DISTANT HETEROTOPIC CALLOSAL CONNECTIONS TO PREMOTOR CORTEX IN NON-HUMAN PRIMATES

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Abstract—Cortico-cortical connectivity has become a major focus of neuroscience in the last decade but most of the connectivity studies focused on intrahemispheric circuits. Little has been reported about information acquired and processed in the premotor cortex and its functional connection with its homotopic counterpart in the opposite hemisphere via the corpus callosum. In non-human primates (macaques) lateralization is not well documented and its exact role is still unknown. The present study confirms in two macaques the existence of homotopic contralateral projections and completes the picture by further exploring heterotopic connections with all the homo- and heterotopic cortical areas located in the contralateral hemisphere. The results showed that PMd and PMv receive multiple low-density labeled inputs from the opposite heterotopic prefrontal, parietal, motor, insular and temporal regions. Such unexpected collection of transcallosal inputs from heterotopic areas suggests that the premotor areas communicate with other modalities through long distance low-density networks which could have important implications in the understanding of sensorimotor and multimodal integration.

Key words: neuroanatomy, cortical connectivity, corpus callosum, premotor cortex, non-human primate, multisensory integration.

INTRODUCTION

In placental mammals the corpus callosum is the main commissural structure (versus the anterior commissure) which connects homotopic and heterotopic regions of the cerebral cortex between the two hemispheres (e.g. Innocenti, 1986; Innocenti, 1994, 1995; Aboitiz et al., 2003). The identification of the topography of these connections is still in progress in humans especially through functional magnetic resonance imaging (Fabri et al., 2011; Phillips and Hopkins, 2012) and diffusion tensor imaging (Hofer and Frahm, 2006; Phillips and Hopkins, 2012). In non-human primates, the majority of brain connectivity data (see datasets established based on the work of Paxinos et al., 2000; Van Essen, 2002; Dubach and Bowden, 2009; Rohlfing et al., 2012; Markov et al., 2014; Calebresi et al., 2015) originate from one hemisphere based on the assumption (though unproven) that lateralization does not play a key role in macaques’. The few available studies (e.g. Pandya and Vignolo, 1971) state that callosal connections predominantly link homotopic cortical regions. This view has been questioned in the last decade (Clarke, 2003) and new evidence of numerous and widespread heterotopic callosal connections have emerged in human studies. For example, in the visual cortex, heterotopic connections to the opposite hemisphere have been identified (Clarke and Miklossy, 1990; Clarke, 1994). Visual connections have also been reported from the inferior temporal cortex (associated with visual recognition) to the contralateral temporoparietal junction referred to as Wernicke’s area in humans (associated with speech comprehension) and to the inferior frontal gyrus (Broca’s area associated with speech production) (Di Virgilio and Clarke, 1997). In the motor cortex, corticocortical connectivity between motor areas and the other hemisphere has been identified in non-human primates (e.g. Pandya and Vignolo, 1971; Jenny, 1979; Rouiller et al., 1994; Liu et al., 2002; Marconi et al., 2003). In 2005, Boussaoud et al. showed that the premotor cortical areas PMd-c (F2) and PMd-r (F7) receive heterotopic inputs from contralateral pre-SMA (F6) and that PMd-r was strongly connected with prefrontal cortex. According to those authors callosal afferent connectivity to PMv-c (F4) was broader than that to PMv-r (F5). Other authors (Marconi et al., 2003) reported that the major heterotopic callosal projection to F7 originated from F2 followed by weaker inputs from pre-SMA (F6), area 8 (FEF) and prefrontal cortex (area 46). The same authors showed that the heterotopic inputs to F2 mainly emanated from F7 followed by a smaller contingent coming from F5, F4, SMA-proper (F3) and F1. These reports suggest that premotor areas connectivity is composed of sets of heterotopic inputs originating from a mosaic of motor areas. Furthermore, in the premotor cortex, touch, vision and/or hearing inputs have been found (Weinrich and Wise, 1982; Weinrich et al., 1984; Graziano et al., 1997, 1999) contributing to sensorimotor transformation (Blanchard et al., 2013). Those heterotopic or multisen-
sory inputs would gain in being further studied since they provide a basis for underlying voluntary actions directed to a goal, more efficiently when more than one sensory modality is engaged (Stein and Meredith, 1993; Giard and Peronnet, 1999; Driver and Noesselt, 2008).

