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Neural processes underlying self- and other-related lies: An individual difference approach using fMRI

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Two hypotheses were tested using a novel individual differences approach, which identifies rate-limiting brain regions, that is, brain regions in which variations in neural activity predict variations in behavioral performance. The first hypothesis is that the rate-limiting regions that support the production of lies about oneself (self-related) are partially distinct from those underlying the production of lies about other individuals (other-related). The second hypothesis is that a cingulate–insular–prefrontal network found to be rate-limiting for interference tasks is involved in both types of lies. The results confirmed both hypotheses and supported the utility of this individual differences approach in the study of deception in particular, as well in the study of complex cognitive phenomena more generally.

INTRODUCTION

Deception is a fundamental and pervasive social behavior that occurs when one individual attempts to convince another to accept as true what the deceiver believes to be false information in order to gain a benefit or avoid punishment (Vrij, 2000). Although in most cases deception has only minor negative results, or even positive ones (e.g., facilitation of social interactions), in some others it can have enormous adverse consequences (e.g., espionage).

Because of the potentially negative consequences of deception, many researchers have sought to understand deception and to devise methods to detect it. The traditional ''emotional approach'' to deception (Vrij, 2000) has led to methods based on observation of arousal-related changes in overt behavior (DePaulo et al., 2003) or on measurement of peripheral psychophysiological variables (e.g., skin conductance). However, such methods have shortcomings, from both a theoretical and an applied standpoint. From a theoretical standpoint, these methods typically

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overlook the fact that deception is not a single, unitary phenomenon; rather there are different types of lies (e.g., those made spontaneously vs. those based on a well-rehearsed and memorized scenario), and different mechanisms may underlie the different types (Ganis, Kosslyn, Stose, Thompson, & Yurgelun-Todd, 2003). From a practical standpoint, such methods are not very precise at detecting deception, in part because they lack specificity (National Research Council, 2003).

In an effort to circumvent these problems, work in the past three decades has emphasized the cognitive processes underlying deception (Buller & Burgoon, 1996; McCornack, 1992, 1997; Zuckerman, DePaulo, & Rosenthal, 1981), laying the foundation for the recent studies on the neurocognitive underpinning of deception. Early research on the neural bases of deception was conducted using event-related potentials (ERPs) (e.g., Allen & Iacono, 1997), but in the past few years there have been numerous functional magnetic resonance imaging (fMRI) studies of deception (Abe et al., 2006; Abe, Suzuki, Mori, Itoh, & Fujii, 2006; Davatzikos et al., 2005; Gamer, Bauermann, Stoeter, & Vossel, 2007; Ganis et al., 2003; Kozel et al., 2005; Kozel, Padgett, & George, 2004; Langleben et al., 2002, 2005; Lee et al., 2002, 2005; Mohamed et al., 2006; Nunez, Casey, Egner, Hare, & Hirsch, 2005; Phan et al., 2005; Spence et al., 2001, 2004).

With the advent of fMRI, researchers are beginning to characterize the mechanisms that underlie not only deception, but different sorts of deception. In a prior fMRI study, we put forward and tested the idea that, like most other complex cognitive processes, partially nonoverlapping neural systems support the generation of different types of lies (Ganis et al., 2003). In that study we documented differences between episodic autobiographical spontaneous lies versus rehearsed lies, and suggested that an additional distinction worth studying is between lies about oneself (self-related) and lies about other individuals (other-related). This distinction is important for at least two reasons. First, there are long-term differences between self-related and other-related lies because the vast majority of lies people tell appear to be self-related (DePaulo & Kashy, 1998); thus, most people are much more experienced in telling self- than other-related lies. Second, there is increasing evidence that the brain networks storing memories about oneself are partially distinct from those storing other types of memories, although the details are still unclear (e.g., Maguire, 2001; Schaefer et al., 2006; Svoboda, McKinnon, & Levine, 2006).

Thus, the first goal of this study was to use the same logic employed in Ganis et al. (2003) to compare the neural processes underlying the production of self-related lies and other-related lies. It is important to note that self-related lies are based on self-related memories, which are the same as autobiographical, but not as episodic, memories (see Roediger & Marsh, 2003): That is, although self-related memories often are about specific episodes (e.g., ''I was at home on Sunday afternoon''), in many cases they are also about semantic or procedural information about oneself that is not associated with a specific episode (e.g., ''I don't like classical music'' or ''I know how to ride a unicycle''). What is in common among all types of self-related memories is that they are about the self. Similarly, what is in common among all types of other-related memories is that they are about another person. Like self-related lies, other-related lies may be about episodic memories (e.g., a specific event during a televised Presidential debate) or about semantic memories not associated with specific episodes (e.g., ''George Bush is a Republican'').

Thus, the distinction between self-related and other-related lies is different from the classic distinction between episodic and semantic memory, and the neural mechanisms that underlie the two sorts of lies are not likely to be the same as those that underlie the two types of memories (cf. Tulving, 2002). Virtually all neuroimaging studies of deception have investigated lies about autobiographical events (either about a person's past life or about laboratory episodes). The only fMRI study that has examined nonautobiographical lies (in this case, not lies about other individuals, but about world knowledge—such as whether New York City is in Ohio) found no differences when comparing directly deceptive and honest responses (Nunez et al., 2005). In the same study, differences were found instead when comparing directly deceptive and honest responses for autobiographical lies (although no direct contrast was reported between the autobiographical and nonautobiographical conditions), including activation differences in the anterior cingulate cortex, and numerous prefrontal cortical foci. This suggests that distinct neural processes underlie these two types of lies.

