# Supplementary data 1

# Methodological Detail

## Data Preprocessing

The SPM12 toolbox (www.fil.ion.ucl.ac.uk/spm) was used for preprocessing the fMRI data. Before further pre-process, the first five volumes were discarded to eliminate the effects of magnetization disequilibrium. Slice timing differences induced by interleaved fMRI acquisition and head motion between volumes were corrected. Then, the fMRI data were normalized using a non-linear registration into 3-mm MNI space. To reduce the effects of physiological processes, WM, cerebrospinal fluid (CSF), linear drift, the global averaged signals and six head motion parameters (R) with their derivatives ([R, diff(R)]) were extracted and were regressed out from the fMRI data of each subject by using a homemade NIT toolbox (http://www.neuro.uestc.edu.cn/NIT.html). Then, the data were temporally filtered using a 0.01–0.08 Hz band-pass filter to remove the physiological noise and potential magnetic field drifts. Besides, as a part of data quality assessment, the difference in frame-wise displacements between two groups were inspected1.

FSL (FMRIB Software Library v5.0.9)2 was used to preprocess the dMRI data. First, toestimatethesusceptibility-induceddistortions,twotypesofunweightedimages,whichwereacquiredusingR/LandA/Pfrequencydirectionrespectively,wereintroducedtotheFSLtopuptool.Subsequently,theeffectsofdistortioninducedbyeddycurrents,inter-volumemovementsandsusceptibilityofthediffusiondatawerecorrectedbyusingeddy3.Thenindividualunweightedimagewas rigidlyalignedwiththestructuralimageusingflirt.Non-linearregisteringwas adopted totransformindividualstructuralimagetoanMNI152standardT1-weightedtemplateusingfnirt.ThentheforwardandbackwardwarpfieldimagesbetweenindividualdMRIandMNIT1spaceswereacquiredbyconcatenating(orinverting)therigidtransformationmatrixandthewarpfieldimagewhichdefinethetransformationbetweenindividualstructuralimagesandMNIstandardspace.DiffusionparametersateachvoxelwereestimatedbyusingMarkovChainMonteCarlosampling4.Inthisstep,upto2possiblefiberpopulationsweremodeledforeachvoxelafter2000iterations.

To control the data quality, we checked the structural image, the average of the non-diffusion-weighted images and an example of the fMRI for each participant. Detailed, a subject would be excluded from further analysis if the signal-noise-rate (SNR) of structural image or unweighted-diffusion image was lower than 800. Also, the results of registering were evaluated by visual inspection. Furthermore, subjects who had a greater than 2 mm frame-wise displacements of the dMRI or fMRI were excluded from further analyses.

## Thalamic Parcellation Combined White Matter Tractography and Functional Connectivity Analysis

Voxels from left (contained 449 voxels, extracted from the Harvard-Oxford atlas with a resolution of 2 × 2 × 2 mm3 in FSL) and right thalamus (contained 444 voxels) were used as seeds in the probabilistic tractography step to acquire WM tractograms. In the tractography process, a total of 10,000 iterations were performed for each seed voxel. To correct the distance-dependent bias, value of each voxel in tractograms was weighted by the streamline length between the seed and this voxel. Next, these tractograms were registered to MNI space at an isotropic resolution of 3-mm according to the previously mentioned warpfield images. A Gaussian kernel with a 4-mm full-width half-maximum was used to smooth the tractogram in seed-space.

Individual tractograms of all subjects were merged into 4D volumes according to left and right hemispheres respectively. And then these two 4D-tractogram datasets (left and right) were fed into a group independent component analysis (ICA). Both datasets were decomposed into several components using tensor-ICA5. Each component represented a spatial pattern of WM connectivity that was consistent across subjects, then was defined as “thalamic origin”. At last, individual weighted thalamic origins were generated by mapping back the normalized “temporal response” of each component onto the seed space in MNI. These dozens of thalamic origins were used to represent the distribution of different WM fibers in the thalamus. At this point, by using the WM connectivity information, the first step of thalamus parcellation was completed.

