Specialization and integration of functional thalamocortical connectivity in the human infant


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Connections between the thalamus and cortex develop rapidly before birth, and aberrant cerebral maturation during this period may underlie a number of neurodevelopmental disorders. To define functional thalamocortical connectivity at the normal time of birth, we used functional MRI (fMRI) to measure blood oxygen level-dependent (BOLD) signals in 66 infants, 47 of whom were at high risk of neurocognitive impairment because of birth before 33 wk of gestation and 19 of whom were term infants. We segmented the thalamus based on correlation with functionally defined cortical components using independent component analysis (ICA) and seed-based correlations. After parcellating the cortex using ICA and segmenting the thalamus based on dominant connections with cortical parcellations, we observed a near-facsimile of the adult functional parcellation. Additional analysis revealed that BOLD signal in heteromodal association cortex typically had more widespread and overlapping thalamic representations than primary sensory cortex. Notably, more extreme prematurity was associated with increased functional connectivity between thalamus and lateral primary sensory cortex but reduced connectivity between thalamus and cortex in the prefrontal, insular and anterior cingulate regions. This work suggests that, in early infancy, functional integration through thalamocortical connections depends on significant functional overlap in the topographic organization of the thalamus and that the experience of premature extrauterine life modulates network development, altering the maturation of networks thought to support salience, executive, integrative, and cognitive functions.

Significance

We investigated the way in which the human thalamus and cortex are functionally connected at the time of normal birth. We found the functional parcellation of the thalamus to be a good facsimile of that found in adult studies. However, although primary cortical regions were almost entirely connected to specific thalamic regions, heteromodal cortex was more widely connected to multiple thalamic regions, giving the potential for an integrative role for these circuits. Development seemed to have been modulated by the experience of premature extrauterine life, with an increase in connectivity to primary sensory cortex, but reduced connectivity between areas of the thalamus and heteromodal cortex known to support higher cognitive functions.


The authors declare no conflict of interest.

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the experience of preterm delivery and premature extrauterine life affect the development of thalamocortical connectivity, and is the effect more marked in rapidly developing heteromodal cortex than in more mature primary cortex?

**Results**

The spatial distribution of the whole-brain BOLD signal in the group, assessed in all subjects at the time of normal birth (gestational age of 38–42 wk) using independent component analysis (ICA), was similar to that described previously (20, 21), including a combined sensory motor component, auditory, visual, and subcortical components, and a fragmented default mode network. It did not differ according to gestational age of the infant at birth (Fig. S1). This finding is in accord with previous data (12, 21–23).

**Functional Parcellation of the Thalamus at the Time of Normal Birth Presents a Near-Facsimile of Known Organization in the Adult.** Hard-thresholding the functional connectivity estimates of nine functionally defined cortical areas selected from the group ICA on neuroanatomical grounds (Table 1) revealed a predominantly symmetrical topographical representation of these cortical regions in the thalamus. Fig. 1 shows this topographical organization of the thalamus defined by the cortical component with which each voxel was most highly correlated (Movies S1–S9).

The primary sensory motor component had the largest territory of thalamic dominance and provided the dominant cortical connectivity for the lateral portion of the thalamus with some posterior extension and an extension toward the medial thalamus. The anterior lateral portion of the thalamus was most highly correlated with sensory motor association areas encompassing the premotor, supplementary motor, and posterior parietal areas and the frontal operculum. The anterior medial thalamus was most strongly connected with the anterior cingulate. The inferior thalamus at this location was dominantly connected with the prefrontal component. Inferiorly, the central portion of the thalamus was dominantly correlated with the frontoparietal insular cortex. The medial thalami were dominated by connections to prefrontal cortex. The posterior medial extent of the thalamus was most correlated with the temporal

