Neural Correlates of Telling Lies:
A Functional Magnetic Resonance Imaging Study at 4 Tesla[1]

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Rationale and Objective. Intentional deception (ie, lying) is a complex cognitive act, with important legal, moral, political, and economic implications. Prior studies have identified activation of discrete anterior frontal regions, such as the ventrolateral prefrontal cortex (VLPFC), dorsolateral prefrontal cortex (DLPFC), dorsal medial prefrontal cortex (DMPFC), and anterior cingulate cortex (ACC) during deception. To extend these findings, we used novel real-time functional magnetic resonance imaging (fMRI) technology to simulate a polygraph experience in order to evoke performance anxiety about generating lies, and sought to ascertain the neural correlates of deception.

Materials and Methods. In this investigational fMRI study done with a 4-T scanner, we examined the neural correlates of lying in 14 healthy adult volunteers while they performed a modified card version of the Guilty Knowledge Test (GKT), with the understanding that their brain activity was being monitored in real time by the investigators conducting the study. The volunteers were instructed to attempt to generate Lies that would not evoke changes in their brain activity, and were told that their performance and brain responses were being closely monitored.

Results. Subjects reported performance anxiety during the task. Deceptive responses were specifically associated with activation of the VLPFC, DLPFC, DMPFC, and superior temporal sulcus.

Discussion. These findings suggest the involvement of discrete regions of the frontal cortex during lying, and that the neural substrates responsible for cognitive control of behavior may also be engaged during deception.

Key Words. Functional magnetic resonance imaging; cognition; emotion; deception; prefrontal cortex.

Engaging in deception, or deliberate falsification (eg, lying), is not an uncommon practice in human social interaction. At times, deception can be personally advantageous to the perpetrator; however, by societal norms, deliberate falsification of facts for the purpose of personal gain is almost always considered "immoral" and/or unlawful behavior given its legal, political, and economic implications (1). Therefore, accurate and reliable detection of deception or lying by objective means poses an interesting challenge to experts in many scientific disciplines. Historically, the polygraph, a multi-channel physiological recording, has been widely used as a lie-detection device. However, because the lie detector relies solely on peripheral measures of anxiety (heart rate, skin conductance, and respiration), presumably evoked by performing a self-perceived wrongful act, little is known about the brain mechanisms involved in generating lies (2,3).

Functional magnetic resonance imaging (fMRI) of the brain affords the unique ability to examine localized changes in event-locked brain activity during both cognitive and emotional operations (4,5), and can be used to

Acad Radiol 2005; 12:164–172

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doi:10.1016/j.acra.2004.11.023

examine the neural substrates of deceptive acts. Interestingly, deception can be conceptualized as a complex cognitive operation involving several processes, including awareness of one's own and others' thoughts (eg, theory of mind), generation of novel responses (lies), inhibition of pre-potent responses (truth-telling), task switching and updating, and motivation for keeping lies covert. Previously, Spence and colleagues (6) showed that lying (vs truth-telling) about autobiographical events was associated with greater activity in the ventrolateral prefrontal cortex (VLPFC) and medial prefrontal cortex (MPFC). Similarly, feigning digital and autobiographical memory loss was associated with widespread activation in the parietal, temporal, and frontal cortices, and particularly the dorsolateral prefrontal cortex (DLPFC) and VLPFC (7). Recently, Ganis and colleagues demonstrated that the anterior prefrontal cortices were engaged during general deception, but that the right anterior prefrontal cortex was more involved in lies that were well rehearsed and that fit into a coherent story about one's own history than in spontaneous, non-coherent lies, and that the anterior cingulate cortex was more active during the spontaneous generation of non-memorized lies (8).

These regions have also been implicated in episodic memory (4), and given that these prior studies of the neural correlates of deception have used episodic memory/recall of autobiographical details as part of the deception task, it remains unclear whether the reported activations of frontal cortical regions are related to episodic memory recall or to the generation of lies. Using a modified version of the Guilty Knowledge Test (GKT)(9–11) with playing cards, Langleben and colleagues (2002) demonstrated greater activity in the anterior cingulate cortex (ACC), MPFC, premotor–motor cortex, and anterior parietal cortex during deceptive than during truthful responses (12) in the absence of episodic memory retrieval.