To document differences between self- and other-related lies, in the current study we used a novel individual differences logic that employs a brain–behavior correlation method (Ganis,

Thompson, & Kosslyn, 2005; Kosslyn, Thompson, Kim, Rauch, & Alpert, 1996; Miller et al., 2002; Ng et al., 2001; Plomin & Kosslyn, 2001; Wager et al., 2005). The core of this logic revolves around the distinction between *rate-limiting* and *minimally sufficient processes*. Rate-limiting processes for a given task are performance bottlenecks; that is, task performance depends greatly on the efficiency of these processes. In contrast, performance is not greatly affected by the efficiency of minimally sufficient processes. An analogy can be used to clarify these concepts. The precision with which one can produce a miniature airplane model depends critically on one's fine motor skills – a rate-limiting process for this task – but not on the strength of one's biceps, assuming the minimal strength required to support the arm. In contrast, the maximum weight one can lift depends crucially on the strength of one's biceps, but only minimally on fine motor skills. This analogy serves to underscore that a rate-limiting process in one task may not be one in another; the key is the role that the process plays in enhancing performance of a specific task. Moreover, we must note that virtually all complex tasks (e.g., throwing darts) depend on combinations of minimally sufficient and rate-limiting processes.

One way to identify the neural substrates of rate-limiting processes for a certain task is to find brain regions where activation predicts task performance across individuals. Such brain regions, by definition, are the ones that mostly affect variations in performance. Although the resulting evidence is still correlational, using information from individual differences to identify rate-limiting processes brings us closer to the goal of defining the sets of component processes that give rise to performance in complex tasks such as deception. Indeed, Sidtis (2007) examined neuroimaging data of speech production, and found that regions revealed by individual differences in performance correspond better to the regions usually found to cause speech problems when disrupted by brain damage than do regions revealed by standard contrast analyses.

Most neuroimaging studies have focused on minimally sufficient processes by finding areas that are consistently more activated in one condition than another. In contrast, only a relatively small number of fMRI studies, none of them investigating the neural bases of deception, have focused on rate-limiting processes and on documenting differences in rate-limiting processes between conditions (e.g., Ganis et al., 2005; Kosslyn et al., 1996; Miller et al., 2002; Ng et al., 2001; Wager et al., 2005).

Among the processes thought to be recruited during the generation of deceptive responses are those required to monitor and resolve interference caused by a ''prepotent'' response—namely, the response that corresponds to the truth (e.g., Johnson, Barnhardt, & Zhu, 2004; Vrij, 2000; Zuckerman, DePaulo, & Rosenthal, 1981). This response must be suppressed in order to produce a lie. To date, there have been no neuroimaging studies that address whether these interference-monitoring and resolution processes are rate-limiting for deception. However, a study of interference-monitoring and resolution processes using the same individual difference logic used here found a network of rate-limiting regions comprising the cingulate, the insula, and portions of the prefrontal cortex (Wager et al., 2005): Activation in these regions predicted the response times (RTs, in this case difference scores between interference and no-interference conditions) in Go/No Go, flanker task, and stimulus–response compatibility tasks, all requiring some form of interference monitoring and resolution.

Thus, the second goal of this study was to test the hypothesis that interference-monitoring and resolution processes are rate-limiting for deception tasks. We expected to find the same network of rate-limiting brain regions found by Wager et al. (2005) when we conducted individual differences analyses of the data from our deception conditions.

MATERIALS AND METHODS

Participants

Sixteen Harvard University undergraduates (8 females, 8 males; mean age 23 years), volunteered to take part in the study for pay. All had normal or corrected-to-normal vision, no history of neurological disease, and were right-handed. All participants gave written informed consent for the study according to the protocols approved by Harvard University and Massachusetts General Hospital Institutional Review Boards. We analyzed data from 14 participants. Data from 2 participants (1 female and 1 male) were not analyzed because they did not complete the study; the demographics of these 2 participants were comparable to those of the entire group.

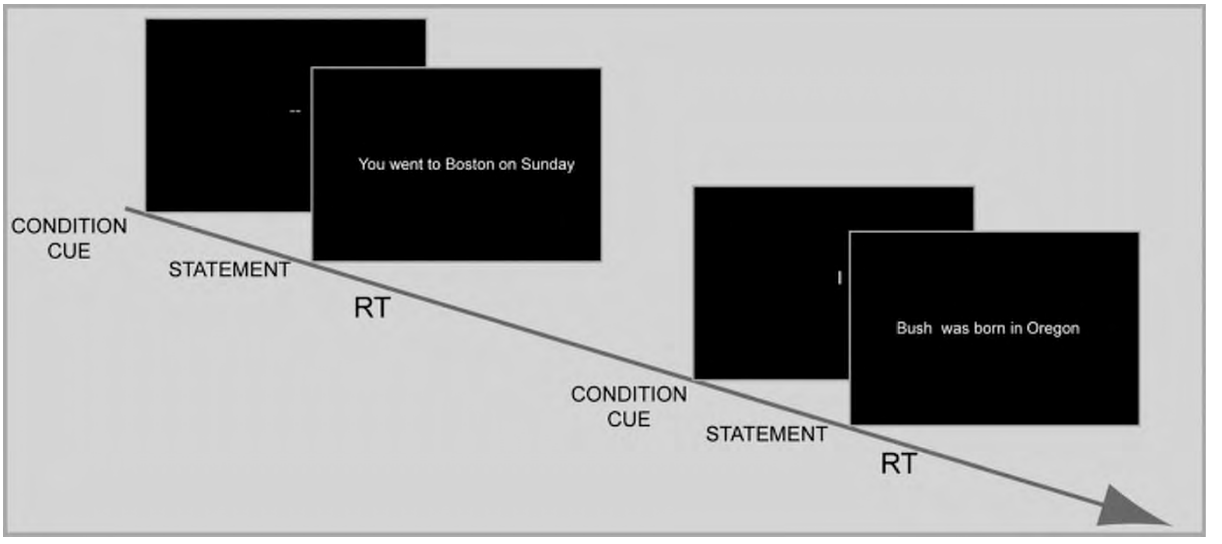

Figure 1. Schematic illustration of two experimental trials. The time between the condition cue and the statement was 4 s. The time between the response (and collection of response time, RT) and the next condition cue was 14 s.

