A cognitive neurobiological account of deception: evidence from functional neuroimaging

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An organism may use misinformation, knowingly (through deception) or unknowingly (as in the case of camouflage), to gain advantage in a competitive environment. From an evolutionary perspective, greater tactical deception occurs among primates closer to humans, with larger neocortices. In humans, the onset of deceptive behaviours in childhood exhibits a developmental trajectory, which may be regarded as ‘normal’ in the majority and deficient among a minority with certain neurodevelopmental disorders (e.g. autism). In the human adult, deception and lying exhibit features consistent with their use of ‘higher’ or ‘executive’ brain systems. Accurate detection of deception in humans may be of particular importance in forensic practice, while an understanding of its cognitive neurobiology may have implications for models of ‘theory of mind’ and social cognition, and societal notions of responsibility, guilt and mitigation. In recent years, functional neuroimaging techniques (especially functional magnetic resonance imaging) have been used to study deception. Though few in number, and using very different experimental protocols, studies published in the peer-reviewed literature exhibit certain consistencies. Attempted deception is associated with activation of executive brain regions (particularly prefrontal and anterior cingulate cortices), while truthful responding has not been shown to be associated with any areas of increased activation (relative to deception). Hence, truthful responding may comprise a relative ‘baseline’ in human cognition and communication. The subject who lies may necessarily engage ‘higher’ brain centres, consistent with a purpose or intention (to deceive). While the principle of executive control during deception remains plausible, its precise anatomy awaits elucidation.

Keywords: lying; deception; executive function; prefrontal cortex; functional magnetic resonance imaging

1. INTRODUCTION

[L]ie...a false statement made with the intention of deceiving... (Chambers 1991)

[D]eception... a successful or unsuccessful deliberate attempt, without forewarning, to create in another a belief which the communicator considers to be untrue. (Vrij 2001)

That deception has long been of salience to human beings is apparent in the religious texts of many civilizations. From the writings of Ptahhotep five millennia ago (Chinweizu 2001), through the Hebrew Old Testament, to later works, humans have been encouraged to be truthful: ‘Thou shalt not bear false witness against thy neighbour’ (Exodus 20: 16, King James version). The presence of such injunctions suggests that humans do indeed bear false witness, not least when they are required to comment upon others. Hence, any consideration of the relationship between the ‘law’ and the ‘brain’ must take account of what humans do when they seek to deceive. Our view is that when humans lie they are probably using some of the ‘highest’ centres of their brains, a proposition that has implications for notions of moral responsibility.

Furthermore, the deception practised in the courtroom or the cell can be seen within a wider context. There are emerging literatures in evolutionary studies (Dunbar 2000; Byrne 2003), normal human child development (Ford 1995) and developmental psychopathology (Sodian & Frith 1992; Hughes & Russell 1993) suggesting that deception is an ability that develops naturally during childhood, and which is ‘normal’. Such behaviours follow a characteristic developmental trajectory (Ford 1995; O’Connell 1998) and are impaired among humans with specific neurodevelopmental disorders (e.g. autism; Sodian & Frith 1992). Hence, there would appear to be an interesting tension between what is supposedly socially undesirable but normal (i.e. telling lies), and that which is said to be commendable but pathological (i.e. total truthfulness). Normal human social interaction may depend upon limited disclosure. Indeed, several authors have pointed out that strictly truthful communication at all times might be rather hard to live with (e.g. Ford 1995; Vrij 2001) and truth itself may be
2. THE USE OF DECEPTION

[One] must know how to colour one’s actions and to be a good liar and deceiver. Men are so simple, and so much creatures of circumstance, that the deceiver will always find someone ready to be deceived.

(Machiavelli 1999, p. 57)

Tactical deception is defined as a behaviour that forms part of the normal repertoire of an animal but which is deployed in such a way that it appears to mislead a conspecific, to the advantage of the index organism (Byrne 2003). Drawing on the available evolutionary evidence, Byrne (2003) speculated that tactical deception probably emerged within the primate lineage some 12 Myr ago, thereby inferring sufficient cognitive capacity among primate species at that time.