The functional connectivity of individual thalamic origin was used for the second step of thalamus parcellation. First, for each thalamic origin, their BOLD time courses were got by a spatial regression on the preprocessed fMRI data. Then, rsFC maps were obtained by correlating the temporal series of these origin with the temporal series of the whole brain voxels. Subsequently, these maps were spatially smoothed by a 6-mm FWHM Gaussian kernel. RsFC t-maps of each thalamic origin was calculated by using a one-sample t-test across rsFC maps of all subjects.

Finally, the parcellation of thalamus was carried out by following strategies: Pearson’s correlation was used to construct a similarity matrix of rsFC t-maps of each thalamic origin. Affinity propagation6 was then introduced to cluster the correlation coefficient matrix. According to the clustered result, the winner-takes-all strategy was used to mark all thalamic origins (whole thalamus) into several subdivisions. Now, the thalamic parcellation was finished7.

After that, Morel atlas8 was used to identify which nuclei were included in each subdivision. After this the composition of each subdivision of thalamus could be defined. Because the patient's thalamic functional connectivity is thought to change, a one-sample t-test was performed on the rsFC maps of controls only, to determine the connectivity pattern of each subdivision.

# Limitation and Scope

Several limitations of this study should be noted. To a certain extent, our work may have a circular logic. But we are more inclined to consider our study as a thalamic parcellation by the integration of structural connectivity and FC, followed by its comparative analysis in FC across groups. To avoid circular logic of the clustering with FC maps of thalamic origins resulted from the DTI analysis and to verify the main results, we induced a conjunction-overlay-like method in this dataset. Briefly, based on the results of affinity propagation clustering, the masks of intergroup difference of thalamic origins belonging to the same cluster were overlaid. These overlays could represent the intergroup difference in FC of thalamic set. Using this method, the calculation of FC of the thalamic set is not needed, which avoids the “circular” analysis. The detail of this method and results can be found in the supplementary material (Supplementary calculation: Conjunction overlay). The results from conjunction overlay were in line with the results from the direct FC of thalamic sets. However, it must be noted that, when a set contains few thalamic origins, the results of the conjunction overlay will be not stable (such as set 5). This is the reason why the results of conjunction overlay have not been reported in the main manuscript.

The findings of the present study demonstrated that compared with the HC, three of the six thalamic subdivision of schizophrenia patients showed specific alterations in FC with the cortex. However, some shortcomings are present in this study. First, several studies have focused on the differences in functional/structural connectivity relative to the HC group in first-episode schizophrenic (FES) patients, even in CHR for psychosis subjects9. A study on FES can avoid the interference of drug effects and obtain a more realistic, reliable relationship between brain network alterations and early clinical symptoms. Furthermore, a focus on CHR could find the potentially damaged brain networks before the onset of clinical symptoms. This may provide guidance for early clinical intervention. Therefore, we expect to recruit patients with FES, or CHR subjects in the future to reveal the early functional changes in specific thalamic nuclei, and to observe the abnormal pattern of the cerebellar-thalamocortical circuit in early stages of schizophrenia.

