Available online at www.sciencedirect.com
SCIENCE DIRECT®
Neuroscience and Biobehavioral Reviews xx (2005) 1–33
NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS
www.elsevier.com/locate/neubiorev

Review

The somatic marker hypothesis: A critical evaluation

Barnaby D. Dunn*, Tim Dalgleish, Andrew D. Lawrence

MRC Cognition and Brain Sciences Unit, 15 Chaucer Road, Cambridge CB2 2EF, UK

Abstract

The somatic marker hypothesis (SMH; [Damasio, A. R., Tranel, D., Damasio, H., 1991. Somatic markers and the guidance of behaviour: theory and preliminary testing. In Levin, H.S., Eisenberg, H.M., Benton, A.L. (Eds.), Frontal Lobe Function and Dysfunction. Oxford University Press, New York, pp. 217–229]) proposes that emotion-based biasing signals arising from the body are integrated in higher brain regions, in particular the ventromedial prefrontal cortex (VMPFC), to regulate decision-making in situations of complexity. Evidence for the SMH is largely based on the performance on the Iowa Gambling Task (IGT; [Bechara, A., Tranel, D., Damasio, H., Damasio, A.R., 1996. Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. Cerebral Cortex 6 (2), 215–225]), linking anticipatory skin conductance responses (SCRs) to successful performance on a decision-making paradigm in healthy participants. These 'marker' signals were absent in patients with VMPFC lesions and were associated with poorer IGT performance. The current article reviews the IGT findings, arguing that their interpretation is undermined by the cognitive penetrability of the reward/punishment schedule, ambiguity surrounding interpretation of the psychophysiological data, and a shortage of causal evidence linking peripheral feedback to IGT performance. Further, there are other well-specified and parsimonious explanations that can equally well model the IGT data. Next, lesion, neuroimaging, and psychopharmacology data evaluating the proposed neural substrate underpinning the SMH are reviewed. Finally, conceptual reservations about the novelty, parsimony and specification of the SMH are raised. It is concluded that while presenting an elegant theory of how emotion influences decision-making, the SMH requires additional empirical support to remain tenable.

Keywords: Emotion; Iowa gambling task; Decision-making; Ventromedial prefrontal cortex; Body

Contents

* Corresponding author. Tel.: +44 1223 355 294; fax: +44 1223 359 062.
E-mail address: barney.dunn@mrc-cbu.cam.ac.uk (B.D. Dunn).

0149-7634/$ - see front matter © 2005 Published by Elsevier Ltd.
doi:10.1016/j.neubiorev.2005.07.001

Perhaps the most influential conceptualisation of the 'emotional brain' over the last century has been the limbic system (MacLean, 1949), an anatomical framework that outlines how emotions and moods are embodied in neural architecture. A core theoretical aspect of the limbic system framework is that emotion experiences arise from the integration of sensations from the external world with information from the body, specifically feedback from the viscera (MacLean, 1949, 1975: for a review, see Dalgleish, 2004). The notion that emotion experience emerges from feedback from the body in tandem with higher level representation of the world partially echoes the seminal theories of emotion put forward by James and Lange (James, 1884, 1894; Lange, 1885). These controversial models argued that emotion experience arises directly from the perception of change in the body: when we run from a bear in the woods, we are afraid because we run rather than we run because we are afraid (for reviews see Mandler, 1990; Laird and Bresler, 1990; Izard, 1990; Lang, 1994; Ellsworth, 1994; Reisenzein et al., 1995; Damasio, 2004; Prinz, 2004).

Antonio Damasio has recently extended the influence of somatic processes to the regulation of decision-making as well as emotion in his influential somatic marker hypothesis (henceforth, SMH; Damasio et al., 1991; Damasio, 1994, 1996, 2004). The SMH proposes that 'somatic marker' biasing signals from the body are represented and regulated in the emotion circuitry of the brain, particularly the ventromedial prefrontal cortex (VMPFC), to help regulate decision-making in situations of complexity and uncertainty (e.g Damasio, 1996; Bechara et al., 2000a,b).

The proposed neural circuitry underlying the SMH departs from the limbic system in an anatomical sense, in that it incorporates a variety of brain regions outside of the classic limbic system structures (MacLean, 1949), including ventromedial prefrontal cortex (VMPFC), somatosensory cortices, insula, and basal ganglia (Damasio, 1998). Further, Damasio extends the function of the limbic system beyond that of a 'visceral brain' (MacLean, 1949) in arguing that multiple sources of feedback from the periphery (visceral, somatosensory, and others) shape decision-making (see Damasio, 2004).

Empirical support for the SMH is largely based on performance on the Iowa Gambling Task (IGT; Bechara et al., 1994, 1996), an experimental paradigm designed to measure decision-making. A correlation has been found between successful IGT performance and the development of somatic marker signals (as indexed by the magnitude of anticipatory skin conductance responses [SCRs]) in healthy control participants. Crucially, these bodily signals were found to be absent in people with VMPFC lesions and this was linked to their poorer performance on the decision-making task.

