Additional Informations for online version only:

Table 2. F and P values from two factors ANOVA statistics

Experiment	Marker	Structure	Main effect of treatment		Main effect of time/genotype		Treatment-time/genotype interaction	
			F(2,47)	***P***	***F(2,47)***	***P***	***F(4,47)***	***P***
Pharmacological blockade of $A_{2A}R$	TH	SNc	90.05	4.47E-18	1.53	n.s.	0.79	n.s.
	GFAP	SNc	826.99	0	0.67	n.s.	3.76	n.s.
		Str	1665.32	0	6.62	0.0025	2.17	n.s.
	CD11b	SNc	107.49	1.87E-18	35.12	4.01E-10	33.60	2.27E-13
		Str	41.01	1.73E-11	36.79	9.56E-11	15.38	2.06E-08
			F(1,26)	***P***	***F(1,26)***	***P***	***F(1,26)***	***P***
fbn$A_{2A}R$ depletion	TH	SNc	5.17	0.032	7.92	0.0092	3.35	n.s.
	GFAP	SNc	20.05	0.00013	23.29	5.32E-05	18.83	0.00019
		Str	248.94	7.85E-15	1634.94	5.29E-25	192.23	1.6E-13
	CD11b	SNc	14.14	0.0013	18.37	0.00040	33.00	1.55E-05
		Str	106.10	1.15E-09	111.57	7.37E-10	108.56	9.39E-10

Table 2 (A.I. for online version): F and *P* values from two-way ANOVA relative to experiments with A_{2A} receptor antagonist (pharmacological blockade of $A_{2A}R$), or in genetically manipulated mice (fbn$A_{2A}R$ depletion). n.s.: not significant.

***Generation of fbnA_{2A}KO mice*:** Postnatal forebrain neuron conditional A_{2A}KO mice were generated using the Cre/*loxP* system based on the specificity of *CaMKII*α promoter. Near congenic (N6, C57Bl/6) male $A_{2A}^{flox/flox}$ mice (i.e., homozygous for the floxed allele of the $A_{2A}R$ gene) with or without a *CaMKII*α*-cre* transgene were used for this study. Tissue-specific postnatal recombination and disruption of the floxed $A_{2A}R$ alleles driven by *CaMKII*α*-cre* expression in forebrain neurons (i.e., fbn A_{2A}KO) were confirmed by PCR and Western blot as described previously (Bastia et al, 2005, Yu et al, 2008).

***Immunohistochemistry*:** For TH, GFAP and CD11b immunostaining, sections were incubated for 1 hr with 5% normal goat serum as blocking agent. The sections were then incubated overnight in primary antibodies (polyclonal rabbit anti-TH, 1:1000, Biomol, United Kingdom; monoclonal mouse anti-GFAP, 1:400, Sigma-Aldrich, Italy; monoclonal rat anti-mouse CD11b, 1:1000, Serotec, United Kingdom). Subsequently the sections were incubated in biotinylated secondary antibodies (goat anti-rabbit IgG for TH, goat anti-mouse IgG for GFAP and goat anti-rat IgG for CD11b, all purchased from Vector, United Kingdom). For visualization, the Avidin-peroxidase protocol (ABC, Vector) was applied, using 3,3'-diaminobenzidine (Sigma-Aldrich, Italy) as the chromogen. Sections were mounted into chrome-alum-gelatin-coated slides dehydrated and coverslipped.

***Stereological counting of TH-immunoreactive and Nissl-stained neurons*:** TH-immunoreactive and Nissl-stained neurons were counted on both hemispheres. All stereological counting was performed using a Leica microscope (DMLB; Leica, Denmark) equipped with a camera (Basler Vision Technologies, Germany) and a stage connected to an *xyz* stepper (PRIOR ProScan) and the newCAST Visiopharm (Denmark) software. The SNc region was outlined at low magnification (5X) for area estimation. The number of labelled neurons was calculated under 63X magnification using randomized meander sampling and optical dissector methods. The cut thickness of sections was 50 μm and the

optical dissector height was 12 μm. The top (13 μm) and bottom (25 μm) layers that shrunk during staining procedure were discarded. The sampling area covered 100% of the region of interest. The counting frame (8302,8 μm^2) applied the exclusion and inclusion lines and unbiased counting was performed by an experimenter blinded to the treatment. Results are presented as the mean of TH-immunoreactive and Nissl-stained neurons per mm^3 ± SEM, calculated using the following formula:

D = 3Q / V sampling

D = density of labelled cells per mm^3

V sampling = area of the region of interest x dissector height

Q = total count of labelled neurons.