In our event-related fMRI study at high-field strength (4 T), we sought to replicate the findings of Langleben and colleagues (12) by using a similar deception paradigm that also minimizes the engagement of autobiographical recall. However, we extended our investigation by using novel real-time fMRI technology (TurboFIRE; Functional Imaging in REaltime) that allows us to obtain reliable brain-activation results while the subject is engaged in the task and is in the scanning device (13–15). By demonstrating to subjects before scanning that their performance and brain activity would be measured and followed in real time, we attempted to simulate the stress on a subject's thought processes and responses that a subject experiences during a polygraph test as a result of the monitoring of peripheral skin conductance. In this study, we tested the hypothesis that prefrontal regions (ACC, MPFC, DLPFC, VLPFC) are involved in the act of deception.

MATERIALS AND METHODS

Subjects

Fourteen healthy, right-handed volunteers (7 males and 7 females; mean age, 32 years; age range, 23–48 years) participated in the fMRI study. All participants were recruited on a volunteer basis, without monetary or other compensation, and no reward was given for their task performance. All subjects were without a history of head injury, learning disability, or neurologic or psychiatric illness, as verified by a semi-structured clinical interview modified from the Structured Clinical Interview from the *Diagnostic and Statistical Manual of the American Psychiatric Association, 4th Revision* (DSM-IV) (16), and had normal or corrected-to-normal visual acuity. After having the experimental protocol explained to them, all participants provided written informed consent for participation in the study as approved by the Wayne State University School of Medicine Human Investigations Committee.

Task Design

The study design was adapted from the "high-motivation" GKT task using playing cards described by Langleben and colleagues (12) (Fig. 1). At the start of the experiment, before scanning began, each subject received the task instructions and was shown the workstation that would be used to analyze the subject's fMRI data in real time, using the TurboFIRE software (13–15). Example scans of previous participants made during the task were displayed on the workstation screen, and subjects were informed that their brain activation would be monitored by the research team while they performed the task in the scanner. Although we used TurboFIRE to monitor brain activation in real-time, the number of trials conducted in this pilot study did not have adequate statistical power for formal data analyses. Subjects were given a response pad and told that their button-press responses would also be monitored while they performed the task in the scanner. In order to make the task simulate a "real-life" experience, each subject was given two playing cards—the 5 of Clubs (5♣) and the 2 of Hearts (2♥)—and was asked to briefly study these cards and then place them

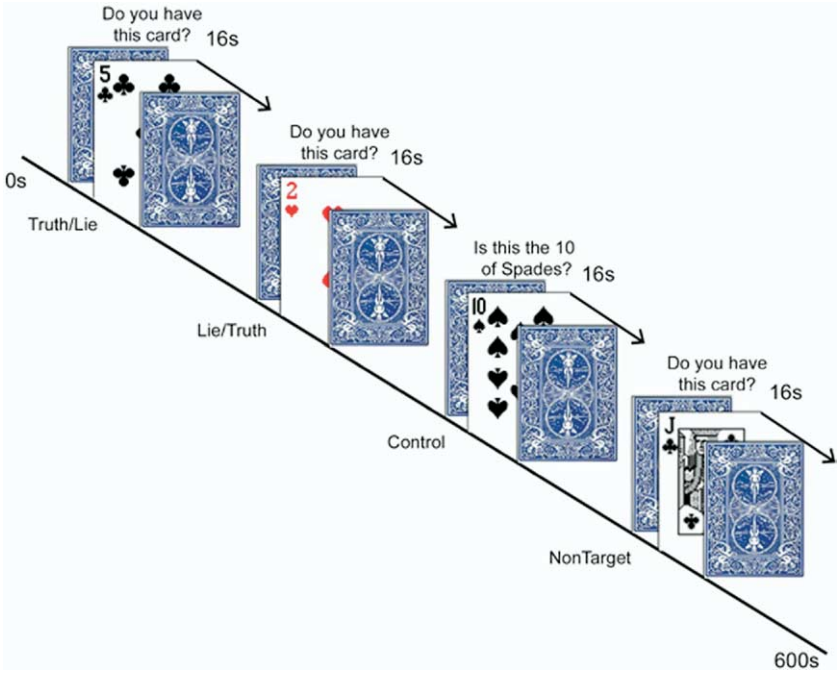

Figure 1. Exemplary segment of the behavioral paradigm for deception. Face card stimuli are presented as 8-second trials (lie, truth, control, and non-target) followed by an 8-second interstimulus interval.