This article will present a review of the extant research testing the central claims of the SMH, particularly focusing on the role of somatic processes in decision-making as measured by performance on the IGT. Section 1 provides a descriptive, non-critical account of the evolution of the SMH and the supports the IGT offers to the framework as described by Damasio's Iowa laboratory. To examine the psychological component of the SMH, Section 2 critically appraises the extent to which the IGT data can validate the SMH. It is proposed that recent empirical findings mean that the IGT, while well validated and extensively applied to a variety of neurological and psychiatric conditions in its behavioural form, will no longer suffice as strong evidence for the SMH. To examine the neural component of the SMH, Section 3 evaluates the extent to which the neural substrate Damasio put forward for the SMH has been empirically validated, concluding that there is reasonable evidence for this aspect of the model. Section 4 raises and evaluates some further conceptual concerns about the novelty and parsimony of the SMH and also discusses how well the theory has been specified. Our review of the evidence will suggest that additional empirical support and

a clearer conceptualisation are needed to validate further this intriguing and potentially useful theoretical framework. Some suggestions for alternative research designs to aid this process will be put forward.

1. Overview of the SMH and its evolution

1.1. Development of the SMH

The SMH grew from attempts to understand the striking emotional and everyday decision-making deficits displayed by patients with damage to VMPFC, the portions of the frontal lobes above the eye sockets. Damasio (1994) reviewed how damage to the VMPFC can have profound effects on work and social function without inducing any obvious impairments in intellect and cognitive performance, focusing particularly on the famous cases of Phineas Gage (see Harlow, 1868 for an overview) and Elliot (EVR; see Damasio, 1979; Eslinger and Damasio, 1985; Damasio et al., 1991).

In 1848, Gage was a successful foreman on the railway and kept a respectable social life. Following a blasting accident, however, a tamping iron went through his eye socket and extensively damaged his frontal cortex before passing out through the top of his skull. Remarkably he survived and at first appearance seemed to have no intellectual impairment. Gage started to display odd decision-making and social behaviours, however. Whereas before he had been a conservative family man, he subsequently could not hold down a job, made risky financial decisions and his family relations broke down. Modern neuroimaging techniques were much later applied to identify the passage of the tamping iron through Gage's skull (Damasio et al., 1994) and it was found to ablate a portion of the frontal lobe centred around the VMPFC (although see MacMillan, 2000 for a critique of Damasio's account of Gage).

Similarly, EVR presented to Damasio's Iowa laboratory after he suffered a brain tumour, which led to bilateral ablation of the VMPFC and related areas (Damasio, 1979, 1994; Eslinger and Damasio, 1985; Damasio et al., 1991). EVR became unable to make decisions, especially in the social and personal domain. He could not plan for the future and tended to choose unsuitable friends, business partners and activities. Other cases of VMPFC damage that produce broadly equivalent impairment have also been documented (e.g. Dimitrov et al., 1999; Barrash et al., 2000). The syndrome suffered by these patients has been documented as 'acquired sociopathy', reflecting the fact that the personality and decision-making effects of damage to this region resemble a milder form of those seen in sociopathy (Damasio et al., 1990, 1991; Tranel, 1994).

The Iowa laboratory conducted an elegant series of studies to attempt to elucidate the cause of the difficulties in day-to-day living displayed by cases such as EVR. Intriguingly, the peculiar choices EVR made in real life were at odds with initial laboratory assessments of his reasoning capabilities, which showed normal, or on many tasks superior, intellectual performance. For example, working memory, attention, cognitive estimation, cognitive flexibility, recency of event judgement and even social knowledge were all unimpaired (Damasio et al., 1991; Saver and Damasio, 1991). Later investigations identified that EVR and other cases with damage to VMPFC had a difficulty in expressing emotion and experiencing feelings. Neuropsychological investigation showed that VMPFC damage altered psychophysiological response and reported emotion experience to emotional but not neutral stimuli (Damasio et al., 1990, 1991; Tranel, 1994). This led Damasio to speculate that these emotional changes were the cause of decision-making difficulties seen following VMPFC damage.

1.2. The somatic marker hypothesis

In his influential book 'Descartes' Error', Damasio (1994) most famously articulated the SMH. Building on his earlier paper on the consequences of VMPFC damage (Damasio et al., 1991), he argued that the decision-making deficits found following VMPFC damage were due to an inability to use emotion-based biasing signals generated from the body (or 'somatic markers') when appraising different response options (see also Damasio, 1996, 2004).

In brief, the SMH postulated that reasoning is influenced by crude biasing signals arising from the neural machinery that underlies emotion. For Damasio, emotion is the representation and regulation of the complex array of homeostatic changes that occur in different levels of the brain and body in given situations. When making decisions, a crude biasing signal (a somatic marker) arising from the periphery or the central representation of the periphery indicates our emotional reaction to a response option. For every response option contemplated, a somatic state is generated, including sensations from the viscera, internal milieu, and the skeletal and smooth muscles (see Damasio, 2004). These somatic markers serve as an indicator of the value of what is represented and also as a booster signal for continued working memory and attention (Damasio et al., 1991; Damasio, 1996). Particularly in situations of complexity and uncertainty, these marker signals help to reduce the problem space to a tractable size by marking response options with an 'emotional' signal. Only those options that are marked as promising are processed in a full, cognitive fashion (Damasio, 1994, 1996; Bechara and Damasio, in press).

