/fl}$ and *Sox2-creER*^{T2}*::Brg1*$^{fl/fl}$ embryos, respectively. High power images (200x magnification) highlight H&E (I, K), clCasp3 (L, N) and BRG1 stainings (M, O) in regions that harbor Brg1 negative cell accumulations in *Sox2-creER*^{T2}*::Brg1*$^{fl/fl}$ embryos. All analyzed embryos received tamoxifen at E7.5. ns: not significant, *p<0.05; Number of animals: n≥3

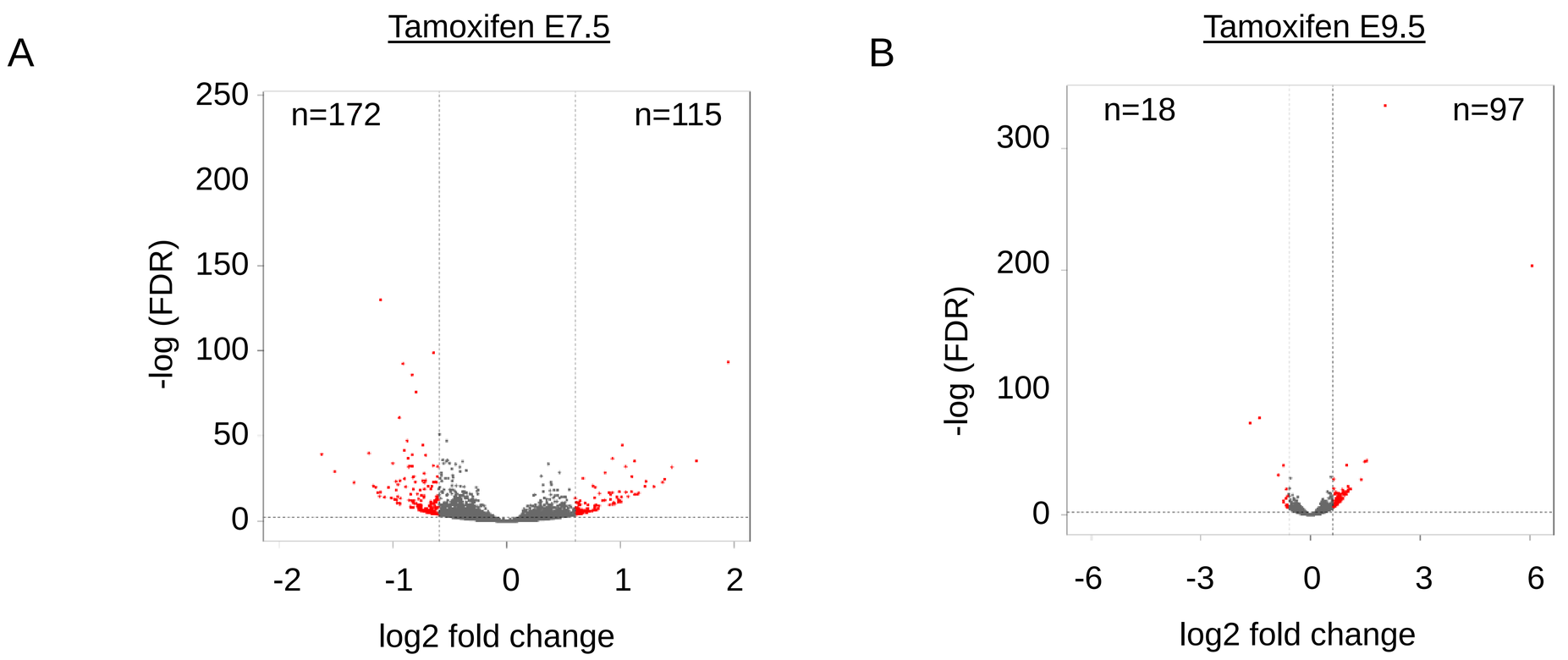

Fig. S3: Volcano Plots of differentially expressed genes. Volcano plots show 172 (deletion E7.5) and 18 (deletion E9.5) downregulated as well as 115 (deletion E7.5) and 97 (deletion E9.5) upregulated genes in *Brg1* deficient compared to *Brg1* competent cells (A, B). Red dots mark differentially expressed genes, defined by a log2 fold change > +/- 0.6 and a FDR <0.1.