in the subject's pocket for the duration of the scan. Subjects were told that they would be asked to lie about possessing one card and to tell the truth about the other, indicating their responses by button-pressing (thumb = "No", index finger = "Yes"); this assignment was counterbalanced across subjects such that half were instructed to lie about the 5 of clubs and half were instructed to lie about the 2 of hearts. This 2-card design was implemented so that the subject, when asked about a card in the subject's possession, had to make a Yes/No decision, without any object-recognition or card-specific (ie, color or number) effect. While in the scanner, subjects were presented with playing cards as separate events within four different categories of cards/events which prompted four different responses: 5♣ (lie/truth), 2♥ (truth/lie), 10 of Spades (10♠; control), and random cards from the rest of the 49-card deck (non-target responses). Screens with the lie, truth, and non-target cards were accompanied by the question, shown above each card, "Do you have this card?" while the screen for the control card carried the question, "Is this the 10 of spades?" The control and non-target cards were intended to promote alertness and attention to the task and to minimize repetition of the lie-truth cards, while the inclusion of the control card forced subjects to read the question posed above all cards rather than provide indiscriminate, automatic "No" responses. For example, if a subject was instructed to lie about the 5♣, then the correct responses for each card type would be as follows: 5♣ = No; 2♥ = Yes; and 10♠ = Yes. Cards other than the 5♣, 2♥, or 10♠ were to be given "No" responses.

On each imaging run (of 2 total runs), subjects saw randomized presentations of 38 separate trials of lie, truth, control, and non-target cards. Each card was presented for 8 seconds, followed by an 8-second interstimulus interval during which the reverse side of the card was shown. Stimuli were presented via MR-compatible LCD goggles (Resonance Technology Inc., Northridge, CA), and button-press responses were recorded using Presentation software (Neurobehavioral Systems, Inc., Albany, CA). It should be noted that in contrast to the task developed by Langleben and colleagues (12), the subjects in our study had actual possession of the test cards, were told to lie about either the 5♣ or 2♥ and received no financial reward or punishment for their performance. They were told that a research investigator blinded to the assignment of truth/lie cards would monitor the accuracy of their button-press responses and their brain activity with real-time fMRI technology (TurboFIRE). In our attempt to simulate a polygraph-like environment, we told subjects that their performance and brain responses were being monitored closely during the course of the experiment.

Table 1
Brain Foci of Activity Related to Voluntary Deception

Brain Region (Brodmann Area)	Lie > Truth					
	Side	X	y	z	k	Z Score
Ventrolateral prefrontal cortex (BA45/47)	L	−54	12	4	75	4.60
	L	−48	16	−6	87	4.05
	L	−52	16	16	9	3.57
	R	60	14	−2	64	3.49
Superior temporal sulcus (BA21/22/37)	R	66	−44	−4	392	4.22
	L	−58	−56	8	15	3.36
Dorsal medial prefrontal cortex (BA8)	B	−6	40	50	391	3.73
Dorsolateral prefrontal cortex (BA9)	R	56	18	36	40	3.65
	L	−36	16	42	60	3.63
Angular gyrus (BA39)	L	−44	−66	14	88	3.58
Supramarginal gyrus (BA40)	L	−44	−26	32	11	3.46
	Lie > Control					
	Side	x	y	z	k	Z Score
Ventrolateral prefrontal cortex (BA45/47)	L	−42	22	−22	286	4.27
	R	60	18	18	41	3.63
Superior temporal sulcus (BA21/22/37)	L	−54	−40	2	38	3.87
	R	62	−34	−2	83	3.58
	L	−48	−56	6	18	3.52
Dorsal medial prefrontal cortex (BA8)	B	−2	20	50	93	3.36
	B	−4	36	50	17	3.22
Dorsolateral prefrontal cortex (BA8/9)	L	−40	4	44	31	3.35

Data shown represent clusters of activation of > 5 contiguous voxels with local maxima of $t > 3.85$, $P < .001$ uncorrected. All values reflect $P < .05$ corrected for multiple comparisons across a small volume of interest. For each maximal activation focus per cluster, laterality, coordinates, k (number of voxels in the cluster), and Z scores are provided. Coordinates are defined in MNI (Montreal Neurologic Institute) stereotactic space in millimeters: x > 0 signifies a position to the right of the midsagittal plane, y > 0 signifies a position anterior to the anterior commissure, and z > 0 signifies a position superior to the plane of the anterior commissure-posterior commissure (AC-PC).

