

## Dissociable Roles of Prefrontal and Anterior Cingulate Cortices in Deception

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**Recent neuroimaging studies have shown the importance of the prefrontal and anterior cingulate cortices in deception. However, little is known about the role of each of these regions during deception. Using positron emission tomography (PET), we measured brain activation while participants told truths or lies about two types of real-world events: experienced and unexperienced. The imaging data revealed that activity of the dorsolateral, ventrolateral and medial prefrontal cortices was commonly associated with both types of deception (pretending to know and pretending not to know), whereas activity of the anterior cingulate cortex (ACC) was only associated with pretending not to know. Regional cerebral blood flow (rCBF) increase in the ACC was positively correlated with that in the dorsolateral prefrontal cortex only during pretending not to know. These results suggest that the lateral and medial prefrontal cortices have general roles in deception, whereas the ACC contributes specifically to pretending not to know.**

**Keywords:** executive function, frontal lobe, lie detection, PET, social interactions

### Introduction

'Never cry wolf!' This well-known saying adapted from Aesop's *Fables* has been used by countless numbers of parents and teachers as a warning against deception. However, since ancient times, humans the world over have repeatedly deceived each other for various reasons. Some types of deception might be relatively trivial, such as those between husband and wife, but others can be serious, such as those between nations. Because deception has a powerful effect on people's lives in various situations, questions concerning why people tell lies, when and where they do, how they do, and whether or not we can detect the deceptions of others are of great interest to many researchers in different disciplines, including psychophysiology, developmental psychology, social psychology and psychiatric medicine.

Some light has been shed on the brain mechanisms involved in deception by the research into its neural basis embarked on by cognitive neuroscientists (Blakemore and Frith, 2004; Blakemore *et al.*, 2004). Using transcranial magnetic stimulation (TMS), Lo *et al.* (2003) found increased cortical excitability of bilateral motor regions related to the generation of deceptive responses, compared with truthful responses. In a recent study using event-related potential (ERP), Johnson *et al.* (2004) argued that the anterior cingulate cortex (ACC) plays a key role in deceptive responses. In addition, several neuroimaging studies using functional magnetic resonance imaging (fMRI)

have reported the involvement of the prefrontal cortex in deception (Spence *et al.*, 2001; Langleben *et al.*, 2002; Lee *et al.*, 2002; Ganis *et al.*, 2003; Kozel *et al.*, 2004a,b). Activation of the ACC has also often been reported, as has prefrontal lobe activity (Langleben *et al.*, 2002; Ganis *et al.*, 2003; Kozel *et al.*, 2004a,b). Although these previous neuroimaging studies indicate a crucial role for the prefrontal and anterior cingulate cortices in human deception, the specific role of each region is still unclear.

Lateral prefrontal activation without ACC activation has been observed in some previous studies on working memory (Courtney *et al.*, 1998; Smith and Jonides, 1999), and ACC activation has been consistently reported during tasks associated with the inhibition of a prepotent response or monitoring of cognitive conflict (Devinsky *et al.*, 1995; Carter *et al.*, 1998; Botvinick *et al.*, 1999). In addition, some researchers have argued for a functional dissociation, i.e. that the lateral prefrontal cortices are directly involved in the implementation of control in situations of conflict, whereas the ACC is mainly associated with signaling the presence of processing conflict (Carter *et al.*, 2000; MacDonald *et al.*, 2000; Badre and Wagner, 2004; Kerns *et al.*, 2004). Based on these findings regarding dissociable roles of the lateral prefrontal and anterior cingulate cortices in conflicting situations, we predicted that the lateral prefrontal cortices are consistently activated during deception tasks in terms of executive function, whereas the ACC is active only in monitoring stronger cognitive conflict in the context of deception.

We examined brain activity focusing on two types of deception for past episodes: deception for experienced events (pretending not to know) and deception for unexperienced events (pretending to know). During two deception conditions and two truth conditions, subjects were presented with old photographs related to experienced events in one and new photographs related to unexperienced events in the other. We expected the prefrontal cortices to be active during the two deception conditions compared to the two truth conditions, because the former necessitate executive functions. In contrast, we anticipated that the ACC would be active only during the deception condition in which subjects were asked to tell lies in response to the old photographs (pretending not to know). The old photographs, compared with the new ones, would elicit stronger conflict for the inhibition of true answers during deception because the memory of experienced events would be vividly recovered by recognition of the old photographs, but not by the new photographs.