# Supplementary tables

|  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Supplementary Table 1 Table s1 | | | | | | | | | | | |
| The composition of the nucleus | | | | | | | | | | | |
| Region 1 | | Region 2 | | Region 3 | | Region 4 | | Region 5 | | Region 6 | | |
| Left | Right | Left | Right | Left | Right | Left | Right | Left | Right | Left | Right | |
| CL | -- | CL | -- | AV | AV | AD | -- | -- | AV | CeM | CeM | |
| LD | -- | CM | CM | -- | CL | AM | AM | Hb | -- | CL | CL | |
| LP | LP | -- | Li | LD | LD | AV | AV | LD | LD | CM | CM | |
| MGN | MGN | LP | LP | -- | LP | CeM | CeM | LP | LP | Hb | Hb | |
| -- | Po | MDpc | -- | PuM | PuM | CL | CL | -- | PuM | Li | -- | |
| -- | PuI | MGN | -- | body | body | -- | LD | body | body | LP | -- | |
| PuL | PuL | Pf | -- | VApc | -- | MDmc | MDmc | VApc | VApc | -- | MDmc | |
| PuM | PuM | Po | Po | VLpd | VLpd | MDpc | MDpc | VLpd | VLpd | MDpc | MDpc | |
| SG | SG | PuA | PuA | VLpv | -- | -- | Pv | VPLp | VPLp | Pf | -- | |
| body | body | PuL | -- |  |  | body | -- |  |  | -- | VAmc | |
| VLpd | -- | PuM | PuM |  |  | VAmc | VAmc |  |  | VApc | VApc | |
| VPLp | -- | SG | SG |  |  | VApc | VApc |  |  | VLa | VLa | |
|  |  | body | -- |  |  | -- | VLpd |  |  | VLpd | VLpd | |
|  |  | -- | VLa |  |  | VLpv | VLpv |  |  | VLpv | VLpv | |
|  |  | VLpd | VLpd |  |  |  |  |  |  | -- | VM | |
|  |  | VLpv | VLpv |  |  |  |  |  |  | VPLp | VPLp | |
|  |  | VPI | VPI |  |  |  |  |  |  | VPM | VPM | |
|  |  | VPLa | VPLa |  |  |  |  |  |  |  |  | |
|  |  | VPLp | VPLp |  |  |  |  |  |  |  |  | |
|  |  | VPM | VPM |  |  |  |  |  |  |  |  | |

Abbreviations: VPL:Ventral posterior lateral nucleus; MD:Mediodorsal nucleus; MDmc:Mediodorsal nucleus Magnocellular part; MDpc:Mediodorsal nucleus Parvocellular part; MV:Medioventral nucleus; CL:Central lateral nucleus; CeM:Central medial nucleus; CM:Centre médian nucleus; Pv:Paraventricular nucleus; Hb:Habenular nucleus; Pf:Parafascicular nucleus; sPf:Subparafascicular nucleus; PuM:Medial pulvinar; PuI:Inferior pulvinar; PuL:Lateral pulvinar; PuA:Anterior pulvinar; LP:Lateral posterior nucleus; MGN:Medial geniculate nucleus; SG:Suprageniculate nucleus; Li:Limitans nucleus; Po:Posterior nucleus; LGN:Lateral geniculate nucleus; VPLa:Lateral geniculate nucleus Anterior part; VPLp:Lateral geniculate nucleus Posterior part; VPM:Ventral posterior medial nucleus; VPI:Ventral posterior inferior nucleus; VL:Ventral lateral nucleus; VLa:Ventral lateral anterior nucleus; VLp:Ventral lateral posterior nucleus; VLpd:Ventral lateral Dorsal part; VLpv:Ventral lateral Ventral part; VA:Ventral anterior nucleus; VAmc:Ventral anterior nucleus Magnocellular part; VApc:Ventral anterior nucleus Parvocellular part; VM:Ventral medial nucleus; AD:Anterior dorsal nucleus; AM:Anterior medial nucleus; AV:Anterior ventral nucleus; LD:Lateral dorsal nucleus; RN:Red nucleus; mtt:Mammillothalamic tract; STh:Subthalamic nucleus;

