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Neuroanatomical Correlates of Malingered Memory Impairment: Event-related fMRI of Deception on a Recognition Memory Task

Jeffrey N. Browndyke¹, James Paskavitz², Lawrence H. Sweet³, Ronald A. Cohen³, Karen A. Tucker¹, Kathleen A. Welsh-Bohmer^{1,4}, James R. Burke⁴, and Donald E. Schmechel⁴

¹Duke University Medical Center, Department of Psychiatry & Behavioral Sciences, Division of Medical Psychology, Durham, North Carolina ²University of Massachusetts Memorial Healthcare, Department of Neurology, Worcester, Massachusetts ³Brown University Medical School, Department of Psychiatry & Human Behavior, Providence, Rhode Island ⁴Duke University Medical Center, Department of Medicine, Division of Neurology, Durham, North Carolina

Abstract

Primary objective—Event-related, functional magnetic resonance imaging (fMRI) data were acquired in healthy participants during purposefully malingered and normal recognition memory performances to evaluate the neural substrates of feigned memory impairment.

Methods and procedures—Pairwise, between-condition contrasts of neural activity associated with discrete recognition memory responses were conducted to isolate dissociable neural activity between normal and malingered responding while simultaneously controlling for shared stimulus familiarity and novelty effects. Response timing characteristics were also examined for any association with observed between-condition activity differences.

Outcomes and results—Malingered recognition memory errors, regardless of type, were associated with inferior parietal and superior temporal activity relative to normal performance, while feigned recognition target misses produced additional dorsomedial frontal activation and feigned foil false alarms activated bilateral ventrolateral frontal regions. Malingered response times were associated with activity in the dorsomedial frontal, temporal, and inferior parietal regions. Normal memory responses were associated with greater inferior occipitotemporal and dorsomedial parietal activity, suggesting greater reliance upon visual/attentional networks for proper task performance.

Conclusions—The neural substrates subserving feigned recognition memory deficits are influenced by response demand and error type, producing differential activation of cortical regions important to complex visual processing, executive control, response planning, and working memory processes.

Keywords

fMRI; MRI, Functional; forensic psychiatry; deception; malingering; Deficits, Memory

INTRODUCTION

Symptom validity testing (SVT) procedures perform reasonably well in distinguishing patients with true cognitive deficits from those intent upon deception (see Bianchini et al. [1] for

review). Due to this clinical utility, SVTs have become standard practise for clinicians, particularly those involved in forensic settings, to assist in the delineation of cognitive change due to organic factors from those fueled by functional etiologies or the prospect of secondary gain. But, what do these tests reference in relation to neural substrates mediating SVT performance or the intentionally poor task effort necessary to feign cognitive impairment? Surprisingly, there is very little research with direct applicability to this question [2], but parallels can be found in efforts attempting to appropriate for event-related potential (ERP) techniques and functional magnetic resonance imaging (fMRI) what has heretofore been the purview of the polygraph.

Early in the 20th century it was coincidentally noted by Jung and Wertheimer that delayed response times could be used to distinguish innocuous stimuli from those with high emotional valence, thereby providing a method for the detection of guilt or deceptive response suppression to stimuli with latent psychological impact (i.e., 'complexes' as defined by Jung) [3]. These early observations of deception-associated reaction time delays hinted at the presence of hidden additional cognitive processes at work when one is attempting to deceive and eventually led to gross interrogation techniques employed prior to the invention of the polygraph [4]. Reaction times have since fallen into some disfavor as reliable interrogation indices of deception, but a resurgence of interest has resurfaced in their possible use as ancillary SVT variables. Vendemia et al. [5] and Willison et al. [6] have both demonstrated consistently greater response times during deceptive cognitive task performances, the application of which is now starting to filter into neuropsychological assessment measures as they become increasingly computerized. While these subtle reaction time increases during cognitive performance may lead to useful clinical or forensic practice applications, they inform little about the neural substrates behind the deceptive behavior associated with the response delays. The same can be said of polygraphic measures deception based upon changes in autonomic arousal, which are additionally known to be fraught with high false positive detection rate problems [7].