In the present PET study, we used real-world events as encoding stimuli (Fujii *et al.*, 2004), which has the advantage of the experimental conditions being close to real life. In particular,

predicted cognitive conflict accompanied by the inhibition of true answers about past memories seems more salient in this paradigm. In addition, we used oral responses as behavioral measures because these seem to be a more normal response type for deception in everyday life, although previous neuroimaging studies of deception have often used button-press responses. Before PET scanning, subjects experienced 20 real-world events. During PET, they were presented with either old photographs related to experienced events or new photographs related to unexperienced events and were instructed to tell either truths or lies (see Materials and Methods for details). This factorial design (the response to stimuli, i.e. truth or lie, and the familiarity of stimuli, i.e. old or new, as factors) enabled us to clarify the main effect of deception and interactions between these two factors. Here we show that the prefrontal cortices are associated with deception regardless of the familiarity of stimuli (both pretending to know and pretending not to know) and that the ACC is associated with deception only for experienced events (pretending not to know).

## Materials and Methods

### Participants

Fourteen male volunteers with no history of neurological or psychiatric disease were paid to take part in this study (age range 18–23 years; mean 20.4 years). There were no pathological findings on the MRI of any of the subjects' brains. All of the subjects were right-handed and had scores above +90 on the Edinburgh Handedness Inventory (Oldfield, 1971). They gave their written informed consent in accordance with the Declaration of Helsinki and the guidelines approved by the Ethical Committee of Tohoku University.

### Stimuli

For the experience phase before PET scanning, we prepared 20 real-world events (e.g. coloring a picture, playing a musical instrument, solving a puzzle, consulting a dictionary), each of which consisted of one of ten kinds of actions involving one of 20 implements. Thus, two events included the same action but differed in the stimuli to which the subjects had to react (i.e. coloring two pictures, playing two kinds of musical instruments, solving two puzzles, consulting two kinds of dictionaries, etc.).

For the later tasks with PET scanning, we prepared as visual stimuli 20 photographs of the implements subjects had used (old stimuli) and 20 photographs of implements they had not used (new stimuli). Corresponding to the experienced events, each pair of new photographs of implements was related to one of 10 kinds of experienced actions. These 40 stimuli were divided into four lists, each consisting of 10 photographs related to 10 kinds of actions. Two lists (old-stimulus-dominant lists) consisted of eight old stimuli and two new stimuli, and the other two lists (new-stimulus-dominant lists) consisted of two old stimuli and eight new stimuli. We employed this eight-to-two proportion in order to get subjects to attend to the stimuli, because if all the presented photographs in each condition had been old or new, the subjects might have been able to respond without looking at the presented stimuli after the first trial.

### Tasks

On the morning of the PET experiment day, the subjects experienced 20 real-world events, using one implement per event, all with one of the experimenters in the same room. Each event lasted ~2 min. The order of the 20 events was randomized over subjects.

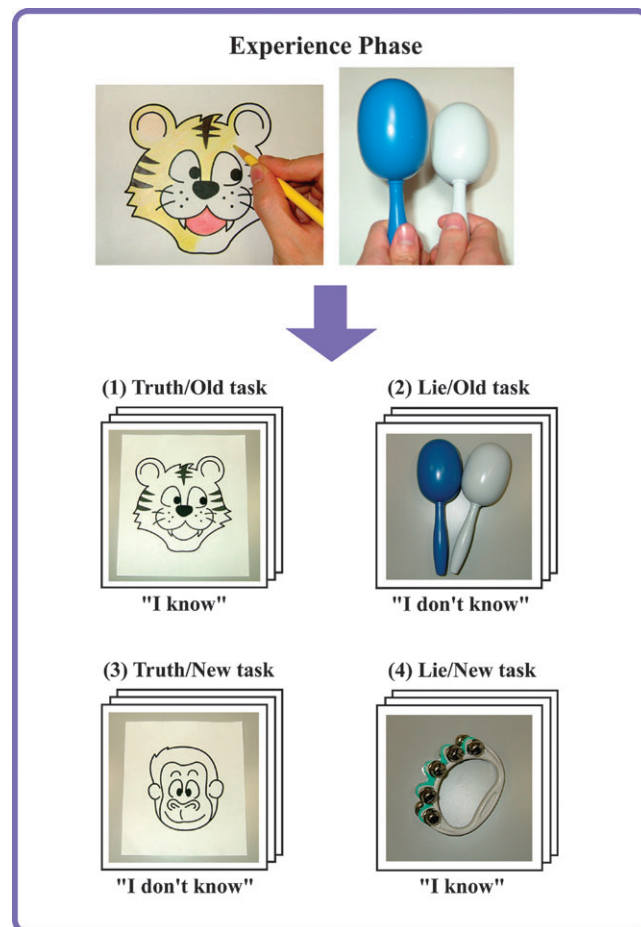
PET measurement was started approximately 120 min (duration range 100–135 min) after the end of the experience phase. During PET, the subjects performed the following four tasks: (i) a Truth/Old task (TO), in which they were instructed to tell the truth in response to one of the old-stimulus-dominant lists; (ii) a Lie/Old task (LO), in which they had to tell lies in response to the other old-stimulus-dominant list; (iii) a Truth/New task (TN), in which they were asked to tell the truth in response to