## Supplementary Table 2

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| Table s2 | | | | | | | |
| FC difference between SZ and HC part 1 (Contrast: SZ > HC) | | | | | | | |
| **Seed** | **Region** | **Hemisphere** | **Cluster size** | **MNI coordinate** | | | **peak t** |
| **X (mm)** | **Y (mm)** | **Z (mm)** |
| ROI3 | Calcarine | L | 28 | -9 | -63 | 3 | 5.9555 |
|  | Lingual | R | 14 | 18 | -57 | -3 | 5.2881 |
|  | Precuneus | R | \ | 20 | -56 | 6 | 5.2051 |
| ROI4 | Postcentral | R | 103 | 54 | -12 | 51 | 5.9137 |
|  | Precentral | R | \ | 42 | -14 | 52 | 5.5116 |
|  | Lingual | R | 372 | 21 | -57 | -3 | 6.2744 |
|  | Cuneus | R | \ | 8 | -77 | 28 | 5.946 |
|  | Occipital\_Sup | R | \ | 23 | -80 | 33 | 6.1932 |
|  | Precuneus | R | \ | 20 | -59 | 8 | 5.481 |
|  | Calcarine | R | \ | 8 | -71 | 5 | 5.4118 |
|  | Occipital\_Inf | L | 20 | -30 | -84 | -6 | 6.3169 |
|  | Occipital\_Sup | L | 126 | -21 | -84 | 27 | 6.4029 |
|  | Postcentral | L | 83 | -51 | -9 | 54 | 5.7227 |
|  | Precentral | L | \ | -52 | -9 | 51 | 5.5518 |
|  | Temporal\_Mid | L | 13 | -60 | -39 | 6 | 5.2164 |
|  | Temporal\_Mid | R | 136 | 51 | -72 | 0 | 6.4484 |
|  | Temporal\_Inf | R | \ | 50 | -70 | -3 | 6.354 |
|  | Temporal\_Sup | L | 51 | -60 | -9 | 0 | 5.5355 |

## Supplementary Table 3

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| Table s3 | | | | | | | |
| FC difference between SZ and HC part 2 (Contrast: SZ > HC) | | | | | | | |
| **Seed** | **Region** | **Hemisphere** | **Cluster size** | **MNI coordinate** | | | **peak t** |
| **X (mm)** | **Y (mm)** | **Z (mm)** |
| ROI5 | Insula | R | 64 | 36 | -12 | 12 | 6.6878 |
|  | Lingual | L | 131 | -9 | -66 | 3 | 6.3681 |
|  | Calcarine | L | \ | -11 | -66 | 7 | 5.7625 |
|  | Lingual | R | 263 | 18 | -51 | -6 | 6.559 |
|  | Precuneus | R | \ | 18 | -56 | 6 | 6.3046 |
|  | Calcarine | R | \ | 8 | -65 | 8 | 5.7858 |
|  | Occipital\_Mid | L | 117 | -48 | -78 | 3 | 6.176 |
|  | Occipital\_Inf | L | \ | -49 | -72 | -2 | 5.747 |
|  | Occipital\_Sup | L | 76 | -24 | -81 | 24 | 5.9653 |
|  | Occipital\_Sup | R | 129 | 18 | -81 | 30 | 5.9573 |
|  | Cuneus |  | \ | 14 | -77 | 26 | 5.7062 |
|  | Parietal\_Sup | R | 15 | 24 | -57 | 57 | 5.6046 |
|  | Postcentral | L | 405 | -57 | -12 | 48 | 6.199 |
|  | Precentral | L | \ | -34 | -17 | 52 | 5.5984 |
|  | Precentral | R | 528 | 51 | -15 | 48 | 6.5801 |
|  | Postcentral | R | \ | 49 | -17 | 49 | 6.1872 |
|  | Rolandic\_Oper | L | 27 | -45 | -15 | 18 | 5.8846 |
|  | Temporal\_Mid | R | 205 | 54 | -69 | 0 | 7.8896 |
|  | Temporal\_Inf | R | \ | 52 | -67 | -4 | 6.9514 |
|  | Temporal\_Sup | L | 67 | -51 | -12 | 3 | 5.498 |
| ROI6 | Insula | R | 21 | 36 | -9 | 9 | 5.5656 |
|  | Lingual | L | 24 | -18 | -54 | 0 | 5.3878 |
|  | Lingual | R | 97 | 21 | -54 | -3 | 6.3342 |
|  | Precuneus | R | \ | 20 | -56 | 8 | 5.8242 |
|  | Occipital\_Sup | L | 37 | -21 | -78 | 30 | 5.8843 |
|  | Cuneus | L | \ | -19 | -78 | 28 | 5.3575 |
|  | Occipital\_Sup | R | 46 | 24 | -72 | 27 | 5.8027 |
|  | Cuneus | R | \ | 15 | -80 | 31 | 5.101 |
|  | Parietal\_Inf | L | 19 | -24 | -51 | 54 | 5.2971 |
|  | Postcentral | L | 797 | -54 | -15 | 36 | 6.8612 |
|  | Precentral | L | \ | -55 | -8 | 34 | 6.0814 |
|  | Temporal\_Sup | L | \ | -65 | -25 | 4 | 5.9539 |
|  | Postcentral | R | 692 | 54 | -12 | 51 | 7.2909 |
|  | Precentral | R | \ | 51 | -8 | 51 | 6.5498 |
|  | Temporal\_Inf | R | 22 | 54 | -66 | -3 | 5.7927 |
|  | Temporal\_Sup | R | 121 | 63 | -9 | -3 | 5.8283 |