Stimuli

The stimuli were 200 written statements, 100 about oneself (self-related) and 100 about George W. Bush (other-related). George W. Bush was chosen for the other-related condition because participants were highly familiar with numerous facts about him and his life from the media, but they had never met him in person. With others, such as close friends or relatives, most other-related knowledge would be contaminated by self-related memories of interactions with these people.[1]

Half of the statements were true and half false. Each true sentence had a companion false sentence (e.g., ''You went to England last week'' vs. ''You went to France last week'') so that the response keys were not systematically associated with honest or deceptive responses: Key 1 (or 2) was the appropriate response for half of the honest responses, and for half of the deceptive responses. For the self-related statements, a week before the fMRI session, the experimenter interviewed each participant about numerous topics so that true and false statements could be constructed (e.g., ''Last year you went to England''). The specific content of these statements was different for each participant. The other-related sentences were well-known facts about George W. Bush (as assessed by polling a group of undergraduates who did not participate in this study), and were identical for all participants. During a second session, also preceding the fMRI session, participants were shown all the statements and asked to cross out the false ones, to ensure they knew the correct answers. Statements were kept simple and short and their average length (5.4 words) was matched across conditions.

Procedure

Tasks were administered on a Macintosh G3 Powerbook computer using Psyscope software (Macwhinney, Cohen, & Provost, 1997). Stimuli were projected via a magnetically shielded LCD video projector onto a translucent screen placed behind the head of the participants. A front-surface mirror mounted on the head coil enabled viewing of the screen. Prior to the MRI session, we administered general health history and Edinburgh Handedness (Oldfield, 1971) questionnaires.

At the outset of the fMRI session, participants read instructions on the computer screen and paraphrased them aloud. We corrected any misconceptions at this time. We then administered 10 practice trials, using simple statements not used in the study (e.g., ''Bush is a singer''). Before each statement a condition cue was shown, either a small horizontal line or a small vertical line (Figure 1).

[1] Data collection for this study ended in the second half of 2005, prior to the widespread negative feelings towards President Bush that arose when the war in Iraq took a turn for the worse. Thus, at the time the study was conducted, the self/other distinction was not clearly confounded with major differences in emotional valence.

The cue instructed participants whether to lie or tell the truth on that trial. The cue type was counterbalanced across participants, so that the horizontal (vertical) line prompted deceptive responses in half of the participants and honest responses in the other half. Participants pressed one key to indicate that the statement was true, and a second key to indicate it was false. They were instructed to respond as quickly as possible without sacrificing accuracy. RTs were measured relative to the onset of each statement.

The fMRI session consisted of eight functional scans. During these scans we presented all 200 statements, in a random order. Each trial began with the condition cue, lasting 4 s. Next, a statement appeared and participants pressed one key to indicate that the statement was true, or a second key to indicate that it was false. After the key press, an intertrial fixation dot was shown for 14 s (to allow the hemodynamic response to return to baseline) before the presentation of the cue for the next trial (Figure 1).

To ensure that participants were actually trying to deceive another person, and gain something for themselves (consistent with our definition of deception), we devised a monetary game. Participants were told that during the fMRI session an investigator would be observing their eye movements and facial expressions by using a camera placed in the scanner, to determine whether they were lying on each trial. Participants would receive an additional 25 cents each time they lied successfully and would lose 25 cents each time their lie was caught by the investigator. All participants received the maximum amount at the end of the study, according to the IRB requirements.

MRI parameters

We used a 3T Siemens Allegra scanner with whole head coil. We collected full-volume structural images using an MPRAGE sequence, before the functional scans (128, 1.3 mm thick sagittal slices, 256×256 matrix). Blood oxygenation changes were assessed with functional scans using a T2*-sensitive sequence (gradient echo, TR = 2000 ms, TE = 30 ms, FOV = 20 cm, flip angle = 90°, 64×64 matrix, voxel size = $3.125 \times 3.125 \times 5$ mm). Each scan resulted in 270 volumes, each composed of twenty-one 5-mm oblique slices (slice gap = 1 mm).

Analyses

Behavioral data

We analyzed mean RTs and error rates (ERs) using a repeated-measures ANOVA with two factors: Deception (lie vs. truth) and Content (self-related vs. other-related).

FMRI data

Data were preprocessed and analyzed with AFNI (Cox, 1996) using the following sequence of steps: (1) slice timing correction; (2) motion correction; (3) spatial smoothing with a Gaussian filter (full-width half-maximum = 4 mm); (4) amplitude normalization, by scaling each time-series to a mean of 100 and calculating the percent change about this mean; (5) spatial normalization to the MNI305 template (Collins, Neelin, Peters, & Evans, 1994); and (6) spatial resampling to a $3 \times 3 \times 3$ mm grid. To model the hemodynamic response function for correct trials in each condition, we used a family of 10 tent functions (covering the period between 4 s before the statement onset to 16 s after it) and estimated the fMRI response at each time point using multiple linear regression. The multiple regression model included fourth-order polynomial trend regressors for each scan to model slow variations in blood-oxygen-level dependent (BOLD) signal over time. In addition, for each condition (self–honest, self–deceptive, other–honest, other–deceptive) there was one regressor for each of the 10 tent functions. Incorrect trials, regardless of the condition they came from, were modeled by using a single set of regressors. Maps of percent signal change for each participant and condition were obtained using the normalized regression coefficients between 6 and 9 s after the onset of each statement, encompassing the peak of the hemodynamic response.

The analyses were similar to those conducted in Ganis et al. (2005). First, we calculated the z-scores of the difference between the deceptive and honest conditions for the RTs, independently for the self- and other-related conditions. Second, the same z-scores were calculated for the fMRI data at each voxel. Difference scores were used because there are large overall individual differences in speed and accuracy, and these would mask any effects of deception *per se* if not

contrasted with a within-individual baseline. The normalization procedure enabled us to compare the findings across regions by using a common scale. Third, we identified which brain regions predicted the behavioral z-scores across participants by conducting a correlation analysis. For this analysis, we used clusters with a 10-voxel extent threshold, all significant at $p < .005$ (corresponding to $|r| > .7$). These parameters provide a good compromise between sensitivity and protection against false positives (Xiong, Gao, Lancaster, & Fox, 1995). To ensure that the results were not due to outliers, for each of the resulting clusters we conducted the correlation analysis a second time using the robust regression algorithm provided by STATA's command ''rreg'', which employs an iteratively reweighted least-squares procedure (StataCorp, 2001). At most one data point for each condition (on about 5% of cases) was given a value of zero by the weighting procedure (i.e., it was flagged as a potential outlier). Finally, we entered the data from identified brain regions into a stepwise forward multiple regression, independently for each condition, to determine which regions were driving the correlations.

