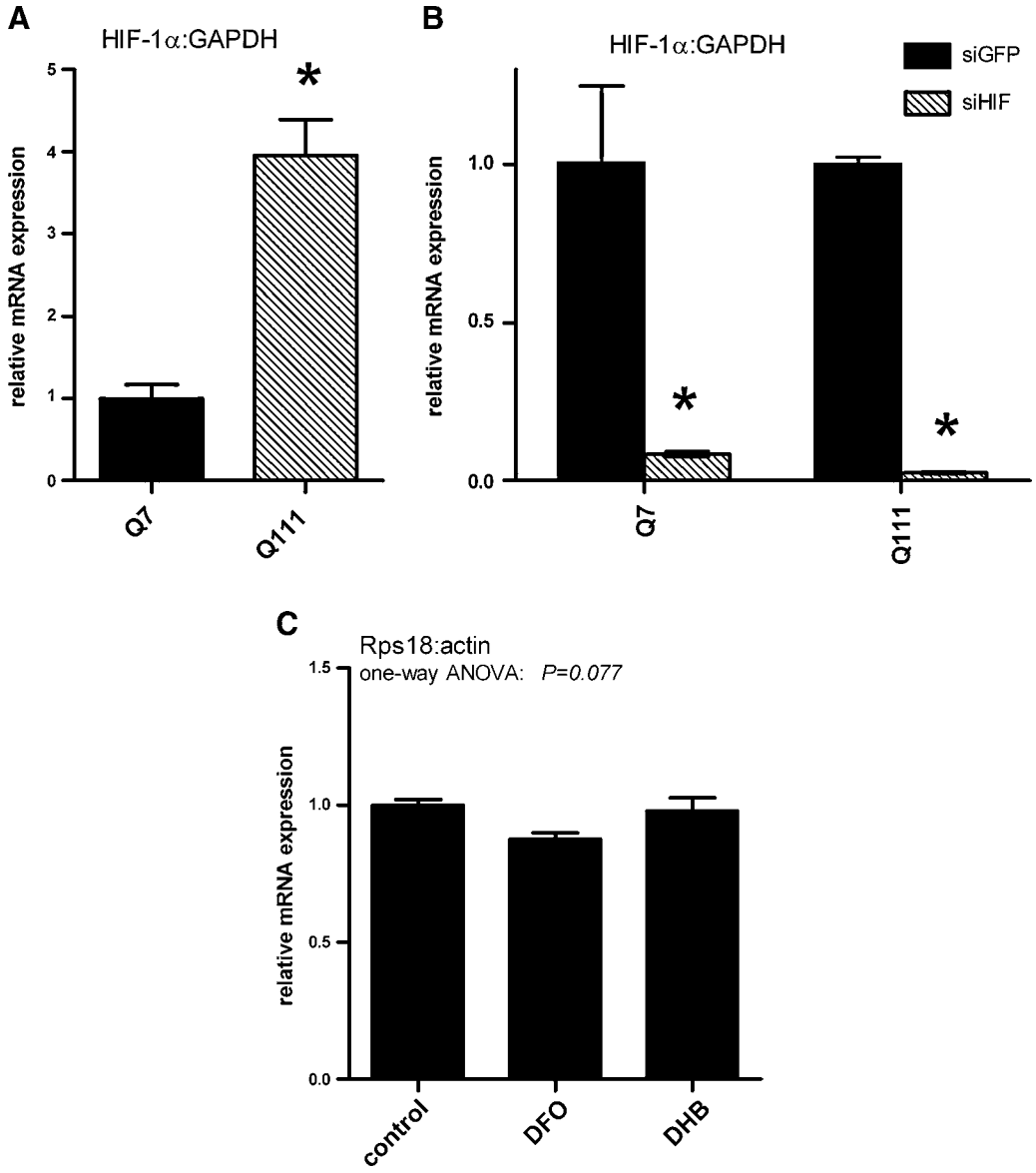
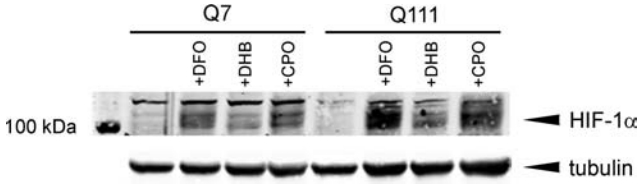
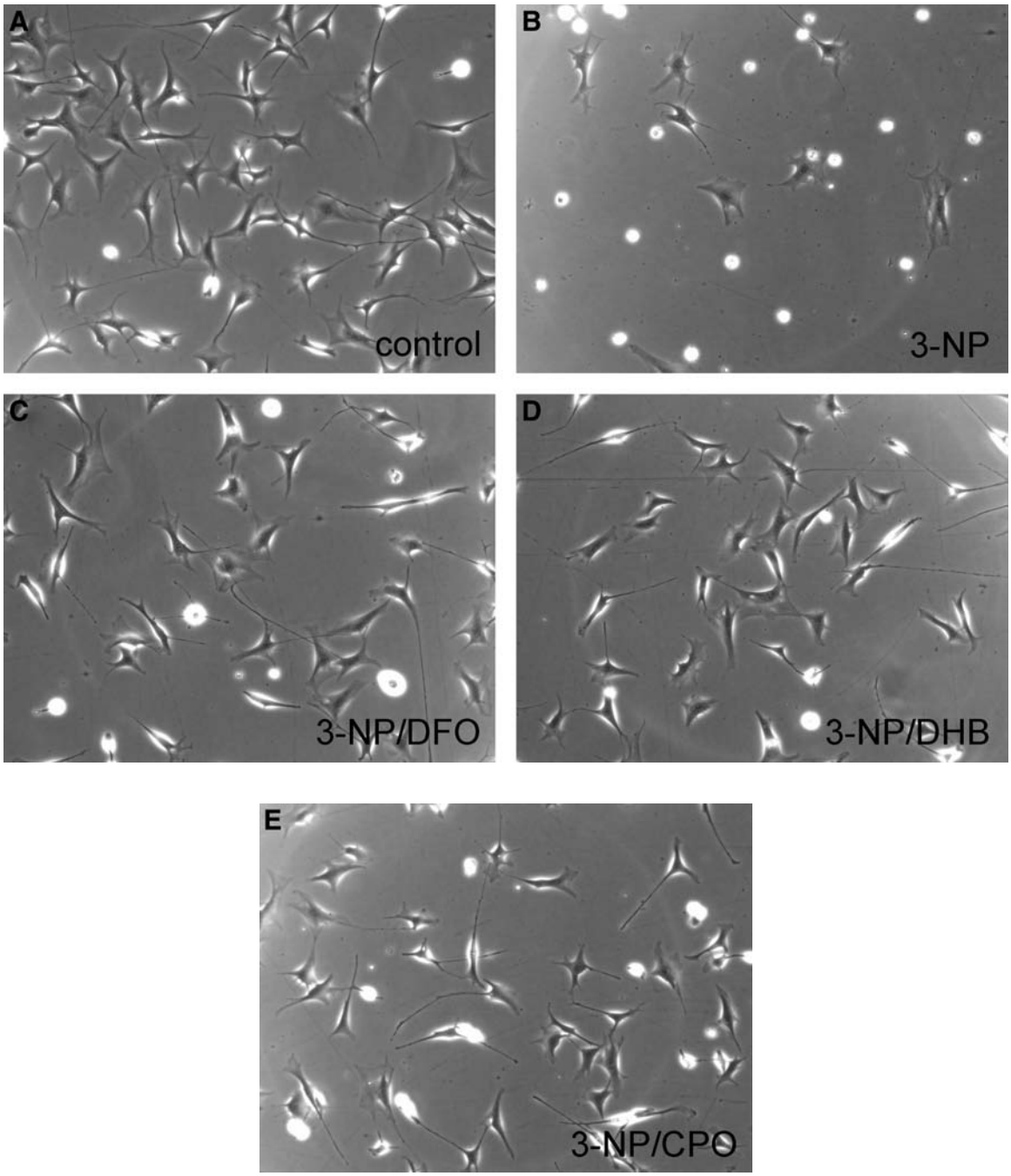