one of the new-stimulus-dominant lists; and (iv) a Lie/New task (LN), in which they had to tell lies in response to the other new-stimulus-dominant list (see Fig. 1). During each task, the 10 photographs of each list were presented one by one every 6 s (stimulus presentation time 4 s; inter-stimulus interval 2 s) on a display controlled by a Windows computer. The subjects were asked to respond orally, i.e. to say 'I know' in the truth conditions if they thought they had used an implement printed on a photograph, or 'I don't know' if they thought they had not – and vice versa in the lie conditions. There is no difference in motor response across the conditions because the number of syllables for 'I know' and 'I don't know' is the same in Japanese (five syllables). The order of the four tasks was counterbalanced across the subjects and the four lists were also counterbalanced over task conditions.

### Data Acquisition

All the subjects' oral responses were tape-recorded and these data were subsequently used for the evaluation of performance accuracy. The reaction times of each trial were also recorded in a computer by the experimenter manually pressing a button.

The rCBF was measured using PET (SET2400W Shimadzu, FWHM 4.0 mm) and  $^{15}\text{O}$ -labeled water (~180 MBq for each injection). The transaxial sampling field of view (FOV) was 256 mm and the axial FOV was 190 mm. The thickness of the slices measured was 3.125 mm. Prior to the PET experiments, subjects had a catheter inserted into the right brachial vein for tracer administration, and their heads were fixed to an air-cushioned headrest apparatus. Each PET data acquisition time was



**Figure 1.** Experimental procedure of the deception task. In the experience phase before PET scanning, subjects experienced 20 real-world events (e.g. coloring a picture, playing a musical instrument). During PET scanning, subjects were presented with photographs related to experienced or unexperienced events and asked to perform the TO, LO, TN and LN tasks. The verbal responses required during each task are shown below the examples of stimuli.

60 s and the start of the acquisition was synchronized with the start of each task. A transmission scan was followed by the experiment, and the data were used to obtain corrected emission images. A T1-weighted MRI scan (1.5 T) was performed on a separate occasion for coregistration.

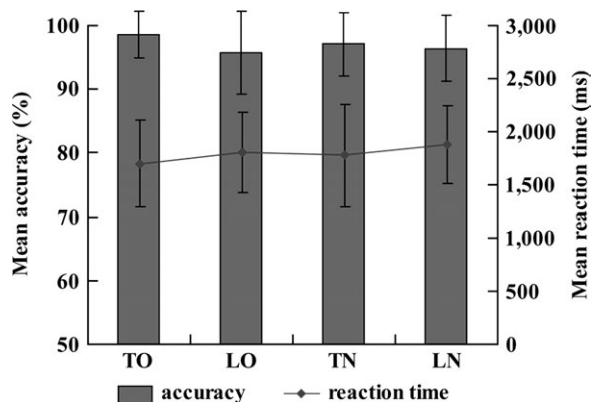
### Data Analysis

The data were analyzed with SPM2 (Wellcome Department of Imaging Neuroscience, UK) executed in Matlab (Mathworks Inc., Sherborn, MA). All rCBF images acquired from each subject were corrected for small movements occurring between scans by realignment to the first image of the experiment. This process generated an aligned set of images and a mean image per subject. A T1-weighted structural MRI was coregistered to this mean PET image. Then, the co-registered T1 image was normalized to the Montreal Neurological Institute (MNI) templates implemented in SPM2. The parameters from this normalization process were applied to each PET image. The PET images were reformatted to isometric voxels ( $2 \times 2 \times 2 \text{ mm}^3$ ) and smoothed with a Gaussian kernel of FWHM of 6 mm. The rCBF-equivalent measurements were adjusted to a global CBF mean of 50 ml/dl/min. Contrast of the main effect of each voxel was assessed using *t*-statistics, resulting in a statistical image (SPM transformed into an SPMz). The threshold of significance was set at  $P < 0.001$  (uncorrected for multiple comparisons). We regarded clusters of 20 or more voxels as significant to reduce the possibility of false-positive results (Type 1 errors). The anatomical identification of activated regions was performed using a standard Talairach and Tournoux space (Talairach and Tournoux, 1988) through the transformation from MNI to Talairach space (Brett *et al.*, 2002).