Studies of contemporary non-human primates suggest that some form of purposeful deception occurs in those closest to man (in terms of their evolutionary lineage), and that, at the level of species, neocortical volume is related to the frequency of such observed deception (Byrne 2003). ‘Simply knowing the ratio of the brain taken up by the neocortex, divided by the volume of the rest of the brain, enables us to predict 60% of the variance in the amount of deception that is observed in the species concerned’ (Byrne 2003, p. 51).

Why might deception arise within primate colonies? Adenzato and Ardito (1999) suggest that deception facilitates individual autonomy within the constraints of group living. ‘To be able to do what he/she wishes, especially in the face of hierarchical restraint, an organism must be able to mislead others.’ Adenzato and Ardito suggest that deceiving organisms rely upon two cognitive psychological mechanisms: ‘theory of mind’, by which they mean the ability of the organism to infer what others are thinking, and ‘deontic reasoning’, by which they mean an appreciation of social rules and the consequences of their transgression. Hence, it only makes sense to speak of ‘deception’ among primates if the animal concerned gives some indication that it understands how the current situation appears to the conspecific it is deceiving, and if there is some advantage to that deception (e.g. avoidance of punishment or access to reward).

Given the normal appearance of lying and deception during childhood (Ford 1995; O’Connell 1998), several authors have speculated upon the (teleonomic) purpose served by such behaviours in human life. These accounts have little to say about the mechanism by which deception emerged during evolution. However, at the level of the individual human child, one speculation has been that deceit delineates a boundary between the ‘self’ and the ‘other’, specifically between the child and her mother (Ford et al. 1988). Learning at the age of 3 or 4 years that he/she can know something that his/her mother does not know (which itself implies a developing theory of mind) establishes for the child the limit of his/her mother’s knowledge, and allows the child some degree of control. Indeed, this experience of control (over information) might drive

the ‘pathological lying’ seen later in life, among dysfunctional adolescents and adults (Ford et al. 1988). Following this argument, the ability to lie is dependent upon the liar’s recognition that his/her thoughts are not known to others; and that different individuals’ understandings of the world may diverge. Hence, deliberate deception is dependent upon the acquisition of a capacity for theory of mind, a capacity that has been the subject of functional neuroimaging studies (Fletcher et al. 1995; Gallagher & Frith 2003)

It is worth noting that lying may be prosocial in certain contexts. It may ease social interaction, by way of compliments and information management. By contrast, precisely truthful communication at all times would be difficult and perhaps rather brutal. Hence, it is unsurprising that ‘normal’ subjects admit to telling lies on most days (Vrij 2001).

Social psychological studies, often of college students, suggest that lying facilitates impression management, especially early on in a romantic relationship (Vrij 2001). Hence, given that theory of mind is a prerequisite for deception, and that deception cases social communication, it is unsurprising that disorders of social interaction (such as autism) are associated with an inability to deceive, and social communication that may appear insensitive (Sodian & Frith 1992; Hughes & Russell 1993; Happe 1994).

Of course, deception is a vital skill in the context of conflict, especially between social groups, countries or intelligence agencies (e.g. Latimer 2001). When practised under these circumstances it might even be perceived as a moral ‘good’ (depending on one’s affiliation). However, when an individual subject is branded a liar, any advantage formerly gained may be lost. Although fluent liars might make entertaining companions (at times), being known as a liar is unlikely to be ultimately advantageous (Vrij 2001).

3. PRINCIPLES OF EXECUTIVE CONTROL

Control of voluntary behaviour in everyday human life is crucial but likely to be constrained by cognitive, neurobiological resources (Baddeley 1966; Passingham 1996; Spence et al. 2002). Control (or executive) functions are not necessarily ‘conscious’, although their contents may access awareness (Badgaiyan 2000; Jack & Shallice 2001). Executive functions include problem solving, planning, the initiation and inhibition of behaviours, and the manipulation of useful data in conscious working memory (e.g. the telephone number about to be dialled, the ramifications of the lie about to be told). These functions engage specific regions of the PFC (see figures 1 and 2), though they also involve distributed brain systems. There seems to be a principle to the cognitive architecture of executive control. ‘Higher’ centres such as the PFC are essential to adaptive behaviour in novel or difficult circumstances, while lower, slave systems, implicating posterior and sub-cortical systems, may be sufficient to perform routine or automated tasks (e.g. riding a bike while thinking of something else; figure 2; Shallice 1988, 2002; Passingham 1996).