SUPPLEMENTAL FIG. 1. (**A**) **TaqMan real-time gene expression assays for HIF-1α show increased basal HIF-1α expression in mutant huntingtin (Q111) cells compared to wild-type (Q7) striatal cells.** Gene expression levels were normalized to GAPDH levels and are expressed as fold increase over Q7 cells. Data represent mean ± SEM of three independent experiments. *$p < 0.05$, Student's *t*-test. (**B**) Q7 and Q111 striatal cells were transduced with a retrovirus delivering hp/GFP (siGFP) or hp/HIF-1α (siHIF-1). HIF-1α expression levels were determined by RT-PCR and were significantly reduced in siHIF cells of either genotype. Gene expression levels were normalized to GAPDH levels and are expressed as fraction of Q7 cells. Data represent mean ± SEM of two independent experiments. *$p < 0.05$, Student's *t*-test. (**C**) Cortical neuron cultures were treated with PHD inhibitors for 24 h and assayed the expression of ribosomal protein s18 (Rps18) and actin. Treatment with PHD inhibitors did not change actin levels when normalized to Rps18.



SUPPLEMENTAL FIG. 2. Structurally diverse HIF PHD inhibitors stabilize HIF-1α protein. HIF-1α protein levels in Q7 and Q111 cells were determined by Western blot analysis following treatment with DFO, DHB, or CPO. All three compounds increased levels of HIF-1α in both cell types.



SUPPLEMENTAL FIG. 3. Prolyl 4-hydrdoxylase inhibitors abrogate 3NP toxicity in Q111 striatal cells. Q7 and Q111 striatal cells were pretreated with the indicated PHD inhibitors for 6 h and then exposed to 3-NP (10 m*M*) for an additional 24 h, after which cell survival was assessed by phase contrast microscopy. (**A**) untreated control cells, (**B**) 3-NP (10 m*M*), (**C**) 50 μM desferrioxamine (DFO) + 3-NP, (**D**) 50 μM 3,4-dihydroxybenzoic acid (DHB) + 3-NP, (**E**) 2.5 μM ciclopirox (CPO) + 3-NP.