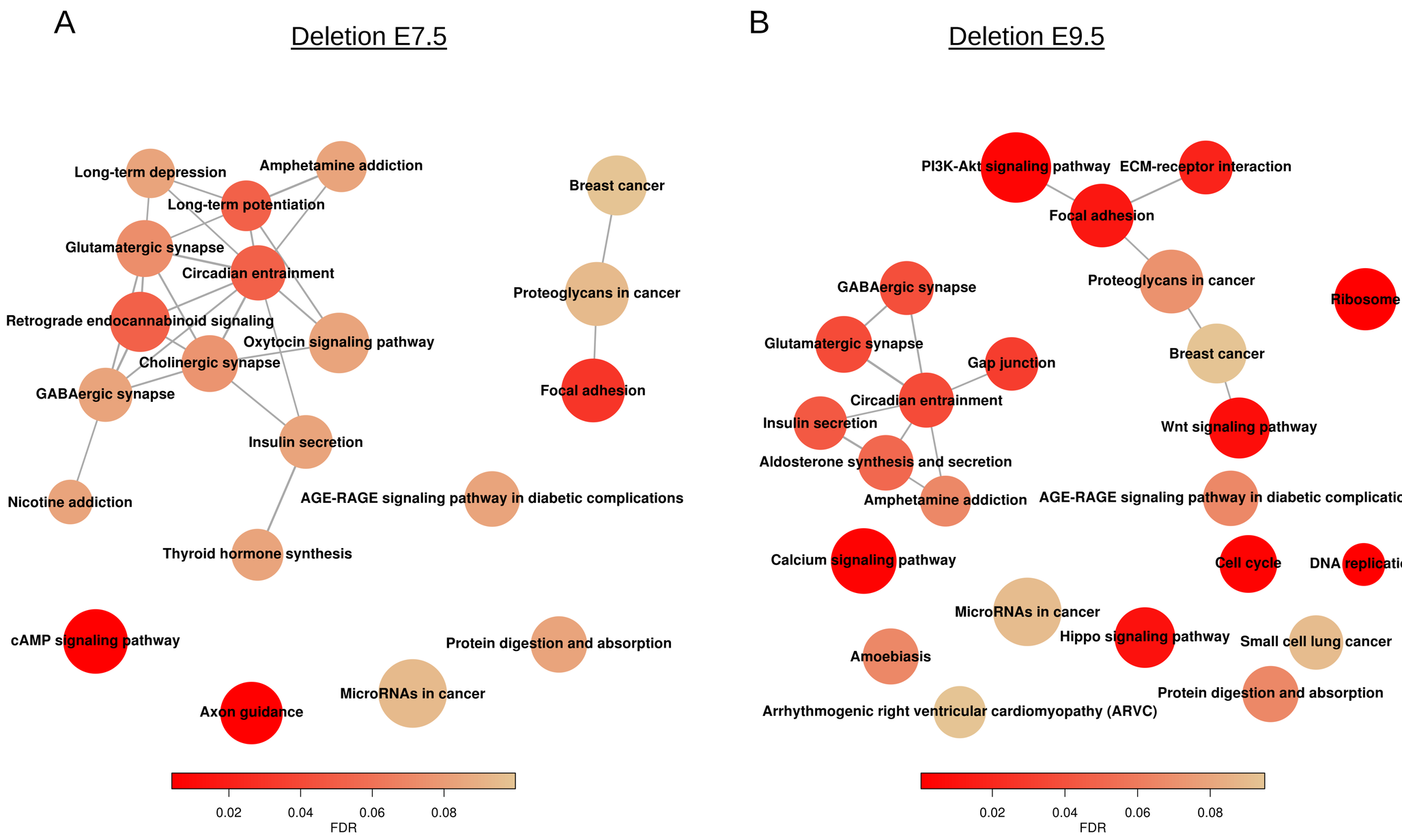

Fig. S4: KEGG pathway analysis of RNA sequencing data. KEGG pathway analyses are depicted after tamoxifen-induced *Brg1* loss at E7.5 (A) and E9.5 (B). Color scale indicates the false discovery rate (FDR).