Most available tracing studies in monkeys (see above) on callosal motor connectivity were based on injections restricted to a specific cortical subarea (e.g. F2, F3, F4 or F5 in PM; F3 or F6 in SMA) or even to a limited body part (hand area in F1 and F3). In order to establish a more comprehensive callosal connectivity pattern, the present study is based on larger tracer injections covering a large part of the dorsal premotor cortex (PMd) and the ventral premotor cortex (PMv), respectively. We tested the hypothesis that both PMd and PMv receive significant direct heterotopic non-motor callosal inputs from the prefrontal, parietal and temporal lobes, which may amount up to 5% of the total callosal projections in each of these lobes.

**EXPERIMENTAL PROCEDURES**

Two non-human primates (Mk-CI *Macaca mulatta* and Mk-R9 *Macaca fascicularis*), 3 and 4 years old and weighing 3 and 4 kg, respectively, were re-used from a previous study on thalamocortical and corticothalamic projections (Cappe et al., 2007, 2009). The study was conducted according to both the guidelines of the National Institute of Health (Guideline for the Care and Use of Laboratory Animals, NIH Publication N°80-23, revised in 1996), those of the European Community (Guidelines for Animals Protection and Use for Experimentation, 86/609/EEC), and approved by local (Swiss) veterinary authorities (authorization N°156/04 and 156/02). All efforts were made to minimize the number of animals used and their suffering. The present work is based on the same injections of four neuroanatomical retrograde tracers (see Table 1) as described in a previous report (Cappe et al., 2009) but considered here for the callosal connectivity. Briefly, the animals were pre-medicated with ketamine (5 mg/kg, i.m.), Carprofen as an analgesic (Rymadil, 4 mg/kg, s.c.), antibiotics (Albipen, ampicillin 10%, 15–30 mg/kg diluted 1:1 in saline, i.m.), atropine sulfate (0.05 mg/kg, i.m.) and dexamethasone (Decadron, 0.02–0.3 mg/kg/day diluted 1:1 in saline, i.m.). Then, the monkeys were anesthetized with propofol (0.1–0.3 mg/kg/min, i.v.) and placed in a stereotaxic frame under aseptic conditions. The skull and the dura mater on the left side were opened over the premotor cortex. In the frontal lobe, PMd and PMv were localized based on the position of the central and arcuate sulci and the boundary between both areas was established based on the genu of the arcuate sulcus (Liu et al., 2002; Morel et al., 2005).

Injections of the tracers were executed by using 5- to 10-μl Hamilton syringes inserted perpendicularly to the cortical surface. Then, the dura mater, muscles and skin were sutured and the monkeys were treated for several days with an analgesic (Rymadil, 5 mg/kg, p.o.) and an antibiotic (Amoxicillin, 10 mg/kg, p.o.). Following a survival period of 2–3 weeks, the animals were deeply anesthetized, given a lethal dose of sodium pentobarbital (Vetanarcol 90 mg/kg i.p.) and were perfused transcardially with first 0.3 L saline (0.9%) then 3 L paraformaldehyde (4% in phosphate buffer 0.1 M, pH = 7.4), with a mixture (2 L) of paraformaldehyde 4% and sucrose 10% (in phosphate buffer) and finally with sucrose 20% and 30% (2 L in phosphate buffer).

The histological processing of the brain has also been described in detail in previous reports by Morel et al. (2005), Cappe et al. (2007) and Cappe et al. (2009). In summary, first the brain was sectioned in the frontal plane (40-µ sections) on a freezing microtome. The sections were collected in five series among which one was immediately mounted on slides and stored in the refrigerator for fluorescent microscopy analysis. The plotting of labeled neurons with fluorescent and/or non-fluorescent tracers was done using the MicroBrightField NeuroLucida System (Colchester, USA). Drawings of cortical contours in Nissl-stained sections were imported in NeuroLucida’s system in order to be overlapped with the analyzed sections to identify the cortical areas. Complete drawings including the plots of labeled cells were then exported to the software CorelDrawX6 (Version 16, 2012) and cell counting was performed.

