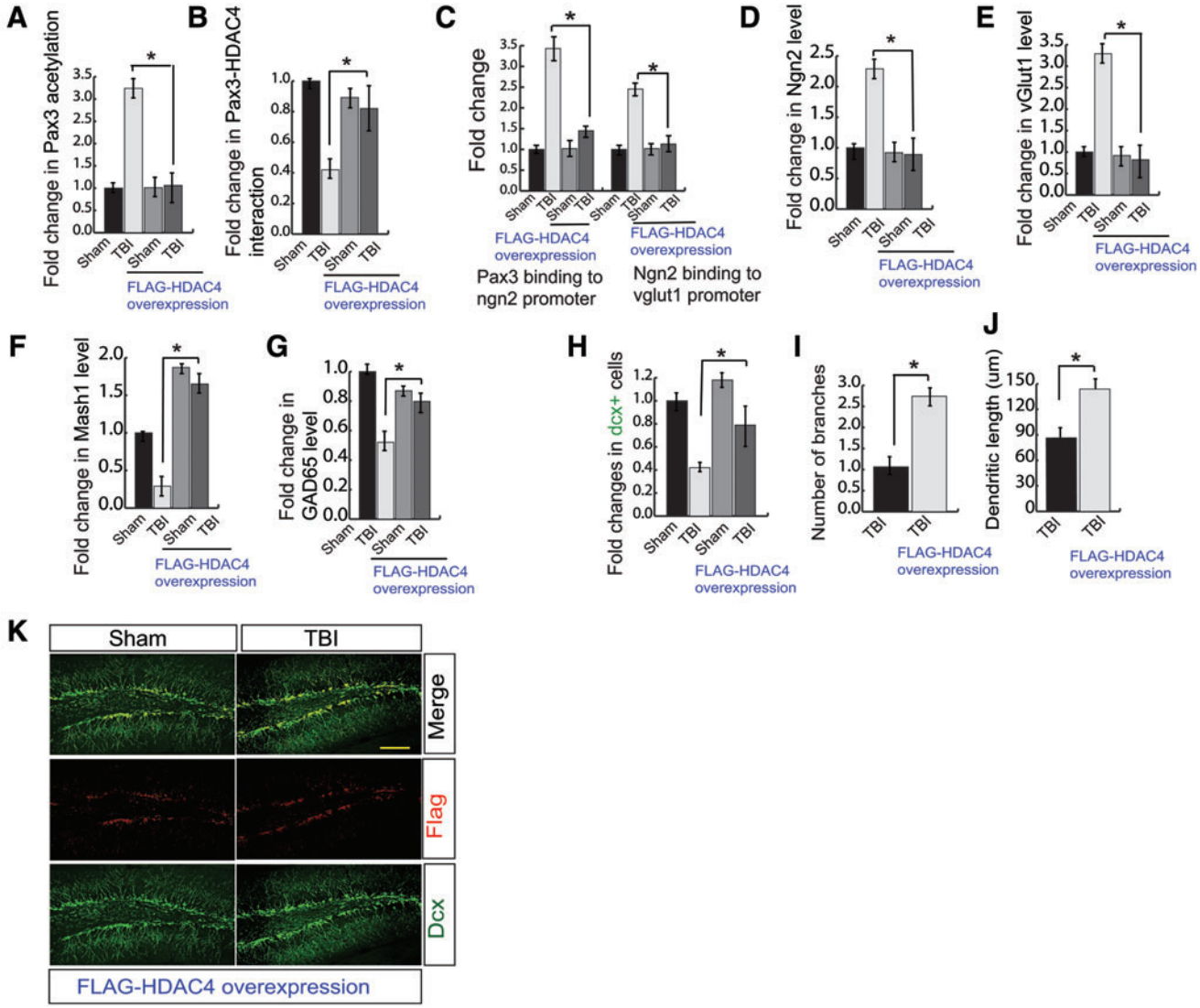

SUPPLEMENTARY FIG. S4. The quantitative analysis of the effect of overexpression of HDAC4 on the transcriptional activity of Pax3, Ngn2, and neurogenesis following TBI. **(A,B)** Overexpression of HDAC4 reduces Pax3 acetylation (A) and the interaction between Pax3 and HDAC4 (B). **(C)** ChIP analysis suggests that the increase in Pax3 binding to the Ngn2 promoter (3.42) was rescued to 1.5 folds and an increase in Ngn2 to the vGlut1 promoter (2.6) was rescued to 1.1 folds. **(D,E)** The fold changes in Ngn2 (D) and in vGlut1 (E) were rescued significantly. **(F,G)** The decrease in Mash1 and GAD65 was blocked after overexpression of HDAC4 and the fold changes in Mash1 (F) and GAD65 (G) were rescued significantly. **(H)** The Dcx-positive cells after overexpression of HDAC4 following TBI were rescued. **(I,J)** The loss of the number of branches (I) and total dendritic length (J) were rescued in mice upon overexpression of HDAC4 in the hippocampus after TBI. **(K)** Confocal microscopic analysis to monitor the overexpression of HDAC4 by staining with Flag antibody along with Dcx. The expression level of HDAC4 remains identical in the DG of hippocampus after sham or 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. Scale bar = 20 μm. ANOVA, analysis of variance; ChIP, chromatin immunoprecipitation; Dcx, Doublecortin; DG, dentate gyrus; GAD65, glutamic acid decarboxylase 65; HDAC4, histone deacetylase 4; SEM, standard error of the mean; TBI, traumatic brain injury; vGlut1, vesicular glutamate transporter 1.