<table>
<thead>
<tr>
<th>Functional cortical component</th>
<th>Description</th>
<th>No. in Fig. S1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary sensory motor</td>
<td>Pre- and postcentral gyrus (pale blue)</td>
<td>1</td>
</tr>
<tr>
<td>Primary auditory</td>
<td>Superior temporal gyrus (red)</td>
<td>2</td>
</tr>
<tr>
<td>Sensory motor association</td>
<td>Superior parietal, superior frontal gyrus (yellow)</td>
<td>3</td>
</tr>
<tr>
<td>Primary visual</td>
<td>Pericalcarine (lilac)</td>
<td>4</td>
</tr>
<tr>
<td>Temporal</td>
<td>Fusiform gyrus (posterior part), medial and inferior temporal gyrus (dark blue)</td>
<td>5</td>
</tr>
<tr>
<td>Prefrontal</td>
<td>Dorsal prefrontal cortex (orange)</td>
<td>6</td>
</tr>
<tr>
<td>Lateral parietal</td>
<td>Postcentral gyrus [lateral portion; pale pink (not represented in Fig. 1)]</td>
<td>7</td>
</tr>
<tr>
<td>Frontoparietal insula</td>
<td>Insula cortex (left) and bilateral lateral frontoparietal junction (dark pink)</td>
<td>8</td>
</tr>
<tr>
<td>Anterior cingulate</td>
<td>Anterior cingulate cortex (green)</td>
<td>9</td>
</tr>
</tbody>
</table>

*Fig. 1. Dominant thalamocortical correlations from nine functionally defined cortical regions using maximum partial correlation coefficient. (A) Midthalamic view. (B) Posterior view. (C) Inferior view. Lateral parietal component does not have any dominant territory. Table 1 has anatomical descriptions and colors. Major cortical projection areas based on ref. 14. D shows cortical targets from specific thalamic nuclei (14). Images are displayed as per radiological convention. VA, ventral anterior; VL, ventral lateral; VPL, ventral posterior lateral.*
cortical component composed of the fusiform gyrus (posterior part) and the medial and inferior temporal gyri. The infero-posterior aspect of the thalamus was most strongly correlated with the anterior cingulate component. The posterolateral thalamus was most strongly correlated with the primary auditory cortex bilaterally and with the primary visual component on the right side only. The lateral parietal component, including the postcentral gyrus, was not found to have an area of dominant connectivity with the thalamus.

**Specialized and Integrated Thalamocortical Connectivity at the Normal Time of Birth.** Using maps of dominant connectivity to characterize thalamic organization underestimates the complexity of thalamocortical connectivity by discarding information about shared or integrated thalamic targets. Consideration of the full distribution of pairwise correlations reveals that, by the time of normal birth, the large-scale neuronal dynamics of the cortex share substantial parts of the anatomical infrastructure of the thalamus (24). Fig. 2 shows each cortical component, and its territory of significant thalamic correlations thresholded at a significance of $P < 0.05$. Visual inspection of these maps shows that thalamocortical connectivity naturally divides into components with widespread connectivity throughout the thalamus (primary sensory motor, temporal, medial prefrontal cortex, anterior cingulate, and frontoparietal insular cortex) and components with connectivity that was mainly limited to their areas of dominance (primary auditory, primary visual, and lateral parietal) (Fig. 2). Thalamic connectivity with the visual cortex is included in Fig. 2 but only reaches a significance of $P < 0.06$, possibly because of its location on the edge of the thalamus. Fig. 3 is a summary of Fig. 2 and shows, for every thalamic voxel, the number of components with significantly correlated BOLD activation. The central portion of the thalamus has significant connectivity with only two cortical components (sensory motor and frontoparietal insular) joined by a third (anterior cingulate component) in the posteroinferior portion. The medial territories of both thalami are significantly connected with six of nine studied cortical components, with no significant correlation with primary visual, primary auditory, or lateral parietal components.

**Thalamocortical Connectivity Is Affected by Premature Birth.** Of nine cortical areas examined, four showed a difference in correlation with the thalamus according to the degree of prematurity experienced by the subject. A decrease in magnitude of thalamocortical connectivity ($P < 0.05$) was found in premature infants in three of nine components investigated (Fig. 4, i–iii). This decreased connectivity was found between the frontoparietal insular component and a widespread area of the thalamus; reduced connectivity with prematurity was also found between thalamus and the anterior cingulate and prefrontal components ($P < 0.05$) (Fig. 4). The difference in connectivity between the anterior cingulate component and the thalamus was observed within the area of its dominant thalamic connectivity, whereas the difference between the medial prefrontal component and the thalamus was detected on the right side only and outside its dominant territory. Thalamocortical connectivity involving the frontoparietal insular cortex, however, was affected by prematurity both in the territory of its dominant connectivity and also outside it in areas dominantly connected with sensory motor and anterior cingulate components. The only cortical area where connectivity increased with prematurity was the lateral parietal component. This small area of the thalamus with increased connectivity is identified from the segmentation in Fig. 1 as being most connected with the sensory motor cortex. There was no relationship between gestational age at birth and motion ($r = 0.055$, df = 64, $P > 0.5$) or age at scan and motion ($r = 0.064$, df = 64, $P > 0.5$).

