Supplementary Methods

Participants

Forty patients with a diagnosis of DSM-IV schizophrenia spectrum disorders experiencing their first-episode of psychosis participated (FEP: 18 males and 22 females; mean age of 22.9 years, SD=5.6). They were from the Seoul Youth Clinic early psychosis cohort from April 2010 to June 2015. All patients had a history of less than a year since their first psychotic episode. They had schizophrenia (N=24), schizophreniform disorder (N=11), schizoaffective disorder (N=3), brief psychotic disorder (N=1) or psychotic disorder not otherwise specified (N=1). Twenty-nine patients were on atypical antipsychotic medication (risperidone, N=8; olanzapine, N=5; paliperidone, N=4; aripiprazole, N=3; ziprasidone, N=1; risperidone and olanzapine N=2; olanzapine and quetiapine N=2; risperidone and aripiprazole N=1; risperidone and quetiapine, N=1; ziprasidone and haloperidol, N=1, or paliperidone and aripiprazole, N=1). The remainder of 11 patients were not on any antipsychotic medication at the time of data collection.

Forty healthy participants individually matched for age (within 2 years) and gender served as a healthy control group (18 males and 22 females, mean age 23.1, SD=5.0). Exclusion criteria for both groups were contraindications to magnetic resonance scanning, neurological disorders (including previous head injury), and learning disabilities. Schizophrenia symptoms were rated using the Positive and Negative Syndrome Scale (PANSS).[1] After complete description of the study to the subjects, written informed consent was obtained. The study was approved by the local research ethics committee.

Functional connectivity analysis

Resting state fMRI data were analyzed using the Statistical Parametric Mapping software (SPM12,

www.fil.ion.ucl.ac.uk/spm) with the Functional Connectivity (CONN, V16) toolbox.[2] All participants met a movement threshold of under 2.5 mm in any direction or 2.5° in any rotation. Pre-processing involved re-alignment, unwarping, slice-time correction, co-registration of the mean functional image with the T1 image of each participant, spatial normalization of images into the Montreal Neurological Institute template with a resampled voxel size of 2x2x2mm, and spatial smoothing using a Gaussian kernel of full-width half-maximum 6 mm.

Masks for striatal and thalamic regions were obtained from the Harvard-Oxford subcortical structural atlas provided with FSL (fsl.fmrib.ox.ac.uk/fsl/fslwiki/Atlases). Masks for deep cerebellar nuclei (DNd, DNv, IN and FN) were based on quantitative cytoarchitecture implemented in the SPM anatomy toolbox.[3] Each ROI was thresholded to contain only voxels that were inside each ROI with a probability above 60%. This was because, we wanted to prevent signal contamination from neighboring regions. This was particularly important in using cerebellar ROIs that are close to each other. When extracting ROI-level signals, we used unsmoothed images to further avoid signal contamination from surrounding areas.[4]