One window into the internal cognitive processes governing deception can be found in the application of event-related potential (ERP) techniques, which reflect the direct electrophysiological characteristics and gross topographical location of information processing events rather than indices of simple arousal. ERPs were first proposed for the detection of deceptive behavior in the mid-1980s [8,9], and since that time, numerous research efforts have examined aspects of the evoked response during covert deception. ERP components, such as the N400 [10], late positive parietal P300 [8,9,11–14], and early medial frontal negativities (MFN) [15], have all demonstrated with some success the ability to distinguish neural signatures associated with the suppression of concealed knowledge. P300 indices of deception are posited to reflect automatic posterior parietal cortical activity associated with the prior visual recognition of presented stimuli; neural activity that is not as strongly elicited if an individual does not have prior recognition or knowledge of a crime-related stimulus [13]. Similar reasoning is associated with deception-related changes in the N400 component [10]; whereas, larger MFN components have been linked to response monitoring and cognitive conflict present during deceptive behaviour [15].

The neuroanatomical correlates of deception, as detected by fMRI techniques, were first reported by Spence et al. [16] who, like subsequent researchers, were intent upon capturing neural activity specific to lying, thereby addressing persistent confounds of traditional polygraphy techniques (i.e., distinguishing innocuous anxiety from guilt), while simultaneously providing greater spatial resolution and localization data than what ERP-based efforts are able to afford. Spence et al. discovered activation in ventrolateral, dorsomedial, and dorsolateral prefrontal cortices (VLPFC, DMPFC, and DLPFC, respectively), as well as the inferior parietal lobule (IPL), during deceptive relative to normal responding for both auditory and visual stimuli. These regions have been commonly associated with deceptive responding

in fMRI studies that followed and appear to reflect the generation and inhibition of responses, increased working memory load, meta-cognition of task performance, and monitoring of social cues necessary to deceive others [17–26]. With the exception of two studies to be discussed below [17,23], the bulk of neuroimaging and ERP investigations into deceptive behavior have focused upon the neuroanatomical correlates involved in the suppression of prior knowledge by employing methodological variations of the Guilty Knowledge Test (GKT) [27]. However, application of the results of these GKT-related investigations is somewhat limited when attempting to characterise the neuroanatomical correlates of feigned cognitive impairment, as the suppression of prior knowledge is only one type of behavior involved in malingering on neuropsychological assessment measures.

To date, two functional imaging studies have examined neural activity under task situations that best approximate the malingering of cognitive deficits; specifically, purposefully poor performance on visual memory tasks, which by virtue of the task situations resulted in responses associated with the suppression of prior knowledge for task target stimuli (i.e., malingered omissive memory errors) and confabulatory-like false endorsement of task foil stimuli (i.e., malingered inclusive memory errors). [17,23]. Both studies reported regions for deceptive responding similar to prior investigations of lying, namely greater DLPFC and DMPFC activity, and in the case of block-averaged BOLD signal [23], greater IPL activity during the feigning of memory impairment. However, these investigators dictated the conditions of discrete deceptive responding; did not provide a risk or reward scenario approximating those motivated by secondary gains; and averaged the neural activity associated with task deception. In contrast, we propose the use of event-related fMRI during proper and malingered performances of a visual object recognition memory task to allow for more direct examination of malingered memory impairment error types relative to normal recognition response counterparts. These response-specific contrasts reflect the discrete neural correlates of task deception involving retrieval suppression and alternate response generation under conditions of stimulus novelty or familiarity relative to proper task performance. Consistent with Abe et al. [17], we hypothesized that deceptive error types may share some functional similarities, such as self-appraisal of performance to maintain deception and working memory load, but they might diverge on neural demands governing response inhibition, effortful retrieval of sensory information for proper performance, and response manipulation.

METHOD

Participants

Participants were recruited from the greater Worcester, MA. area. Project oversight and approval was provided by the University of Massachusetts Memorial Healthcare Institutional Review Board and written informed consent was obtained prior to screening and enrollment. Subject screening was conducted by a board certified neurologist (J.P.) and two clinical neuropsychologists (J.B., L.S.). Nine individuals volunteered to participate, but only seven met normal inclusion criteria or MR safety considerations (e.g., no major medical conditions, lack of neurological, psychiatric, and/or substance abuse history, free of ferrous implants, ability to tolerate closed space, and 20/20 corrected vision). Three male and four female right-hand dominant participants were enrolled (age 33.2 $\pm$ 10.8 yrs.; educ. 14.8 $\pm$ 3.0 yrs.). There were no statistically significant differences in fMRI task performance (i.e., task accuracy and reaction time) as a function of subject demographics.