## Results

### Behavioral Data

The mean accuracy and reaction time were 98.6% (SD = 3.6) and 1699 ms (SD = 411) for TO, 95.7% (SD = 6.5) and 1807 ms (SD = 380) for LO, 97.1% (SD = 5.0) and 1776 ms (SD = 484) for TN, and 96.4% (SD = 5.1) and 1876 ms (SD = 362) for LN. These data were analyzed using two-way repeated-measures analysis of variance (ANOVA), with the response to stimuli (Truth, Lie) and the familiarity of stimuli (Old, New) as factors (Fig. 2). ANOVA showed no significant main effects of either the response to stimuli [correct response,  $F(1,13) = 1.349$ ,  $P = 0.266$ , ns; reaction time,  $F(1,13) = 4.552$ ,  $P = 0.053$ , ns] or the familiarity of stimuli [correct response,  $F(1,13) = 0.085$ ,  $P = 0.775$ , ns; reaction time,  $F(1,13) = 3.027$ ,  $P = 0.106$ , ns], and no interaction between these two factors [correct response,  $F(1,13) = 0.455$ ,  $P = 0.512$ , ns; reaction time,  $F(1,13) = 0.007$ ,  $P = 0.934$ , ns].

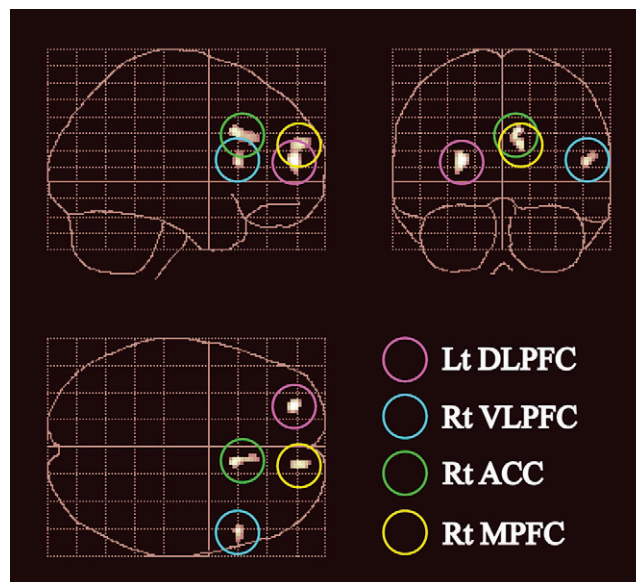


**Figure 2.** Mean accuracy of responses and mean reaction times across the four tasks. Error bars indicate standard deviation. Two-way ANOVA showed no significant difference.

### Brain Activation

To identify the neural correlates of deception, the functional imaging data were first analyzed for the main effect of deception [(LO - TO) + (LN - TN)]. This analysis revealed significant activations in the left middle frontal gyrus [BA 10/46; the most anterior part of the dorso-lateral prefrontal cortex (DLPFC)], right inferior frontal gyrus [BA 45; ventro-lateral prefrontal cortex (VLPFC)], right ACC (BA 24/32) and right medial prefrontal cortex (BA 9; MPFC). These activated regions are shown in Figure 3. Table 1 summarizes these data for anatomical structures and Brodmann's area, MNI coordinates, Z-values and cluster size of peak activations. In this stage of analysis, the main effect of truth telling [(TO - LO) + (TN - LN)] was also calculated, but no significant activation was found.

Second, to examine the influence of the familiarity of stimuli on rCBFs in each activated region and whether or not an interaction occurred, the rCBF values measured at each maximum were analyzed using two-way ANOVA with the response to stimuli (Truth, Lie) and the familiarity of stimuli (Old, New) as factors. The results are illustrated in Figure 4. Results of the ANOVA for the left DLPFC showed a significant main effect of the 'Lie' [ $F(1,13) = 23.470$ ,  $P < 0.001$ ], but showed neither a main effect of the familiarity of stimuli [ $F(1,13) = 0.172$ ,  $P = 0.685$ , ns] nor an interaction between the two factors [ $F(1,13) = 0.173$ ,  $P = 0.684$ , ns]. ANOVA for the right VLPFC yielded similar results: a significant main effect of the 'Lie' [ $F(1,13) = 24.857$ ,  $P < 0.001$ ], without a main effect of the familiarity of stimuli [ $F(1,13) = 1.879$ ,  $P = 0.194$ , ns] or an interaction [ $F(1,13) = 1.087$ ,  $P = 0.316$ , ns]. Results for the right ACC showed a significant main effect of the 'Lie' [ $F(1,13) = 20.895$ ,  $P < 0.001$ ], without a main effect of the familiarity of stimuli [ $F(1,13) = 1.301$ ,  $P = 0.275$ , ns]. In this region, interaction between the two factors was significant [ $F(1,13) = 14.828$ ,  $P < 0.005$ ]. Post-hoc test (Scheffe) revealed that in the right ACC the effect of 'Lie' was significant between the LO tasks and TO tasks (LO > TO,  $P < 0.001$ ), but was not significant between the LN tasks and TN tasks ( $P = 0.756$ , ns), and the effect of 'Old' was