## Supplementary Table 4

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Table s4 | | | | | | | | | |
| FC difference between SZ and HC (Contrast: SZ < HC) | | | | | | | | | |
| **Seed** | **Region** | **Hemisphere** | **Cluster size** | | | **MNI coordinate** | | | **peak t** |
| **X (mm)** | **Y (mm)** | **Z (mm)** |
| ROI2 | Thalamus | L | | 38 | -6 | | -15 | 6 | 5.753 |
|  | Thalamus | R | | 57 | 9 | | -15 | 6 | 6.1305 |
|  | Vermis\_9 | \ | | 38 | 0 | | -57 | -30 | 5.5456 |
| ROI3 | Cerebelum\_Crus2 | L | | 49 | -9 | | -84 | -33 | 6.6004 |
|  | Cerebelum\_Crus2 | R | | 17 | 36 | | -81 | -51 | 5.3283 |
| ROI4 | Cerebelum\_8 | L | | 22 | -36 | | -57 | -45 | 5.9087 |
|  | Cerebelum\_Crus1 | R | | 17 | 33 | | -57 | -39 | 5.6897 |
|  | Cerebelum\_Crus2 | L | | 41 | -9 | | -84 | -33 | 6.0331 |
|  | Cingulum\_Ant | L | | 30 | 0 | | 30 | 30 | 5.433 |
|  | Inferior Semi-Lunar Lobule | \ | | 68 | 39 | | -75 | -57 | 5.4856 |
|  | Inferior Semi-Lunar Lobule | \ | | 40 | -30 | | -81 | -54 | 5.9511 |
|  | Thalamus | R | | 222 | 9 | | -12 | 9 | 6.8387 |
|  | Thalamus | L | | \ | 8 | | -9 | 7 | 6.4915 |
|  | Vermis\_6 | \ | | 33 | 0 | | -57 | -24 | 5.9458 |
| ROI5 | Cerebelum\_7b | R | | 161 | 36 | | -75 | -54 | 6.0747 |
|  | Cerebelum\_Crus2 | L | | 937 | -9 | | -87 | -36 | 7.5186 |
|  | Cerebelum\_Crus2 | R | | \ | 27 | | -79 | -46 | 6.9018 |
|  | Cerebelum\_Crus2 | L | | 242 | -33 | | -78 | -51 | 6.8645 |
|  | Cerebelum\_Crus2 | L | | 89 | -9 | | -81 | -30 | 6.4833 |
|  | Cerebelum\_Crus2 | R | | 45 | 12 | | -84 | -30 | 5.7483 |
|  | Thalamus | L | | 378 | -12 | | -12 | 9 | 7.4835 |
|  | Thalamus | R | | 303 | 2 | | -3 | 3 | 7.0914 |
|  | Vermis\_9 | \ | | 63 | 3 | | -57 | -33 | 6.1034 |
|  | Vermis\_9 | \ | | 161 | 3 | | -57 | -33 | 7.1096 |

# Supplementary figures

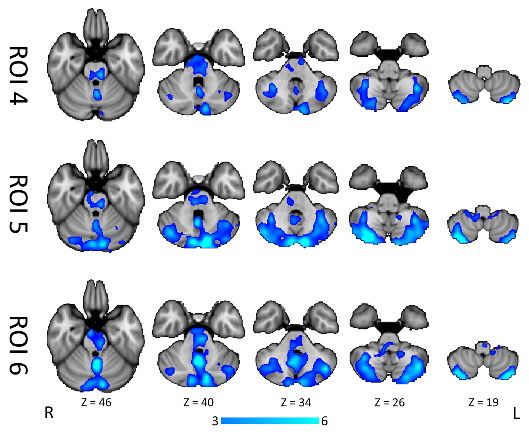
**Supplementary Figure 1** The head motion matching. These two groups were matched according to their head motion level. a. There’s no significant intergroup difference (p>0.05) in relative framewise displacement of resting-state fMRI. b. Also, there’s no significant intergroup difference (p>0.05) in relative head rotation of resting-state fMRI.