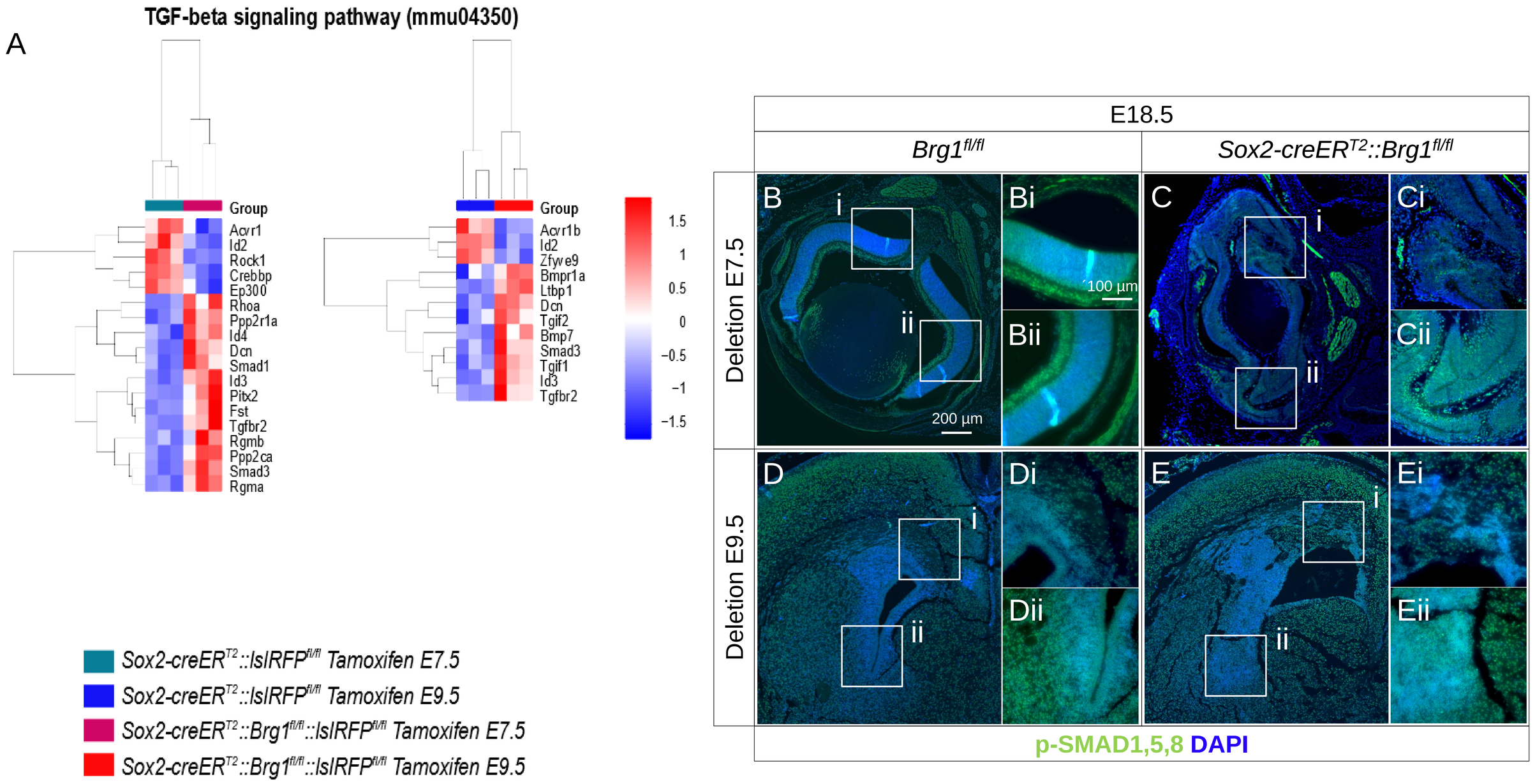

Fig. S5: Alterations in TGFβ signaling after loss of *Brg1* at E7.5 or E9.5. Genes of the TGFβ signaling pathway as defined by KEGG (mmu04350) that differ significantly (FDR<0.1) between RFP positive cells derived from *Sox2-creER^{T2}::lslRFP$^{fl/fl}$* and *Sox2-creER^{T2}::Brg1$^{fl/fl}$::lslRFP$^{fl/fl}$* E14.5 brains show clustering according to group (A). IF-P of p-Smad1,5,8 show no alterations in the eyes of mutants and controls with *Brg1* deletion at E7.5 (B, C). Likewise, there are no differences in the neocortex (D, Di, E, Ei) or the SVZ (D, Dii, E, Eii) after *Brg1* loss at E9.5 in mutants and controls. Scale bar in B corresponds to C, D and E and the scale bar in Bi is representative for all other high power images. All images were taken from E18.5 embryos representative for at least 3 animals per group in a 100x magnification (B,C,D,E) or 200x magnification (Bi,ii,Ci,ii,Di,ii,Ei,ii) .