Somatic markers can reflect actions of the body proper (the 'body' loop) or the brain's representation of the action expected to take place in the body (the 'as-if' loop). In other words, the brain can construct a forward model of changes it

expects in the body, allowing the organism to respond more rapidly to external stimuli without waiting for that activity to actually emerge in the periphery. This is similar to accounts of how motor control of the periphery is regulated by advance modelling (e.g. Wolpert and Ghahramani, 2000). Perception of somatic state information makes us more likely to approach or withdraw from a situation. These signals can function at an overt level (where the individual is consciously aware of the emotions and bodily changes associated with a particular response option) or at a covert level (where the individual is unaware of his/her emotions and bodily activity).

Decision-making can be viewed as a combination of 'high reason', carrying out a logical cost-benefit analysis of a given action, and marker signals, indicating how rewarding or punishing an action is likely to be in complex situations where more detailed cost-benefit analysis is not possible (Damasio et al., 1991; Damasio, 1994, 1996, 2004).

Damasio has argued that damage to the VMPFC and other structures involved in the representation and regulation of body-state (including amygdala, insula, somatosensory cortex, cingulate, basal ganglia and brain-stem nuclei) leads to impaired decision-making because the somatic marking system can no longer be activated (see Fig. 1 which outlines the proposed 'somatic marker' network and Section 3 evaluating the proposed neural substrate of the SMH). The VMPFC is believed to be the crucial area of the brain that integrates actual or predicted bioregulatory state representations with potential response options, so is central to the generation of somatic markers. Cases such as Gage and EVR would therefore only be able to make decisions based on a logical cost-benefit analysis and would be unable to utilise prior emotion experience to guide them towards previous advantageous choices and away from disadvantageous choices. In uncertain situations, where a complete logical analysis of the situation is not possible, such a profile would lead to a marked decision-making impairment characterised by either extreme procrastination or the selection of inappropriate response options that would be immediately dismissed by people with intact marker signals. This profile has been characterised as 'myopia for the future', where the individual is unable to predict long-term punishments and rewards based on previous experience (e.g. Bechara et al., 1994).

1.3. The Iowa gambling task

As noted already, a major plank of empirical evidence supporting the SMH stemmed from the IGT, an experimental paradigm designed to mimic real life decision-making situations in the way it factors uncertainty, reward and punishment (see Bechara et al., 1994, 1996, 1999, 2000a for full details). A key feature of this task is that participants have to forego short-term benefit for long-term profit.

The task requires participants to select from one of four decks of cards that are identical in physical appearance for 100 trials. Each card choice leads to either a variable financial reward or a combination of a variable financial reward and penalty. Unknown to participants, the rewards and punishments on the decks have been fixed by the experimenter. For each selection from decks A and B participants win $100 and from each selection from decks C and D participants win $50. Every so often variable punishment is also given. On deck A, five in ten trials generate a penalty ranging from $35 to $150. On deck B, one in ten trials incurs a penalty of $1250. On deck C, five in

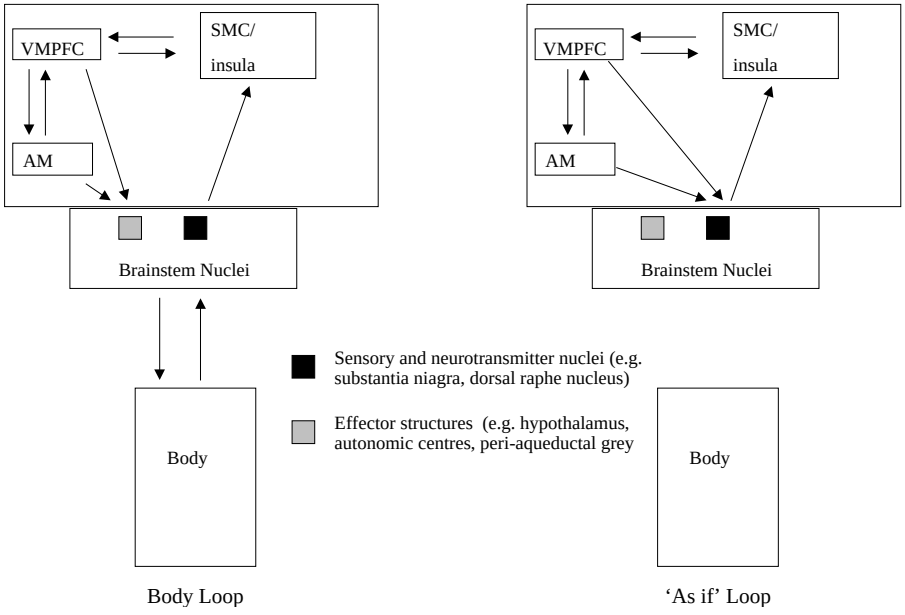
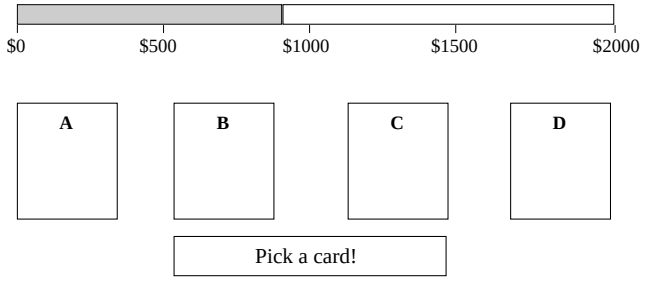