The quantitative analysis was conducted by calculating for each tracer the percentage of cells labeled in one cortical area against the total number of cells labeled with this particular tracer in the whole hemisphere. Such percentage distribution as a function of a cortical area was represented first in histograms and grouped according to morphological location. Second, the strength of the callosal connections between the multipolar areas was represented in the form of a color weighted connectivity matrix as done by others (e.g. Markov et al., 2014). This matrix used a logarithmic scale which was then translated into a positive scale where values covered 3 equal ranges from 0 to 1.25 corresponding to sparse connections, from 1.25 to 2.5 corresponding to moderate connections and, greater than 2.5 corresponding to strong connections.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Injection site</th>
<th>Tracer</th>
<th>Volume (µ)</th>
<th>Number of sites injected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mk-R9</td>
<td>PMd</td>
<td>Fluoroemerald (FE)Molecular Probes, Eugene, OR</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>PMv</td>
<td>Fast Blue (FB)Fluka, Switzerland</td>
<td>3.5</td>
<td>7</td>
</tr>
<tr>
<td>Mk-CI</td>
<td>PMd</td>
<td>Diamidino Yellow (DY)Sigma Aldrich, France</td>
<td>5.1</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>PMv</td>
<td>Cholera toxin B subunit (CB)List Biological Laboratories, Campbell, CA</td>
<td>1.9</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 1. Summary of injection sites, tracers, volumes and number of sites injected in the two macaques Mk-R9 and Mk-CI. Representations of the injection sites are available in Figs. 1 and 2. Due to an error of transcriptions in Cappe et al. (2009), the tracer injected in PMv in Mk-CI is indeed CB and not WGA, as indicated by mistake in Cappe et al. (2009): in their legend of Fig. 1, “WGA” should be replaced by “CB”. The data are however not affected, as the Fig. 1 of Cappe et al. (2009) indeed describes the data for PMv, derived from CB injection.
RESULTS

Injection sites

In the present study, two out of the six cortical areas injected in Cappe et al. (2009) were reinvestigated for callosal connectivity: the dorsal and the ventral premotor cortices (PMd and PMv). Repeated injections of different retrograde tracers extended anteriorly from the genu of the arcuate sulcus (Figs. 1–3) to the caudal end of the spur of the arcuate sulcus. The injections sites were in F7/F2 (learning-related area [Brasted and Wise, 2004] modulated by eye movement [Boussaoud, 1985]/guiding reaching area [Cisek and Kalaska, 2005]) and in F4/F5 (sensory guidance of movement [Graziano et al., 1994] and peripersonal space [Fogassi et al., 1996]/hand shaping during grasping, vocalization [Coudé et al., 2011] and mirror neurons [Kohler et al., 2002]). Examples of the general distribution of retrogradely labeled neurons with FB, FE, DY and CB are presented in Fig. 3. The relative position of each coronal section (as well as their estimated stereotaxic level in mm from interaural axis) is indicated on a schematic brain map based on the monkey brain atlas of Saleem and Logothetis (2007).

Injections in PMd

Fig. 4, upper panel, shows the distribution of retrograde labeling in the hemisphere contralateral to the injection of tracers FE and DY in PMd. The distribution of callosal inputs to PMd with respect to their lobe of origin is indicated in Table 2. In Mk-R9 the most abundant retrograde labeling was found in motor areas (69.1%). Less dense labeling was observed in the temporal lobe (areas TE + TPO; 9.1%), the parietal lobe (3a/b, 1–2, SII; 9.7%), the prefrontal cortex (8.4%, in particular areas 44 and 45) and the insular cortex (2.9%). In Mk-CI the main labeling has been obtained in motor areas (94.3%) followed by the prefrontal cortex (5.7%, area 9). In terms of origin of the callosal cortical projections, callosal inputs to PMd originate mainly from motor cortical areas. To a lesser extent, moderate transcallosal projections came from the parietal and the parietal lobe in one animal (Mk-R9) and from the prefrontal cortex (Mk-R9 & Mk-CI). Sparse callosal projections were identified coming from the insula (Fig. 4).