**Discussion**

This study confirms that, at the time of normal birth, the infant brain has robust, predominantly symmetrical network architecture. We show for the first time, to our knowledge, that, already by the time of normal birth, the topographical organization of functional thalamocortical connectivity defined by strongest connectivity is consistent with current information on the adult brain using tracer methods (25–30), diffusion studies (15, 16), and functional imaging in adult subjects (18). These results add to the growing evidence of the maturity of the human brain by the time of normal birth (12, 31, 32).

Although the topography shown in Fig. 1 is commensurate with adult thalamic topography derived using other imaging modalities, there are subtle differences with the adult functional parcellations produced by Zhang et al. (18) and the tractography
Thalamocortical components significantly altered by preterm birth. Reduced gestational age at birth was associated with reduced connectivity between thalamus and (i) frontoparietal insular, (ii) anterior cingulate, and (iii) prefrontal cortex and (iv) increased connectivity with lateral parietal cortex. Significant changes in thalamocortical connectivity were observed in regions of heteromodal cortex, where it may mediate experience-dependent plasticity (48). Cortical areas receiving inputs from first-order thalamic relays also mature earlier with regards to myelin formation and cortical thickness (49), whereas thalamocortical units found in this study to be affected by prematurity involve cortex which takes the longest to reach peak cortical thickness (50). In older children and young adults, Fair et al. (19) found that the development in motor and sensory thalamocortical interactions showed limited change over time compared with other thalamic regions. This finding suggests that primary thalamocortical interactions are already well-established by early childhood.

Because the BOLD imaging used in this study is very motion-sensitive (51), only datasets displaying very minimal motion were included (33). Although the neonatal thalamus does not have sufficient connectivity with thalamocortical connectivity have used adult-defined cortical regions of interest and analyzed thalamic correlations to these areas in neonates and children rather than using areas of cortical activity that are coherent at the time of normal birth as in this study. This methodological difference, perhaps, accounts for the difference in results found (33).

A more detailed analysis of thalamic connectivity revealed overlapping connectivity profiles that share links with multiple cortical territories. The thalamus was differentiated between regions with connectivity to multiple cortical components and regions with connectivity to only one or two areas. The thalamic areas connected to multiple cortical regions were not connected to primary visual, primary auditory, or lateral parietal components. These different connectivity profiles seem to reflect the functional role of the respective thalamocortical units. With the exception of primary sensory motor connectivity, thalamocortical units involving primary cortex seemed more restricted than those with connections to heteromodal cortex. Given the age of the subjects, this result may represent a developmental stage, or may be a feature of mature thalamocortical connectivity. It is notable, however, that analysis of adult data shows that human association cortices participate in multiple networks and provide functionally specialized and flexible regions, whereas somatomotor and early visual cortices participate in single networks (35, 36). Cortical regions with reduced thalamic connections in preterm infants were areas receiving higher-order thalamic input: circuits involving corticothalamic cortical information. These cortical regions are less mature but develop rapidly during the preterm period (37), suggesting their increased vulnerability to premature extrauterine life and the importance of this developmental window for the establishment of thalamocortical connections.

The regions of frontoparietal, prefrontal, and anterior cingulate cortex affected form the basis of the salience network described using task-based functional imaging in adults (38), which may have relevance to the high incidence of difficulties experienced by children born preterm (39), especially with regards to inattentive attention deficit (40, 41), anxiety, and autistic spectrum disorders (42, 43) that persist into adulthood (44). Areas where we do not see a difference with prematurity are thalamic areas highly correlated with primary cortex described as first-order thalamic relays, because they are reported to be innervated exclusively and homogeneously by subcortical drivers, receiving large excitatory inputs from no secondary sources (45).