Fig. 1. Neural architecture implicated in the SMH. The left-hand side illustrates a schematic of the 'body' loop and the right-hand side illustrates a schematic of the 'as-if' loop. VMPFC, ventromedial prefrontal cortex; AM, amygdala; SMC, somatosensory cortex. Figure reproduced from Bechara and Damasio (2005).

ten trials involve a penalty ranging from $25 to $75. Finally, on deck D, one in ten trials gives a penalty of $250. Overall, the high reward decks (A and B) give higher levels of punishment (so leading to a net loss of $250 every 10 trials), whereas the low reward decks (C and D) give lower levels of punishment (so leading to a net gain of $250 every 10 trials). Thus, successful task performance relies on sampling more from decks C and D than from decks A and B. There is no advantage for participants in selecting more from the frequent punishment (A and C) versus infrequent punishment (B and D) decks, or vice versa. Crucially, it is argued that this reward/punishment schedule is opaque, such that participants are unlikely to be able to perform an exact calculation of net gains and losses. To do well, it is therefore claimed that participants must rely on more 'intuitive' decision-making processes, in particular the activation of somatic marker biasing signals (Bechara et al., 1994, 1996). Fig. 2 presents an overview of the rewards and punishments on each deck of the IGT.

Prior to completing the task, participants are instructed that the game requires them to choose cards from any one of the four decks until they are told to stop. They are not given information about how long the task will go on nor when it will stop and it is explicitly stated that they can change deck whenever they wish. The goal of the game is defined as to win as much money as possible and to avoid losing money as far as possible. Participants are told that while there is no way for them to work out when they will lose money, they will find that some decks are worse than others and that to do well they need to stay away from the worst decks.

In the original manual version, participants are seated in front of four decks of cards of identical physical appearance and are given a $2000 loan of money (facsimile US$ bills).



	Reward	Punishment	Net Profit (over 10 trials)
A	Win $100 (100% trials)	Lose $150 to $35 (50% trials)	- $250
B	Win $100 (100% trials)	Lose $1250 (10% trials)	- $250
C	Win $50 (100% trials)	Lose $25 to $75 (50% trials)	+ $250
D	Win $50 (100% trials)	Lose $250 (10% trials)	+ $250

Fig. 2. Graphical representation of IGT. On each of 100 trials, participants must select one of four decks of cards, from which they receive either a reward or a combined reward and punishment. Decks A and B offer short-term rewards but long-term punishments, leading to a net loss. Decks C and D offer reduced immediate rewards but smaller long-term punishments, leading to a net profit. Acquisition of the task is measured by number of selections from the advantageous decks relative to the disadvantageous deck on each block of 20 trials.

A computerised version of the task displaying the cards on a computer screen and using a visual representation of profits and losses (a green bar increases or decreases in size on the screen to record total money held by the participants) has also been subsequently developed.

A central aspect of the IGT is that SCRs have been found to be associated with successful learning. When recording SCR data, a modified version of the task is used that has a longer interval between trials (typically greater than 15 s), thus allowing SCR activity to return to baseline before the next selection. Analysis of these data has looked at whether the SCRs generated in anticipation of deck selection, following reward, and following combined reward and punishment vary as a function of which deck is chosen (e.g. Bechara et al., 2000a).

1.4. Review of Iowa laboratory studies using the IGT in support of the SMH

Bechara et al. (1994) first described the performance of patients with damage encompassing but not restricted to VMPFC on the task. The VMPFC patients ($n=6$) were significantly worse at the IGT than healthy control volunteers ($n=44$). Over time the control group learned to select more from the 'safe', winning decks (C and D) than the 'risky', losing decks (A and B), whereas the VMPFC lesion group continued to prefer the disadvantageous decks for the duration of the task. Interestingly, patient EVR was tested on multiple occasions of the task and failed to learn, whereas control participants performance improved on subsequent testing. The authors concluded that the VMPFC lesion group decisions were driven more by the immediate reward than the delayed punishment available on the disadvantageous decks ('myopia for the future' Bechara et al., 1994).