A recurring theme in the psychology of deception is the difficulty of deceiving in ‘high stake’ situations: information previously divulged must be recalled, emotions and behaviours ‘controlled’, information managed (Vrij 2001). The latter resemble executive tasks. Hence, much of the liar’s behaviour may be seen, from a cognitive neurobiological perspective, as falling on a continuum with other
That the orbitofrontal cortex may be involved in successful deception, or at least in withholding information, has been implied by Ford (1995). Drawing on the example of the ‘pseudopsychopathic personality’ syndrome observed after orbitofrontal lesions, Ford points out that though these patients may exhibit certain features of psychopathy (such as impulsiveness and aggression), they tend not to lie. Instead they exhibit a callous disregard for social convention and an ‘honesty’ that may be extremely insensitive to decorum and the feelings of others. They may be inappropriately truthful (i.e. ‘tactless’). Hence, it is possible that the presence of an intact orbitofrontal cortex facilitates the telling of lies (perhaps as a consequence of response inhibition; in this case the inhibition of truthful responses). Lesions of this brain region in non-human primates produce deficits on conditional response tasks (including certain forms of the ‘go, no-go’ task) that may elicit perseveration (contextually inappropriate response repetition; Iversen & Mishkin 1970; Butters et al. 1973). In humans, lesions may also be associated with perseveration and a failure to inhibition pre-potent responses (Starkstein & Robinson 1997). Hence, from a cognitive neurobiological perspective, the pseudopsychopath of Ford (1995) utters pre-potent truths, tactlessly, because they are ‘released’ by orbitofrontal lesions.

5. IMAGING DECEPTION

(a) Spence (2001)

We proposed that the inhibition of relatively pre-potent responses (‘truths’) would be associated with greater activation of ventral prefrontal regions (systems known to be implicated in response inhibition; see §4 and figure 1). We also proposed that the concomitant generation of ‘lie’ responses (in contrast to ‘truths’) would be associated with greater dorsolateral prefrontal cortical activity (this area being implicated in the generation of novel responses; Frith et al. 1991; Spence et al. 1998).

We used a simple computerized protocol in which subjects answered questions with a ‘yes’ or a ‘no’, pressing specified single computer keys. All the questions concerned activities that subjects might have performed on the day that they were studied. We had previously acquired information from each of them, concerning their activities, when they were first interviewed. However, there was an added feature of the method applied in that subjects performed these tests in the presence of an investigator who was a ‘stooge’, who would be required to judge afterwards whether the subjects’ responses were truths or lies. The computer screen presenting questions to the subjects also contained a ‘colour rule’, subjects responded with truthful responses in the presence of one colour and lie responses in the presence of the other. All questions were presented twice, once each under each colour condition, so that finally we were able to compare response times and brain activity during ‘truth’ and ‘lie’ responses. We have published studies from three cohorts of subjects ‘outside the scanner’ (30–48 subjects in each; Spence et al. 2001, 2003; Farrow et al. 2003) and one sample of 10 subjects ‘inside the scanner’ (Spence et al. 2001), each cohort performing two variants of our experi-

Figure 1. Cartoon illustrating two regions of PFC implicated in behavioural control. The brain is viewed from the left side. DLPFC has been particularly implicated in the generation of behaviours (especially novel or ‘internally generated’ behaviours; Frith et al. (1991); Spence et al. (1998)); VLPFC has been implicated in response inhibition and reversal (Starkstein & Robinson 1997). VLPFC is markedly activated in our experimental lying protocols (see figures 3 and 4).

