Supplementary Figure 1. Analysis of Cre-mediated recombination in Olig1<sup>cre/lox</sup> mice. A, EGFP expression within GFAP<sup>+</sup> astrocytes and NeuN<sup>+</sup> neurons in the hippocampus of O-NR1<sup>/3</sup> mice; yellow arrows highlight several GFAP<sup>+</sup>GFP<sup>+</sup> cells and red arrows highlight several NeuN<sup>+</sup>GFP<sup>+</sup> cells. B, Several GFAP<sup>+</sup>EGFP<sup>+</sup> cells and NeuN<sup>+</sup>EGFP<sup>+</sup> cells from E shown at higher magnification. C, EGFP expression within ALDH1<sup>+</sup> astrocytes in the cortex of O-NR1<sup>/3</sup> mice. D, Graph showing the percentage of NeuN<sup>+</sup> neurons and ALDH1<sup>+</sup> astrocytes that express EGFP in the cortex of O-NR1<sup>/3</sup> and O-NR1<sup>/0</sup> mice.
Supplementary Figure 2. Basic membrane properties of NMDAR-deficient OPCs following embryonic NMDAR ablation. Response of representative callosal OPCs in O-NR1^{+/+} and O-NR1^{+/null} mice to depolarization. Red trace = injection of 640pA.
Supplementary Figure 3. NMDAR-deficient OPCs in gray matter display normal membrane properties and synaptic connectivity. **A-B**, Membrane capacitance ($C_m$) and resting membrane potential ($V_m$) of OPCs in CA1 hippocampus (HC) and cortex (Ctx) of mature (P40-45) O-NR1$^{+/+}$ and O-NR1$^{fl/fl}$ mice. Differences between genotypes were not significant ($C_m$ HC, $p = 0.09$; $C_m$ Ctx, $p = 0.15$; $V_m$ HC, $p = 0.10$; $V_m$ Ctx, $p = 1$). $n$ indicated at the base of each column. **C**, Density of voltage-gated sodium channels in hippocampal OPCs from mature O-NR1$^{+/+}$ and O-NR1$^{fl/fl}$ mice ($p = 0.92$). **D**, Membrane resistance of OPCs in HC and Ctx of mature O-NR1$^{+/+}$ and O-NR1$^{fl/fl}$ mice (Ctx, $p = 0.37$; HC, * $p = 0.03$). **E**, Response of hippocampal OPCs from O-NR1$^{+/+}$ and O-NR1$^{fl/fl}$ mice to application of hypertonic solution (HS, indicated by black bar). Asterisk indicates HS-evoked mEPSC shown at expanded time scale to the right. **F**, Quantification of the number of HS-evoked mEPSCs in hippocampal OPCs from O-NR1$^{+/+}$ and O-NR1$^{fl/fl}$ mice ($p = 1$).
Supplementary Figure 4. Spontaneous EPSCs persist in NMDAR-deficient OPCs.  

A. Two minute long recordings from representative callosal OPCs in mature (P40-45) O-NR1\(^{+/+}\) and O-NR1\(^{+/−}\) mice. Spontaneous excitatory postsynaptic current (sEPSC) highlighted with red asterisk shown at expanded time scale to the right.  

B. Quantification of sEPSC frequency in callosal OPCs from control (O-NR1\(^{+/+}\), P-NR1\(^{+/+}\)), constitutive (O-NR1\(^{+/−}\)), and inducible (P-NR1\(^{+/−}\)) NMDAR ablation mice. Differences between genotypes were not significant (\(p = 0.054\), K-W ANOVA).  

C. Quantification of sEPSC frequency in OPCs from hippocampus (HC) and cortex (Ctx) of O-NR1\(^{+/+}\) and O-NR1\(^{+/−}\) mice. Differences between genotypes were not significant (HC \(p = 1\), Ctx \(p = 0.77\)).