**Figure 3.** Brain regions showing a significant main effect of deception.



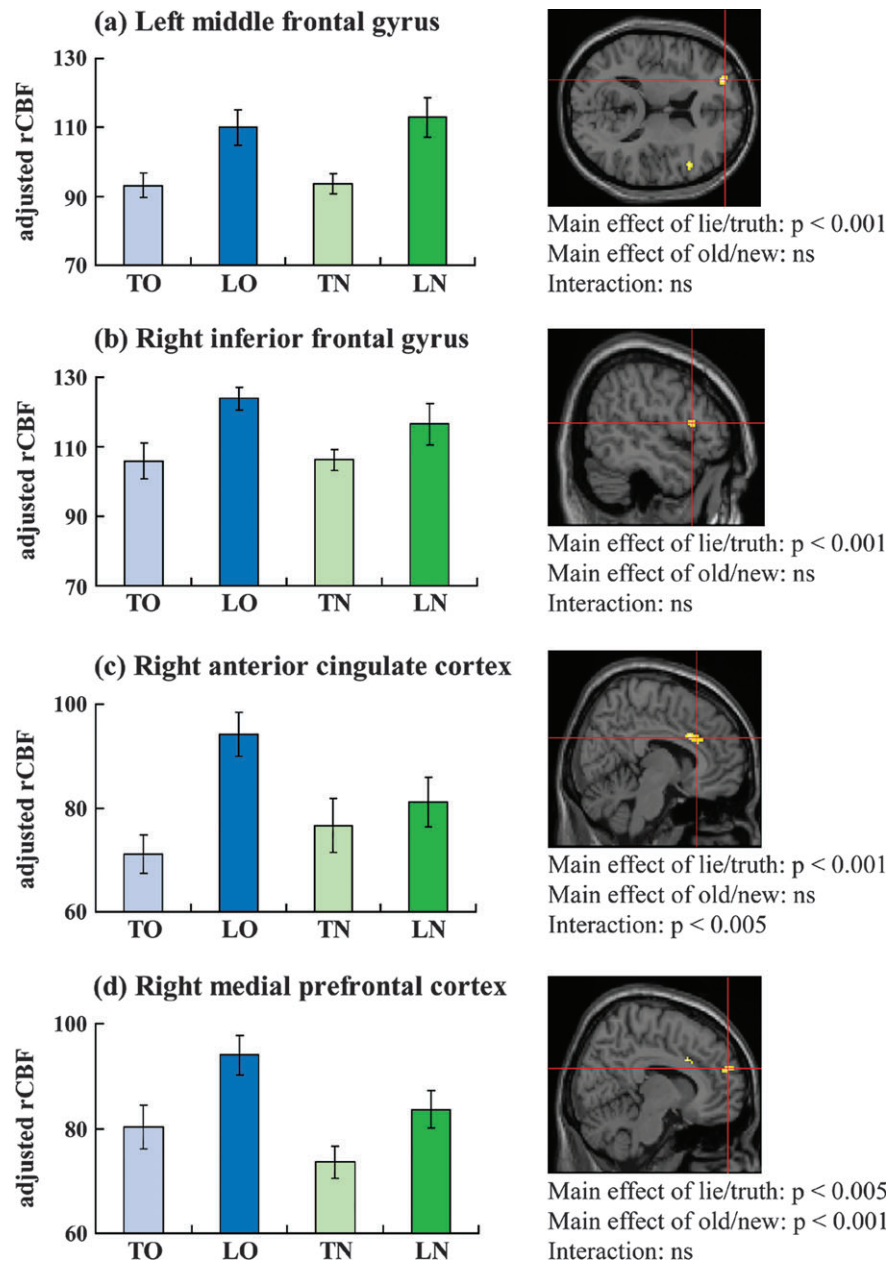
**Table 1**

Brain regions showing activation in a main effect of deception

Region (Brodmann's area)	MNI coordinates			Z-value	Cluster size
	x	y	z		
Left middle frontal gyrus (10/46)	-26	54	14	4.39	51
Right inferior frontal gyrus (45)	52	18	12	4.07	22
Right anterior cingulate cortex (24/32)	10	16	32	4.16	34
Right medial prefrontal cortex (9)	10	56	24	4.04	26

significant between the LO tasks and LN tasks (LO > LN,  $P < 0.05$ ), but not between the TO tasks and TN tasks ( $P = 0.631$ , ns). Results for the right MPFC showed a significant main effect of 'Lie' [ $F(1,13) = 16.336$ ,  $P < 0.005$ ] and a main effect of 'Old' [ $F(1,13) = 18.692$ ,  $P < 0.001$ ], without an interaction [ $F(1,13) = 0.404$ ,  $P = 0.536$ , ns].