**Supplementary Figure 2** Reordered correlation matrix by affinity propagation clustering between spatial functional connectivity maps of each white matter connectivity-defined thalamic region.



**Supplementary Figure 3** The altered thalamic-cerebellar interaction in schizophrenia. These three pictures demonstrate that the functional connectivity of ROIs 4, 5 and 6 in the cerebellum were altered in schizophrenia compared with the healthy controls.



## Supplementary Figure 4 The decreased intra-thalamic integration in schizophrenia. These four pictures exhibit that the functional connectivity of ROIs 2, 4, 5 and 6 were altered in schizophrenia compared with the healthy control.

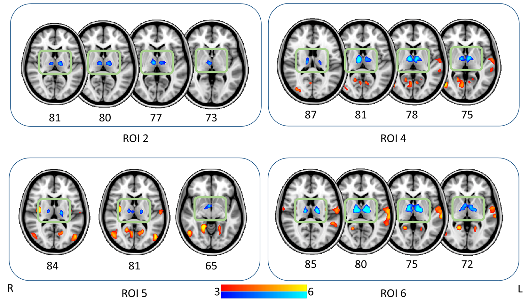


Figure s4.

## Supplementary Figure 5



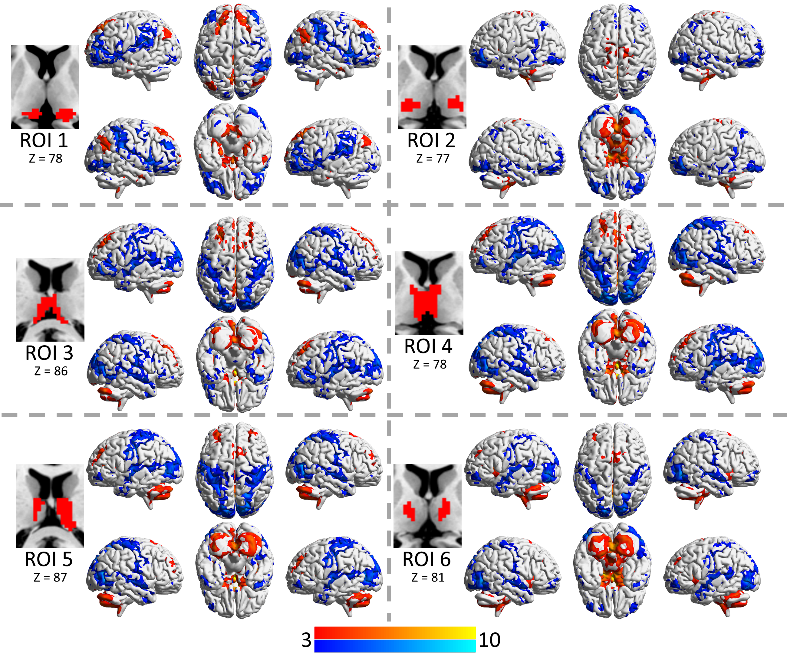
These correlation maps demonstrate that the thalamic functional integration of ROI 4 and ROI 5 negatively correlated with the duration of disease (q<0.05, FDR corrected).

## Supplementary Figure 6



These maps demonstrate the correlation between FC and chlorpromazine equivalent dose (CPE). Only FC of ROI 5 was found correlating with CPE. The upper row demonstrates that FC in right crus I and II of cerebellum of ROI 5 negatively correlates with CPE (p<0.005, uncorrected). The middle row and the lower row demonstrate that FC in the right lateral occipital cortex(LOC), the inferior division and the right primary somatosensory cortex (SM1) of ROI 5 positively correlate with CPE (p<0.005, uncorrected).