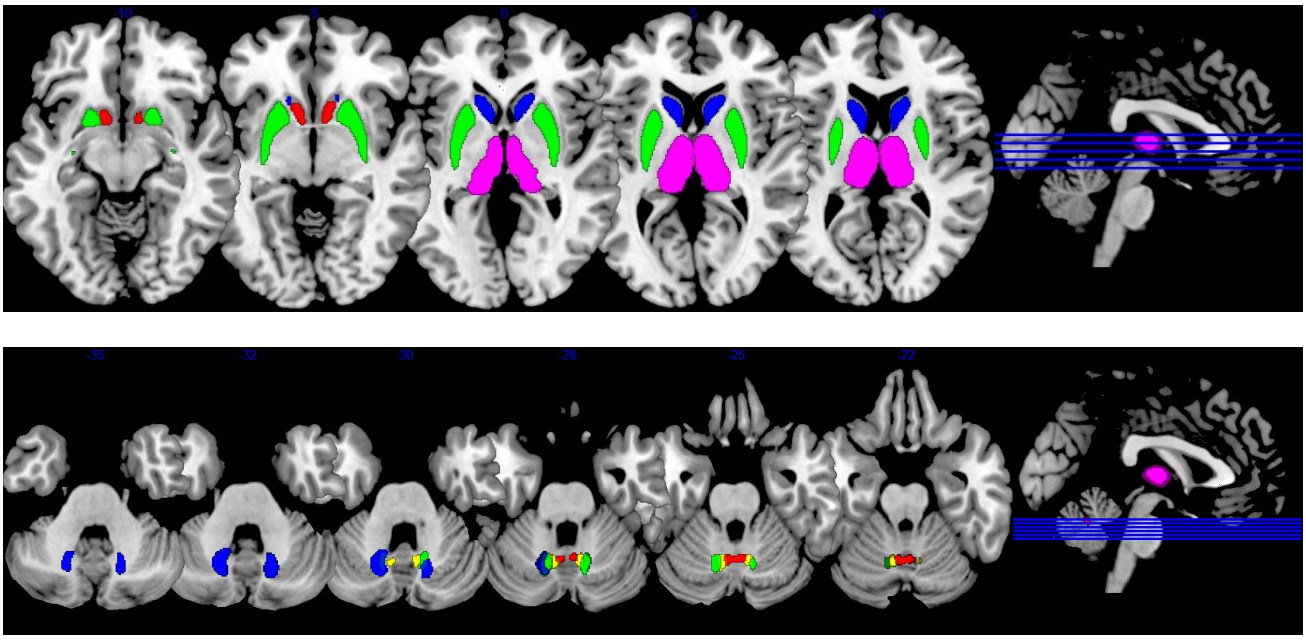

Figure S1. Striatal, thalamic and cerebellar regions of interest (ROIs) used in the study overlaid onto the MNI-152 T1-weighted brain image. Caudate (blue), putamen (green), nucleus accumbens, (red), and thalamus (pink) are shown in the upper panel. Ventral dentate nucleus (Blue), dorsal dentate nucleus (green), interpositus nucleus (yellow) and fastigial nucleus (red) are shown in the lower panel.

We took care to account for head motion artifacts, although there were no between-group differences in any of the translation parameters calculated during realignment (See Table S1). We used Friston's 24 head motion parameters in minimizing motion related artifacts (6 head motion parameters, 6 head motion parameters for difference between one time point and its preceding time point, and the 12 corresponding squared parameters).[5] The use of Friston's 24 parameters was superior to the use of smaller sets of parameters in removing motion artifacts for resting state fMRI analysis.[6,7] Further, five principal components from each of white matter and CSF masks were used in regressing out physiological artifacts.[8] This component-based method is critical in obtaining valid anti-correlations that are not an artifact produced by global signal regression.[9] The resulting time series were bandpass-filtered between 0.008 and 0.09 Hz to reduce the effect of low frequency drifts and high-frequency noise, as implemented in the CONN toolbox. As suggested, data scrubbing was not performed.[7]

Table S1. Group-averaged head motion parameters. Standard deviations are given in brackets.

Metric	Group	
	FEP	HCs
Translation		
X	-0.0008 (0.05392)	-0.0394 (0.21887)
Y	0.0367 (0.07415)	0.034 (0.13962)
Z	0.0023 (0.18843)	-0.0024 (0.33575)

In our ROI analysis, there was a 16x16 ROI-to-ROI connectivity matrix for each subject. We conducted both individual connection-level and network-level functional connectivity analyses. The individual connection level analysis represents the identification of between group differences in

individual connections with a statistical threshold correction procedure for inflated Type I error such as false discovery rate (FDR) correction.[10] The network-based statistic (NBS) is used to identify interconnected structures that are associated with group-differences.[11] The use of these complementary methods is important in identifying damaged connections and compensatory network activity in abnormal brain function.[12]

We used FDR corrected p-values in our individual connection-level analysis with a threshold of $p < .05$. In our network-level functional connectivity analysis, we used network intensity, which is the sum of test statistic values across all connections. As suggested,[11] we first selected individual connections with a lenient threshold ($p < .1$), and then we applied family-wise error (FWE) correction with a threshold of $p < .05$. For the seed-to-voxel analysis, regions of significant difference were defined by the voxel-level height threshold of uncorrected $p < .001$ and the cluster-level extent threshold of $p < .05$, corrected for FWE.

