Figure legends to supplementary figures

Figure S1. Effects of different antagonists on dendritic branching and dendritic spine density of MSNs in the NAc and Cpu. Mice (n=4-6) were treated with saline or the D1 receptor inhibitor SCH23390 (0.5mg/kg), the D3 receptos antagonist NGB2904 (1mg/kg), the MEK antagonist SL327 (30mg/kg) and the NMDA receptor inhibitor MK801 (0.1mg/kg) once daily for 4 weeks and then the dendritic morphological analysis were performed. (A) Representative dendritic branching images of MSNs for the NAc and CPu in the indicated groups of mice. Scale bar, 10 μ m. (B) Representative dendritic spine images of MSNs for the NAc and CPu in the indicated groups of mice of dendritic branching (C) and dendritic spine density (D) in different groups of mice (for dendritic branching, F_(4,235) =1.681 and *p*=0.155 for NAc, F_(4,235)=0.597 and *p*=0.665 for CPu; for spine density, F_(4,220)=1.047 and *p*=0.384 for NAc, F_(4,220)= 0.560 and *p*=0.692 for CPu). Data represent mean ± SEM.

Figure S2. Effects of cocaine and D1/D3 inhibitor pretreament on dendritic branching and spine density of pyramidal neurons in basilar dendrites of Layer III somatosensory cortex. Mice were treated with cocaine (20 mg/kg) or saline once a day for 4 weeks and then the dendritic morphological analyses were performed. The D1 receptor inhibitor SCH23390 (0.5mg/kg), the D3 receptor antagonist NGB2904 (1mg/kg) and saline were administrated 30 min before cocaine or saline administration. (A) Diagram of a coronal section indicating

the area of somatosensory cortex analyzed for dendrite analysis (left). Representative dendritic branching and spine images of pyramidal neuron in basilar dendrites of Layer III somatosensory cortex (right). Scale bar, 10μ m. (B and C) Quantification of dendritic branching (B) and spine density (C) in the indicated groups of mice (for dendritic branching, $F_{(3,188)} = 0.94$, *p*=0.422; for spine density, $F_{(3,188)}=1.236$, *p*=0.298). Data represent mean ± SEM.

Figure S3. Effects of NGB 2904 (0.1-5mg/kg) on cocaine-induced increase of the number of dendrite and spine density. Mice were treated with cocaine (20 mg/kg) once a day for 4 weeks and then the dendritic morphological analyses were performed. The D3 receptor antagonist NGB2904 (0.1-5mg/kg) were administrated 30 min before cocaine administration. (A) Representative dendritic spine images of MSNs for the NAc and CPu in the indicated groups of mice. Scale bar, 10μ m. (B) Quantification of dendritic branching and dendritic spine density in the indicated groups of mice. Data represent mean ± SEM. **p*<0.05 compared with cocaine-treated mice (0 dose NGB2904 group).

Figure S4. Effects of different antagonists on MEF2 phosphorylation at Ser408 in the NAc and CPu after repeated cocaine treatment. Mice (n=4-6) were treated with saline or the D1 receptor inhibitor SCH23390 (0.5mg/kg), the D3 receptor antagonist NGB2904 (1mg/kg), the MEK antagonist SL327 (30mg/kg) or the NMDA receptor inhibitor MK801 (0.1mg/kg) once daily for 7 consecutive days.

Coronal sections were stained with a phosphorylation site-specific antibody to P-Ser408 MEF2A. P-MEF2-immunoreactive cells in the NAc and CPu were counted 24 hr after the last injection. Scale bar, $200 \,\mu$ m. Data represent ratio of the number of P-Ser408 MEF2A positive cells in $1 \,\text{mm}^2$ (mean ± SEM) area in inhibitor-treated mice versus saline treated mice. Saline-treated levels were set at 1 for quantifications.







Figure S2.



Dose NGB 2904 (mg/kg)



Figure S3.



Figure S4.