4. LYING AS A COGNITIVE PROCESS

Deceiving another human subject is likely to involve multiple cognitive processes, including theory of mind concerning the victim’s thoughts (their ongoing beliefs) and the analysis of responses made by both the liar and the victim in the context of their interaction. In the light of the above, we may posit that in the normal situation the liar is called upon to do at least two things simultaneously. He must construct a new item of information (the lie) while also withholding a factual item (the truth), assuming that he knows and understands what constitutes the ‘correct’ information. Within such a theoretical framework it is apparent that the truthful response comprises a form of baseline, or pre-potent response. We would predict that such a response would be made by an honest subject answering the same question or by the liar were he to become distracted or fatigued (indeed, from this perspective it is understandable why inebriation or sedation might ‘release’ the truth via disinhibition: in vino veritas). We might, therefore, propose that responding with a lie demands some form of additional cognitive processing, that it will engage executive, prefrontal systems (more so than telling the truth). Hence, we have a hypothesis that may be tested using functional neuroimaging (Spence et al. 2001).

mental protocol. The brain imaging technique applied was fMRI.

Our analyses revealed that whether subjects were studied inside or outside the scanner there was a statistically significant effect of lying upon response time (it being ca. 200 ms longer during ‘lying’ compared with responding truthfully). In the scanned sample, lie responses were associated with increased activation in bilateral ventrolateral prefrontal and anterior cingulate cortices (together with medial prefrontal and left inferior parietal cortices; figure 3). These data support the hypothesis that prefrontal systems exhibit greater activation when subjects are called upon to generate experimental ‘lies’ and they demonstrate (at the level of groups of subjects) that longer processing time is required to answer with a lie. However, our predictions of which prefrontal regions would be most activated during deception were only partly confirmed. The presence of consistent activation in ventrolateral PFCs and the minimal activation of DLPFC suggested to us that that the inhibition of the pre-potent (truthful) response, inherent in our task, contributed most to the pattern of activity seen. While ‘lying’ comprised only a reversal of the pre-potent response (e.g. ‘yes’ for ‘no’) rather than an elaboration of a ‘new lie’, it may have been insufficiently demanding for there to be marked activation of dorsolateral prefrontal regions (see the Ganis et al. (2003) study).

Notwithstanding these and other limitations (described in § 5c), our finding of increased response time during lying is congruent with a recent report of a convicted murderer, filmed while lying and telling the truth (Vrij & Mann 2001). Although recounting similar material on both occasions, this subject exhibited slower speech with longer pauses and more speech disturbance when lying. He also exhibited fewer ‘illustrators’ (bodily movements). Previous meta-analyses of behavioural lying studies have also pointed to speech disturbance, increased response latency and a decrease in other motor behaviours in the context of attempted deception (Ekman & Friesen 1972; Vrij & Mann 2001). Although responses on our (computerized) tasks were non-verbal, the behavioural and functional anatomical profile revealed (above) may indicate a common process underlying these findings and others; namely, an inhibitory mechanism that is used by those attempting to withhold the truth (a process associated with increased response latency).

It is noteworthy that the difference between lying and truth times for all groups in our studies was ca. 200 ms (Spence et al. 2001, 2003; Farrow et al. 2003). This is consistent with behavioural data acquired by other authors, studying the ‘guilty knowledge’ test (Farwell & Donchin 1991; Seymour et al. 2000; see § 5b).

(b) Langleben (2002)

Other groups have also used fMRI and found the PFC to be implicated in deception. Langleben and colleagues used the guilty knowledge paradigm, to test the hypothesis that subjects would activate executive, inhibitory brain regions while withholding a truthful response. Subjects were studied in a MR scanner while they made motor responses to a sequence of playing cards presented visually. The subjects each held one card, which was known to them and which comprised their ‘guilty knowledge’). Subjects used a button box to respond manually ‘yes’ or ‘no’ regarding the identity of the card they held. They also answered control questions, some requiring truthful responses, and other ‘non-target’ questions to confirm their attention to the protocol. Denying possession of a target playing card (the ‘lie’) was associated with greater activation in the anterior cingulate cortex (brain coordinates 4,26,42, in a region very similar to that identified in Spence et al. (2001): 3,28,43; Talairach & Tournoux 1988) and left parietal cortex (in a region medial to that identified in Spence et al. (2001)). There were no brain regions that exhibited greater activation during truthful responding relative to the lie condition. Response times were not reported.