Verbal fluency and clinical ratings

Verbal fluency was assessed with the Controlled Oral Word Association Test (COWA),[13] including a letter-fluency task and a category-fluency task. Each task required the participants to produce as many words as possible, beginning with a given letter (letter fluency: 3 letters) or from a category (category fluency: 2 categories) in 60 seconds. The total number of correct responses was scored.

Patients' functional outcome was measured using the Global Assessment of Functioning (GAF) scale of the DSM-IV. Schizophrenia symptoms were rated using the Positive and Negative Syndrome Scale (PANSS).[1] We grouped PANSS items to derive the three core psychopathological dimensions of 'psychomotor poverty' (blunted affect, emotional withdrawal, social withdrawal, and poor rapport), 'disorganization' (conceptual disorganization, stereotyped thinking, difficulty in abstract thinking, and excitement) and 'reality distortion' (suspiciousness/persecution, hostility, grandiosity, and delusions)

as previously.[14]

Supplementary Results

DTI findings

The means and standard deviations of DTI variables for three pairs of cerebellar peduncles were represented in supplementary results for documentary purpose (Table S2). The significant 3 DTI indices for the left SCP were all negatively correlated with current GAF scores at a trend level (AD: r=-.288, p=.071; RD: r=-.300, p=.060; MD: r=-.305, p=.056). In addition, left SCP RD values were negatively correlated with category fluency performance at a trend level (r=.282, p=.078). In contrast, right SCP FA values were positively correlated with current GAF scores (r=.332, p=.036) and category verbal fluency scores at a trend level (r=.282, p=.078), but negatively correlated with disorganization scores at a trend level (r=-.291, p = .068).

Table S2. Means and standard deviations (in brackets) for DTI variables in FEP and control participants.

	FEP				Controls			
	Left		Right		Left		Right	
SCP								
AD	0.00155	(0.00017)	0.00156	(0.00014)	0.00146	(0.00009)	0.00155	(0.00013)
RD	0.00080	(0.00015)	0.00080	(0.00013)	0.00075	(0.00009)	0.00082	(0.00013)
MD	0.00105	(0.00015)	0.00105	(0.00013)	0.00099	(0.00009)	0.00106	(0.00013)
FA	0.45986	(0.04682)	0.46752	(0.04825)	0.45342	(0.03361)	0.45119	(0.03428)
MCP								
AD	0.00125	(0.00013)	0.00124	(0.00013)	0.00122	(0.00007)	0.00122	(0.00007)
RD	0.00068	(0.00014)	0.00066	(0.00012)	0.00066	(0.00008)	0.00065	(0.00007)
MD	0.00087	(0.00013)	0.00085	(0.00012)	0.00084	(0.00007)	0.00084	(0.00007)
FA	0.42255	(0.04025)	0.43156	(0.03880)	0.42164	(0.03230)	0.43203	(0.03588
ICP								
AD	0.00137	(0.00013)	0.00134	(0.00010)	0.00134	(0.00008)	0.00132	(0.00006)
RD	0.00079	(0.00015)	0.00074	(0.00011)	0.00075	(0.00008)	0.00071	(0.00006)
MD	0.00098	(0.00014)	0.00094	(0.00010)	0.00095	(0.00008)	0.00091	(0.00006)
FA	0.39036	(0.03463)	0.40290	(0.03571)	0.39277	(0.02007)	0.41007	(0.02218)

Significant between-group differences were found in SCP variables as described in the main text. However, none of variables were significantly different between-groups for the MCP or ICP.

Volumetric findings

Table S3. Means and standard deviations (in brackets) for volumetric variables in FEP and control participants.

	FEP				Controls			
Intracranial Volume	1572223.0 (176210.5)				1560360.4 (158042.3)			
	Left		**Right**		**Left**		**Right**	
Thalamus	8787.7	(1252.3)	7867.5	(846.4)	8682.4	(783.5)	7946.1	(719.9)
Caudate	3898.6	(536.5)	3630.4	(513.0)	3869.0	(513.2)	3662.1	(558.9)
Putamen	5530.6	(623.4)	5423.2	(704.0)	5861.8	(775.0)	5638.2	(730.9)
Accumbens	493.1	(99.3)	531.7	(94.5)	522.4	(90.6)	535.8	(85.1)
Ant. cerebellum	8042.9	(931.0)	8698.6	(1027.1)	8391.7	(1016.7)	8831.8	(998.5)
Superior /posterior cerebellum	38581.5	(5233.0)	38756.6	(5586.9)	39512.6	(3899.6)	39489.0	(4588.9)
Inferior/posterior cerebellum	14373.5	(2168.5)	14184.3	(2148.5)	14551.3	(1792.0)	14245.7	(1785.9)
Vermis	5845.5 (762.4)				5919.7(574.7)			

Left putamen volume and left anterior cerebellar volume (trend) were significantly reduced as described in the main text. None of other variables were significantly different between-groups.