## Supplementary Figure 7



The results of parcellation and the group FC map of each ROI. According their spatial distribution, the six subdivisions were designated as the ventroposterior part (ROI 1), the ventral postmedian part (ROI 2), the dorsomedial part (ROI 3), the ventromedial part (ROI 4), the dorsolateral part (ROI 5) and the ventral anterior part (ROI 6) of thalamus. Positive interactions with the ventroposterior subdivision of thalamus were found in bilateral precuneus, hippocampus, parahippocampal gyrus, superior frontal gyrus, medial frontal gyrus, cingulate gyrus, caudate, putamen, pallidum, angular, cuneus and cerebellum. Negative interactions with the ventroposterior subdivision of thalamus were found in bilateral inferior temporal gyrus, middle temporal gyrus, fusiform gyrus, middle occipital gyrus, inferior occipital gyrus, lingual gyrus, superior frontal gyrus, middle frontal gyrus, inferior frontal gyrus, superior temporal gyrus, inferior parietal lobule, precentral gyrus, postcentral gyrus, insula, supplementary motor area and cerebellum. Positive interactions with the ventral postmedian part subdivision of thalamus were found in bilateral precentral gyrus, postcentral gyrus, cingulate gyrus, caudate, putamen, pallidum, hippocampus, parahippocampal gyrus and cerebellum. Negative interactions with the ventral postmedian part subdivision of thalamus were found in bilateral frontal pole, temporal pole, middle temporal gyrus, inferior temporal gyrus, lateral occipital cortex and occipital pole. The FC of the other four thalamic subdivisions were similar. Positive interactions with those FCs were found in bilateral frontal pole, superior frontal gyrus, cingulate gyrus, precuneus, parahippocampal gyrus, inferior temporal gyrus, basal ganglia network and cerebellum. Negative interactions with those FCs were found in bilateral dorsal attention network, SM1, auditory network, visual network and insula.

# Supplementary data 2

# Supplementary calculation

## FC difference of the whole thalamus

### Methods:

Functional connectivity (FC) of the whole thalamus was acquired by using bilateral thalamus as ROI. In detail, first, the preprocessing pipeline of functional MRI (fMRI) was consistent with the description of the main body, including slice timing, realign, normalization, artifacts removing, regressed out signal of white matter/cerebrospinal fluid/non-brain/whole brain/head movement, and temporally filtering. The time series of thalamus was extracted from the preprocessed fMRI data based on the thalamic mask in the Harvard-Oxford sub-cortical atlas. Second, the FC was defined as the Fisher’s Z value of Pearson’s correlation coefficient between mean time series of thalamus and time series of all other voxels in brain. Then the FC map of each subjects was spatially smoothed by a 6-mm FWHM Gaussian kernel. Third, the intergroup difference in FC was calculated by using two-sample t-test. Specially, the results of two-sample t-test were restricted in the voxels whose absolute one-sample t value were greater than 2 in both groups. This restriction reduced the false positive ratio of the intergroup results. At last, the intergroup differences were demonstrated at a significance level of p <0.001 uncorrected.

### Results:

The intergroup difference in FC of the whole thalamus was showed in Figure X1. Similar to the results described in the main body, the thalamic FC with the anterior cingulate cortex, with sensory system, with primary somatomotor cortex (SM1), and with cerebellum were found impacted in patients with schizophrenia.

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Figure X1. The intergroup difference in FC of the whole thalamus. The different voxels shown in the figure were significant p <0.001 uncorrected. The color of red-yellow indicates the thalamic FC (absolute Z value) in patients with schizophrenia is higher than that of healthy controls. The blue-lightblue indicates the thalamic FC (absolute Z value) in patients with schizophrenia is lower than that of healthy controls.

### Explanation:

The results were in line with the results from the segmented thalamus, indicating that the differences in FC obtained from the segmented thalamus were relatively reliable. But the results from segmentation could provide further information about the pattern of changes in FC of different subdivisions of the thalamus.

