

SUPPLEMENTARY FIG. S2. Quantitative analysis of the influence of Ngn2 on vGlut1 and Mash1 expression. (A) The fold changes in the intensity of red fluorescent for Ngn2 staining was monitored between TBI and sham mice. (B) The ratio of Ngn2/Dcx-positive cells was increased in TBI mice as the number of Ngn2-positive cells were increased along with a decrease in Dcx-positive cells. (C) The fold change in the increase in binding of Ngn2 on vGlut1 promoter was increased significantly. Statistical significance was measured by one-way ANOVA with a Tukey-Kramer post hoc correction, n=7, *p<0.05. All data are expressed as mean±SEM. ANOVA, analysis of variance; Dcx, Doublecortin; DG, dentate gyrus; SEM, standard error of the mean; TBI, traumatic brain injury; vGlut1, vesicular glutamate transporter 1.



SUPPLEMENTARY FIG. S3. The quantitative analysis of the influence of HDAC4 on Pax3 acetylation and transcription of Ngn2. (A) The fold change in binding of Pax3 to Ngn2 promoter was increased significantly after TBI. (B) The fold change in Pax3 acetylation was increased after TBI. (C) TBI leads to a decrease in Pax3-HDAC4 interaction to 0.3 folds. (D) TBI leads to a decrease in the expression level of HDAC4 to 0.35 folds. (E) The fold changes in the intensity of red fluorescent for HDAC4 staining was monitored between TBI and sham mice. (F) The ratio of Dcx/HDAC4-positive cells was increased in TBI mice as the number of Ngn2-positive cells was increased along with a decrease in Dcx-positive cells. (G) The induction of pospho-HDAC4 level was decreased after pre-treatment with lithium (5 mg/kg). (H) The reduction in the level of HDAC4 was rescued after treatment with lithium prior to TBI. Statistical significance was measured by one-way ANOVA with a Tukey-Kramer post hoc correction, n=7, *p<0.05. All data are expressed as mean ± SEM. ANOVA, analysis of variance; Dcx, Doublecortin; HDAC4, histone deacetylase 4; SEM, standard error of the mean; TBI, traumatic brain injury.