Finally, to examine whether the rCBF increases in the right ACC are correlated with those in the three regions of the prefrontal cortices, we performed two correlation analyses between the differences in rCBF values measured at maxima in the ACC and three prefrontal regions: one between the



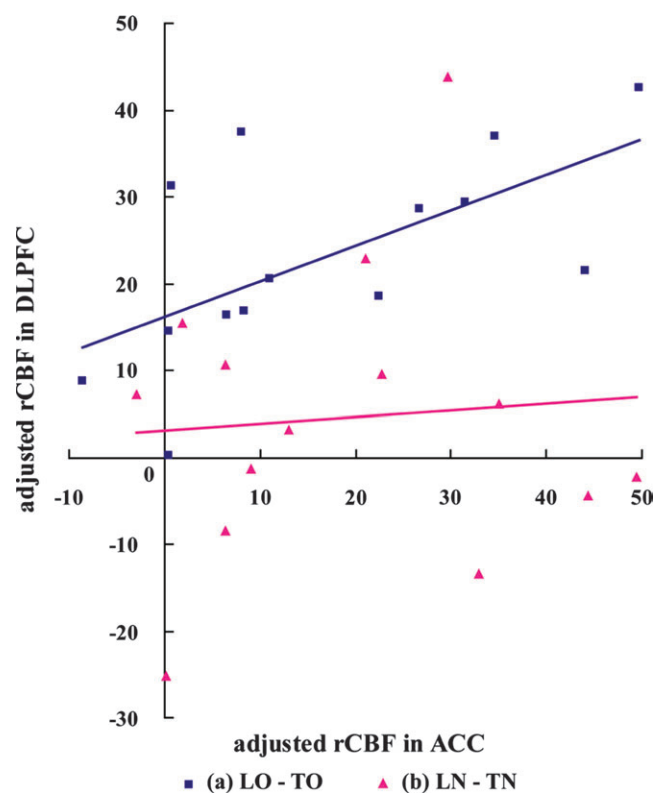
**Figure 4.** Four regions showing a significant main effect of deception. The activations are superimposed onto MRIs of Montreal Neurological Institute (MNI) templates. Histogram bars represent the mean rCBF values adjusted by global normalization during the four tasks, Truth/Old (TO), Lie/Old (LO), Truth/New (TN) and Lie/New (LN). Error bars indicate standard error. (a) Left middle frontal gyrus (-26, 54, 14; BA 10/46); (b) right inferior frontal gyrus (52, 18, 12; BA 45); (c) right anterior cingulate cortex (10, 16, 32; BA 24/32); (d) right medial prefrontal cortex (10, 56, 24; BA 9). The left middle frontal gyrus and right inferior frontal gyrus showed a significant main effect of the response to stimuli (truth/lie) only. An interaction between the two factors, in addition to a significant main effect of the response to stimuli (truth/lie), was found in the right anterior cingulate cortex. The right medial prefrontal cortex showed significant effects of both the response to stimuli (truth/lie) and the familiarity of stimuli (old/new).

differences in rCBF values during LO and TO, and the other between those during LN and TN. We found a significant positive correlation only between the differences in rCBF values in the right ACC and those in the left DLPFC ( $r = 0.622$ ,  $P < 0.05$ ; Fig. 5), which is based on the increase in rCBF values during LO compared with TO. We found no significant correlation between the differences in rCBF values in the ACC and the three prefrontal regions during LN and TN.

## Discussion

The present study examined behavioral and functional anatomical responses associated with deception. The subjects performed our deception tasks with high accuracy. Although there were no significant differences in any of the behavioral data between the deceptive and truthful responses, reaction times showed a marginal effect of lie/truth ( $\sim 100$  ms,  $P = 0.053$ ). To some extent, this trend replicates that found for response times in previous neuroimaging studies of deception (Spence *et al.*, 2001; Ganis *et al.*, 2003), suggesting that the cognitive processes associated with deception are more complex than those associated with truth telling.

The functional imaging data revealed that practicing deception concerning past episodes was associated with activation in some brain regions, including the lateral prefrontal cortices (dorsolateral and ventrolateral activations) and ACC. These activations are consistent with those found in several previous neuroimaging studies of deception (Langleben *et al.*, 2002; Ganis *et al.*, 2003; Kozel *et al.*, 2004a,b). Other than these areas, the main effect of deception was associated with



**Figure 5.** Scatter plot showing correlations of the increase in rCBF values between the right ACC and left DLPFC. (a) The correlation in the 'Old' condition (LO – TO) was significant ( $r = 0.622$ ,  $P < 0.05$ ). (b) The correlation in the 'New' condition (LN – TN) was not significant.

activation in the MPFC. It should be noted that, in line with our prediction, the ACC showed an interaction between the two factors and was only associated with the processes related to pretending not to know.