## Conjunction overlay

### Methods:

To avoid circular logic of this method and to verify the main results, we induced a conjunction-overlay-like method in the data. In detail, first, the intergroup differences in FC of 55 weighted thalamic origins were acquired in the preprocessed fMRI data by spatially regressing. Then, as same as above-mentioned, the intergroup difference in FC was calculated by using one-sample restricted two-sample t-test. The significant different masks in FC of each thalamic origins were extracted by using the significant p <0.001 uncorrected. Each thalamic source had two masks, one indicated that FC of patients was higher than that of, and the patient FC value is lower than controls, another indicated the lower part. Subsequently, based on the results of affinity propagation clustering, the two kinds of masks of thalamic origins belonging to the same cluster were overlaid, respectively. After all, the overlays of the difference of thalamic origins could represent the intergroup difference in FC of thalamic set.

### Results:

The difference overlays of the thalamic sets were showed in Figure X2-4. It’s found that the results were highly consistent with the results reported in the main body, which demonstrated that the altered thalamic FC with SM1, the occipital regions and the temporal regions were located in ROI 4 (figure X2), ROI 5 (figure X3) and ROI 6 (figure X4) of the thalamus in patients with schizophrenia.

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Figure X2. The overlaid map of difference in FC of thalamic origins belonged to ROI 4. The lower threshold was set to half the number of origins that makes up this set. The color of red-yellow indicates the number of origins which showed significant higher FC in patients with schizophrenia comparing with that of healthy controls. The blue-lightblue indicates the number of origins showed significant lower FC in patients with schizophrenia.

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Figure X3. The overlaid map of difference in FC of thalamic origins belonged to ROI 5. The lower threshold was set to half the number of origins that makes up this set. The color of red-yellow indicates the number of origins which showed significant higher FC in patients with schizophrenia comparing with that of healthy controls. The blue-lightblue indicates the number of origins showed significant lower FC in patients with schizophrenia.

C:\Users\Administrator\AppData\Local\Microsoft\Windows\INetCache\Content.Word\FigureX4_FC_set_5.tif

Figure X4. The overlaid map of difference in FC of thalamic origins belonged to ROI 6. The lower threshold was set to half the number of origins that makes up this set. The color of red-yellow indicates the number of origins which showed significant higher FC in patients with schizophrenia comparing with that of healthy controls. The blue-lightblue indicates the number of origins showed significant lower FC in patients with schizophrenia.

### Explanation:

The results from conjunction overlay were in line with the results from FC of thalamic sets. However, it must be noted that, when a set contains less thalamic origins, the results of the conjunction overlay will be not stable (such as set 5). This is also the reason why the results of conjunction overlay have not been in the main body.

## The signal stability in each set

### Methods:

To ensure that all sets contain enough voxels to overcome partial volume effects, the signal in and between sets were investigated. For each set, randomly selected 70% voxel in it. Iterated 1000 times, and calculated the average time series of the chosen voxels for each iteration, then calculate the correlation between these average time series and the original time series of this set. The mean value and standard deviation of these Pearson’s correlation coefficients were used to measure the consistency of the signals within the ROI. Besides, the Pearson’s correlation coefficients were also used to measure the temporal correlations between the six sets.

### Results:

As showed in figure X5 (A), the mean temporal correlation coefficients between iterations and original set were around 99 percent. The figure X5 (B) demonstrated the time series of each thalamic set. The figure X6 showed the Pearson’s correlation coefficients between the six sets.



Figure X5. A. The grey scatter in boxplot demonstrate the correlation coefficients between time series of each iteration and time series of the original set. The boxes and the yellow error bar demonstrate the mean values and the standard deviations of correlation coefficients respectively. B. The time series of each thalamic set.

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Figure X6. The matrix of Pearson’s correlation coefficients between the time series of six sets.

### Explanation:

According to the figure X5(A), the mean temporal correlation coefficients between iterations and original set were high. This implied that the time series of voxels inside a thalamic set were stable. As showed in figure X5(B) figure X6, the Pearson’s correlation coefficients between the time series of six sets were relatively different. This may indicate that different thalamic sets could be involved in different information processing processes.

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