The specificity of the results was tested using three methods. First, we determined the extent to which the fMRI data predicted the behavioral data in one condition, by using the brain regions defined by the analysis in the other condition. That is, the brain regions found in the correlation analysis for the self-related condition were used to attempt to predict the data in the other-related condition (and vice versa). If the brain regions found in one condition are specific, activation in them should not predict the RTs in the other condition. Furthermore, the correlation coefficients in the two cases should be significantly different when compared directly with each other. Second, to ensure that the results were not due to global correlations between brain activation and behavior, we used the average brain activation (after removing the brain regions found in the correlation analyses) as a covariate in a partial regression analysis. Third, to test the temporal specificity of the results, we conducted the same correlation analyses in an earlier time window (0–4 s after condition cue presentation), before the neural sequelae elicited by deception processes were evident in the hemodynamic response.

RESULTS

Behavioral data

On average, participants responded correctly to 94.7% of the statements, and responded more quickly on correct trials (Figure 2) in the honest than in the deceptive condition (2073 ms and 2302 ms, respectively; $F(1, 13) = 16.4$, $p < .001$), consistent with the behavioral results reported by Morgan, Tolley, & Kosslyn (in press) with a very similar paradigm that measured voice onset. In addition, participants responded more quickly in the self- than other-related condition (2117 ms and 2258 ms, respectively; $F(1, 13) = 18.7$, $p < .001$). The size of the deception effect (honest minus deceptive) was numerically smaller for self- than other-related conditions (216 ms and 244 ms, respectively), but the difference was not significant, $F(1, 13) = 0.23$, $p > .5$.

In addition, the participants made fewer errors (Figure 3) in the honest than in the deceptive condition (3.7% and 6.9%, respectively; $F(1, 13) = 6.9$, $p < .05$), but they made comparable numbers of errors in the self- and other-related conditions (5.2% and 5.4%, respectively; $F(1, 13) = 0.67$, $p > .1$). Similarly to the RTs, the size of the deception effect in the ERs did not differ between self- and other-related conditions (3.2% and 3.3%, respectively; $F(1, 13) = .98$, $p > .1$). The largest correlation between RTs and ERs was in the self-related honest condition, but this correlation was not significant ($r = .37$, $p = .19$). All other correlations between RTs and ERs were close to

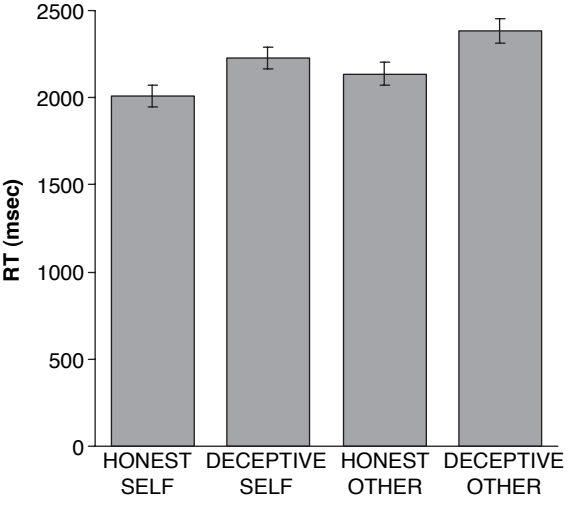

Figure 2. Average RTs in the four experimental conditions. Error bars indicate between-participant variability.

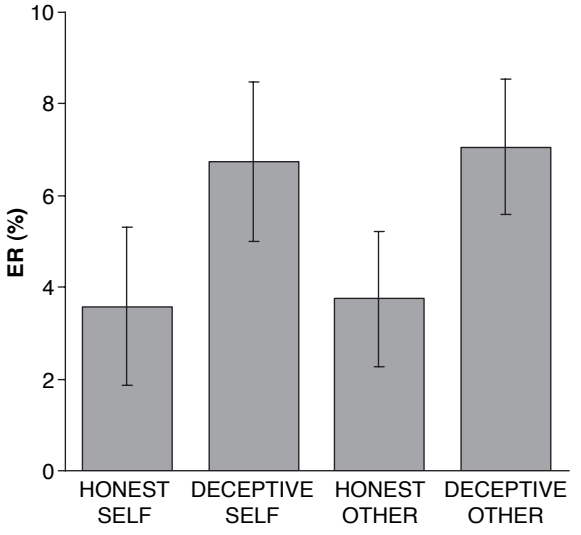

Figure 3. Average error rates (ERs) in the four experimental conditions. Error bars indicate between-participant variability.

zero (all r values $< .15$), indicating that the results were not due to speed–accuracy trade-offs. We did, however, find that participants who tended to have a large deception effect in the self-condition also tended to have a large effect in the other-condition, as indicated by a positive correlation over participants between the RT deception effect in the two conditions ($r = .58$, $p < .05$). The correlation between the ER deception effect in the two conditions was not significant ($r = .2$, $p > .4$).

FMRI data

Given the focus of this article, we only report the results of the analysis of rate-limiting processes. The results of the main correlation analyses are given in Tables 1 and 2 and shown in Figures 4 and 5. Brain activation predicted the RTs in numerous regions during the self-related condition. In many cases, activation in single clusters predicted over 65% of the variance in the RTs. In the frontal lobe, we found clusters in the cingulate gyrus (GC), the medial, inferior, middle, and superior frontal gyri (GFd, GFi, GFm, and GFs, respectively), and the insula (INS). In the occipital and temporal lobes, we found clusters encompassing portions of the lingual gyrus (GL), the inferior and middle occipital gyri (GOi and GOm, respectively), the fusiform gyrus (GF), and the parahippocampal gyrus (Gh) and hippocampus (Hi). In the parietal lobe, we found clusters in the precuneus (PCu) and the cuneus (Cun). Finally, we also found clusters in the right thalamus (Th) and in the left cerebellum (Cer). The correlation between brain activation and behavior for all clusters was positive (Table 1 and Figures 4 and 5); that is, high deception z-scores for brain activation were associated with high deception z-scores for the RTs.