(c) Lee et al. (2002)

On the basis of behavioural experiments examining the ways in which healthy subjects would set about feigning memory impairment, Lee and colleagues suggested that a
real ‘feigner’ would take account of their response performance as they went along, so that they would not perform ‘too badly’ all the time (in case this provoked suspicion). To remain credible, such a malingering would wish to perform no worse than chance when answering questions relating to their feigned deficit (e.g. their autobiographical memory). In a MR scanner subjects performed two forced-choice tasks, one relating to identifying three-digit numbers they had seen previously, the other to items of autobiographical information, for example, where they had been born (Lee et al. 2002). Subjects made manual responses to indicate their answers. When compared with truthful responding (on both tasks) malingering was associated with increased activation in bilateral dorsolateral prefrontal, inferior parietal, middle temporal and posterior cingulate cortices, together with bilateral caudate nuclei. The authors did not report any areas where truthful responding elicited greater activation. Response times were not reported.

(d) Ganis (2003)

In this study the authors made a novel distinction, not emphasized in earlier studies, between lies that form part of a well-rehearsed and coherent scenario and those that are spontaneous and need not fit into such a larger narrative framework. Subjects were studied while they gave motor (button press) and vocal responses, comprising both forms of ‘lie’. Their findings were that both types of lie were associated with greater activation in bilateral anterior prefrontal cortices and bilateral hippocampal gyri, while there were no reported areas of greater activation during truthful responding. On a sub-group of subjects for whom behavioural response measures were available, the authors did not find a significant difference in response times during lying and truthful responding. However, it is interesting to note that their raw data do suggest a difference of ca. 200 ms (whereas the mean response time for memorized-scenario lies was 838 ms, and that for spontaneous, isolated lies was 859 ms, the mean response time for truthful responses was 613 ms; Ganis et al. 2003). This study may have been underpowered to detect significant differences between deceptive and truthful response time.

These authors also reported some other similarities to the foregoing work. Anterior cingulate gyri exhibited greater activation during spontaneous lies, at a focus (4, 6, 39) 20 mm posterior to that seen in the studies by Spence et al. (2001) and Langleben et al. (2002; See §5b). Also, they found an area of activation associated with the telling of spontaneous, isolated lies in the right Brodmann Area 47 ‘in a spherical (region of interest) centred at the coordinates reported in Spence et al. (2001)’ (p. 835).

With respect to the proposed distinction between rehearsed and spontaneous lies, Ganis and colleagues report greater right frontal activation in the former and greater anterior cingulate cortex and visual cortex activity in the latter.

(e) Future directions

The scanning studies to date, including our own, have been subject to behavioural and task-related limitations: a certain artificiality, the frequent use of a non-vocal signal to transmit the deception, and the ‘low stake’ nature of the ‘lying’ involved. Some of these limitations have involved compromises imposed by the scanning technology itself. Future experimentation should seek to use other paradigms for testing the neurological components of deception, in part by expanding the kinds of tasks used.

In a current, unpublished fMRI study, we have begun to explore this kind of variation, using ‘silent periods’ in the scanner to allow auditory stimuli and vocal responses to be used. By studying vocal lies and by adding a ‘defy/comply’ condition, we posited that the following cognitive subtraction would reveal those brain regions specifically activated by lying, rather than by memory of the index event or the mere reversal of a pre-potent response:

\[
\text{brain activations specific to lying} = (\text{lie-truth}) - (\text{defy-comply}).
\]

This approach appears promising. Preliminary data analysis using this subtraction suggests that lying was specifically associated with activation of the following regions: right ventrolateral and orbitofrontal cortices (BA 47 and 11, respectively), right medial (BA 6) frontal gyrus, right inferior parietal lobule (BA 40) and left premotor cor-

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**Table 1. Areas activated during lie condition in vocal lying study (relative to truthful responding and following subtraction of ‘defy/comply’ activations).**

\[(p < 0.05, \text{corrected for multiple comparisons.})\]