Procedure

Task stimuli, composed of 2-dimensional black-and-white line drawings of nameable objects from the Test of Memory Malingering [28] and the Snodgrass & Vanderwart visual object

stimuli set [29], were selected to construct a set of 200 unique object stimuli, some of which were similar only in superordinate category membership (i.e., animals, tools, etc.).

Object encoding and recognition task runs were constructed and presented using E-Prime experimental software (Psychology Software Tools, Inc.). Two encoding task runs, each composed of 50 items, and two recognition task runs individually composed of 50 targets and 50 novel foils were presented in the following order; (1) object encoding, (2) normal recognition, (3) object encoding, and (4) malingered recognition. There were 5 min. retention intervals between encoding and recognition runs. All stimuli were presented for 2 sec. against a stark white background, followed by a static visual crosshair inter-stimulus interval of varying duration (mean duration 3 sec.).

Task instructions for encoding runs stipulated that participants were to learn and remember all object pictures for later recognition. Recognition run instructions asked participants to respond by button press whether the current stimuli was one seen in the immediately preceding encoding run ("old") or a novel foil ("new"). Additional instructions were given prior to the malingered recognition run telling participants to respond as though they were slightly injured in an automobile accident and to feign a memory impairment for financial gain, but not obviously so or risk detection and punishment for fraud. Prior to these additional instructions, participants were unaware of the switch in task demands and were not coached on how to feign impairment. After the procedure, participants were asked about any strategies employed during their performances of both recognition runs.

All task instructions and stimuli were presented to the participants by back-projecting stimuli on a screen placed at the foot of the scanning gurney to a mirror built into the RF head coil positioned above the subject and angled out the magnet bore. A two-button MR-compatible response box (Cedrus Corp.) placed under participants' right hand was attached to the presentation computer for recording of participant responses. Responses were recorded during recognition runs and were used to derive task estimates, error rates, and response times.

Analyses

All MR data were acquired with a 1.5T GE Signa-LX scanner using a standard RF quadrature head coil. High-resolution [(256 × 256, 24 cm × 24 cm), 1.5 mm slice thickness, 100 slices], T₁-weighted sagittal plane images were acquired with a SPGR sequence (TR/TE = 22/20 msec, $\Phi = 22^\circ$) for anatomic localization of functional activation. Functional data were acquired using a GRE-EPI sequence (TR/TE = 3000/60 msec, $\Phi = 90^\circ$) with contiguous axial slices positioned to cover the entire brain volume [(64 × 64, 24 cm × 24 cm), 5 mm slice thickness, 27 slices]. GRE-EPI voxel size = 3.75 × 3.75 × 5 (~ 70 μ L).

Image preprocessing was performed using Statistical Parametric Mapping software implemented in Matlab (SPM2; Wellcome Institute, London, UK). After discarding initial volumes to allow for signal stabilization, EPI images were slice-time and motion-corrected; spatially normalized via 5th degree B-spline interpolation to the Montreal Neurological Institute (MNI) EPI template; and resampled to 2 mm³ isomorphic voxels. The resulting normalized data were then spatially smoothed using an 8 mm FWHM Gaussian kernel. Event-related signal associated with stimuli onset was modeled using 128 high-pass filter and autocorrelation correction to a canonical hemodynamic response function (HRF) with time derivative and movement regressors. Both normal encoding runs were modeled in a similar fashion.

Single-subject, fixed-effect results were entered into group-level, non-parametric paired *t*-test permutation analyses (SnPM) [30] of a priori between-condition recognition response contrasts. Contrasts were conducted to detect differences between-conditions for recognition

memory task responses with shared stimulus type presentation (i.e., activity that varies as a function of truthfulness or deception while holding stimulus familiarity or novelty constant). As such, prior target stimuli contrasts reflect dissociable brain activity associated with normal recognition hits (NH) and malingered recognition misses (MM), while novel foil stimuli contrasts illustrate dissociable activity associated with normal recognition correct rejections (NCR) and malingered recognition false alarm errors (MFA). Encoding runs were also compared for differences that could impact interpretation of the recognition run results. Non-parametric interrogation was selected for examination of second-level statistical inference given the conservative nature and general robustness of the procedure with small sample sizes [30]. SnPM permutations were set to the maximum, and proportional scaling was conducted to allow for comparison between conditions/runs. For all second-level, paired t-test permutation analyses, statistical significance was determined via a cluster-wise threshold of $t \geq 3.50$ and a conservative spatial extent of $k_E \geq 40\text{mm}^3$ ($P < .01$, corrected) [31,32].