Key support for the hypothesised role of somatic markers in performance on the IGT derived from identification of a physiological correlation of success on the decision-making task (Bechara et al., 1996). SCRs were measured in seven patients with frontal lobe damage encompassing VMPFC and 12 normal controls during task performance. Both patients and controls showed reward and punishment SCRs. After a short period of time, however, the control group also started to develop anticipatory SCRs, which were larger for selections from the 'risky' decks than the 'safe' decks. The absence of anticipatory SCRs in the VMPFC lesion group correlated with impaired task performance. It was speculated that patients were failing to activate somatic markers to help them to distinguish between good and bad outcomes in situations of uncertainty (Bechara et al., 1996). In particular, failure to activate a negative marking signal for the disadvantageous decks based on previous punishment history would make the VMPFC lesion group insensitive to the possibility of further future punishment on these decks. Fig. 3 presents the typical behavioural and skin conductance

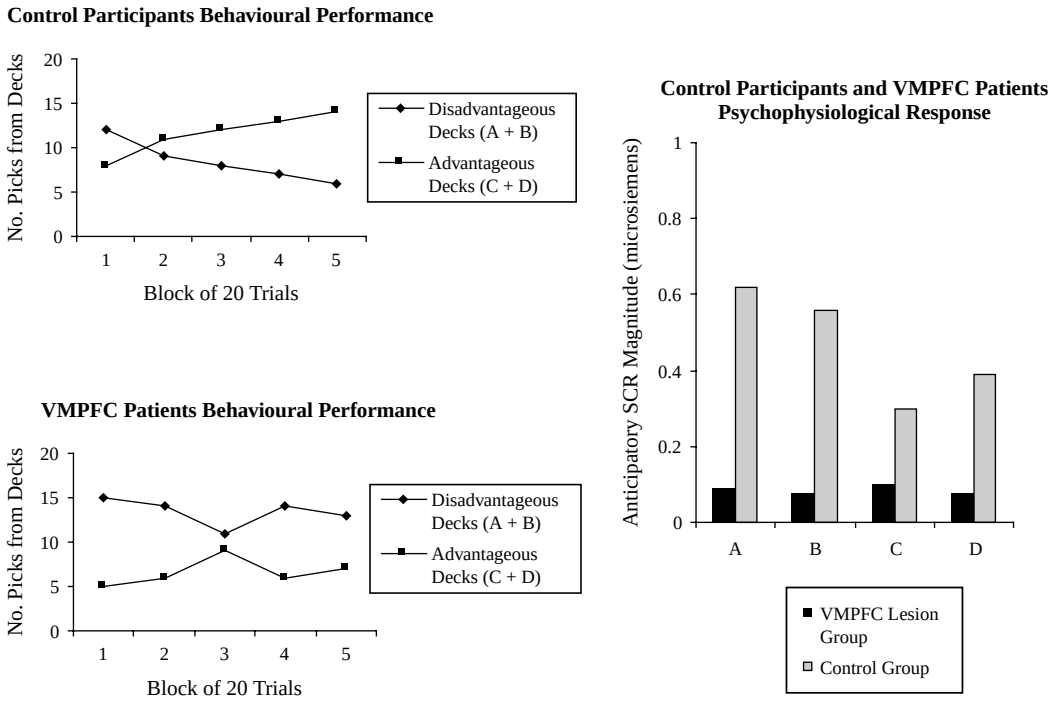


Fig. 3. Typical behavioural and psychophysiology results on the IGT. Healthy control participants (top left panel) increasingly select from the advantageous decks over time, whereas VMPFC lesion patients (bottom left panel) opt for the disadvantageous decks for the duration of the task. Healthy control participants show a greater anticipatory SCR to the disadvantageous relative to advantageous decks, but this effect is absent in the VMPFC lesion patients (right panel). Data reproduced from Bechara et al. (1994, 1996, 1997) and Bechara and Damasio (2005).

profiles found for VMPFC lesion patients and healthy control volunteers on the IGT.

As noted earlier, an important claim is that the reward/punishment schedule of the IGT is opaque, so learning is therefore taking place at a non-declarative, largely implicit level. To test this assertion, Bechara et al. (1997) asked ten healthy participants and six VMPFC lesion patients to complete the task and stopped them after every 10 trials to see whether they knew consciously what was going on in the game. Analysis indicated that participants went through four phases: pre-punishment (no punishment yet encountered), pre-hunch (no understanding of what was going on), hunch (hypotheses generated on which were 'good' and 'bad' decks), and the conceptual period (clear idea of what was going on). Around 30% of control participants did not reach the conceptual period, despite performing normally on the task. Anticipatory SCR activity and increased selection from the good decks began to take place for the control group in the pre-hunch period and was sustained throughout the task. This was taken to suggest that implicit learning was taking place prior to explicit understanding of the paradigm. Notably, 50% of the VMPFC patients did reach the conceptual period but still performed disadvantageously on the task.

