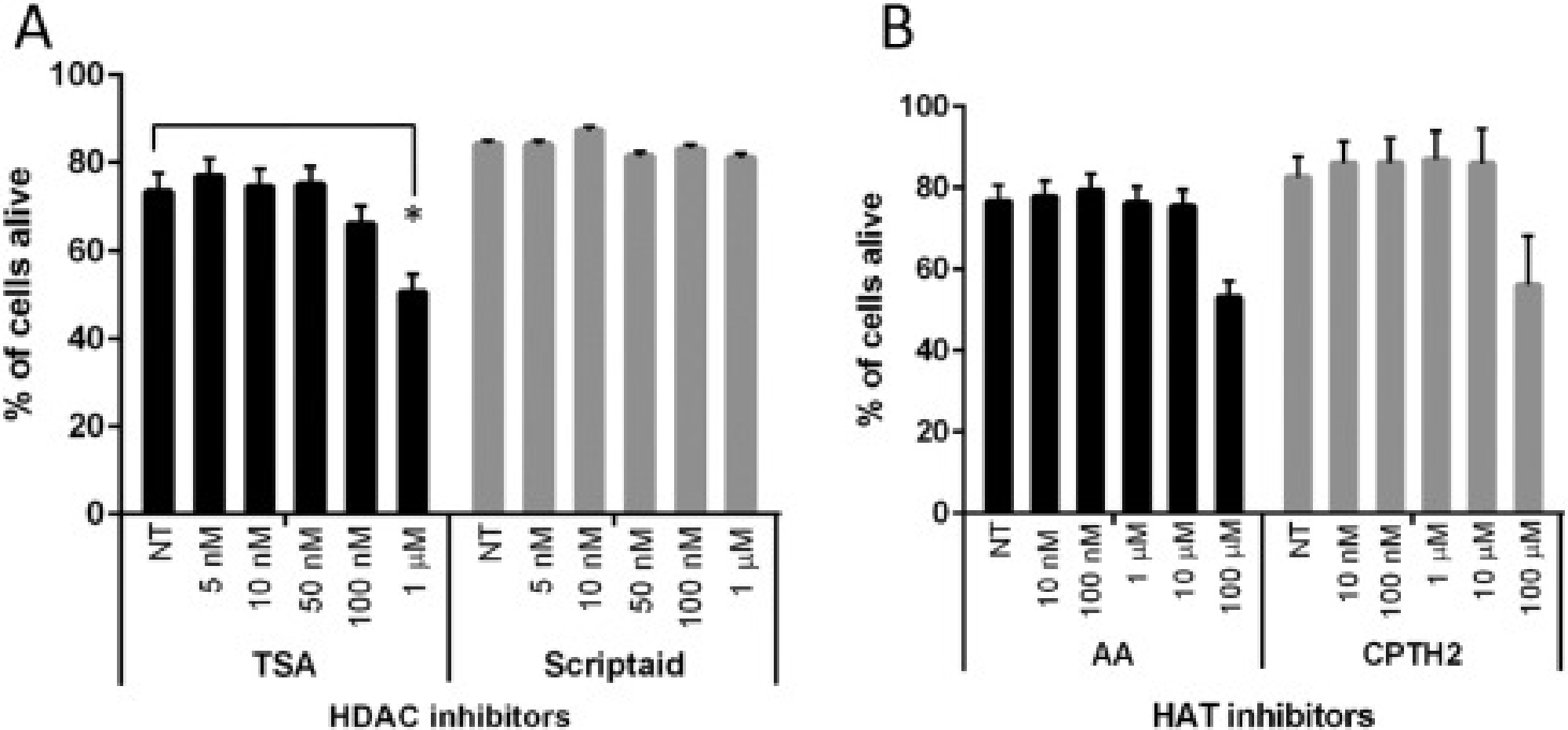
A
100
80
60
40
20
0
% of cells alive
*
NT
5 nM
10 nM
50 nM
100 nM
1 μM
TSA
Scriptaid
HDAC inhibitors
B
100
80
60
40
20
0
% of cells alive
NT
10 nM
100 nM
1 μM
10 μM
100 μM
AA
CPTH2
HAT inhibitors

Fig. S1.

Pharmacological inhibition of HDACs or HATs does not affect cell viability. P3 cortical neurons were transfected with control plasmids (EBFP) cultured on laminin substrates and Class I and II HDAC pharmacological inhibitors Trichostatin A (TSA) and Scriptaid were added 1DIV in increasing concentrations (0,5,10,50,100 nM and 1 μM) (A) or HAT pharmacological inhibitors Anarcardic acid (AA) and CPTH2 were added 1DIV in increasing concentrations (0,10,100 nM, 1 μM, 10 μM and 100 μM) (B) and allowed to incubate for 2 days. At 2DIV, cells were treated with hoescht stain to identify nuclei and Yo-Pro-1-Iodide and/propidium iodide to identify dead cells. Images were acquired through automated microscopy for three channels-nuclear (Hoechst 33342), cell death stain (Yo-Pro-1-Iodide or propidium iodide), and reporter (EBFP plasmids) and compartmental analysis quantified intensities for each channel. Bars show % cells alive [percent dead cells (Hoechst +/Yo-Pro-1-Iodide + or Hoechst +/propidium iodide +) subtracted from 100%]. Cells treated with TSA at concentrations between 5 and 50 nM remain viable, with higher concentrations (100 nM, 1 μM) leading to cell death (p-value < 0.1, two-way ANOVA with post-hoc Dunnett's). This concentration range was chosen for neurite outgrowth experiments. Scriptaid treatment does not affect cell viability at any of the concentrations tested. Cells treated with HAT inhibitors at concentrations between 10 nM and 10 μM remain viable, with higher concentrations (100 μM) leading to cell death. This concentration range was chosen for neurite outgrowth experiments. HAT inhibitor treatment was not statistically different from no treatment group at any of the concentrations tested (two-way ANOVA with post-hoc Dunnett's). For all experiments, $n > 1000$ cells (three biological replicates), error bars represent SEM.