Non-parametric, multi-subject simple regression analyses were carried out to investigate whether reaction times for each recognition response type were correlated with hemodynamic signal intensity in brain regions noted as being significantly different in our primary contrasts (i.e., NH vs. MM, and NCR vs. MFA). The results from the paired t-test non-parametric permutation analyses were used as inclusive masks, which constrained the regression analyses to only those regions shown to be significantly different between conditions. For these post-hoc, limited small-volume SnPM regression analyses, statistical significance was lowered and established as active voxel groupings with signal intensities SnPM $t_p > 2.80$ and $k_E \geq 10\text{mm}^3$ spatial extent ($P < .05$).

RESULTS

Behavioural

Task performances were significantly different between conditions. Recognition discrimination (i.e., $Pr = \text{Hit}_{\text{rate}} - \text{False Alarm}_{\text{rate}}$) was $0.83 (\pm 0.07)$ for the normal condition and $0.02 (\pm 0.23)$ for the malingered condition ($\underline{S} = -2.37$, $p < .05$). Recognition response bias [i.e., $Br = \text{False Alarm}_{\text{rate}} / (1 - (\text{Hit}_{\text{rate}} - \text{False Alarm}_{\text{rate}}))$] with liberal bias reflecting $Br > 0.5$, was also significantly different between conditions [i.e., mean normal Br $0.59 (\pm 0.11)$; mean malinger Br $0.46 (\pm 0.05)$; $\underline{S} = -2.21$, $p < .05$]. Mean NH and MM response reaction times (msec.) were $875.70 (\pm 95.19)$ and $1297.55 (\pm 212.46)$, respectively ($\underline{S} = -2.37$, $p < .05$), while NCR and MFA mean times were $1045.82 (\pm 131.96)$ and $1495.01 (\pm 284.29)$, $\underline{S} = -2.37$, $p < .05$. Comparison of recognition response reaction times within condition failed to yield any significant differences (i.e., NH vs. NCR, and MM vs. MFA).

Recognition discrimination was in the markedly impaired range for the malingered condition and superior for the normal condition. A significant, though less disparate, difference was noted for response bias, and malingered reaction times were uniformly longer, but less than 0.5 sec., than normal recognition response counterparts.

Neuroimaging

No suprathreshold differences were detected between the two normal encoding runs. These runs were subsequently combined, and significant voxel-wise ($p < .001$) object encoding-related maxima were observed in the right (MNI $44_x, -62_y, -20_z$; $t = 10.05$, $2704 k_E$) and left (MNI $-38_x, -70_y, -18_z$; $t = 8.63$, $1389 k_E$) fusiform regions (BA 37 / 19), left precuneus (BA 18 / 31; MNI $-22_x, -76_y, 14_z$; $t = 5.66$, $120 k_E$), right cuneus (BA 7; MNI $24_x, -80_y, 32_z$; $t = 4.73$, $155 k_E$), and right parahippocampal region (BA 28; MNI $24_x, -22_y, -14_z$; $t = 4.56$, $38 k_E$).

Normal Target Recognition vs. Malingered Errors of Omission—Dissociable activation associated with prior encoding stimulus presentation (i.e., recognition task target stimuli) was observed between conditions in the right lingual and cingulate regions, and left cuneus and precuneus for normal target hits (NH) relative to their malingered target miss (MM) counterparts (see Table 1 & Figure 1). The most significant NH > MM contrast clusters were in the right lingual and left precuneus. Conversely, deceptive target misses (MM) were associated with large clusters of activity in the right IPL and left DMPFC. Additional, though smaller, significant clusters were observed in the right middle temporal gyrus and posterior cingulate and left middle frontal gyrus, supramarginal, and amygdalar regions.

Normal Foil Correct Rejection vs. Malingered Errors of Inclusion—Recognition task responses to novel foil stimuli (i.e., NCR and MFA) produced dissociable results between conditions. Correctly rejecting novel foils (NCR) was associated with large clusters of activity in the left lingual and cuneus regions. NCR-associated activation was additionally observed in the left middle temporal gyrus and posterior cingulate regions. Malingered errors of inclusion (MFA) were notable in the production to left and right VLPFC activation; the right cluster being significantly larger in extent than the left. Additional large MFA activation clusters were observed in the right middle temporal gyrus and a smaller region of activity in the right middle occipital gyrus region.