We also found increased connectivity with prematurity between the thalamus and a single cortical component, the lateral parietal component, which in adults is involved in processing signals from face, lips, jaw, tongue, and throat (46). This finding raises the hypothesis that premature exposure to activities, such as breastfeeding and bottle feeding, may serve to increase functional connectivity to regions of cortex with more mature microstructure.

This anatomical distribution of areas affected by prematurity may reflect known developmental programming: microstructural maturation of primary sensory cortex seems to occur earlier than heteromodal cortex (37), and growth association protein, which is present in growing axons but lost when stable connections are made (47), is absent from axonal connections to first-order relays (44) but persists in regions of heteromodal cortex, where it may mediate experience-dependent plasticity (48). Cortical areas receiving inputs from first-order thalamic relays also mature earlier than with regards to myelin formation and cortical thickness (49), whereas thalamocortical units found in this study to be affected by prematurity involve cortex which takes the longest to reach peak cortical thickness (50). In older children and young adults, Fair et al. (19) found that the development in motor and sensory thalamocortical interactions showed limited change over time compared with other thalamic regions. This finding suggests that primary thalamocortical interactions are already well-established by early childhood.

Because the BOLD imaging used in this study is very motion-sensitive (51), only datasets displaying very minimal motion were
chosen for inclusion in this study. This choice reflects the perceived need for lack of motion to accurately study the synchrony of BOLD signals between cortical areas and a structure as small as the neonatal thalamus. In addition, the premature infants studied were without brain lesions to exclude those infants in whom severe motor impairment could already be predicted from structural MRI scans. We find effects of prematurity on thalamocortical connectivity in our analysis, while acknowledging that our study group has been selected in this way.

Materials and Methods

Subjects. The study was reviewed and approved by the National Research Ethics Service, and all infants were studied with written consent from their parents. All 66 infants were scanned once at the estimated time of completed gestation (defined as 38–42 wk from the last menstrual period); 47 infants had been born prematurely (mean = 30 wk, range 24–32 wk), and 19 infants had been born at full term (mean = 40 wk, range = 36–42 wk). Additional details are in Table S1. All MRI studies were supervised by an experienced pediatrician, and pulse oximetry, temperature, and electrocardiography data were monitored throughout; ear protection of silicone-based putty placed in the external ear (President Putty, Coltele; Whaledent) and Mini-muffs (Natus Medical Inc.) was used. Parental consent was obtained, and chloral hydrate sedation (25–50 mg/kg) was administered in all but one term-born infant.

High-resolution anatomical scans (T1- and T2-weighted MRI scans) were reviewed by an expert in perinatal MRI: none had major focal destructive parenchymal lesions, nine of the infants born prematurely had small punctate lesions, which are common in preterm infants and of uncertain significance (52), and one infant had a single small white matter cyst.

Imaging Methods. All images were acquired on a 3-T Philips Achieva MRI Scanner. Whole-brain functional imaging was performed using T2* gradient echo planar image acquisition (sequence parameters: repetition time = 1.5 s; echo time = 45 ms; flip angle = 90°; 256 volumes; slice thickness = 3.25 mm; in-plane resolution = 2.5 mm²; 22 slices; scan duration = 6.4 min) with an eight-channel phased array head coil. T2-weighted fast-spin echo MRI was acquired using TR = 8,670 ms, TE = 160 ms, flip angle = 90°, slice thickness = 2 mm, field of view = 220 mm, matrix = 256 × 256 (voxel size = 0.86 × 0.86 × 1 mm).