Injections in PMv

Fig. 4, lower panel, illustrates the histogram distribution of retrograde labeled cells in the hemisphere contralateral to the injection of two tracers (FB and CB) in PMv of the two monkeys. The distribution of callosal inputs to PMv with respect to their lobe of origin is indicated in Table 3. In Mk-R9 these injections have labeled cells mainly in motor areas (78%): area 24, SMA-proper (F3), F2, F4 and F5. A moderate labeling was found in the prefrontal (9.8%), parietal (7.0%) and insular (5.1%) regions. No retrograde labeling was observed in the temporal lobe. In Mk-CI, the main (homotopic) labeling was found in motor areas (83.4%): F5, F4, frontal eye field area 8A, F3, F2 and in the cingulate cortex (area 24). Less dense labeling was noticed in the other lobes: prefrontal, parietal, insula and temporal lobes. Put into connectivity perspective callosal inputs to PMv mainly originated from contralateral premotor cortices. To a lesser extent, moderate to weak callosal inputs were identified from the FEF, the precentral operculum, somatosensory cortices (SII and 1–2), the area G (terminal plexus in gustatory cortex) and the insula. Finally, sparse callosal connections originated from a large palette of cortical areas (Fig. 4).

Connectivity matrices

In order to assess more quantitatively the respective cortical areas of origin, connectivity matrices were established. Fig. 5 shows the individual connectivity matrices for Mk-R9 and Mk-CI. This matrix has been obtained based on the calculated ratio of labeled neurons with one marker in a specific cortical area relative to the total number of labeled neurons with the same marker over the hemisphere opposite to the injection site. These values have been turned into logarithms then translated to a positive scale in order to quantify connection weights. In Fig. 5, each column gives the calculated connection weight for each animal per area (PMv and PMd) and each row the origin of its transcallosal inputs originating from 41 separate cortical areas. According to the colorbar used, bright colors represented strong connections whereas dark colors represented weaker connections. The results show strong callosal homotopic connections especially at F4, F5, F2 levels. Other strong links with PMv and PMd were observed with heterotopic areas F2, F3, F4, F5 and 24. Moderate non-motor heterotopic connections are also well present in both monkeys but show less homogeneity in relation with PMd. Therefore only areas 8A and 8Bs can be noted as moderately connected to PMd whereas PrCo, the insula, the area G, the parietal and the prefrontal lobes show moderate connections to PMv. As far as sparse heterotopic connections are concerned, the sources to PMv originate essentially from the parietal and the prefrontal lobes in both animals. In contrast, for PMd, only one animal (Mk-R9) displays sparse heterotopic connections with the parietal and the prefrontal lobes but also with the temporal lobe.

DISCUSSION

Our results are in agreement with the hypothesis that both PMd and PMv receive multiple heterotopic non-motor callosal inputs from the contralateral prefrontal, parietal and possibly the temporal lobes. Those projections are few in quantity compared with the homotopic ones but represent a non-negligible amount (up to 5–7%) and therefore may be functionally relevant.

Connectivity of PMd and PMv

Across the two animals of different species, it appears that the homotopic projections (Figs. 4 and 5) are relatively consistent, suggesting that injections are comparable. Those repeated injections were executed in two different cortical subregions (F2/F7 and F4/F5;
see Figs. 1 and 2) and revealed strong homotopic corticocortical connections with the opposite hemisphere as described earlier by others (Pandya and Vignolo, 1971; Rouiller et al., 1994; Marconi et al., 2003). In addition to these major homotopic inputs linking bilaterally the F3 areas, the cingulate motor areas (Rouiller et al., 1994), F7, F2, F4, F5 (Boussaoud et al., 2005), smaller contingent of callosal inputs to PMd or PMv originating from

