

CORRECTION

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Correction: Neural stem cells genetically-modified to express neprilysin reduce pathology in Alzheimer transgenic models

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The original article contains an artifact in Fig. 6H that obscures the view of the lower-middle portion of the image. The correct original image for Fig. 6H can be viewed in this Correction article.

The original article can be found online at <https://doi.org/10.1186/scrt440>.

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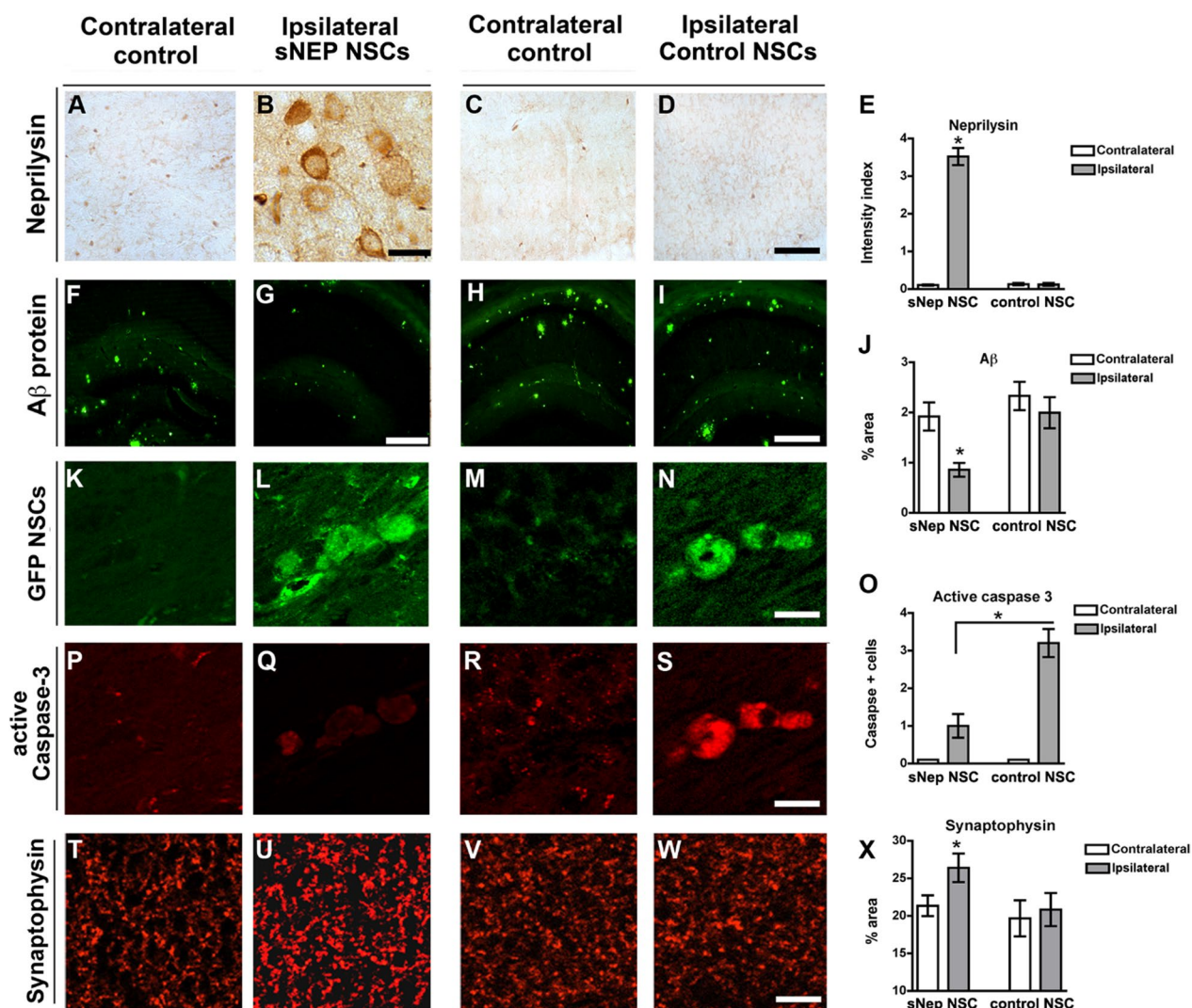


Fig. 6 sNEP-NSCs reduce plaque pathology and resist degeneration in a second transgenic AD model. Nprilysin immunoreactivity in the contralateral (A) and ipsilateral (B) hippocampus of sNEP-NSC transplanted transgenic mice reveals high levels of NSC nprilysin expression in vivo. (C-D) Control NSCs, in contrast, produce little to no nprilysin following transplantation, quantified in (E). At 10 months of age, Thy1-APP mice exhibit considerable amyloidosis (6E10 labelling, green) within the hippocampus (F). However, transplantation of sNEP-NSCs significantly reduced Aβ pathology within the ipsilateral hippocampus (G). Control NSCs by comparison have no effect on Aβ levels (H-I), quantified in (J). GFP labelling (green) reveals examples of NSCs engrafted into the ipsilateral hippocampus (L, N), but not within the contralateral vehicle-injected side of the brain (K, M). In line with in vitro findings, caspase activation is reduced by expression of nprilysin (O). Little active caspase-3 immunoreactivity (red) is detected within the ipsilateral hippocampi of transgenic mice (P, R). However, caspase-3 activation (red) within sNEP-NSCs (Q) is significantly reduced versus control NSCs (S). Furthermore, levels of the presynaptic terminal marker synaptophysin (T-X) are significantly increased by sNEP-NSC transplantation (U), suggesting that nprilysin expression can reduce Aβ-induced synaptotoxicity. N = 6/group, error bars represent standard error of the mean (SEM). Scale Bar = 30 μm in A-D, 350 μm in F-I, 14 μm in K-S, 45 μm in T-W. Aβ, beta-amyloid; AD, Alzheimer’s disease; NSCs, neural stem cells; sNEP, secreted nprilysin.

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