DTI and volumetric contributions to functional connectivity

To evaluate the contributions of DTI and volumetric abnormalities to functional connectivity results reported above, we repeated all functional connectivity analyses, controlling for variables producing significant or near-significant between-group differences (left SCP MD values, right SCP FA values, left putamen or left anterior cerebellar grey-matter volumes). Our functional connectivity finding at individual connection level (left FN and right putamen connectivity) became a trend ($T(77) = -2.57$, $p=.0828$, corrected), when left putamen volume was used as a covariate. For our NBS results, the significant between-group difference became a trend, only when left anterior cerebellar volume was

entered as a covariate (p=.099, corrected). For our unique functional connectivity results, the left IN connectivity with right Broca's area (BA 45) was not changed by any covariates. However, the right DNv and left frontopolar connectivity became non-significant when left SCP MD values were used as a covariate. For right IN connectivity with the left inferior parietal cortex (BA39) and right premotor cortex (BA6), these became non-significant, when left putamen volume was used as a covariate. With right SCP FA covariate, right IN connectivity with the left inferior parietal cortex became non-significant. Finally, the right IN and premotor connectivity became non-significant with left SCP MD values or left anterior cerebellar volume as a covariate.

Table S4. Factor structure from a principal component analysis for variables associated with abnormalities in FEP

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
Variables					
Right_putamen volume	**.928**	-.005	.040	-.027	.021
Left putamen volume	**.918**	-.068	-.149	.153	.143
Left anterior cerebellar volume	**.622**	-.307	.005	-.278	.032
Left IN/right Broca unique connectivity	-.036	**.831**	.106	.128	.092
Left NAc volume	.274	**-.636**	-.017	.225	.180
Left NAc network activity	.088	**.494**	**-.445**	-.258	**.453**
Right IN/left IPC unique connectivity	.102	-.027	**.822**	-.078	-.080
Right SCP FA	-.325	.221	**.648**	.200	.182
Left FN/right putamen connectivity	-.064	-.146	-.114	**.804**	.197
Right_DNv/left frontopolar unique connectivity	-.041	-.093	-.196	**-.678**	.289
Right IN/right premotor unique connectivity	-.287	.065	.075	-.069	**-.749**
Left_SCP_MD	**.405**	-.093	-.318	.129	**-.562**
Eigenvalue	2.54	1.53	1.48	1.41	1.31
Variance explained (%)	21.20	12.75	12.39	11.74	10.88

Factor loadings greater than 0.40 are in bold. Factor 3 significantly negatively correlated with disorganization syndrome scores (r=.445, p=.004). GAF scores were positively associated with Factor 3 (r=.338, p=.033) and Factor 4 (r=.400, r=.011). In addition, Factor 1 was positively correlated psychomotor poverty scores (r=.300, p=.060) and negatively associated with category fluency performance (r=-.297, p=.063).

Supplementary discussion

Patients exhibited decreased RSFC between the left FN and right putamen at individual connection level. As RSFC is based on temporal correlations between activities of two different brain areas, it is uncertain which area is primarily disturbed in case of disordered left FN and right putamen RSFC. Nonetheless, it should be noted that the FN has never been a subject of empirical investigation in schizophrenia research, although investigating the FN was suggested as important in understanding schizophrenia pathophysiology over two decades ago.[15] In addition to FN's role in cerebello-thalamic projections, the FN is the starting point of the cerebello-hypothalamic pathway involved in autonomic function including cardiovascular function,[16] and is connected to locus coeruleus (LC) norepinephrine neurons to influence alertness and attention.[17] The FN also plays a key role in the generation and control of saccadic and smooth-pursuit eye movements.[18] Considering that autonomic and eye movements abnormalities have been widely recognized in schizophrenia research, further research is warranted to establish the role of the FN in this illness.