Fig. 1. (A) Upper left: the photomicrograph shows the PMd region of the left hemisphere of Mk-R9 injected with Fluoroemerald (FE). Below that, sections S-15 to S-41 are examples of consecutive coronal slices through the PMd of the same animal displaying a reconstruction of the injected site. One can note that the injections covered an area from anteriorly to the genu of the arcuate sulcus (F7) till midway of the spur of the arcuate sulcus (F2). (B) The second photomicrograph from the same animal shows the PMv region of the left hemisphere injected with Fast Blue (FB). Examples of corresponding consecutive coronal sections through PMv are displayed as a reconstruction of the injected site. The injections started at the level of the caudal end of the principal sulcus and finished midway over the spur of the arcuate sulcus (therefore in F4 and F5). See list of abbreviations for the lettering.
contralateral heterotopic areas were described with PMd connected with area 46 of the prefrontal cortex, F6, F5, F4, F3, F2, F1 and area eight (Marconi et al., 2003). The present study reports the same patterns of homo and heterotopic callosal connections to PMd and PMv (see Figs. 4 and 5) and completes it with some other non-motor contingent of callosal projections to PMv (1–2, 3a/b, SII, 44, 45, area 12, 24, G, Insula, PrCo) identified in both animals and to PMd (area 45, 24). The present observations mean that globally PM receives more contralateral non-homotopic projections than expected (Marconi et al., 2003; Boussaoud et al., 2005). We can add to these results the identification of sparse heterotopic connections originating from the parietal cortex.
In the motor system, reports demonstrated that intrahemispheric connectivity is formed by a series of interconnected areas working hierarchically (Keele et al., 1990; Grafton and Hamilton, 2007) and/or in parallel (Rizzolatti et al., 1998; Rizzolatti and Luppino, 2001). For example the neurons projecting to F1 were shown to originate mainly from PMd, PMv, SMA according to a somatotopic organization (Godschalk et al., 1984; Ghosh et al., 1987) and from other networks like the somatosensory areas 3a, 1–2, SII, the posterior parietal cortex and the cingulate cortex (Morecraft et al., 2012). Similarly PMd and PMv were shown to be connected to many other cortical areas located in the same hemisphere in the frontal and parietal lobes and to a lesser extent in the temporal lobe (Matelli et al., 1984; Barbas and Pandya, 1987; Kurata, 1991; Morecraft et al., 2012). Interestingly these two premotor areas have already been reported to receive inputs from associative “sensory” areas like MIP, AIP, 7a and 7b (Matelli et al., 1986; Ghosh and Gattera, 1995; Wise et al., 1997; Tanne-Gariepy et al., 2002) known to have visual properties. Therefore these premotor areas were further investigated recently from a multisensory perspective (Lanz et al., 2013) in order to better understand their involvement in sensory-motor transformations. However, although these pathways and processes were generally found in one hemisphere because motor projections are mostly crossed when they reach the cortex (see Van der Knaap and Van der Ham, 2011 about the inhibitory theory through the corpus callosum to facilitate brain lateralization) it remains that during
bimanual tasks (e.g. opening a peanut) each hemisphere receives an afferent copy from the opposite hemisphere in order to confront inputs from both sides and perform accurate actions (Brinkman, 1984; Geffen et al., 1994; Andres et al., 1999; Wahl and Ziemann, 2008; Liuzzi et al., 2011). In this context, a large connection via the corpus callosum with the opposite cortex was described by Rouiller et al. (1994) between both SMAs (F3) which are consistent with the functional data (Kermadi et al., 1997, 1998) showing that these structures play a role in the control of bimanual coordinated movements (see Kermadi et al., 2000 for similar conclusions with the cingulate motor cortex, the posterior parietal cortex, F1 and PMd). The anatomical basis of such results have been confirmed in the present work (Figs. 4 and 5) but some other additional projections originating from contralateral heterotopic areas state that a broader range of information (most likely inhibitory and excitatory) is transmitted to both PMd and PMv suggesting that PM might be part of a large sensorimotor and multisensory network stretched over the opposite hemisphere. For instance, a recent investigation in humans (Rousseau et al., 2016) concluded that the execution of a vertical hand movement activates a network that includes the contralateral primary motor and somatosensory cortices, PM, SMA, the anterior cerebellum, the cingulate cortex, the prefrontal cortex, the temporal gyrus, the hippocampi (bilateral), and the insula. These anatomical findings help describe the cortical network connected through the corpus callosum which is in position to contribute to sensorimotor and multisensory integration across the two hemispheres.

Multisensory processes

The present data when brought together in a connectivity matrix (Fig. 5) demonstrate that there is a general trend among the two animals characterized by a predominance of homotopic callosal links innervating PMv and PMd. However moderate and sparse projections form a heterotopic network which can be used as a substrate for multisensory integration. Indeed, recent studies support this view and showed various sensory inputs to the two studied premotor areas. Examples include visuomotor behavior (Nelissen et al., 2011; Takahara et al., 2012; Limanowski and Blankenburg, 2016), somatosensory representation (Disbrow et al., 2003; Wardak et al., 2016) and auditory discrimination (Lemus et al., 2009). Premotor fields seem

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**Table 2. Summary of distribution of retrogradely labeled callosal inputs to PMd**