To support the 'myopia for the future' interpretation of the deficit displayed by VMPFC lesion patients on the IGT, Bechara et al. (2000b) examined performance of healthy control volunteers and lesion patients on two modified forms of the task. In the first of these, the reward and punishment contingencies were reversed such that differences in reward determined the optimal response. The advantageous decks yielded higher immediate punishment but even greater delayed reward, whereas the disadvantageous decks gave low immediate punishment but even lower future reward. VMPFC lesion patients were impaired on this version of the task also relative to control volunteers, indicating that the mechanism underpinning task impairment is unlikely to be loss of sensitivity to either punishment or reward cues. This study did not report anticipatory psychophysiology data, however, so it is unclear if a similar SCR profile emerged in the variant task. The second variant task investigated whether decision-making deficits in VMPFC lesion patients could be normalised by increasing the adverse future consequences associated with the 'risky' decks. Even after increasing the delayed punishment and decreasing the delayed reward on the disadvantageous decks, the VMPFC lesion patients were impaired on the task relative to controls (Bechara et al., 2000b).

In summary, work from the Iowa laboratory has found that VMPFC lesions impair the ability to select from the advantageous decks on the IGT and that this behavioural deficit is associated with the absence of anticipatory SCR signals that are known to differentiate between the good and bad decks in healthy control volunteers. Manipulation of the reward/punishment schedule used supports the conclusion that this behavioural deficit following VMPFC lesions

reflects 'myopia for the future' (Bechara et al., 1994) rather than altered sensitivity to reward and punishment. Further, early performance on the IGT appears to be regulated by non-declarative learning systems, since healthy control participants have been shown to select from the advantageous decks prior to being able to explicitly report the reinforcement contingencies of the decks. All of these findings are consistent with the SMH (see Section 3 for a more detailed review of studies from the Iowa laboratory investigating the exact neural substrate underpinning these somatic markers).

2. Evaluation of the Iowa gambling task as key evidence for the SMH

Work from the Iowa laboratory at first glance provides strong support for the SMH. On closer examination, however, a number of issues with the IGT come to light that potentially undermine this evidence. The strengths and weaknesses of the IGT will now be systematically reviewed in order to determine the extent to which IGT data can support the SMH.

2.1. Strengths of the IGT

The IGT, both as a source of evidence for the SMH and as a measure of broader decision-making, has a number of strengths. First, impairments on the task following brain damage and normative performance on the task in healthy controls have been extensively validated within the Iowa laboratory. Forty-five patients with different kinds of frontal lobe lesions and 35 patients with lesions to the lateral temporal or occipital cortex have performed the IGT. Of these patients, only those with damage to neural regions implicated in the SMH (e.g. VMPFC, amygdala) appear to be impaired on the task (Bechara et al., 2000a,b). Further, over 80 healthy control participants have reportedly completed the task and produced a similar profile of results as in the original data (Bechara et al., 1994, 1996). In addition, studies conducted outside the Iowa laboratory more often than not replicate the behavioural findings on the task, although few of these included psychophysiological measurements (see Table 1 for a summary of studies using the IGT published before July 2005).

Second, it has been demonstrated that the IGT is robust in the face of certain changes in its parameters. A similar performance pattern emerges when the nature of the incentive used is varied, for example, when giving real financial rewards or facsimile money (Bowman and Turnbull, 2003). Further, behavioural data have been replicated when using different time delays on the task (Bowman et al., 2005) or when using a manually administered or computerised form of the task (Bechara et al., 2000a).

Third, lifespan developmental changes in performance on the IGT have been examined. Performance on the paradigm improves with increasing age until adulthood (Crone and van der Molen, 2004; Overman et al., 2004; Kerr and Zelazo, 2004; Hooper et al., 2004) and then appears to tail off in older age (Denburg et al., 2001, 2005; Lamar and Resnick, 2004). Gender differences have also been demonstrated, which appear to be mediated by age. Adolescent women were found to select cards associated with both immediate wins and long-term outcome, whereas adolescent men picked decks on the basis of long-term outcome only (Overman, 2004). Garon and Moore (2004) report that girls perform better than boys between 3 and 6 years of age, whereas other studies found that adult men showed superior behavioural performance to adult women on the task (Reavis and Overman, 2001; Bolla et al., 2004). Evans et al. (2004) found that higher levels of education and intelligence are associated with poorer performance on the IGT. Non-university educated female participants showed double the improvement in the last 40 trials of the IGT compared to those with a university education. These individual difference findings aid interpretation of the IGT at the single case level.

Fourth, the behavioural form of the IGT has been shown to be a highly sensitive measure of impaired decision-making in a variety of neurological and psychiatric conditions known to be characterised by real world decision-making impairments (e.g. pathological gambling, obsessive compulsive disorder [OCD]; see Table 1). Further, there is increasing evidence that the task has reasonable predictive validity. For example, IGT performance has been associated with response to pharmacotherapy in OCD (Cavedini et al., 2004b) and safety of sexual practices in substance abusing HIV-positive males (Gonzalez et al., 2005). These factors offer good support for the validity of the paradigm and also illustrate that the task has stimulated a considerable body of research.

Despite its strengths, there are a number of issues concerning the IGT that undermine the extent of the support it can offer to the SMH. These will now be discussed in turn.