A reduction in white-matter integrity in the left superior cerebellar peduncle (SCP), in terms of increased axial diffusivity (AD) and mean diffusivity (MD), was found in patients. Accordingly, a number of previous studies have reported abnormalities that are specific to the SCP in chronic schizophrenia patients.[19-22] The majority of these reported predominant left SCP abnormalities. Furthermore, adolescents at high risk for psychosis progressively exhibited reduced FA values in the SCP (left more than right) over 12 months.[23] However, the middle cerebellar peduncle (MCP) which carries input fibers from the contralateral cerebral cortex to the cerebellar cortex via the pons was not significantly lowered in adolescents with first-admission or chronic schizophrenia patients.[24,25] We also documented that the MCP was not disordered in our patients (See Table S2). It should be noted that the cerebellar white matter atlas we used defined the SCP as white matter tracts starting from the ipsilateral dentate nucleus to the contralateral thalamus.[26] In order to fully investigate structural connectivity between the cerebellum and striatum, the end point would need to be extended to the

striatum. Furthermore, as well as the DN, both the IN and FN should be included as starting regions of the SCP in such a DTI investigation. We are presently undertaking this work.

Previous studies with chronic schizophrenia patients have found either increased (with 884 patients)[27] or statistically not different (with 2,028 patients)[28] putamen volume compared with HCs. Antipsychotic medication might play a role in increasing caudate and putamen volumes.[29] Instead, we found decreased left putamen volume (and right putamen volume to a lesser extent) in FEP. Our finding is consistent with studies demonstrating smaller left putamen volume in individuals at high clinical-risk,[30] and bilateral putamen volume reduction (left more than right) along with increased variability in motor responses in non-clinical participants with a high score on a psychotic experience questionnaire.[31] We suggest that the effect of long-term antipsychotic medication in increasing putamen volume may mask smaller putamen volume at an early stage of, or even well before the onset, of schizophrenia. In addition, we found that putamen volume was highly positively correlated with left anterior cerebellar volume in patients, but not in HCs. Normally, there would not be a correlation between a sub-cortical area's volume with cerebellar volume (when head-size was corrected), as the cerebellum increased in size along with neocortical size while relative subcortical volume size remained unchanged.[32] Thus, it is highly likely that the same pathological process underlies this closely related volume loss. That is, "neurons that fire together, wire together and survive together" to form a network,[33] but this networking is lost in patients.

We found a strong trend of left anterior cerebellar grey-matter volume reduction, which is consistent with a meta-analysis of grey-matter deficit in first-episode schizophrenia.[34] Studies published after this meta-analysis have also reported a prominent grey-matter volume deficit in the anterior cerebellum using a whole-brain VBM approach[35] as well as using a cerebellar ROI approach[36] in patients with first-episode schizophrenia. Neuroimaging and neuropsychological studies link the anterior cerebellum with sensorimotor function.[37] It is well established in animal studies that the anterior cerebellum with its projection to the interpositus nucleus (IN) plays an important role in the

timing of delay eye blink conditioning.[38] Accordingly, delay eye blink conditioning and timing functions are significantly impaired in both patients with schizophrenia and their first-degree relatives.[39-41] A voxel-based morphometry study in schizophrenia patients further showed that eye blink conditioning performance was significantly correlated with anterior cerebellar volume,[42] and a functional neuroimaging study showed that the anterior cerebellum along with thalamic and frontal areas was under-activated in patients.[43] Together, these results suggest that anterior cerebellar volume reduction is an early neurobiological sign of schizophrenia.