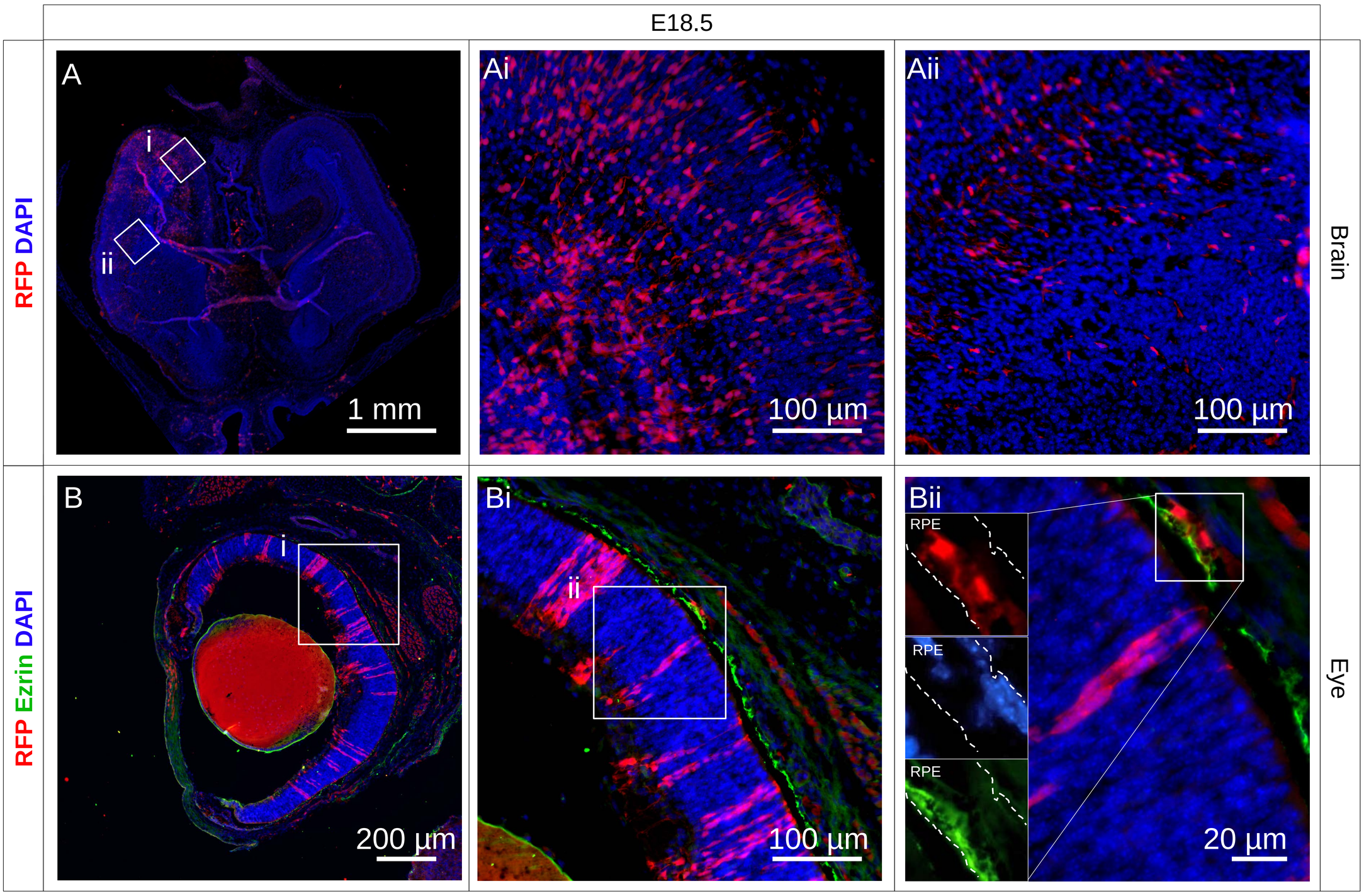

Fig. S7: Cre reporter pattern at E18.5 identifies progenies of cells in which the Cre recombinase became active at E7.5. RFP positive cells (red) descend from cells in which tamoxifen treatment at E7.5 activated the Cre recombinase. These cells are detected in the brain (A, Ai, Aii), in the neural retina (B, Bi, Bii), and in the retinal pigment epithelium (RPE) marked by Ezrin stainings (green). Nuclei are counterstained with DAPI (blue). 40x magnification (A), 100x magnification (B), 400x magnification (Ai,ii,Bi), 400x magnification with ROI (Bii).