Data Selection. Acknowledging the sensitivity of functional data to motion (reviews are in refs. S1 and S3) and with the aim of investigating a small structure such as the thalamus, only datasets with very low motion were eligible for inclusion. Within the premature cohort, 150 datasets were examined. Of the 47 met the criteria for inclusion. In this study, acceleration/curvature head motion correction with FSL MCMFLIRT (54) (and exclusion of the first six volumes in every subject), only scans with 200 contiguous volumes with motion of ≤0.08 mm relative mean displacement were included. ICA was then performed on individual datasets using FSL MELODIC (55), because an ICA-based identification of artifacts, including head motion, has been shown to be a very sensitive approach (56, 57). In addition to 47 included subjects, 5 additional subjects who met the motion criteria (mean = 0.08 mm relative mean displacement) were excluded at this point, because it was found in these subjects that there was still significant motion as defined by either component with spectra in the high-frequency range and/or spatial representations around the edge of the brain (58). Nineteen term subjects were selected using the same motion criteria, providing a dataset with minimal observed head motion. There was no difference in motion between the infants born preterm and those born at the normal time. Motion parameters based on the relative mean displacement were 47 infants born preterm mean = 0.052 mm (range = 0.03–0.08 mm) and 19 infants born at the normal time of birth mean = 0.050 mm (range = 0.03–0.08 mm).

Data Analysis. Functional. After removal of nonbrain structures from the T2-weighted structural image using a neonatal tissue segmentation algorithm (59), fMRI preprocessing was carried out using tools from the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain Software Library (60). Prestatistical processing consisted of removal of the first six functional volumes, correction for head motion (54), spatial smoothing by using a Gaussian kernel of FWHM of 5 mm, and high-pass temporal filtering (200 s). Functional volumes were registered to the subject’s T2-weighted structural image (54), with boundary-based registration (61) optimized for neonatal tissue contrasts. It is not possible to reliably identify single voxels of cerebrospinal fluid from the echo planar image of an infant at the time of normal birth, which is done in adult studies to model time course regressors, because ventricles are too small to avoid partial volume effects. Therefore, cerebrospinal fluid was identified in the subject’s T2-weighted structural image, and data from voxels corresponding to these areas were discarded. The remaining data were then transformed to a population-based neonatal template (62) using nonlinear registration (60).

To allow for plasticity and acknowledging that cortical areas cannot be defined from task-based paradigms at this age, cortical regions of interest were defined using components defined by ICA. Preprocessed functional data contained 200 time points per subject, and motion correction was applied on a subject-by-subject basis to each subject to produce a single 4D dataset, and resting-state components common to the group were defined using MELODIC (55) with a fixed dimensionality of 25, which achieved a good balance between interpretability and robustness, similar to that reported in adults (63). ICA maps were thresholded using an alternative hypothesis test based on fitting a Gaussian/γ-mixture model to the distribution of voxel intensities within spatial maps and controlling the local false discovery rate at P < 0.05 (55). The resulting maps and full ICA decomposition are shown in Fig. S1.

Cortical component selection. Nonoverlapping cortical component masks were created by assigning each cortical voxel to a specific resting-state component depending on which network had the highest z score at that voxel. For analysis of thalamocortical connectivity, we selected nine bilateral cortical areas based on prior anatomical knowledge and previous work in adults (63), term infants (12), and animals (Table 1). From 25 ICA components, we discarded subcortical components (thalamus, cerebellum, brainstem, and basal ganglia), components where the power spectra (in the frequency domain) in individuals was in the high-frequency range in more than 10% of subjects, and unilateral components. The exception was the primary visual component: the anatomical position of the primary visual cortex leaves it vulnerable to noise caused by dense vasculature and motion, and this component was included, despite being characterized by high-frequency power spectra in 7 of 66 subjects (10.6%). Of the remaining components, spatial correlation with adult networks reported in the work by Smith et al. (63) was tested using cross-correlation (Table S2). Visual and auditory components were represented more than one time: in this case, we selected the network best corresponding with the anatomical position of primary visual and auditory cortex.

Within a group-defined cortical functional area, there is likely to be some heterogeneity at the subject level. For each individual subject, each component identified at the group level was mapped back to each subject’s dataset through a spatial regression of the group ICA maps on the individual fMRI dataset followed by a regression of the resulting time series on the same dataset (64). To ensure that the first Eigen time series at the subject-specific level best represented the function determined by the group analysis rather than another functional area within the same group-defined cortical region, for each subject, the components were thresholded at z > 1.96, and the remaining voxels inside the group-defined mask were used as the cortical target from which the first Eigen time series was taken. The resulting component maps in individual subjects derived using this dual-regression approach were used as regions of interest, and partial correlation scores were calculated with the series of each thalamic voxel (65).