Associations with Reaction Time by Recognition Memory Response Type—NH and NCR reaction times were neither positively nor negatively correlated with hemodynamic activity in regions noted as being significantly different in the primary task condition contrasts. MFA reaction time was positively associated with activity in the right middle temporal gyrus (BA 39; MNI 52_x, -58_y, 10_z; $t = 3.20$, 10 k_E); one of the regions noted as being significantly more active during deceptive endorsement (i.e., malingered inclusive memory errors) than normal responding to novel foil recognition memory stimuli. MM reaction time was positively associated with activity in the right and left supplementary motor area (BA 32; MNI -8_x, 12_y, 42_z; $t = 3.42$, 49 k_E & BA 32; MNI 6_x, 16_y, 42_z; $t = 3.22$, 25 k_E, respectively) and right inferior parietal lobule (BA 40; MNI 52_x, -42_y, 24_z; $t = 4.07$, 99 k_E); regions noted as being significantly more active during deceptive denial of recognition for previously presented memory stimuli (i.e., malingered omissive memory errors).

DISCUSSION

Our results are largely consistent with other deception research efforts examining lying, which tend to show significant activation of the posterior MTL, IPL, DLPFC, DMPFC, and VLPFC during deceptive responding. However, similar to the Abe et al. [17] and Ganis et al. [18], we were specifically interested in the neural correlates of feigned cognitive impairment and found that some of the aforementioned regions active during lying behaviour may be specific to the type of feigned impairment response, which in turn may be dictated by stimulus type and associated response demands (i.e., stimulus familiarity vs. novelty). Our comparisons of deception-associated event-related fMRI data suggest that on cognitive task or situations involving visual episodic memory, the DLPFC, DMPFC and VLPFC correlates of deception may be further refined depending upon whether the deceptive response primarily requires response production or suppression in the context of stimulus novelty (i.e., malingered inclusive memory errors) or familiarity (i.e., malingered omissive memory errors). Malingered inclusive responding to novel recognition memory stimulus items, requiring deceptive response production and false item endorsement, engaged the VLPFC (BA 47/13); whereas, DLPFC (BA 9) and DMPFC (BA 32) activity was associated with deceptive omission and response suppression to familiar recognition memory stimuli. This latter association is reinforced by the positive association between DMPFC activity and increased response times for malingered target miss errors. DMPFC regions proximal to the anterior cingulate and

supplementary motor area (SMA) are known for their role in mediating response conflict, inhibition, and complex motor planning. Similar deception-related response conflict processes have been observed in association with the medial frontal negativity (MFN) ERP component generated in this region [15]. Additionally, the observed association between reaction time increases during these malingered omissive errors and BOLD activity in the DMPFC suggests that this area may be one of the regions involved in the malingered-related reaction time increases noted by Vendemia et al. [5] and Willison et al.[6].

In addition to regional cortical activity differing as a function of deceptive response type, our event-related fMRI investigation is one of two investigations to observe greater activation in the normal response condition relative to deception [21]. Similar to that investigation, our findings of greater dorsomedial parietal and occipital activation during normal recognition may reflect alternate visual object recognition and attentional resources that are otherwise not called into action or are suppressed during purposefully deceptive response modes. Other researchers have posited that the general lack of greater activity for truthful responding relative to deception across studies is evidence that 'truthfulness' is the native, pre-potent state prior to deceptive behaviour [33], but the bases for this pre-potency is unclear, and, in the case of malingered visual episodic memory, our results suggest that the pre-potency may vary with response or task demands. Spence et al.'s [33] suggestion that bilateral VLPFC activity may be necessary to suppress pre-potent truthful response appears only to hold in our data for deceptive errors of inclusion (i.e., malingered foil false alarms). Whether this suggests that the pre-potency described by Spence et al. is tied more strongly to novel stimulus conditions remains unanswered.

The notable posterior MTL/IPL (BA 39/40) activity observed and associated with both deceptive error types is consistent with Lee et al. [23] and Spence et al. [16,33], who attribute this activation to continuous monitoring of prior probabilities and response counting to better feign impairment or lie less obviously. This functional attribution is bolstered by results of our post-procedural debriefing, in which all participants responded that they attempted to gauge the proportion of their true recognition and feigned impairment responses during the malingered condition to make their deception less detectible. Activity in this region was positively associated with the increased reaction times for MM and MFA errors relative to their normal recognition memory counterparts, reinforcing the additional cognitive processing load necessitated by prior probability monitoring.