References

1. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophr Bull.* 1987;13(2):261-276.
2. Whitfield-Gabrieli S, Nieto-Castanon A. Conn: a functional connectivity toolbox for correlated and anticorrelated brain networks. *Brain Connect.* 2012;2(3):125-141.
3. Tellmann S, Bludau S, Eickhoff S, Mohlberg H, Minnerop M, Amunts K. Cytoarchitectonic mapping of the human brain cerebellar nuclei in stereotaxic space and delineation of their co-activation patterns. *Front Neuroanat.* 2015;9:54.
4. Mishra A, Rogers BP, Chen LM, Gore JC. Functional connectivity-based parcellation of amygdala using self-organized mapping: a data driven approach. *Hum Brain Mapp.* 2014;35(4):1247-1260.
5. Friston KJ, Williams S, Howard R, Frackowiak RS, Turner R. Movement-related effects in fMRI time-series. *Magn Reson Med.* 1996;35(3):346-355.
6. Yan CG, Cheung B, Kelly C, et al. A comprehensive assessment of regional variation in the impact of head micromovements on functional connectomics. *NeuroImage.* 2013;76:183-201.
7. Power JD, Mitra A, Laumann TO, Snyder AZ, Schlaggar BL, Petersen SE. Methods to detect, characterize, and remove motion artifact in resting state fMRI. *NeuroImage.* 2014;84:320-341.
8. Behzadi Y, Restom K, Liau J, Liu TT. A component based noise correction method (CompCor) for BOLD and perfusion based fMRI. *NeuroImage.* 2007;37(1):90-101.
9. Chai XJ, Castanon AN, Ongur D, Whitfield-Gabrieli S. Anticorrelations in resting state networks without global signal regression. *NeuroImage.* 2012;59(2):1420-1428.
10. Hochberg Y, Benjamini Y. More powerful procedures for multiple significance testing. *Stat Med.* 1990;9(7):811-818.
11. Zalesky A, Fornito A, Bullmore ET. Network-based statistic: identifying differences in brain networks. *NeuroImage.* 2010;53(4):1197-1207.
12. Simpson SL, Laurienti PJ. Disentangling Brain Graphs: A Note on the Conflation of Network

and Connectivity Analyses. *Brain Connect.* 2016;6(2):95-98.

13. Benton AL. Development of a multilingual aphasia battery. Progress and problems. *J Neurol Sci.* 1969;9(1):39-48.
14. Lee KH, Williams LM, Loughland CM, Davidson DJ, Gordon E. Syndromes of schizophrenia and smooth-pursuit eye movement dysfunction. *Psychiatry Res.* 2001;101(1):11-21.
15. Martin P, Albers M. Cerebellum and schizophrenia: a selective review. *Schizophr Bull.* 1995;21(2):241-250.
16. Dietrichs E. Cerebellar autonomic function: direct hypothalamocerebellar pathway. *Science.* 1984;223(4636):591-593.
17. Schwarz LA, Miyamichi K, Gao XJ, et al. Viral-genetic tracing of the input-output organization of a central noradrenaline circuit. *Nature.* 2015;524(7563):88-92.
18. Kheradmand A, Zee DS. Cerebellum and ocular motor control. *Front Neurol.* 2011;2:53.
19. Okugawa G, Nobuhara K, Minami T, et al. Neural disorganization in the superior cerebellar peduncle and cognitive abnormality in patients with schizophrenia: A diffusion tensor imaging study. *Prog Neuropsychopharmacol Biol Psychiatry.* 2006;30(8):1408-1412.
20. Liu H, Fan G, Xu K, Wang F. Changes in cerebellar functional connectivity and anatomical connectivity in schizophrenia: a combined resting-state functional MRI and diffusion tensor imaging study. *J Magn Reson Imaging.* 2011;34(6):1430-1438.
21. Magnotta VA, Adix ML, Caprahan A, Lim K, Gollub R, Andreasen NC. Investigating connectivity between the cerebellum and thalamus in schizophrenia using diffusion tensor tractography: a pilot study. *Psychiatry Res.* 2008;163(3):193-200.
22. Huttlova J, Kikinis Z, Kerkovsky M, et al. Abnormalities in myelination of the superior cerebellar peduncle in patients with schizophrenia and deficits in movement sequencing. *Cerebellum.* 2014;13(4):415-424.
23. Mittal VA, Dean DJ, Bernard JA, et al. Neurological soft signs predict abnormal cerebellar-thalamic tract development and negative symptoms in adolescents at high risk for psychosis: a longitudinal perspective. *Schizophr Bull.* 2014;40(6):1204-1215.

24. Wang F, Sun Z, Du X, et al. A diffusion tensor imaging study of middle and superior cerebellar peduncle in male patients with schizophrenia. *Neurosci Lett.* 2003;348(3):135-138.