Brain activation also predicted the RTs in numerous regions during the other-related condition (Table 2 and Figures 4 and 5). Similarly to the self-related case, activation in some clusters predicted over 65% of the variance in the RTs. In the frontal lobes, clusters included the GC, the INS and the claustrum (Cl), the GFm and the GFs. In the temporal lobe, we found a cluster in the left Gh and Hi. Finally, in the parietal lobe we found clusters in the left PCu and inferior parietal lobule (LPi). The GC, INS, and GFm/GFs clusters were very close to, but did not overlap with, those found in the self-related condition. The correlation between brain activation and behavior for all clusters was negative (Tables 1 and 2 and Figures 4 and 5); that is, larger deception z-scores for brain activation were associated with low deception z-scores for RTs.

To determine which brain regions were driving the pattern of correlations with the RTs, we entered the identified brain regions (hereafter, regions of interest, ROIs) into a stepwise forward multiple linear regression analysis, independently for the two conditions, as in prior work (Ganis et al., 2005; Kosslyn et al., 1996). However, due to collinearity (i.e., most variables were highly correlated with one another), it was not possible to obtain reliable estimates of the relative contribution of the different variables. This suggests that all regions may function together as a tightly coupled network: The average correlation among areas was .76 and .78 for the self-related and other-related conditions, respectively). To partially circumvent this problem, we used an alternative approach. We calculated multiple linear regression models for all possible subsets of three areas and determined which subset explained the largest amount of variance in the RTs. For the self-related lies, the results indicated that the subset comprising the GC/GFd/GFs, the GL/GOi/Cun/GF/GOm, and the Thal clusters was the most predictive (Table 1): Together, this subset of regions explained 94.9% of the variance in the RTs. For the other-related lies, the subset encompassing the GFm/GFs, the GC (BA 32/24), and the GFm clusters was the most predictive (Table 2): Together, this subset of regions explained 81% of the variance in the RTs.

TABLE 1
Brain areas that had significant correlations between BOLD activation and response times (self-related deception minus honest responses for both measures)

Regions within cluster			*Correlation*	*Talairach coordinates (center of mass)*			*Talairach coordinates (range)*					
Name	*BA*	*Volume (mm^3)*	*r*	*x*	*y*	*z*	*Min x*	*Max x*	*Min y*	*Max y*	*Min z*	*Max z*
GC/GFd/GFs	32/24/8	3024	.84	−3	17	38	−14	8	8	32	33	48
GC/GFd	32/24/9	675	.85	6	34	21	2	11	29	38	12	30
GC/GFd	32/9	486	.82	5	41	9	2	11	41	44	3	15
GFi/INS/Cl	47/13	945	.83	34	21	−3	26	44	17	26	−16	6
INS/Cl/GFi	13/47/45	918	.81	−30	19	4	−35	−26	14	26	−4	12
GFs/GFm	10/9	2511	.87	31	49	18	20	41	41	53	6	30
GFm/GFs	9/10	270	.78	−32	40	26	−35	−32	38	44	21	30
GL/GOi/Cun/GF/GOm	18/17	3618	.84	21	−96	−8	11	32	−104	−89	−22	12
GOi/GF/GL/Cun	18/17	2430	.90	−26	−93	−8	−35	−20	−101	−86	−16	3
GL/Cun	18/17	2430	.91	7	−88	−1	−2	14	−95	−80	−16	18
Cun/GL	18/17	2214	.88	9	−74	−5	−2	17	−83	−68	−22	9
Cun	19/18	1053	.75	−11	−86	27	−17	−8	−92	−80	18	36
GL	18	999	.88	−10	−73	−8	−14	−5	−77	−68	−19	3
PCu/Cun	31/18	378	.74	12	−71	24	8	14	−74	−68	21	27
Gh/Hi	36	205	.92	−29	−26	−10	−32	−26	−29	−23	−13	−7
Th	N/A	297	.80	3	−20	7	2	5	−23	−20	3	12
Cer	N/A	567	.87	−11	−78	−27	−17	−5	−80	−77	−31	−25
Cer	N/A	378	.77	−10	−62	−5	−17	−5	−65	−62	−7	−4
Cer	N/A	297	.75	−28	−75	−20	−32	−23	−77	−74	−22	−19

Notes: Abbreviations: BA, Brodmann's area; GC, cingulate gyrus; GFd, medial frontal gyrus; GFs, superior frontal gyrus; GFi, inferior frontal gyrus; INS, insula; Cl, claustrum; GOi, inferior occipital gyrus; GF, fusiform gyrus; GOm, middle occipital gyrus; GL, lingual gyrus; Cun, cuneus; PCu, precuneus; Gh, parahippocampal gyrus; Hi, hippocampus; GFm, middle frontal gyrus; Th, thalamus; Cer, cerebellum. Correlations were calculated with a robust regression routine from the software package STATA (StataCorp, 2001). Coordinates: *x*, left/right; *y*, anterior/posterior; *z*, inferior/superior. Shown in italics are the three regions that, together, explained variance in the RTs better than any other subset of three regions.