MRI Acquisition

The subjects were scanned with a 4-T MedSpec MRI scanner (Bruker, Ettlingen, Germany) on a Siemens Syngo platform (Siemens Medical Systems, Erlangen, Germany) with a standard RF coil. After a T_1-weighted, high-resolution anatomical scan, fMRI data were acquired through single-shot multi-echo echoplanar imaging (EPI) (17,18) with 7 evenly spaced TEs ranging from 11–78 ms (TR = 2000 ms; FOV = 192 mm; 32 x 32 matrix; 16 slices; 6-mm slice thickness; 0.6-mm slice gap; flip angle = 90°) (17). Slices were oriented axially or nearly axially along the AC–PC line at the level of the amygdala.

fMRI Data Analysis

Data sets from all 14 subjects met our criteria for high quality and scan stability with minimum motion correction (< 2 mm displacement in any one direction), and were subsequently included in fMRI analyses. Image processing and data analysis was done with the statistical parametric mapping software package SPM99 (Wellcome Department of Cognitive Neurology, London; www.fil.ion.ucl.ac.uk/spm). Standard pre-processing was applied, comprising slice-time correction, realignment, and spatial normalization to the Montreal Neurological Institute (MNI) high-resolution T_1 template. Images were resampled into this space with 2-mm isotropic voxels, and were smoothed with a gaussian kernel of 6 mm full-width at half-maximum to minimize noise and residual differences in gyral anatomy, resulting in an effective spatial resolution of 12.8 x 14.4 x 14.9 mm. Each normalized image was bandpass-filtered (high-pass filter = 32 seconds) to remove low-frequency noise.

For the statistical parametric mapping (SPM) analysis, a general linear model was applied from which statistical inferences were based on the theory of random gaussian fields, and changes relative to the experimental conditions were modeled by convolution with the canonical hemody-

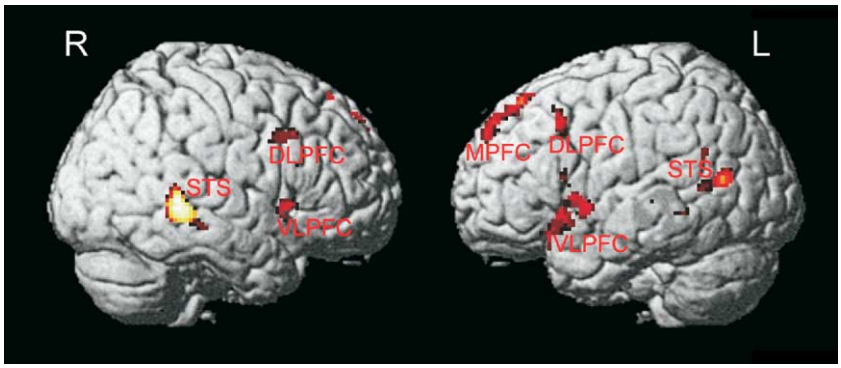

Figure 2. Group random effects SPM of the contrast lie > truth ($P < .001$ uncorrected) overlaid on a 3D canonical MNI brain rendering, showing bilateral activation in the dorsal medial prefrontal cortex (MPFC), dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), and superior temporal sulcus (STS) associated with deception. For maximum foci, see Table 1.