While this investigation was consistent with findings from prior fMRI deception research efforts, a few limitations should be addressed. While no functional activity differences were found between encoding runs, it is possible that some of the deception-related activity is compounded by proactive interference. The task design required a surprise condition change from normal to malingered recognition, which did not lend itself to a counterbalancing of runs. And, lastly, while helpful in boosting confidence for our between-condition findings, normal performance errors were too sparse to reliably model for comparison with similar, though feigned, errors. Comparisons of this type may determine whether brain activation differences exist between 'honest' memory errors or actual impairment and those purposefully feigned, which, in turn, may serve to improve the sensitivity and specificity of fMRI for malingered detection.

Our results highlight the importance of evaluating not only deceptively omissive responding (i.e., falsely denying prior knowledge), but also responding that may be deceptively inclusive (i.e., falsely endorsing materials for which prior knowledge is not present). Prior neuroimaging investigations of deception have focused almost exclusively on the former behavior given its saliency to law enforcement scenarios in which the detection of guilty knowledge is the target. However, as we have demonstrated, examination of deceptive endorsement may be equally

important, particularly when this sort of responding has the potential to dilute confidence in the neural substrates revealed by simple contrasts of guilty vs. lack of prior knowledge and has been shown to be an effective counter-measure in some ERP-based deception detection efforts [14]. Additionally, using an event-related fMRI methodology, we have demonstrated the presence of response-specific and task dependent anatomical targets for further examination into the neural substrates of feigned cognitive impairment. However, future coincident examination of 'honest' and deceptive memory errors using single-trial type analyses with larger participant samples, both with documented brain injury and feigned impairment, will be necessary to reliably translate these findings of regionally specific malingering differences in fMRI activity to clinical or forensic application.

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REFERENCES

- [1]. Bianchini KJ, Mathias CW, Greve KW. Symptom validity testing: a critical review. *Clin Neuropsychol* 2001;15:19–45. [PubMed: 11778576]
- [2]. Allen MD, Bigler ED, Larsen J, Goodrich-Hunsaker NJ, Hopkins RO. Functional neuroimaging evidence for high cognitive effort on the Word Memory Test in the absence of external incentives. *Brain Inj* 2007;21:1425–1428. [PubMed: 18066945]
- [3]. Wertheimer M, King DB, Peckler MA, Raney S, Schaef RW. Carl Jung and Max Wertheimer on a priority issue. *J Hist Behav Sci* 1992;28:45–56. [PubMed: 11612657]
- [4]. Goldstein ER. Reaction times and the consciousness of deception. *Am J Psychol* 1923;34:562–581.
- [5]. Vendemia JM, Buzan RF, Simon-Dack SL. Reaction time of motor responses in two-stimulus paradigms involving deception and congruity with varying levels of difficulty. *Behav Neurol* 2005;16:25–36. [PubMed: 16082077]
- [6]. Willison J, Tombaugh TN. Detecting simulation of attention deficits using reaction time tests. *Arch Clin Neuropsychol* 2006;21:41–52. [PubMed: 16280230]
- [7]. Lykken DT. Psychology and the lie detector industry. *Am Psychol* 1974;29:725–739. [PubMed: 4451301]
- [8]. Farwell LA, Donchin E. Can the P300 be used in the detection of deception? *Psychophysiology* 1986;23:434. Abstract.
- [9]. Rosenfeld JP, Nasman VT, Whalen R, Cantwell B, Mazzeri L. Late vertex positivity in event-related potentials as a guilty knowledge indicator: a new method of lie detection. *Int J Neurosci* 1987;34:125–129. [PubMed: 3610500]
- [10]. Boaz TL, Perry NW, Raney G, Fischler IS, Shuman D. Detection of guilty knowledge with event-related potentials. *J Appl Psychol* 1991;76:788–795.
- [11]. Farwell LA, Donchin E. The truth will out: interrogative polygraphy ("lie detection") with event-related brain potentials. *Psychophysiology* 1991;28:531–547. [PubMed: 1758929]
- [12]. Mertens R, Allen JJ. The role of psychophysiology in forensic assessments: Deception detection, ERPs, and virtual reality mock crime scenarios. *Psychophysiology* 2008;45:286–298. [PubMed: 17995914]
- [13]. Rosenfeld JP, Reinhart AM, Bhatt M, Ellwanger J, Gora K, Sekera M, et al. P300 correlates of simulated malingered amnesia in a matching-to-sample task: topographic analyses of deception versus truth-telling responses. *Int J Psychophysiol* 1998;28:233–247. [PubMed: 9545659]
- [14]. Rosenfeld JP, Soskins M, Bosh G, Ryan A. Simple, effective countermeasures to P300-based tests of detection of concealed information. *Psychophysiology* 2004;41:205–219. [PubMed: 15032986]