<table>
<thead>
<tr>
<th>brain area</th>
<th>AB</th>
<th>X</th>
<th>Y</th>
<th>Z</th>
<th>t</th>
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</thead>
<tbody>
<tr>
<td>right orbitofrontal PFC</td>
<td>11</td>
<td>38</td>
<td>40</td>
<td>-15</td>
<td>4.50</td>
</tr>
<tr>
<td>right VLPFC</td>
<td>47</td>
<td>34</td>
<td>31</td>
<td>-2</td>
<td>4.22</td>
</tr>
<tr>
<td>right premotor cortex</td>
<td>4</td>
<td>4</td>
<td>1</td>
<td>57</td>
<td>4.32</td>
</tr>
<tr>
<td>right inferior parietal lobule</td>
<td>40</td>
<td>50</td>
<td>-60</td>
<td>44</td>
<td>4.97</td>
</tr>
<tr>
<td>left premotor cortex</td>
<td>6</td>
<td>-10</td>
<td>22</td>
<td>66</td>
<td>4.40</td>
</tr>
</tbody>
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At a less conservative statistical threshold \((p < 0.001, \text{uncorrected for multiple comparisons})\), activity in orbitofrontal regions was seen to be bilateral (figure 4), similar in location though not identical to that of our previous study (Spence et al. 2001).

**6. COMMENT**

From a cognitive perspective the telling of lies resembles an executive process. On behavioural measures there is an activation of ‘defy/comply’ activations.

From a biological perspective, our findings and those of others suggest that deception engages the higher centres of the human brain and places certain demands on the cognitive capacities of the individual who is lying. It would be naive to imagine that such a body of work might not impact upon that other ‘real world’ of forensic practice, at least at a theoretical level (at the moment). The question of whether or not societies should resort to lie detection is one deserving of broad societal debate and is not in itself a scientific question. However, the development of means of lie detection that are (physically) harmless to the individual concerned might be regarded as a moral good, if contrasted with the more traditional means of information extraction alluded to by Simic (2004).

Nevertheless, the right to silence and the value of non-coerced confessions as desirable elements of human behaviour are also deserving of respectful consideration and continue to attract thoughtful review (e.g. Brooks 2000). We do not have the space to do sufficient justice to these issues in the current paper so we offer the following as cautions to the premature application of brain imaging technology to the problem of lie detection. The problems we foresee include the following.

(i) **Ecological validity:** the experiments that we have reviewed have generally involved compliant subjects telling trivial lies. They have not involved the high-stake situations that might be expected to pertain in the forensic arena.

(ii) **Experimental design:** it is clear that all experimenters to date (ourselves included) have devised simple experiments of simulated deception, which facilitate analyses using simple contrasts (i.e. lie versus truth) with the theoretical assumptions inherent in such designs. Making a categorical distinction between truth and lie suggests a certain clarity but may not cohere in the
‘real world’, where information may be imprecise, motives mixed and elements of truthfulness contained within the lie that the subject tells. Additionally, no study reported to date has demonstrated a distinct physiological signature to ‘truth’, merely that ‘lies’ activate the brain more, particularly in executive regions.

(iii) Statistical power: the studies we have reviewed concern the averaged brain activities of groups of subjects and we are aware of no study to date that has provided convincing evidence of a physiology of deception at the level of the single subject. Hence, there may well be a range of individual differences and it would be premature to extrapolate from the sorts of data we have considered to the individual suspect in the courtroom or the cell.

(iv) Can lying be ‘pathological’?: while deception is by definition a deliberate act, we are aware of conditions in which it may be conceptualized as ‘pathological’ (e.g. Abed 1995; Tyrer et al. 2001). Future theoretical work might focus upon the question of whether such a deliberate act can be pathological in nature, or whether it is instead the motivations, the reasons, driving the act that are the locus of implied pathology. It might well be that those who lie habitually are not ‘abnormal liars’ (i.e., they are not ‘lying abnormally’), merely people who use a normal strategy to excess.

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**Glossary**

**BA:** Brodmann area

**DLPFC:** dorsolateral prefrontal cortex

**fMRI:** functional magnetic resonance imaging

**MR:** magnetic resonance

**PFC:** prefrontal cortex

**VLPFC:** ventrolateral prefrontal cortex

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