namic response function (HRF) in order to approximate the activation patterns (19). Statistical parametric maps (SPMs) representing the association between the observed time series (eg, blood-oxygenation-level-dependent [BOLD] signal) and one or a linear combination of the regressors were generated for each subject. Within-subject contrasts were derived for brain activity related to the following comparisons: lie > truth, lie > control, truth > lie, and truth > control. These contrast images were then entered into a one-sample *t*-test across the 14 subjects in a second-level, random-effects analysis to allow for inferences applying to the general population (20). This produced statistical parametric maps of the *t* statistic at each voxel, which were subsequently transformed to the *Z* distribution. From voxel-wise comparisons, activation foci were considered significant in regions in which we had an *a priori* hypothesis (ACC, MPFC, DLPFC, VLPFC), and whose activation surpassed a height threshold of $P < .001$ uncorrected ($t > 3.85$), with an extent of at least 5 contiguous voxels. These thresholds are commonly applied in the literature, and were intended to strike a balance between rates of type I and type II error. Reported activations outside these *a priori* regions had to exceed a threshold of $P < .05$, corrected for multiple comparisons. We also analyzed activations within regions for which we had an *a priori* hypothesis by extracting the raw time courses of BOLD signals for lie and truth trials (expressed as percentage signal changes from Control trials, normalized to the start of each trial) from functionally derived local maxima (peak voxel of activation) for each subject, using MarsBar software (http://marsbar.sourceforge.net).

RESULTS

Behavioral Results

The rates of correct responses were 97.5% for the lie trials, 99.2% for the truth trials, and 100% for the control trials; the overall mean rate of correct responses for all trials was 98.0%. There was no significant difference in error rates between lie and truth conditions (*t*-test, $P > .1$). For the lie, truth, and control trials collectively, no subject made more than 4 mistakes across the two imaging runs. In post-scan debriefing, subjects denied noticing anxiety about the lie trials, but most (10 of 14) reported that they felt some anxiety when thinking about whether their response would be detected by changes in their brain activity (eg, performance anxiety), suggesting that our real-time fMRI protocol successfully engendered some aspects of a polygraph-like experience (2,3).

Imaging Results

The lie > truth and lie > control contrasts yielded nearly identical results (Table 1). Relative to both truth and control trials, lie trials were associated with increased activity in bilateral VLPFC (Brodmann Area [BA] 45/47), bilateral posterior superior temporal sulcus (BA 21/22/37), bilateral dorsal MPFC (BA8), and bilateral DLPFC (BA8/9) (Figs 2 and 3a). The lie > truth comparison also yielded activations within angular (BA39) and supramarginal gyri (BA40). Among these regions, activation of the MPFC (BA8/9) was detected in individual analyses in all 14 of the study subjects (Fig 3**a**). Neither the truth > lie nor truth > control contrasts produced a significant BOLD signal change in any brain area at the *a priori* sig-

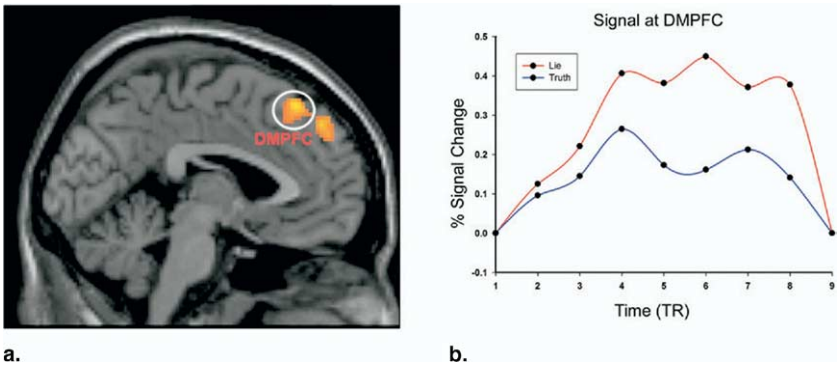

Figure 3. **(a)** Group random effects SPM of the contrast lie > truth ($P < .001$ uncorrected) overlaid on a sagittal section (x = −2) of an MNI canonical brain, showing activation in the dorsal medial prefrontal cortex (DMPFC). **(b)** Time-course of BOLD signal change within the DMPFC for Truth and Lie trials, relative to Control, averaged across all subjects. For maximum foci, see Table 1.

nificance threshold. The time-course extraction of the BOLD signal change from the control condition showed a greater activity during lie vs. truth trials (averaged across 14 subjects), peaking from approximately 8–12 seconds after the start of the trial (Fig 3**b**).