A

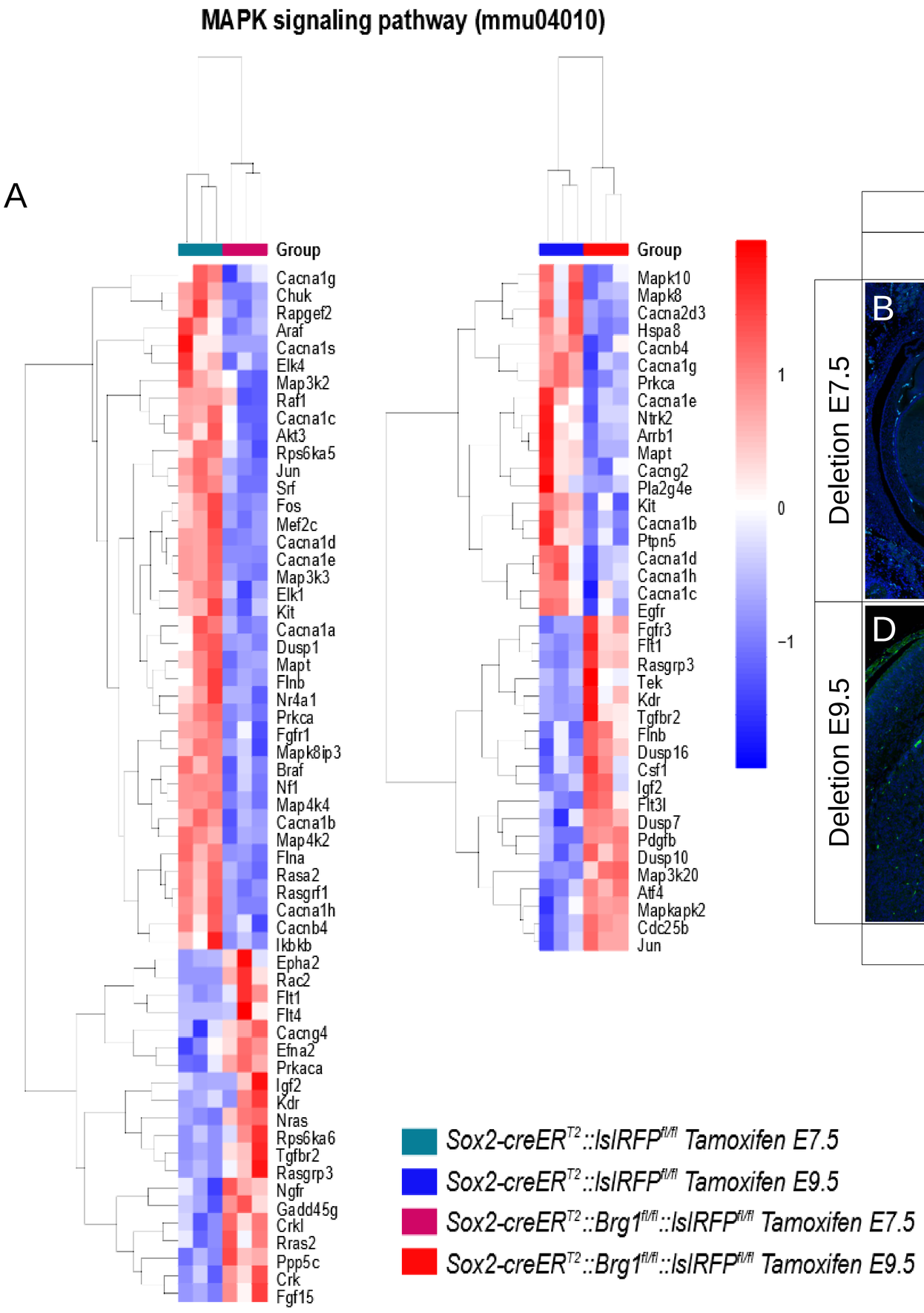

Fig. S8: Alterations in MAPK signaling after loss of *Brg1* at E7.5 or E9.5. Genes of the MAPK signaling pathway as defined by KEGG (mmu04010) that differ significantly (FDR<0.1) between RFP positive cells derived from *Sox2-creER^{T2}::lslRFP$^{fl/fl}$* and *Sox2-creER^{T2}::Brg1$^{fl/fl}$::lslRFP$^{fl/fl}$* E14.5 brains show clustering according to group (A). IF-P of p-ERK 1,2 shows alterations in the eyes of E7.5 mutants and controls (B, C). Likewise, there are differences in the SVZ (D, E) after *Brg1* loss at E9.5 in mutants and controls. Scale bar in B corresponds to C, D and E and the scale bar in Bi is representative for all other high power images. All images were taken from E18.5 embryos representative for at least 3 animals per group in a 100x magnification (B,C,D,E) or a 400x magnification (Bi,Ci,Di,Ei).