2.2. Cognitive penetrability of the IGT reward/punishment schedule

The cognitive impenetrability of the IGT is central to the claim that it can only be successfully completed through recourse to emotion-based learning via somatic marker signals in the early stages. Damasio states that: "the key ingredient that distinguishes the task of Bechara and colleagues from other tasks of probabilistic reasoning is that subjects discriminate choices by feeling; they develop hunches that certain choices are better than others… subjects with damage to VMPFC fail this task and they fail it precisely because they are unable to represent choice bias in the form of an 'emotional hunch'" (Damasio et al., 2003, p. 84). Further, other articles from the Iowa laboratory make the claim that: "in normal individuals non-conscious biases guide reasoning and decision-making behaviour

Table 1
Summary of studies examining IGT performance in clinical groups organised by presenting condition (published before July 2005)

Authors	Groups studied	SCR used?	Deficit found in clinical group?	Control data replicated?
Clark et al. (2001)	Acutely manic inpatients ($n=15$); non-psychiatric participants ($n=30$)	N	Slower learning in manic patients	Y
Wilder et al. (1998a,b)	Schizophrenic patients ($n=12$); healthy control volunteers ($n=30$)	N	None	N, control group preferred infrequent punishment (decks B and D) to frequent punishment (decks A and C)
Beninger et al. (2003)	Schizophrenic patients on typical medication ($n=18$); schizophrenic patients on a-typical medication ($n=18$)Healthy control volunteers ($n=18$); healthy control volunteers ($n=18$)	N	Patients on atypical medication showed disadvantageous deck preference, whereas those on typical medication performed normally on the task	Y
Ritter et al. (2004)	Chronic schizophrenic patients ($n=20$); non-psychiatric participants ($n=15$)	N	Disadvantageous deck preference in schizophrenic patients	Y
Shurman et al. (2005)	Schizophrenic patients ($n=39$); healthy control volunteers ($n=10$)	N	No preference for advantageous decks, preference for infrequent punishment (decks B and D) rather than infrequent punishment (decks A and C)	Y
Turnbull et al. (in press)	Schizophrenic patients ($n=21$); healthy control volunteers ($n=21$)	N	None on original task, but schizophrenic patients with marked negative symptoms showed deficit on subsequent reversal learning	Y
Bark et al. (2005)	Catatonic schizophrenic patients ($n=8$); paranoid schizophrenic patients ($n=19$); healthy control volunteers ($n=26$)	N	Disadvantageous deck preference in schizophrenic patients	Y
Nielen et al. (2002)	OCD patients ($n=27$); healthy control volunteers ($n=26$)	N	None	Y
Cavedini et al. (2002)	OCD patients ($n=34$); healthy control volunteers ($n=34$); panic disorder control patients ($n=16$)	N	Disadvantageous deck preference in OCD, related to poor response to drug treatment	Y
Whitney et al. (2004)	Schiozophrenia with obsessive symptoms ($n=26$); schizophrenic patients without obessive symptoms ($n=28$); OCD patients ($n=11$)	N	Trend for both schizophrenic groups to select more from disadvantageous decks, compared to OCD patients	No standard control group used
Cavedini et al. (2004)	Patients with OCD ($n=34$) treated with fluvoxamine only or fluvoxamine plus risperidone	N	Patients with good IGT performance responded to fluvoxamine alone, where patients with impaired IGT performance responded to fluvoxamine plus risperidone	No standard control group used
Cavedini et al. (2002)	Pathological gambling patients ($n=20$); healthy control volunteers ($n=40$)	N	Disadvantageous deck preference, increasing over time in pathological gambling patients	Y
Goudriaan et al. (2005)	Pathological gambling patients ($n=48$); alcohol-dependent patients ($n=46$); Tourette syndrome patients ($n=47$); healthy control volunteers ($n=49$)	N	The pathological gambling and alcohol-dependent patients selected less from advantageous decks overall, relative to both control participants and Tourette syndrome patients	Y
Bechara et al. (2001)	Substance-dependent individuals (SDI: $n=41$); VMPFC patients ($n=5$); healthy control volunteers ($n=40$)	N	Slower learning in SDI group, disadvantageous deck preference in VMPFC group	N, 31% of controls performed in the range of VMPFC patients
Bechara and Damasio (2002)	Substance-dependent individuals (SDI: $n=41$); VMPFC patients ($n=10$); healthy control participants volunteers ($n=49$)	Y	Slower learning in SDI group, lower anticipatory SCR in SDI group relative to controls	N, 37% of controls performed in the range of VMPFC patients
Verdejo et al. (2004)	Substance abusing individuals ($n=104$)	N	76% of addiction patients showed disadvantageous deck preference	No standard control group used
Fein et al. (2004)	Abstinent alcoholics ($n=44$); healthy control participants ($n=58$)	N	Abstinent alcoholics made significantly fewer selections from advantageous decks	Y
Fishbein et al. (2005)	Abstinent drug abusers ($n=21$); healthy control volunteers ($n=20$)	Y	Trend for abstinent drug abusers to select less from the infrequent punishment advantageous deck. No group differences on SCR or heart rate	Not reported
Whitlow et al. (2004)	History of heavy marijuana use ($n=10$); healthy control participants ($n=10$)	N	Marijuana group showed preference for disadvantageous decks	Y