<table>
<thead>
<tr>
<th></th>
<th>Mk-R9%</th>
<th>Mk-CI%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parietal</td>
<td>9.7%</td>
<td>0%</td>
</tr>
<tr>
<td>Prefrontal</td>
<td>8.4%</td>
<td>5.7%</td>
</tr>
<tr>
<td>Motor</td>
<td>69.1%</td>
<td>94.3%</td>
</tr>
<tr>
<td>Insula</td>
<td>2.9%</td>
<td>0%</td>
</tr>
<tr>
<td>Temporal</td>
<td>9.1%</td>
<td>0%</td>
</tr>
</tbody>
</table>
therefore good candidates for passing information from one system to another and hence perform integrative processes. Moreover, we suggest that cortico-cortical callosal pathways could be added to the originally described multisensory integration mechanisms formed by intrahemispheric cortico-cortical loops (Ghazanfar and Schroeder, 2006) and thalamocortical loops (Cappe et al., 2009). However, if we want to further understand the functional intricacy significance of these cortico-cortical callosal connections it will be necessary to examine neural activity in situ and simultaneously on both sides of the brain.

Applied clinical relevance

The neurophysiological mechanisms of task-related modulation of neural networks have been studied by Merchant et al. (2014). A cognitive task with maximum interactions shows local field potential changes in the corresponding hemisphere as well as in the opposite hemisphere. A longer time lag was observed in the opposite hemisphere. Time lags for negative interactions were longer than for positive interactions in keeping with neuroanatomical measurements (Merchant et al., 2014).

In the context of recovery from a cortical lesion, following a focal lesion in F1, it has been reported in non-human primates that adjacent cortical territories (Nudo and Milliken, 1996; Friel and Nudo, 1998) as well as interconnected regions (e.g. premotor cortex, Frost et al., 2003; Dancase et al., 2005) may play a role. Furthermore, when the monkeys were treated with anti-Nogo-A antibody (neutralizing axon growth inhibitors), the callosal connectivity of the premotor cortex was reorganized (Hamadjida et al., 2012). Our anatomical findings support the notion that post-lesional plasticity taking place in one hemisphere might trigger some adaptive changes of the callosal connectivity in case of a unilateral lesion of the premotor area (e.g. apraxia symptoms, Watson and Heilman, 1983).

In the context of sensory deprivation in humans suffering from chronic deafness, recent morphometric studies (Penhune et al., 2003; Kara et al., 2006) have reported an increase in the volume of the hand motor area, suggesting cross modal plasticity involving either hemispheres. One could deduce that this compensatory phenomenon of sensory substitution (see for example Rauschecker, 1995; Von Melchner et al., 2000; Finney et al., 2003; Lomber et al., 2010; Barone et al., 2013) points toward the role of the corpus callosum in cortical plasticity. However the speculative nature of these phenomena in humans warrants further experiments to become relevant in clinical practice.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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REFERENCES


<table>
<thead>
<tr>
<th>Code</th>
<th>Area Description</th>
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<tbody>
<tr>
<td>1–2</td>
<td>somatosensory areas 1 and 2</td>
</tr>
<tr>
<td>3a/b</td>
<td>somatosensory areas 3a and 3b</td>
</tr>
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<td>5</td>
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<tr>
<td>7b</td>
<td>visual areas 7b</td>
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<tr>
<td>7op</td>
<td>area 7op (parietal operculum)</td>
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<tr>
<td>B4/B5</td>
<td>frontal eye field areas</td>
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<tr>
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<td>gustatory cortex</td>
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<td>agranular, dysgranular and granular insula</td>
</tr>
<tr>
<td>IPa</td>
<td>area in the superior temporal sulcus</td>
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LIP: lateral intraparietal area
M1: primary motor cortex (F1)
ML: middle lateral, belt region of the auditory cortex
PGa: area in the superior temporal sulcus
PMd: dorsal premotor cortex (F2/F7)
PMv: ventral premotor cortex (F4/F5)
PCo: precentral opercular area
Pre-SMA: pre-supplementary motor area (F6)
R: rostral, core region of the auditory cortex
RM: rostromedial, belt region of the auditory cortex
RTL: lateral rostrottemporal, belt region of the auditory cortex
RTM: medial rostrottemporal, belt region of the auditory cortex
SII: secondary somatosensory area
SMA-proper: supplementary motor area (F3)
STG: superior temporal gyrus
STGr: rostral superior temporal gyrus
TAa: area in the dorsal bank of the superior temporal sulcus
TE: area in the ventral bank of the superior temporal sulcus
TPO: area in the dorsal bank of the superior temporal sulcus
VIP: ventral intraparietal area