Data supplement



Fig. DS1 Putamen and ventral striatum functional connectivity in the control and the schizophrenia groups.

Statistical parametric maps of putamen (upper row) and ventral striatum (lower row) functional connectivity of the two control groups and the schizophrenia group (voxelwise one-sample *t*-tests, *P*_{FWE}<0.05 corrected for familywise error (FWE)). Voxelwise functional connectivity is measured by individual β-maps based on multiple regressions on averaged seed functional magnetic resonance imaging time course.



Fig DS2 Reduced putamen and ventral striatum functional connectivity in the schizophrenia group.

Statistical parametric maps of group differences in putamen (upper row) and ventral striatum (lower row) functional connectivity (voxelwise *post hoc t*-tests control group > schizophrenia group for ANOVA with factors group (control group, schizophrenia group) and seed side (left, right), *P*_{FWE} < 0.05 corrected for familywise error (FWE)). Voxelwise functional connectivity is measured by individual β-maps based on multiple regressions on averaged seed functional magnetic resonance imaging time course.

Voxel-based morphometry (VBM) analysis

To control for potential effects of striatal structural changes on intrinsic functional connectivity, we included regional grey matter volume as covariate-of-no-interest in functional connectivity analyses. As described recently,6 we used the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm.html) to analyse brain structure. T1-weighted images were corrected for bias-field inhomogeneity, registered using linear (12-parameter affine) and nonlinear transformations, and tissue-classified into grey matter (GM), white matter, and cerebrospinal fluid within the same generative model. The resulting GM images were modulated to account for volume changes resulting from the normalisation process. Here, we only considered non-linear volume changes so that further analyses did not have to account for differences in head size. Finally, images were smoothed with a Gaussian kernel of 8mm (FWHM). Regional VBM volumes of ventral dorsal striatal region-of-interests were averaged and included in corresponding ANOVAs as described in the main text.

Table DS	1 Patients' individual medication protocol, dosages
Patient ID	Medication
1	20 mg olanzapine
2	100 mg clozapine, 80 mg ziprasidone
3	30 mg olanzapine, 15 mg aripiprazole
4	10 mg olanzapine, 5 mg risperidone
5	30 mg olanzapine, 5 mg risperidone
6	NO medication
7	12.5 mg olanzapine, 6 mg paliperidone
8	NO medication
9	20 mg olanzapine
10	400 mg Quetiapine, 9 mg paliperidone
11	25 mg olanzapine
12	30 mg olanzapine, 50mg clozapine
13	30 mg olanzapine, 5 mg risperidone
14	400 mg quetiapine, 5 mg risperidone
15	25 mg olanzapine, 50 mg clozapine
16	400 mg amisulpride, 5mg risperidone
17	NO medication
18	200 mg amisulpride, 15 mg aripiprazole
19	30 mg olanzapine, 400 mg quetiapine
20	15 mg olanzapine
21	200 mg clozapine, 12mg paliperidone

Table DS2	Extra-striatal intrinsic functional connectivity in the independent healthy control group sample							
Striatal seed	Anatomical regions	side	k	MNI	z-score	P-value*		
Putamen	MidFG, IFG (opercular, triangular, orbital) SFG Precentral gyrus, SMA Insula Putamen Pallidum Caudate Rolandic operculum Cingulum (anterior, middle)	R+L	6127	21 12 – 3	>7	<0.001		
Ventral striatum	Caudate Putamen IFG (orbital) SFG (medial) Insula Pallidum Cingulum (anterior) MidFG (orbital)	R	1432	12 15 – 12	>7	<0.001		
	Caudate Putamen Insula IFG (orbital) SFG (medial) Pallidum Cingulum (anterior) MidFG (orbital)	L	1337	-159-15	>7	<0.001		
Intrinsic functional connectivity of striatal subcomponents as displayed and described in more detail in Fig. DS1, (one-sample t-tests, P < 0.05 FWE-corrected for multiple comparisons).								

MNI = peak voxel coordinates in MNI-space (x y z).