DISCUSSION

This trial-related fMRI was designed to further isolate the neural correlates of deception by using novel real-time fMRI technology in order to simulate a polygraph experience. All subjects performed the study task well, and although they denied experiencing anxiety about lying, the subjects did generally report anxiety about their performance and about whether consequent changes in brain activity would be observable by the study investigators. Using a modified version of the GKT, we observed specific activation of the dorsal MPFC, the VLPFC, and the right STS during lying, relative to both truth and control trials. These findings support the findings in prior functional anatomical studies of deception, and support the notion that discrete prefrontal cortical regions play an important role in the generation of lies, the suppression of truth, or both.

Before discussing our findings, we note several limitations of the present study. First, we did not collect psychophysiological (eg, skin conductance, heart rate) or event-related–potential data during fMRI scanning, which would have allowed us to examine whether any association exists between the BOLD response and other central and peripheral physiologic measures. Because of problems with the button-press hardware used in the study, we were also unable to collect reliable response-time measures during scanning; we did not obtain quantitative measurements of subjects' anxiety (eg, numerical ratings) related to the study task. Therefore, this study should be regarded as preliminary and as only approximating a polygraph environment. Although our post-scan debriefing suggested some success in this regard, additional work is required to substantiate our findings. Our paradigm was relatively constrained in that it allowed only 2 responses for each presented card stimulus, and consequently may have engaged other cognitive processes such as response reversal (alternation learning), which was previously shown to also activate VLPFC (21). Third, the paradigm used in our study was limited in that it tested only one type of lie (eg, withholding information about an item in one's possession), and ignored several other forms and aspects of deception, as recently explored by Ganis and colleagues (8). Fourth, and perhaps most importantly, our study had a major limitation in not reflecting "real-life" instances of deception. Although we minimized monetary reward and autobiographical details in our paradigm in order to isolate the act of lying, the scenario we created may have lacked interest, elements of guilt, personal gain, and the psychological stress that often accompany the generation and enactment of a lie. Future studies will be needed to address these important issues.

Our results are consistent with prior observations that the DLPFC, VLPFC, and DMPFC are specific neural correlates of the act of deception. Lying about one's own historical events has been associated with bilateral activation of the VLPFC (BA47) and DMPFC (BA8)(6), while faking memory loss has been associated with activation of

the DLPFC, VLPFC, and DMPFC (7). In their prior study in which they used a playing-card version of the GKT, Langleben and colleagues reported activation within the superior frontal gyrus/dMPFC (BA8), but not the DLPFC or VLPFC (12). Anatomically, these regions are closely connected and share local projections (22). At a functional level, they have been collectively implicated in a variety of cognitive tasks, but most commonly in higher executive functions such as working memory, planning, task switching and updating, and cognitive control (4,23). It has been particularly conjectured that this circuit is critical for inhibitory control over pre-potent responses (24,25) and for monitoring conflicting response tendencies (23,26,27). Such a process would match well with deception if the latter can be conceptualized as intentional negation or withholding of truth, which is considered to be a pre-potent, learned response (28).

Because the act of deception involves complex cognitive processes, and cognitive–emotional interactions, including processes such as response inhibition, cognitive control of behavior, and executive function, it is not surprising that brain regions that subserve these processes are also engaged during voluntary deception. Lesions of these regions impair the extinction of conditioned responses in rodents (29), and performance on conditional response tasks, such as go/no-go paradigms, and result in preservative errors in non-human primates (30,31); similarly, lesions and/or dysfunction in ventral and dorsal prefrontal cortex in humans are associated with impulsivity, perseveration, and failure to control pre-potent response tendencies (30,32–34). It would be of great interest to confirm that activation of these frontal cortical regions represent the neural signatures of deception, and that fMRI can be used to reliably test for the activation of deception in single participants in single trials, and on a test–retest basis. These questions are currently being investigated in our laboratory with real-time fMRI (13–15).