Our results indicate that the left DLPFC was associated with deception irrespective of the familiarity of stimuli, because only a main effect of deception was significant. The activity in the left DLPFC might be interpreted as a neural correlate of the implementation of executive function (Duncan, 2001; Miller and Cohen, 2001; Spence, 2004; Spence *et al.*, 2004), because participants had to inhibit their subjective true answers and make deceptive answers in response to the presented stimuli. Inhibition of the prepotent responses and making deceptive responses seem to be closely linked to working memory in terms of the maintenance of subjective true answers and manipulation of the true answers to deceptive answers. A neuropsychological study reporting a patient with bilateral damage to the DLPFC showed clear evidence that the DLPFC is indispensable for central executive function of working memory (Fujii *et al.*, 1997). The selective involvement of the DLPFC in such executive function has also been suggested by neuroimaging studies (D'Esposito *et al.*, 1995; Cohen *et al.*, 1997).

Another possible explanation for the lateral prefrontal activity is that left DLPFC activation might be associated with the generation of deceptive responses, and right VLPFC activation with inhibition of the true answers, as stated below. In line with this idea, it is widely reported that the DLPFC is associated with action generation. In particular, and consistent with our finding of left-lateralized DLPFC activation during deception, left dominant DLPFC activation has been found in the free selection of response behaviors such as random number generation (Jahanshahi *et al.*, 2000), word stem completion (Desmond *et al.*, 1998) and verbal fluency (Frith *et al.*, 1991; Phelps *et al.*, 1997).

The present study detected significant DLPFC activation only in the left hemisphere. However, previous fMRI studies of deception have reported DLPFC activation in the right hemisphere (Kozel *et al.*, 2004a), bilateral hemisphere (Lee *et al.*, 2002), and the more anterior part of the prefrontal cortex with right dominance (Kozel *et al.*, 2004b) or bilaterally (Ganis *et al.*, 2003). This inconsistency of laterality might result from the differences in stimuli or response forms employed in our own and the previous studies. Alternatively, the threshold in our statistical analysis might have prevented us from detecting bilateral activation in the DLPFC. Whatever the explanation, the challenging issue of laterality in relation to task differences or other factors should be carefully considered in future functional neuroimaging studies on deception.

As well as the left DLPFC, the right VLPFC also showed only a main effect of deception. Some fMRI studies of deception have also found VLPFC activation with right dominance (Kozel *et al.*, 2004b) or bilaterally (Spence *et al.*, 2001; Kozel *et al.*, 2004a). There is a possibility that the right VLPFC activation in our deception tasks reflects the process of the inhibition of honest answers to the presented stimuli. A recent neuropsychological study reported that damage to the right inferior frontal gyrus evoked disruption of a stop signal task (Aron *et al.*, 2003). In the functional neuroimaging studies using the go/no-go task or related paradigm, activation of the right VLPFC has been found during the no-go condition (Konishi *et al.*, 1999; Rubia *et al.*, 2001) or during the generation of the stop signal (Garavan *et al.*, 1999; Rubia *et al.*, 2003).

The right ACC showed interaction and was only associated with pretending not to know. Although some previous studies of deception have reported activation of the ACC (Langleben *et al.*, 2002; Ganis *et al.*, 2003; Kozel *et al.*, 2004a,b), its involvement might not always be necessary for all types of deception. Phan *et al.* (2002) reported in their review of PET and fMRI that ACC activation was often observed during emotional tasks. However, in the present study, emotional processing is unlikely to have caused activation of the ACC, which was associated with only one of the deception tasks. Instead, in the processes related to pretending not to know, ACC activity might be associated with conflict monitoring (Carter *et al.*, 2000; MacDonald *et al.*, 2000; Badre and Wagner, 2004; Kerns *et al.*, 2004), because vivid memory traces of events were automatically recruited by looking at the old stimuli in response to which the subjects had to lie. On the other hand, in the processes related to pretending to know, the ACC might not be involved in the detection of such a conflict, because vivid memory traces of events were not recovered by encountering the new stimuli.

A previous fMRI study reported stronger activation of the ACC during spontaneous lies that do not fit into a story than during well-rehearsed lies that fit into a coherent story (Ganis *et al.*, 2003). On the face of it, this finding seems to be the opposite of our finding, but the nature of the memories associated with practicing deception was different between the two studies. Ganis *et al.* asked their subjects to memorize a false scenario (i.e. deceptive responses *per se*) in the condition of well-rehearsed lies, whereas we manipulated the novelty/familiarity of events associated with deception, not the deceptive response itself. Although there seem to be qualitative differences in terms of the memories associated with deception, our interpretation of conflict monitoring on ACC activation during pretending not to know is essentially similar to the interpretation of Ganis *et al.* that this region is associated with conflict monitoring and the inhibition of competing responses.

