Hippocampal functions are not impaired in L7-PKCI.

Although the L7/PCP2 promoter restricts the expression of the PKCI transgene to Purkinje cells, it was conceivable that the unidentified integration site of the transgene may affect the expression of genes required for hippocampal plasticity and function. We directly assessed hippocampal plasticity by examining long-term potentiation (LTP) at the Schaffer collateral-CA1 synapse in acute hippocampal slices. We focused on the Schaffer collateral pathway because many previous studies have demonstrated that LTP at this synapse is crucial for learning contextual and spatial information. (a) LTP was first induced using a commonly used 100 Hz stimulation protocol. We found no significant differences between LTP induced in wild-type (WT) mice as compared to PKCI littermate mice (WT: 11 slices, 4 mice; PKCI: 17 slices, 5 mice; ANOVA $F_{1,26} = 0.65, P = 0.42$). (b) We then assessed the level of LTP induction using five theta-burst stimulation (TBS), which mimics the in vivo activity of hippocampal neurons during exploratory behavior and might be more sensitive to reveal changes in plasticity. Again, LTP in control mice was indistinguishable from LTP in PKCI mice (WT: 12 slices, 4 mice: PKCI: 13 slices, 5 mice; ANOVA $F_{1,23} = 0.01, P = 0.93$). At the bottom of (a, b), representative traces are shown at baseline (red line) and at 60 min after LTP induction (black line) (left, WT; right, PKCI). (c) Finally, also synaptic transmission was not significantly different between mutants and controls (WT: 17 slices, 4 mice; PKCI: 18 slices, 5 mice; presynaptic fiber volley: $F_{4,132} = 0.04, P = 0.99$; postsynaptic fEPSP: $F_{4,132} = 1.21, P = 0.31$). Plots show the fEPSP as a function of the evoked presynaptic fiber volley, at 20, 40, 60, 80, 100 μA stimulation. The line represents the calculated trend line.