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MicroRNA-mediated switching of chromatin-remodelling complexes in neural development

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In the print issue of this Letter, Fig. 3 was incorrectly printed as a black and white image. The correct image is shown below.

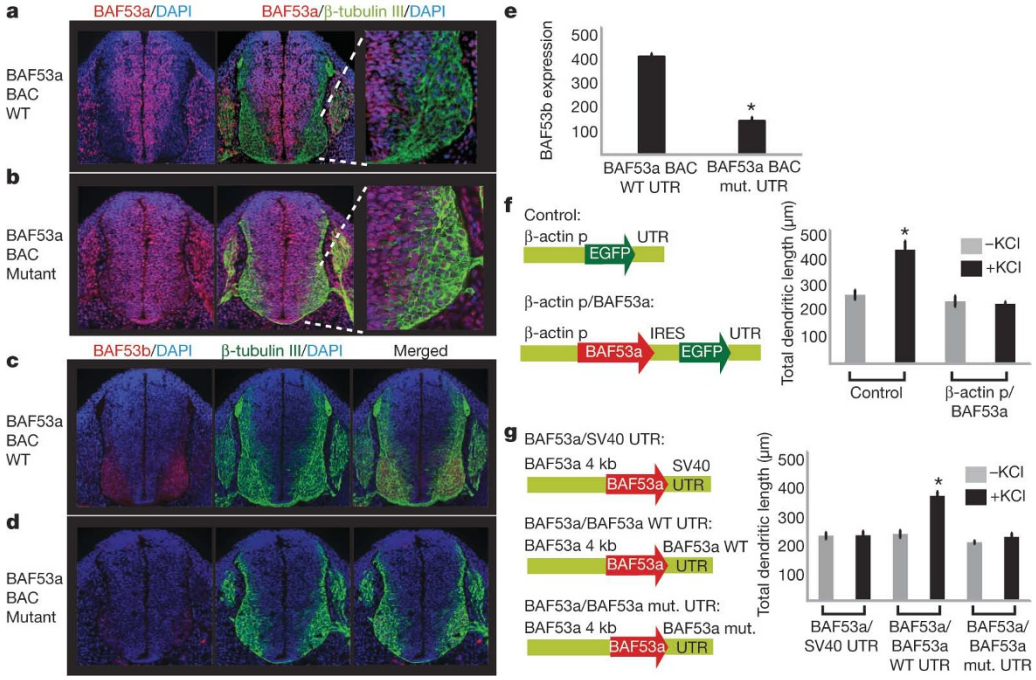

Figure 3 | BAF53a repression is essential for activity-dependent dendritic outgrowth in neurons. **a**, Normal downregulation of BAF53a in post-mitotic neurons in transgenic embryos with wild type BAF53a BAC. The rightmost panel shows the lower-right quadrant of the neural tube. **b**, Persistent expression of BAF53a in neurons seen with BAF53a BAC containing point mutations in the miRNA-binding sites. **c**, Normal expression of BAF53b (red) in β-tubulin-III-positive (green) neurons in transgenic embryos with wild-type BAF53a BAC. **d**, Reduced BAF53b expression with persistent expression of BAF53a in neurons. **e**, Quantification of BAF53b expression: ratio of BAF53b level (arbitrary units) and β-tubulin-III-positive neurons. Average values are from eight sections of the neural tube. Error bars, s.e. $*P < 0.005$, Student's *t*-test. **f**, Constructs to overexpress BAF53a in cultured hippocampal neurons and quantification of dendritic outgrowth of GFP-positive neurons upon stimulation using KCl. The average values are from five individual coverslips from two independent experiments, with each coverslip containing 50–100 scored neurons. Error bars, s.e. $*P < 0.005$, Student's *t*-test. p, promoter; IRES, internal ribosome entry site. **g**, Schematic diagrams of BAF53a expression constructs using different 3′ UTRs and quantification of dendritic outgrowth of transfected neurons upon stimulation using KCl. In independent experiments, we found that the 4-kb upstream region of BAF53a (illustrated) was sufficient to drive expression of GFP reporters that could be repressed by endogenous miR-9* and miR-124. Error bars, s.e. $*P < 0.001$, Student's *t*-test.