We observed that the MPFC (BA8/9) was consistently activated during Lie trials across all subjects, suggesting that it may be a neural signature for the generation of lies. The dorsal MPFC is considered to be part of the "paralimbic" heteromodal association cortex with strong connections to both the DLPFC and VLPFC, but also to the limbic cortex (ACC, amygdala) (22). Although it is not often activated by pure cognitive studies (4), this region is commonly activated across several types of emotional tasks (5). We attempted to simulate the stress of a polygraph experience by informing subjects that their brain activation would be monitored closely in real time. Behavioral results revealed that subjects felt anxious while in the MRI scanner, suggesting that certain elements of lying involving emotion/anxiety (eg, fear of being detected) may have been evoked (1). Also, it is possible that subjects are instinctively aware that the act of lying is wrong and/or immoral on the basis of societal and cultural expectations (28), in which case it would be likely to evoke some affective and/or somatic response that may not be consciously perceived, such as the responses detected by conventional polygraphy (2). Interestingly, the dorsal MPFC has been implicated in awareness of one's own emotions (35,36) and in self-referential processing and evaluating the intentions of others (eg, theory of mind) (36–38), and the magnitude of its activity has been associated with subjective and physiological arousal (39,40). All of these emotional and meta-cognitive processes characterize some elements of lying or withholding of the truth.

Unexpectedly, we also observed activation of the posterior superior temporal sulcus (STS) during deception (lie) relative to both truth and control trials. This region has been observed to be active during feigned memory loss/malingering (7), but not in other studies of deception (6,12). Although its activation is not often reported in studies of cognitive control and/or response inhibition (4), the STS has been conjectured to be critical to social cognition, and particularly the formation of social judgments (41). Activation of the STS has also been observed during explicit judgment of trusthworthiness in faces, and its activation was greater in an untrustworthy- versus-trustworthy contrast. Additionally, the STS has been implicated in intention detection and theory of mind (37,38). Taken together with the results of our study, these findings suggest that the STS may not only have a role in social appraisals, but also in the complex evaluation of intention, trust, and cooperation, all of which are components of the interaction between the perpetrator and the victim of deception. Although this is an intriguing possibility, these initial findings warrant caution for investigation in future studies.

It is important to note that we did not detect activation of the anterior cingulate gyrus during deception, a finding consistent with that in some prior studies of deception (8,12), but not in others (6,7). Besides the prefrontal regions noted above, the dorsal ACC has also been implicated in neurobiological models of cognitive control, inhibition of competing/pre-potent responses, and mediation of conflict (23,24,26,30). The

present study employed a deception paradigm similar to the one used by Langleben and colleagues, who observed activation of the dorsal ACC (in addition to the DMPFC) during voluntary lying (12). Subjects in that study were told that they would win a $20 prize if they "succeeded in concealing the identity of their card from a 'computer'," but would forfeit the $20 if they "lied about any other card other then the one hidden in their pocket," thereby giving them a monetary incentive to lie well and to perform the study task accurately under scrutiny. Such a task involves reward and motivation, which have been shown to consistently activate the ACC (42,43). Therefore, it is possible that the deception task used in our study might not activate the ACC, since it lacked any tangible reward or motivation for performance. Alternatively, our conservative random effects analysis, small sample of subjects and lie trials, or inherent individual variability may have influenced the detection of activation of the ACC during lying. This may be plausible for two reasons: (1) when the uncorrected significance threshold was lowered to $P < 0.05$, the foci of activation extended inferiorly to portions of the ACC, specifically BA32, and (2) 9 of 14 subjects in our study showed activation of the ACC in the lie > truth contrast. Future studies are needed to clarify the involvement of the ACC in deception.

In conclusion, we used novel real-time fMRI technology to simulate the polygraph experience (eg, provoke performance anxiety) in order to ascertain the neural correlates of lying. Our findings demonstrate engagement of the ventrolateral, dorsolateral prefrontal, and dorsal medial prefrontal cortices, in the act of deception, all of which are regions implicated in earlier studies of deception. The consistency of these findings across a number of studies of deception suggests that fMRI, coupled with psychophysiologic techniques, may have potential as a reliable lie-detection device.

ACKNOWLEDGMENTS

We gratefully acknowledge Oliver Speck and Juergen Hennig of the University of Freiburg, Germany, for their work in developing the multi-echo EPI sequence for our 4T MR scanner, and Stefan Posse of Wayne State University and the University of New Mexico for the use of TurboFIRE software. This work was supported by the State of Michigan through the Joe F. Young Sr. Foundation, and by the National Defense University.

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