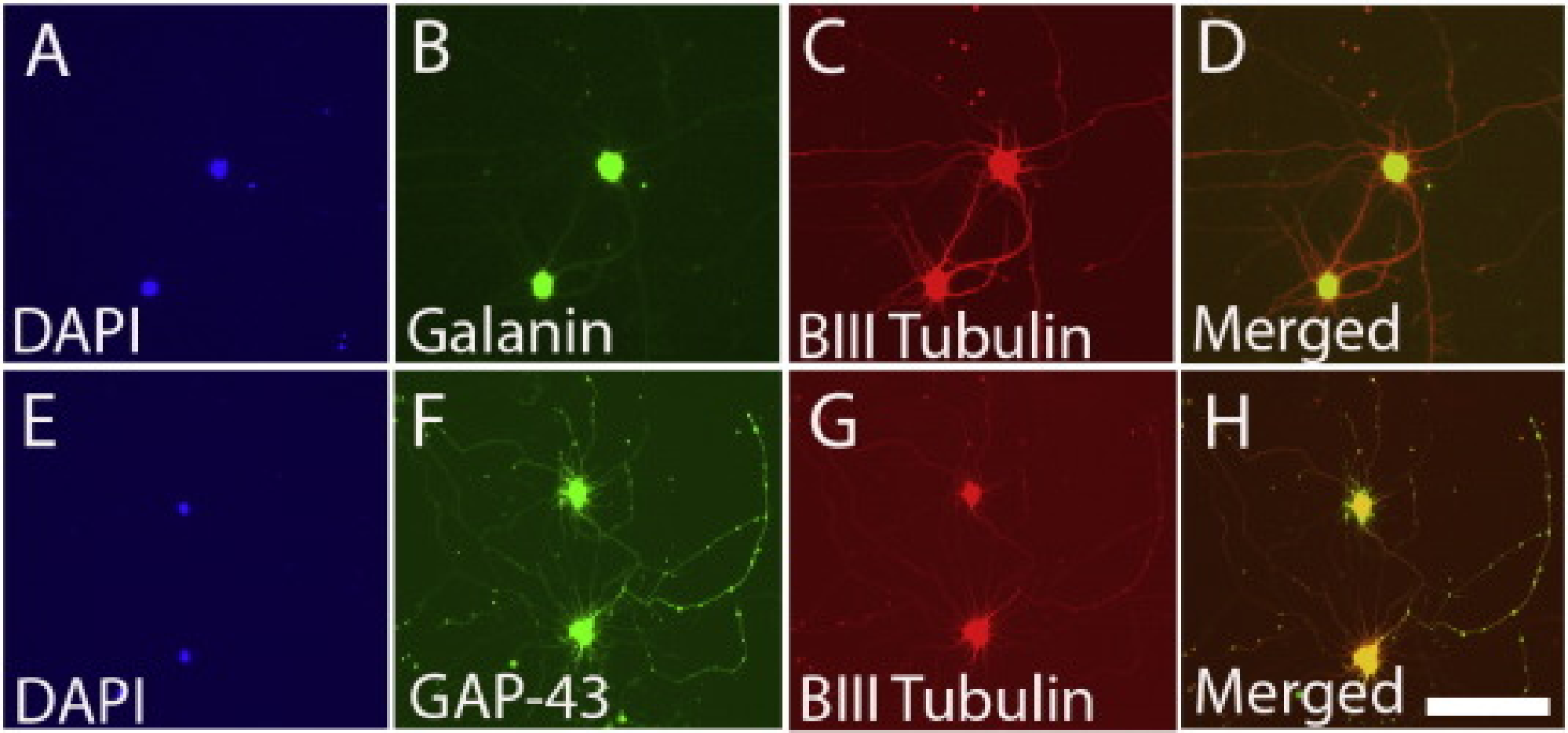
A
DAPI
B
Galanin
C
BIII Tubulin
D
Merged
E
DAPI
F
GAP-43
G
BIII Tubulin
H
Merged

Fig. S2.

Regeneration-associated gene expression in post-natal cortical neurons maintained *in vitro*. P3–P5 cortical neurons were cultured on laminin substrates for 3 days. Cells were then fixed and stained with antibodies targeting classical regeneration-associated genes GAP-43/Galanin. Cells were also stained with antibodies against BIII tubulin (red) to identify neurons (C, G) and DAPI nuclear stain (blue) (A, E). P3–P5 cortical neurons maintained *in vitro*, show clear expression of regeneration associated genes Galanin (B) and GAP-43 (F).