Supporting Information

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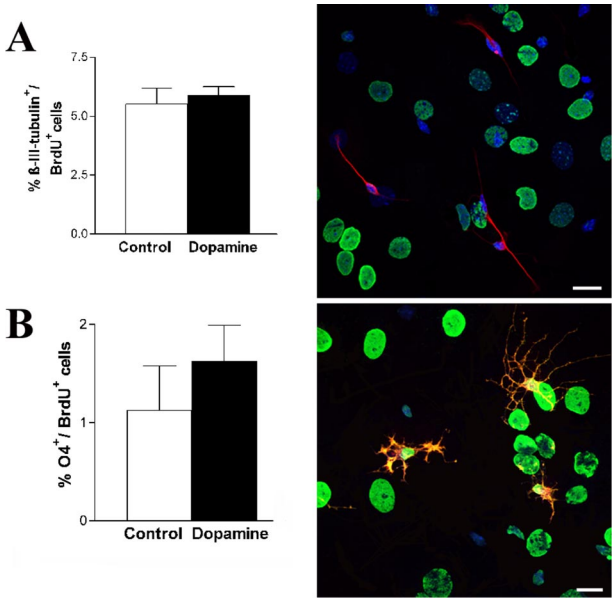

Fig. S1. Dopamine stimulation did not affect the number of newborn neurons or oligodendrocytes in vitro. To determine the effect of dopaminergic stimulation on the differentiation of adult NPCs, control and dopamine-treated cultures were pulsed with BrdU for 24 h and differentiated for 4 days. Phenotypic analysis of newly-generated cells immunostained for β-III-tubulin (*A*) and O4 (*B*) showed no difference between the dopamine-treated and untreated cultures. Nuclei are stained with Hoescht 33258. (Scale bar: 60 μm.)

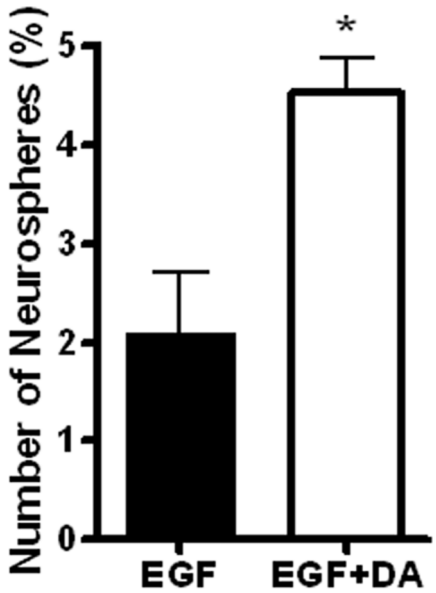

Fig. S2. Clonal efficiency is increased in the presence of dopamine. Adult SVZ NPCs were grown in EGF alone or in combination with dopamine for 7 days. They were then seeded as single cells per well for another 7 days after which the number of wells containing neurospheres was counted. Graph shows that there are a greater percentage of neural stem cells in the presence of dopamine. *, $P < 0.05$.

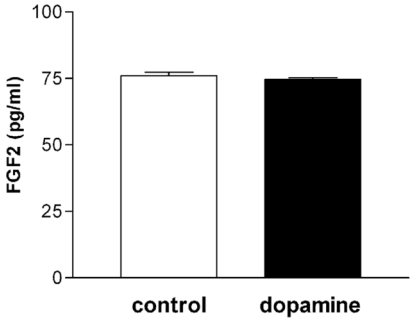

Fig. S3. Dopamine stimulation had no effect on the release of FGF2 from adult SVZ-derived NPCs. FGF2 release from adult SVZ-derived precursor cells after 24 h of treatment with dopamine compared with control samples by using an ELISA.

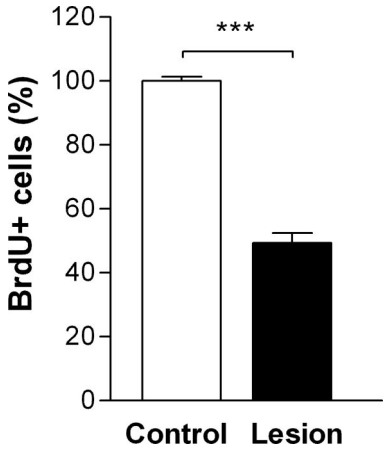

Fig. S4. Elimination of dopaminergic fibers via a 6-OHDA lesion significantly decreases proliferation in the olfactory bulb as a consequence of reduced local EGF production. Stereologically determined number of $BrdU^+$ cells in coronal sections of the olfactory bulb 21 d after the last BrdU injection. ***, $P < 0.001$ lesion vs. control group.

Table S1. The concentration of dopamine and its metabolite DOPAC in 6-OHDA lesioned animals treated with L-DOPA was significantly increased compared with untreated 6-OHDA lesioned animals

Animal	Dopamine	DOPAC
6-OHDA lesion	0.097169 ± 0.025451	0.033587 ± 0.012712
6-OHDA lesion + L-DOPA	0.385473 ± 0.078219*	0.206715 ± 0.034478**

To determine whether L-DOPA is converted to dopamine in the 6-OHDA lesioned animal, we used the experimental paradigm outlined in Fig 4 but animals were killed 2 h after the last L-DOPA injection. The striatum was immediately removed and analyzed by HPLC for dopamine and DOPAC concentration. Tissue concentrations in all animals are expressed in pmol/mg striatal tissue (mean ± SEM) of 5 animals from each group.
*$P < 0.05$ lesion versus lesion + L-DOPA.
**$P < 0.01$ lesion versus lesion + L-DOPA.

Table S2. Primary antibodies concentrations used, species antibodies were raised in, and suppliers

Antibody	Species	Concentration used	Supplier
BrdU	Sheep	1:1,000	ABCAM
	Mouse	1:1,000	Covance
GFAP	Rabbit	1:500	DAKO
O4	Mouse	1:5	S. Chandran, CBR Cambridge
β-III-Tubulin	Mouse	1:500	Covance
NeuN	Mouse	1:100	Chemicon
EGFR	Rabbit	Prediluted	Chemicon
	Mouse	1:100	R&D Systems
TH	Mouse	1:100	Chemicon
D2DR	Rabbit	1:200	Santa Cruz

Hoescht 33258 nuclear stain was used 1:5,000 (Sigma). Live staining for O4 was carried out before fixation by using anti-O4 for 1 h at 37 °C. For BrdU immunolabelling, sections were preincubated in 2 M HCl for 30 min followed by 3 washes with 0.1 M borate buffer, pH 8.5. Paraffin-embedded human sections were deparaffinized by heating them to 65 °C for 45 min and immersing them in xylene overnight. Sections were then rehydrated through a series of ethanol solutions before being immersed in citrate buffer (10 mM + 0.05% Tween 20, pH 6) and boiled in a microwave for 10 min.