Supplemental Material to:

Giovanni Marchetti, Adèle De Arcangelis, Véronique Pfister, and Elisabeth Georges-Labouesse

α 6 integrin subunit regulates cerebellar development

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Figure S1.Normal cortical lamination in $\alpha^{6fl/finestin-Cre}$ mice. Immunostaining for cortical layer-specific markers Cdp/Cux (red) and Ctip2 (green) on P21 coronal sections of control and mutant cerebral cortex. Neurons in layers II-IV expressed Cdp/Cux, while neurons in layer V expressed Ctip2. No differences in cortical laminar organization were observed comparing the control and mutant brains. Scale bar, 100 μ m.



Figure S2. Granule cell precursor proliferation is not affected in the $\alpha 6^{fi/f_{inestin-Cre}}$ mice. (**A**) Sagittal sections of P10 mouse cerebellum stained with DAPI. The asterisks indicate the primary fissure (pf, *), the secondary fissure (sf, **), and prepyramidal fissure (ppf, ***). Scale bar, 200 µm. (**B**) Mice were injected with BrdU and sacrificed 2 h later. Representative sagittal sections of P10 control and mutant cerebellum stained with BrdU antibody. Scale bar, 40 µm. (**C**) Quantitative analysis of BrdU incorporation at P10. The quantification of the BrdU incorporation was performed within the fastest growing folia, respectively pf (control: 16.3 ± 3.4%; mutant: 15.5 ± 6.9%), sf (control: 17.5 ± 3.5%; mutant: 21.4 ± 11.5%), and ppf (control: 20.7 ± 11.8%; mutant: 20 ± 3.6%) fissures, where the proliferation rate is presumably the highest. Three sections were counted per animal (3 controls and 3 mutants). Histogram depicting the percentage of BrdU-positive cells to total DAPI-positive cells in the fissure indicated in (**A**). No statistically significant difference was detected in the number of BrdU-positive cells between control and mutant animals in all three fissures. Error bars indicate the SD. EGL, external granular layer.