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Supplemental Information

Modulation of cAMP and Ras Signaling Pathways Improves Distinct Behavioral Deficits in a Zebrafish Model of Neurofibromatosis Type 1

Marc A. Wolman, Eric D. de Groh, Sean M. McBride, Thomas A. Jongens, Michael Granato, and Jonathan A. Epstein

SUPPLEMENTAL INFORMATION

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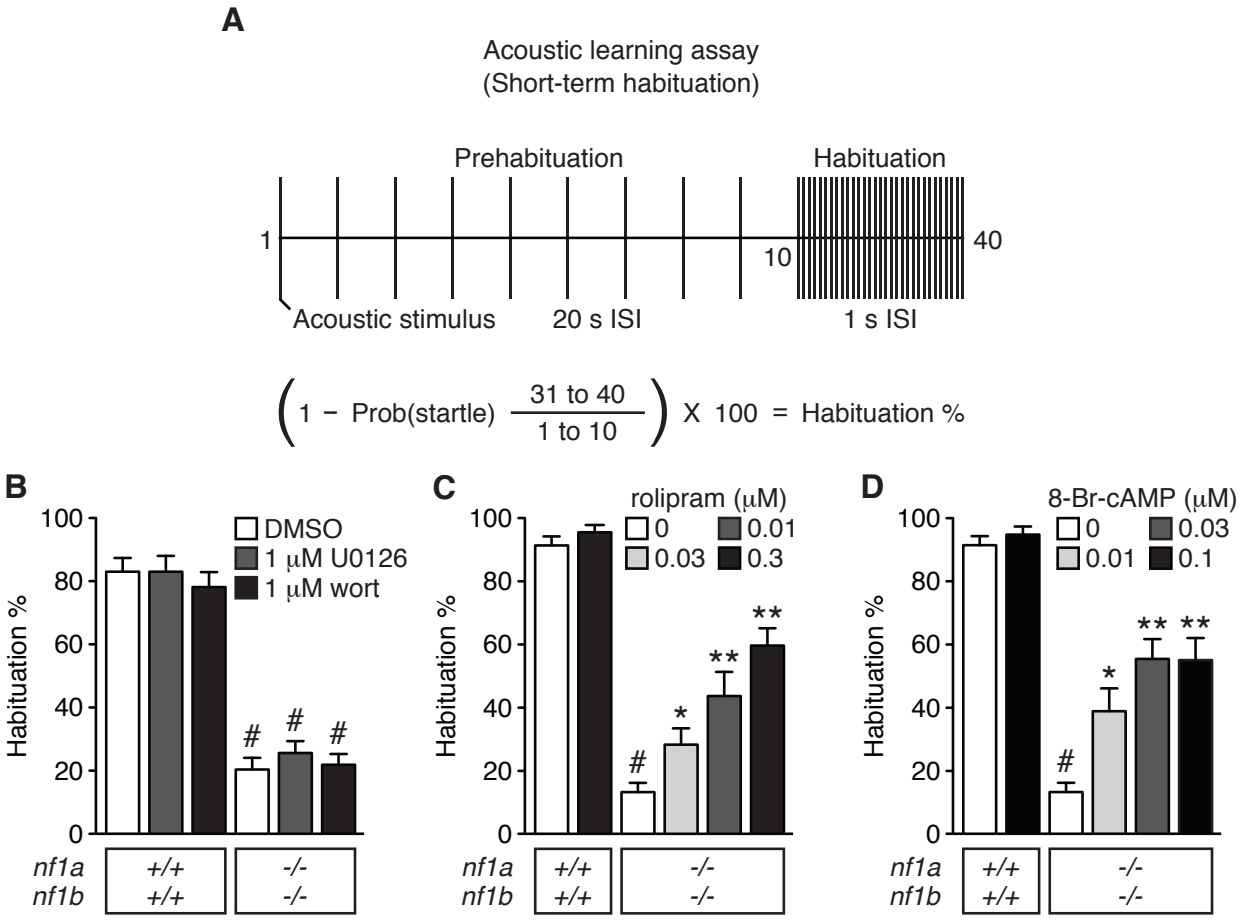

Figure S1, Related to Figure 2. cAMP Signaling Mediates *nf1*-Dependent Acoustic Learning

(A) Schematic representation of acoustic learning assay. (B-D) Mean habituation percentage to repeated acoustic stimulation (*n* = 12-32 larvae per genotype/treatment). #*P* < 0.001 versus DMSO-treated wild-type larvae. **P* < 0.01, ***P* < 0.001 versus DMSO-treated *nf1a*$^{-/-}$; *nf1b*$^{-/-}$ larvae. One-way ANOVA. Error bars denote SEM.