TABLE 2
Brain areas that had significant correlations between BOLD activation and response times (other-related deception minus honest responses for both measures)

Regions within cluster			*Correlation*	*Talairach coordinates (center of mass)*			*Talairach coordinates (range)*					
Name	*BA*	*Volume (mm^3)*	*r*	*x*	*y*	*z*	*Min x*	*Max x*	*Min y*	*Max y*	*Min z*	*Max z*
GC	24	324	−.80	5	−4	39	2	8	−5	−2	36	45
GC	32/24	297	−.87	13	9	39	11	17	8	11	36	45
INS/Cl	13	729	−.86	−35	8	9	−41	−32	5	14	6	18
INS/Cl	13	459	−.82	33	0	3	32	35	−2	2	−4	9
INS	13/41	378	−.80	−44	−23	18	−47	−41	−26	−20	15	21
GFm/GFs	10/46	351	−.70	−42	49	18	−47	−41	47	53	15	21
GFm	8/9	270	−.78	−33	31	38	−38	−32	29	35	36	42
PCu	7	432	−.86	17	−51	50	8	23	−53	−50	45	54
LPi	40	270	−.79	34	−37	42	32	38	−41	−35	39	45
Gh/Hi	36	513	−.78	−28	−12	−19	−32	−26	−17	−8	−22	−19

Notes: Abbreviations: BA, Brodmanns area; GC, cingulate gyrus; INS, insula; Cl, claustrum; GFm, middle frontal gyrus; GFs, superior frontal gyrus; PCu, precuneus; LPi, inferior parietal lobule; Hi, hippocampus; Gh, parahippocampal gyrus. Correlations were calculated with a robust regression routine from the software package STATA (StataCorp, 2001). Coordinates: *x*, left/right; *y*, anterior/posterior; *z*, inferior/superior. Shown in italics are the three regions that, together, explained variance in the RTs better than any other subset of three regions.

The effect of global brain activation on the results was assessed by measuring the partial correlation between the RTs and the brain activation in the ROIs, holding the global brain activation variable constant. The sign and significance of the partial correlation coefficients for all ROIs were the same as in the original analysis, indicating that global brain activation was not a factor.

Next, to test the specificity of the results, for each ROI we compared the correlation coefficient we found in one condition with that calculated by using the same ROI to predict the RTs in the other condition. If the ROIs in one condition were not specific then they would also predict the RTs in the other condition. The results were clear: No correlation coefficient was significant when trying to predict the RTs for one condition using the ROI from the other condition (all $|rs| < 0.3$). Furthermore, all correlations between the ROIs and the RTs in the corresponding condition were significantly larger than those with the RTs from the other condition ($p < .01$ in all cases).

To provide evidence for the temporal specificity of the correlations, we conducted the same analyses on an earlier time window of the data, between 0 and 4 s after the onset of the sentence. Given the typical hemodynamic delay (e.g., Kruggel & von Cramon, 1999), we did not expect any ROIs to predict behavior in this time window. The results confirmed this prediction: With the same threshold and cluster size used in the main analysis, activation in none of the brain regions predicted performance. This result also confirmed that the parameters used in these analyses were unlikely to produce false positives.

Several brain regions found in the self-related condition overlapped with those of the cingulate–insular–prefrontal network for interference monitoring and resolution found in the study by Wager et al. (2005). These included the main GC cluster ($x = 4$, $y = 11$, $z = 45$ in Wager et al.'s study), the two INS clusters ($x = -34$, $y = 19$, $z = 5$, and $x = 34$, $y = 19$, $z = 5$ in Wager et al.'s study), and the two GFm/GFs clusters ($x = -30$, $y = 49$, $z = 20$, and $x = 30$, $y = 41$, $z = 15$ in Wager et al.'s study). A cingulate–insular–prefrontal network was also found for the other-related condition, but the coordinates of the brain regions involved did not overlap exactly with those found in the self-related lie condition. These comparisons provide additional evidence that the two types