- [15]. Johnson R Jr, Henkell H, Simon E, Zhu J. The self in conflict: the role of executive processes during truthful and deceptive responses about attitudes. *Neuroimage* 2008;39:469–482. [PubMed: 17919934]
- [16]. Spence SA, Farrow TF, Herford AE, Wilkinson ID, Zheng Y, Woodruff PW. Behavioural and functional anatomical correlates of deception in humans. *Neuroreport* 2001;12:2849–2853. [PubMed: 11588589]
- [17]. Abe N, Suzuki M, Tsukiura T, Mori E, Yamaguchi K, Itoh M, et al. Dissociable roles of prefrontal and anterior cingulate cortices in deception. *Cereb Cortex* 2006;16:192–199. [PubMed: 15858160]
- [18]. Ganis G, Kosslyn SM, Stose S, Thompson WL, Yurgelun-Todd DA. Neural correlates of different types of deception: an fMRI investigation. *Cereb Cortex* 2003;13:830–836. [PubMed: 12853369]
- [19]. Kozel FA, Padgett TM, George MS. A replication study of the neural correlates of deception. *Behav Neurosci* 2004;118:852–856. [PubMed: 15301611]
- [20]. Kozel FA, Revell LJ, Lorberbaum JP, Shastri A, Elhai JD, Horner MD, et al. A pilot study of functional magnetic resonance imaging brain correlates of deception in healthy young men. *J Neuropsychiatry Clin Neurosci* 2004;16:295–305. [PubMed: 15377736]
- [21]. Langleben DD, Loughhead JW, Bilker WB, Ruparel K, Childress AR, Busch SI, et al. Telling truth from lie in individual subjects with fast event-related fMRI. *Hum Brain Mapp* 2005;26:262–272. [PubMed: 16161128]
- [22]. Langleben DD, Schroeder L, Maldjian JA, Gur RC, McDonald S, Ragland JD, et al. Brain activity during simulated deception: an event-related functional magnetic resonance study. *Neuroimage* 2002;15:727–732. [PubMed: 11848716]
- [23]. Lee TM, Liu HL, Tan LH, Chan CC, Mahankali S, Feng CM, et al. Lie detection by functional magnetic resonance imaging. *Hum Brain Mapp* 2002;15:157–164. [PubMed: 11835606]
- [24]. Mohamed FB, Faro SH, Gordon NJ, Platek SM, Ahmad H, Williams JM. Brain mapping of deception and truth telling about an ecologically valid situation: functional MR imaging and polygraph investigation--initial experience. *Radiology* 2006;238:679–688. [PubMed: 16436822]
- [25]. Nunez JM, Casey BJ, Egner T, Hare T, Hirsch J. Intentional false responding shares neural substrates with response conflict and cognitive control. *Neuroimage* 2005;25:267–277. [PubMed: 15734361]
- [26]. Phan KL, Magalhaes A, Ziemlewicz TJ, Fitzgerald DA, Green C, Smith W. Neural correlates of telling lies: a functional magnetic resonance imaging study at 4 Tesla. *Acad Radiol* 2005;12:164–172. [PubMed: 15721593]
- [27]. Lykken DT. The GSR in the detection of guilt. *J Appl Psychol* 1959;43:385–388.
- [28]. Tombaugh, T. *Test of Memory Malingering Manual*. MultiHealth Systems; New York: 1996.
- [29]. Snodgrass JG, Vanderwart M. A standardized set of 260 pictures: norms for name agreement, image agreement, familiarity, and visual complexity. *J Exp Psychol [Hum Learn]* 1980;6:174–215.
- [30]. Nichols TE, Holmes AP. Nonparametric permutation tests for functional neuroimaging: A primer with examples. *Hum Brain Mapp* 2001;15:1–25. [PubMed: 11747097]
- [31]. Hayasaka S, Nichols TE. Combining voxel intensity and cluster extent with permutation test framework. *Neuroimage* 2004;23:54–63. [PubMed: 15325352]
- [32]. Forman SD, Cohen JD, Fitzgerald M, Eddy WF, Mintun MA, Noll DC. Improved assessment of significant activation in functional magnetic resonance imaging (fMRI): use of a cluster-size threshold. *Magn Reson Med* 1995;33:636–647. [PubMed: 7596267]
- [33]. Spence SA, Hunter MD, Farrow TF, Green RD, Leung DH, Hughes CJ, et al. A cognitive neurobiological account of deception: evidence from functional neuroimaging. *Philos Trans R Soc Lond B Biol Sci* 2004;359:1755–1762. [PubMed: 15590616]
- [34]. Lancaster JL, Summerlin JL, Rainey L, Freitas CS, Fox PT. The Talairach Daemon: A database server for Talairach Atlas labels. *Neuroimage* 1997;5:S633.