With respect to the functional relationship between the ACC and the three prefrontal cortices, we found a positive correlation only between the differences in rCBF values (in LO - TO, but not in LN - TN) in the right ACC and left DLPFC ( $r = 0.622$ ,  $P < 0.05$ ; Fig. 5). It is unlikely that this correlation is simply due to anatomical connections between these two areas (Goldman-Rakic, 1988), because it was significant for the differences between the 'Old' condition (LO - TO), whereas it was not significant for the differences between the 'New' condition (LN - TN). This finding suggests that the ACC is functionally connected to the DLPFC and that conflict associated with activity in the former during pretending not to know might enhance the executive processes related to the DLPFC. In fact, possible functional modulation from the ACC to the DLPFC has been reported in a recent neuroimaging study of cognitive control (Kerns *et al.*, 2004).

In the present study, the right MPFC was more activated in both lie conditions than in both truth conditions, without an interaction. Some previous neuroimaging studies have also reported MPFC activity during lie conditions (Spence *et al.*, 2001; Langleben *et al.*, 2002). Our finding indicates that as well as the lateral prefrontal areas, this medial prefrontal region might also play a general role in practicing deception. One possible interpretation is that this activation might reflect an emotional response of the subjects in our deception tasks, since the MPFC is probably associated with emotional processing

(Phan *et al.*, 2002). Consistent with our finding of right-dominant medial prefrontal activation, it has been reported that activity in the right MPFC was related to afferent representation of skin conductance responses (Critchley *et al.*, 2000), which have been used as one of the physiological indices of lie detection.

In addition to the main effect of deception, this region also showed a main effect of 'Old', which suggests that the MPFC might be more sensitive to old than to new stimuli in the setting of deception tasks — as may be indicated by the results of a previous psychophysiological study of lie detection using the galvanic skin response (Kugelmass *et al.*, 1967). This study revealed that during lie detection, only the recognition of old stimuli (critical stimuli for the liars in the experimental condition) caused subtle changes in the measured physiological values that were sufficient to indicate deception, regardless of answering 'yes' or 'no' (telling a lie or not). Thus, the recognition of old stimuli might evoke more robust emotion than encountering new stimuli in the context of deception tasks, and this strong emotion might be related to activity in the MPFC. Alternatively, the main effect of 'Old' in MPFC activation might simply reflect the retrieval of experienced events. A recent fMRI study examining the neural basis of autobiographical memory recollection reported activation of the medial prefrontal region (Gilboa *et al.*, 2004).

It is worth commenting here on the relationship between memory and deception. Our experimental paradigm seems to be associated with the Guilty Knowledge Test (GKT), first described by Lykken (1959) in terms of the specific memories underlying deception. The GKT utilizes a series of multiple-choice questions, each presenting one 'relevant' answer that would be known only to a suspect involved in the crime and several 'neutral' (control) answers that are chosen so that an innocent suspect would not be able to discriminate them from the relevant answer. Therefore, the GKT procedure detects specific memories related to the crime rather than deceptive responses *per se*. The tasks of pretending to know in our experiment were not affected by prior knowledge of experienced events, whereas the tasks of pretending not to know were closely associated with prior knowledge of experienced events, which appears to be similar to the interrogation used in the GKT. Thus, our finding of cingulo-prefrontal network activation during pretending not to know might be partly related to the findings of the GKT paradigm (Langleben *et al.*, 2002). More detailed research into the relationship between memory and deception is therefore required. Additionally, in studies of deception, whether we examine the memories under the specific condition or deception *per se* should be taken into account.

## Conclusion

Our results demonstrate the possibility of dissociable roles of the prefrontal and anterior cingulate cortices in human deception. The prefrontal cortices, including the DLPFC, VLPFC and MPFC, are associated with giving deceptive responses regardless of the familiarity of the stimuli, although the precise role of each prefrontal cortex needs to be clarified in future studies. The ACC is associated only with giving deceptive responses to old (experienced) stimuli. When individuals have to give deceptive responses to experienced events, the ACC probably detects strong cognitive conflict and may have



a specific role in the inhibition of memories about experienced events.

There is a limitation of the present study that should be borne in mind for future studies into the brain mechanisms underlying deception. Although we employed real-world event tasks, simulated deception in laboratory experiments cannot be viewed as being the same as deception in real life. In particular, the emotional components of tasks dealing with deception are often not sufficient to allow one to investigate the effect of the emotion itself. A further refined experimental design is needed to deal with this problem and to enable us to understand the complex biological mechanisms of human social interactions.

## Notes

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