25. Henze R, Brunner R, Thiemann U, et al. The optic radiation and the cerebellar peduncles in adolescents with first-admission schizophrenia--a diffusion tensor imaging study. *J Neuroimaging.* 2014;24(2):111-116.

26. van Baarsen KM, Kleinnijenhuis M, Jbabdi S, Sotiropoulos SN, Grotenhuis JA, van Cappellen van Walsum AM. A probabilistic atlas of the cerebellar white matter. *NeuroImage.* 2016;124(Pt A):724-732.

27. Okada N, Fukunaga M, Yamashita F, et al. Abnormal asymmetries in subcortical brain volume in schizophrenia. *Mol Psychiatry.* 2016;21(10):1460-1466.

28. van Erp TG, Hibar DP, Rasmussen JM, et al. Subcortical brain volume abnormalities in 2028 individuals with schizophrenia and 2540 healthy controls via the ENIGMA consortium. *Mol Psychiatry.* 2016;21(4):585.

29. Jorgensen KN, Nesvag R, Gunleiksrud S, Raballo A, Jonsson EG, Agartz I. First- and second-generation antipsychotic drug treatment and subcortical brain morphology in schizophrenia. *Eur Arch Psychiatry Clin Neurosci.* 2016;266(5):451-460.

30. Hong SB, Lee TY, Kwak YB, Kim SN, Kwon JS. Baseline putamen volume as a predictor of positive symptom reduction in patients at clinical high risk for psychosis: A preliminary study. *Schizophr Res.* 2015;169(1-3):178-185.

31. Mittal VA, Orr JM, Turner JA, et al. Striatal abnormalities and spontaneous dyskinesias in non-clinical psychosis. *Schizophr Res.* 2013;151(1-3):141-147.

32. Barton RA. How did brains evolve? *Nature.* 2002;415(6868):134-135.

33. Post RM, Weiss SR. Emergent properties of neural systems: how focal molecular neurobiological alterations can affect behavior. *Dev Psychopathol.* 1997;9(4):907-929.

34. Leung M, Cheung C, Yu K, et al. Gray matter in first-episode schizophrenia before and after antipsychotic drug treatment. Anatomical likelihood estimation meta-analyses with sample size weighting. *Schizophr Bull.* 2011;37(1):199-211.

35. Lei W, Deng W, Li M, et al. Gray matter volume alterations in first-episode drug-naive patients with deficit and nondeficit schizophrenia. *Psychiatry Res.* 2015;234(2):219-226.

36. Cohen M, Rasser PE, Peck G, et al. Cerebellar grey-matter deficits, cannabis use and first-episode schizophrenia in adolescents and young adults. *Int J Neuropsychopharmacol.* 2012;15(3):297-307.

37. Stoodley CJ, Schmahmann JD. Evidence for topographic organization in the cerebellum of motor control versus cognitive and affective processing. *Cortex.* 2010;46(7):831-844.

38. Perciavalle V, Apps R, Bracha V, et al. Consensus paper: current views on the role of cerebellar interpositus nucleus in movement control and emotion. *Cerebellum.* 2013;12(5):738-757.

39. Bolbecker AR, Kent JS, Petersen IT, et al. Impaired cerebellar-dependent eyeblink conditioning in first-degree relatives of individuals with schizophrenia. *Schizophr Bull.* 2014;40(5):1001-1010.

40. Lee KH, Bhaker RS, Mysore A, Parks RW, Birkett PB, Woodruff PW. Time perception and its neuropsychological correlates in patients with schizophrenia and in healthy volunteers. *Psychiatry Res.* 2009;166(2-3):174-183.

41. Penney TB, Meck WH, Roberts SA, Gibbon J, Erlenmeyer-Kimling L. Interval-timing deficits in individuals at high risk for schizophrenia. *Brain Cogn.* 2005;58:109-118.

42. Edwards CR, Newman S, Bismark A, et al. Cerebellum volume and eyeblink conditioning in schizophrenia. *Psychiatry Res.* 2008;162(3):185-194.

43. Parker KL, Andreasen NC, Liu D, Freeman JH, O'Leary DS. Eyeblink conditioning in unmedicated schizophrenia patients: a positron emission tomography study. *Psychiatry Res.* 2013;214(3):402-409.