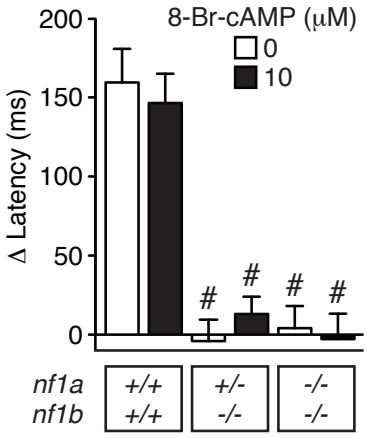

Figure S2, Related to Figure 1. Enhanced cAMP Signaling Does Not Improve Memory Recall in *nf1* Mutants

Mean O-bend latency change 1 h after spaced training (test) versus untrained controls (*n* = 35 to 66 O-bend maneuvers per genotype/treatment). #*P* < 0.001 versus DMSO-treated wild-type larvae. One-way ANOVA. Error bars denote SEM.

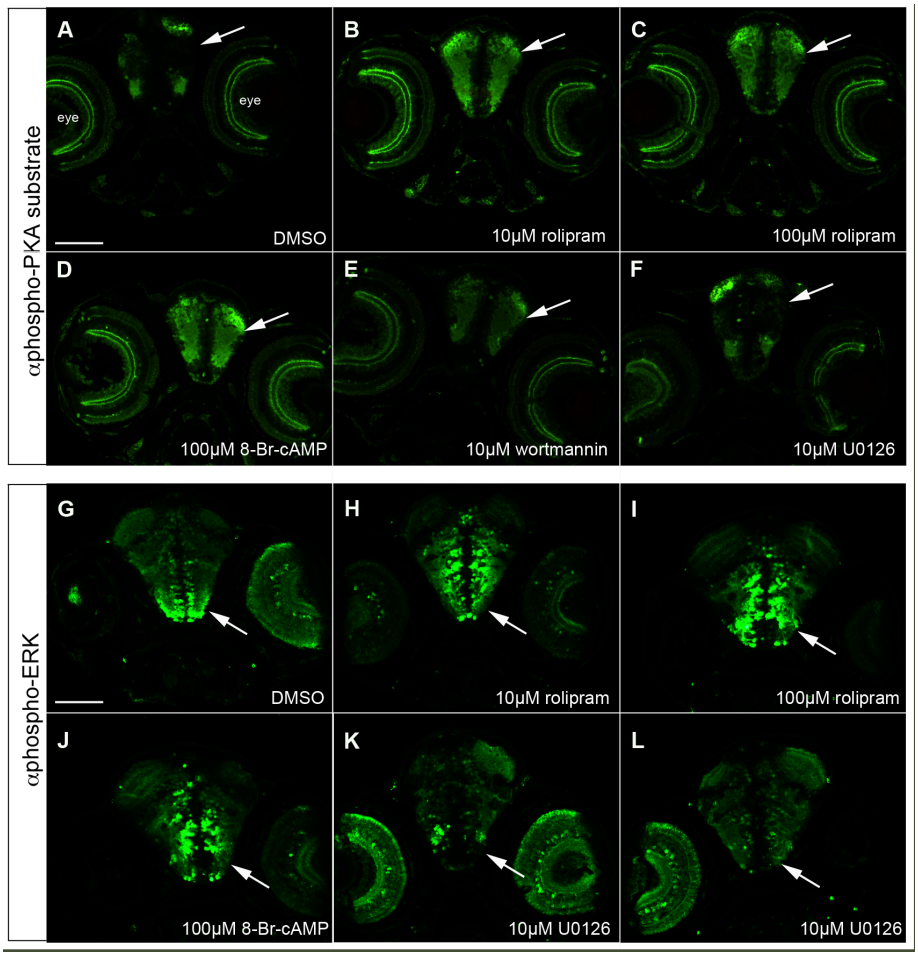

Figure S3, Related to Figures 1-3. Pharmacologic Specificity of Reagents Used in Behavioral Assays

Wild-type larvae were treated with DMSO or small molecules (at the concentrations noted) for 30 min, fixed, and sectioned for immunohistochemistry with (A-F) anti-phospho-(Ser/Thr) PKA substrate antibody or (G-L) anti-phospho-ERK antibody. Longitudinal sections through the brain (arrow) and eyes are shown. Scale bar represents 100 μM.

Movie S1, Related to Figure 1. Zebrafish Larva Performs O-Bend Response to Dark Flash Stimulus