Group analysis. Correlation scores for each component were combined using fixed effects analysis, and the results were used to parcellate the thalamus (Fig. 1 and Movies S1–S9), assigning thalamic voxel membership according to the cortical component with which it had the highest average correlation score in the group (18). In addition to this fixed effects model to show dominant connectivity, significance (P < 0.05 corrected for multiple comparisons after threshold-free cluster enhancement) was assessed using nonparametric permutation testing (66), and results are shown in Fig. 2. To illustrate which regions of the thalamus are significantly connected with more than one cortical area, the resulting nine significant thalamic maps (Fig. 2) were binarized and summed (Fig. 3). Finally, using a general linear model of gestational age at birth, the thalamocortical correlation maps shown in Fig. 2 were tested voxelwise for statistically significant associations, with gestational age at birth using nonparametric permutation testing (66). The results were spatial maps characterizing the effect of prematurity on thalamocortical connectivity (Fig. 4A, Upper) and scatterplots showing the correlations coefficients in each infant according to gestational age at birth (Fig. 4B, Lower).

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Fig. S1. Temporal concatenation ICA-estimated resting pattern in the group of 66 subjects. Sagittal, coronal, and axial views of the spatial map for each component. Images are z statistics overlaid on the template 41-week brain. Red to yellow indicates z values ranging from 2 to 12. The right hemisphere of the brain corresponds to the left sides of the coronal and axial images. Components 1–9 correspond to functional cortical components described in Table 1.

Table S1. Demographic information—gestational age at birth, scan, and sex

<table>
<thead>
<tr>
<th></th>
<th>Premature cohort</th>
<th>Term cohort</th>
<th>66 Infants</th>
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<tr>
<td>Mean gestational age</td>
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<tr>
<td>(in weeks), at birth</td>
<td>29.86 (24.71–32.86)</td>
<td>39.91 (36.29–41.86)</td>
<td>32.76 (24.71–41.86)</td>
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<tr>
<td>(in weeks), at scan</td>
<td>42.38 (40–44.56)</td>
<td>43.47 (40.29–48.28)</td>
<td>42.71 (40–48.28)</td>
</tr>
<tr>
<td>Sex, % male</td>
<td>43</td>
<td>22</td>
<td>40</td>
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Table S2. Cross-correlation analysis between cortical components derived from group ICA of infant data at the time of normal birth and resting-state networks in adults

<table>
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<tr>
<th>Infant component</th>
<th>Adult resting state network</th>
<th>Correlation</th>
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<tbody>
<tr>
<td>Primary sensory motor</td>
<td>5</td>
<td>0.43</td>
</tr>
<tr>
<td>Primary auditory</td>
<td>6</td>
<td>0.38</td>
</tr>
<tr>
<td>Sensory motor association</td>
<td>9</td>
<td>0.34 (RSN5 = 0.3)</td>
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<tr>
<td>Primary visual (occipital pole)</td>
<td>1</td>
<td>0.56</td>
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<tr>
<td>Temporal</td>
<td>12</td>
<td>0.12</td>
</tr>
<tr>
<td>Prefrontal</td>
<td>15</td>
<td>0.30 (RSN18 = 0.25)</td>
</tr>
<tr>
<td>Lateral parietal</td>
<td>12</td>
<td>0.28 (RSN8 = 0.24; RSN11 = 0.22)</td>
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<tr>
<td>Frontoparietal insular</td>
<td>7</td>
<td>0.45</td>
</tr>
<tr>
<td>Anterior cingulate</td>
<td>9</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Data derived from ref. 1.

Movie S1. Dominant thalamocortical correlations from nine functionally derived cortical regions using maximum partial correlation coefficient. Cortical components derived from group independent component analysis (as per Fig. S1) are shown with areas of dominant thalamic correlation. Primary sensory motor component.

Movie S1

Movie S2. Primary auditory component.

Movie S2
Movie S3. Sensory motor association component.

Movie S4. Primary visual component.
Movie S5. Temporal component.

Movie S6. Prefrontal component.
Movie S7. Lateral parietal component.

Movie S8. Frontoparietal insular component (NB rendered in pink in Figs. 1 and 2).