Figure 1. Brain activity differences associated with normal and malingered memory performances
Note: Regions of dissociable event-related fMRI activity for normal and malingered recognition memory responses under shared target and novel foil stimuli conditions (i.e., NH vs. MM and NCR vs. MFA, respectively). SnPM threshold $t \geq 3.50$ and extent $k_E \geq 20$ (40 mm³), $P < .01$ (corrected).

Cluster location, spatial extent, and local maxima of fMRI signal differences between normal and malingered recognition memory responses

Table 1

Contrasts ^a	Activation Locus (<i>Brodmann Area</i>) ^b	SnPM _(t)	k _E	Local Maxima ^c		
				x	y	z
NH > MM	R. Hemisphere	4.10	367	2	-72	-6
	Cingular Gyrus (BA 30)	3.93	23	26	-48	6
L. Hemisphere	Precuneus (BA 7)	5.18	169	-2	-74	46
	Cuneus (BA 19)	4.08	33	-16	-84	34
MM > NH	R. Hemisphere	5.41	720	50	-50	30
	Middle Temporal Gyrus (BA 37)	4.22	111	46	-44	0
L. Hemisphere	Posterior Cingulate Gyrus (BA 23)	4.03	24	10	-28	34
	Anterior Cingulate / SMA (BA 32)	4.76	571	-4	14	38
NCR > MFA	Amygdala / Parahippocampal Gyrus	4.49	33	-32	-2	-22
	Supramarginal Gyrus (BA 40)	4.23	44	-34	-40	32
L. Hemisphere	Middle Frontal Gyrus (BA 9)	3.70	67	-34	30	36
	Lingual Gyrus (BA 19)	4.05	60	28	-78	-4
MFA > NCR	Lingual Gyrus (BA 18)	4.68	540	-26	-76	-10
	Cuneus (BA 19/7)	4.44	153	-16	-84	34
L. Hemisphere	Middle Temporal Gyrus (BA 19/22)	4.04	49	-34	-58	18
	Posterior Cingulate Gyrus (BA 30)	3.74	26	-12	-62	14
MFA > NCR	Middle Temporal Gyrus (BA 39)	5.82	115	58	-58	8
	Inferior Frontal / Peri-insular (BA 47/13)	4.82	131	46	14	-4
L. Hemisphere	Middle Temporal Gyrus (BA 39)	4.21	109	48	-68	28
	Middle Occipital Gyrus (BA 18)	4.08	26	40	-76	-16
MFA > NCR	Inferior Frontal / Peri-insular (BA 47/13)	3.96	45	-42	18	-6

Note: Target stimulus events (NH = normal condition target hits; MM = malingered condition target misses) and foil stimulus events (NCR = normal condition foil correct rejection; MFA = malingered condition foil false alarm).

^a Statistical non-parametric mapping (SnPM) paired t-test contrast of recognition response type x condition [cluster-wise inference, threshold $t \geq 3.50$ and extent $k_E \geq 20$ (40 mm³), $P < .01$ (corrected)].

^b Anatomical and Brodmann area labels based upon 7 mm³ search range of the Talairach Daemon Database [34] using MNI-to-Talairach nonlinear transform coordinates.

^c Montreal Neurological Space (MNI) coordinates.