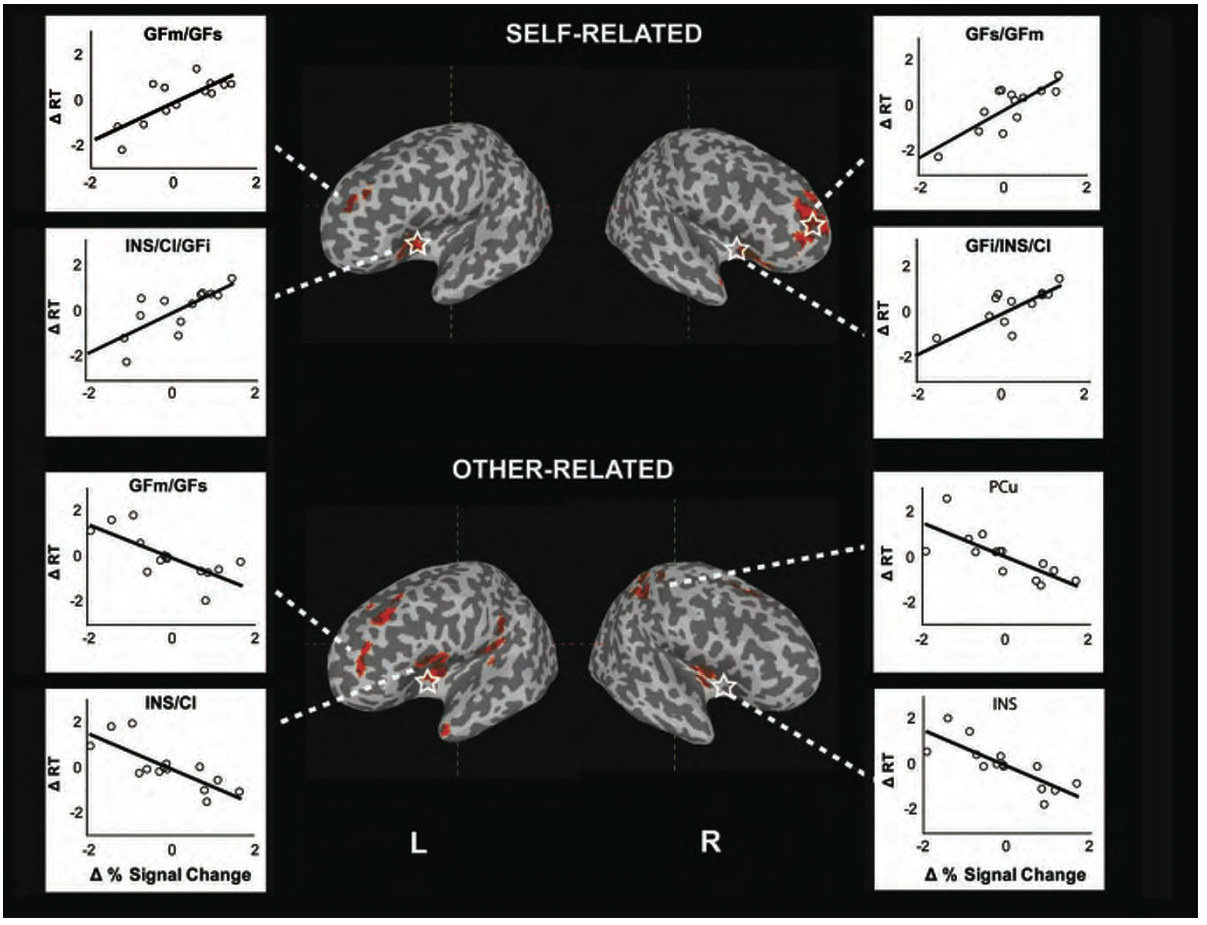

Figure 4. Lateral view of the results of the main correlation analysis identifying regions that predicted RTs across participants. Only clusters with more than 10 adjacent voxels at $p < .005$ (corresponding to $|r| > .7$) were considered significant (Tables 1 and 2). To illustrate clearly regions in which there was significant activation, the clusters shown here were drawn with a lower threshold ($p = 0.01$). Regions that predicted the RTs in the self-related (top) and other-related (bottom) conditions are shown in red. The stars on the insula and on the prefrontal cortex indicate the coordinates of the center of mass of the corresponding rate-limiting regions found by Wager et al. (2005) during three interference tasks. The scatterplots show RTs' z-scores as a function of brain activation z-scores for representative regions.

of lies, about the self vs. about another person, rely on distinct neural systems.

DISCUSSION

First and foremost, we found that the self-related and other-related conditions engaged different sets of rate-limiting regions. Although some subsets of these regions are similar, the two sets are clearly distinct. Crucially, these differences cannot be attributed to differences in RTs or ERs across conditions because the behavioral results were similar in the two conditions.

As predicted, both conditions engaged the anterior cingulate, the insula, and prefrontal cortex (Tables 1 and 2), regions thought to be involved in interference monitoring and resolution (e.g., Carter et al., 1999; Fan, Flombaum, McCandliss, Thomas, & Posner, 2003). However, the foci of activation in the two conditions were not exactly the same. For instance, the centers of mass of the insular activation in the two conditions were 13 and 21 mm apart in the left and right hemispheres, respectively. This result suggests that there is some content (or process) domain specificity within these regions, consistent with findings such as those reported by Fan et al. (2003), in which adjacent portions of the anterior cingulate and prefrontal cortex were activated by different interference tasks (Stroop, flanker, and spatial compatibility tasks). The left hippocampus, another region that implements a rate-limiting process, showed a similar effect: Both

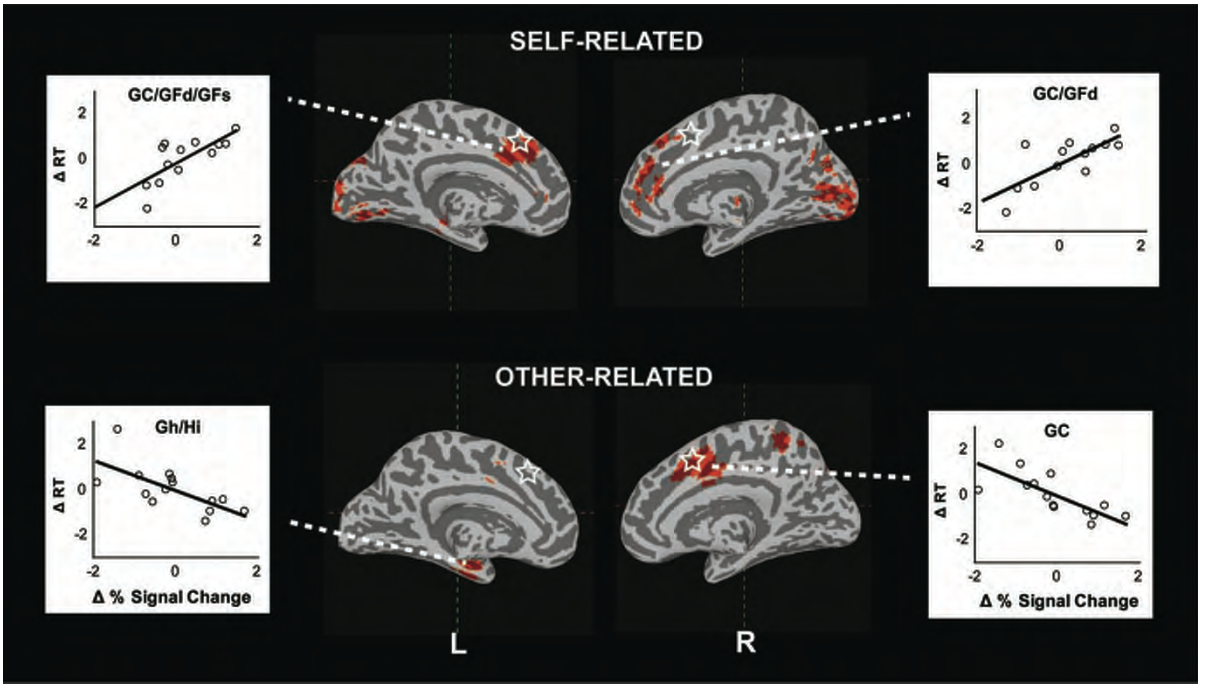

Figure 5. Medial view of the results of the main correlation analysis identifying regions that predicted the RTs across participants. Only clusters with more than 10 adjacent voxels at $p < .005$ (corresponding to $|r| > .7$) were considered significant (Tables 1 and 2). To illustrate clearly regions in which there was significant activation, the clusters shown here were drawn with a lower threshold ($p = 0.01$). Regions that predicted the RTs in the self-related (top) and other-related (bottom) conditions are shown in red. The stars on the cingulate and adjacent medial frontal cortex indicate the coordinates of the center of mass of the corresponding rate-limiting regions found by Wager et al. (2005) during three interference tasks. The scatterplots show RTs' z-scores as a function of brain activation z-scores for representative regions.

conditions activated the left hippocampus, but the center of mass of the focus of activation in the self-related condition was 14 mm more posterior than that in the other-related condition. The hippocampus was not a rate-limiting region in the interference tasks used by Wager et al. (2005), possibly because their tasks did not involve encoding information into, or retrieving it from, long-term memory (Schon, Hasselmo, Lopresti, Tricarico, & Stern, 2004; Svoboda et al., 2006). In contrast, such operations are necessary in both deception conditions: In our task, deceptive responses require the incidental retrieval and encoding of truthful information (as do honest responses).

A number of additional rate-limiting regions were found only in the self-related condition, including occipito-parietal and temporal regions (GOi, GOm, PCu, Cun, GL, and GF), the right thalamus, and the left cerebellum. Performance-related activation in these occipito-parietal and temporal regions may be related to visual mental imagery processes associated with retrieving autobiographical information (Ganis et al., 2003). One possibility is that individuals who produced more vivid mental images in response to the statements experienced more interference during deception. In addition, because we used written sentences as stimuli, it is likely that some of these effects were caused in part by the visual stimuli themselves, or by differential patterns of eye movements, or both. However, the fact that these regions were not found in the other-related condition suggests that an explanation based entirely on a visual stimulation artifact or on differential patterns of eye movements (e.g., more eye movements during the deceptive than honest conditions) cannot explain the pattern of results.

We also found that activation in the right thalamus predicted RTs in the Go/No-Go task used in Wager et al. (2005), which suggests that this activation may be related to motor inhibition, possibly via thalamocortical loops (Guillery & Sherman, 2002a, 2002b). Consistent with this explanation, there is single-case evidence that right thalamic dysfunction is associated with pathological lying (Modell, Mountz, & Ford, 1992). The left cerebellum activation is consistent with that found in Ganis et al. (2003), and may be reflect differences in the amount or type of

memory retrieval in the two conditions (cf. Andreasen et al., 1999). Finally, a small cluster in the right inferior parietal lobule (LPi) predicted the RTs only in the other-related condition. This region may be part of the ''scaffolding'' network required to generate other-related lies, as discussed below.

The second result of this study is that the same cingulate–insular–prefrontal network that Wager et al. (2005) found to predict performance during three interference tasks (all of which require interference monitoring/resolution) predicted performance in the deception conditions (relative to the honest one): The centers of mass of the rate-limiting brain regions found in Wager et al. (2005) matched exactly those found in this study for the self-related condition and were very close to those found for the other-related condition. This finding provides strong evidence that these brain regions implement interference-monitoring and resolution processes that are rate-limiting not only for the interference tasks used in Wager et al. (2005), but also for the deception tasks used here. The very high correlation among these regions across individuals suggests that they may act together as a network. As in Wager et al. (2005), for self-related lies, the correlation between brain activation and the RTs was positive: Larger brain activation z-scores were associated with larger behavioral z-scores; that is, the worst liars (those who took the longest to tell a lie compared to the truth) also showed the highest brain activity during deception (compared to the truth).

Another piece of evidence that the two sorts of lies have different neural underpinnings is the fact that the correlation between brain activation and RTs had different signs in the two conditions: Unlike the self-related condition, in the other-related condition larger brain activation z-scores were associated with smaller behavioral z-scores (the worst liars showed the lowest brain activation). We can only speculate about how to interpret this unexpected result. As noted earlier, in their daily lives, people overwhelmingly lie about themselves (compared to others), as shown by studies of college students (e.g., DePaulo & Kashy, 1998). Thus, over the course of their lives most people have become practiced at lying about themselves, and practice often increases the efficiency of the neural resources required to carry out a task at the same level of performance. For instance, practice on interference tasks reduces activation in the anterior cingulate and prefrontal regions so that, after practice, it takes less neural work to achieve the same performance (e.g., Chein & Schneider, 2005; Milham, Banich, Claus, & Cohen, 2003).

Thus, the rate-limiting regions engaged during self-related lies may reflect the variability in performance among experts in this domain: The best experts have the lowest activation scores whereas the worse experts have the highest activation scores. In contrast, lying about others is a much more infrequent activity for which people may not develop expertise. Lying about others may require the effortful task of configuring a ''scaffolding'' brain network on the fly rather than activating an expert network already in place (Petersen, van Mier, Fiez, & Raichle, 1998). If so, then the rate-limiting regions found for other-related lies may reflect the amount of effort participants put into setting up such a network for the purpose of lying: The negative sign of the correlation for other-related lies shows that the participants with the highest brain activation scores (i.e., those who tried harder) were also the best liars.

It is important to point out some limitations of this study. First, although we used a self- vs. other-related manipulation, there are potential variables that may be correlated with this manipulation and that may be causing some of the differences. For example, there could be some differences in the emotional content of the statements used in the two conditions. Since we did not obtain ratings of the statements on emotional scales, future work will be required to address this issue. Second, the sample size ($N = 14$) is rather small for an individual differences analysis. Thus, the study had limited statistical power and we may have missed important regions with relatively low correlations between brain activation and RTs.

SUMMARY

In summary, this study is a first step toward using an individual differences approach in neuroimaging research on deception. This approach specifically identifies brain regions that support rate-limiting processes. The results show that self-related and other-related lies are supported by partially nonoverlapping networks of rate-limiting regions. Of particular note, we documented that a cingulate–insular–prefrontal network, shown in other studies to be involved in interference

monitoring and resolution, implements a rate-limiting process for self-related lies. A similar network was also engaged by the other-related lies; however, the activation foci were close but not overlapping with those found for the self-related lies, which suggests some regional segregation. Finally, we found that increased activation was associated with slower responses for self-related lies (relative to honest responses), but with slower responses for other-related lies (relative to honest responses). Overall, this study provides further evidence that deception is a multifaceted phenomenon, and the findings suggest that neuroimaging methods to detect deception may need to be fine-tuned for the specific types of lie to be detected. Furthermore, the findings indicate that neuroimaging studies of deception may need to take account of individual differences.

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