(*continued on next page*)

Table 1 (*continued*)

Authors	Groups studied	SCR used?	Deficit found in clinical group?	Control data replicated?
Rotheram--Fuller et al. (2004)	Opiate-dependent smokers ($n=9$); opiate-dependent non-smokers ($n=9$); control smokers ($n=9$); control non-smokers ($n=10$)	N	Methadone maintained smokers selected less from advantageous decks compared to all other groups	N, control non-smokers performed at chance levels
Schmitt et al. (1999)	Psychopathy offenders classified as low ($n=51$), medium ($n=68$), or high ($n=38$) on psychopathy checklist	N	All offender groups showed disadvantageous deck preference, but this was related to anxiety and not psychopathy levels	No standard control group used
van Honk et al. (2002)	Low psychopathic group ($n=16$); high psychopathic group ($n=16$)	N	High psychopathic group showed disadvantageous deck preference	Y
Best et al. (2002)	Intermittent explosive disorder patients (IED: $n=24$); healthy control volunteers ($n=22$)	N	Disadvantageous deck preference in IED patients	Y
Martin et al. (2004)	HIV-positive substance-dependent males ($n=46$); HIV-negative substance-dependent males ($n=47$)	N	HIV-positive group made fewer selections from advantageous decks, relative to HIV-negative group	No standard control group used
Gonzalez et al. (2005)	HIV-positive substance-dependent males ($n=109$); HIV-negative substance-dependent males ($n=154$)	N	Greater sensation seeking and better performance on the IGT was associated with more risky sexual practices	No standard control group used
Cavedini et al. (2004a,b)	In-patients with anorexia ($n=59$); healthy control volunteers ($n=82$)	N	Patients with binge/purge anorexia did not develop preference for advantageous decks over time. Patients with restrictive anorexia showed preference for disadvantageous decks	Y
Davis et al. (2004)	Body mass index (BMI) in; healthy adult women ($n=41$)	N	Higher BMI related to disadvantageous performance on the task	Y
van Honk et al. (2004)	Women given testestorene versus placebo (within-subjects cross-over design; $n=12$ in each condition)	N	Less advantageous performance found in testosterone condition, relative to placebo condition	Y
Dalgleish et al. (2004)	Psychosurgery for depression (stereotactic subcaudate tractotomy): recovered Psychosurgery group ($n=10$); depressed psychosurgery group ($n=10$); recovered medication group ($n=9$); healthy control participants ($n=9$)	N	Recovered psychosurgery group insensitive to negative feedback	Not reported
Campbell et al. (2004)	Huntington's disease group ($n=15$); healthy control volunteers ($n=16$)	Y	Huntington's patients selected less from advantageous decks in last 20 trials only and showed normal anticipatory but impaired feedback SCRs	Y, both behavioural and psychophysiological
Levine et al. (2005)	Patients with Traumatic Brain Injury (TBI; $n=71$)	N	Gambling task was sensitive to TBI in general, but not severity or quantified chronic phase atrophy of injury	No standard control group used
Mongini et al. (2005)	Women with history of chronic migraine ($n=23$); healthy female control volunteers ($n=23$)	N	None	Not reported
Apkarian et al. (2004)	Chronic back pain (CBS: $n=26$); complex regional pain syndrome (CRPS: $n=12$); healthy control volunteers ($n=26$)	N	Slower learning in CBP group, disadvantageous deck preference in CRPS group	Y
Kleeberg et al. (2004)	Patients with multiple sclerosis ($n=20$); healthy control participants ($n=16$)	Y	Slower learning and reduced anticipatory SCRs in patients with multiple sclerosis	Y
Ernst et al. (2003a)	Adolescents with externalising behavioural disorders ($n=33$); healthy control adolescents ($n=31$)	N	No group differences when first administered task. Adolescents with externalising behavioural disorder showed less marked improvement on second administration, relative to control adolescents	Y
Ernst et al. (2003b)	Adults with ADHD ($n=10$); healthy control participants ($n=12$)	N	None	Not reported
Toplak et al. (2005)	Adolescents with attentional-deficit-hyperactivity disorder (ADHD: $n=44$); healthy control adolescents ($n=34$)	N	Adolescents with ADHD made more selections from the disadvantageous high magnitude of punishment decks (B) and fewer selections from the advantageous high magnitude decks (D). Deck selection behaviour in ADHD correlated with hyperactivity/impulsivity symptoms	Y

(*continued on next page*)

Table 1 (*continued*)

Authors	Groups studied	SCR used?	Deficit found in clinical group?	Control data replicated?
Harmsen (in press)	Nicotine-dependent smokers ($n=61$); not nicotine-dependent smokers ($n=47$)	N	No difference between groups	No standard control group used
Jollant et al. (2005)	Patients with history of violent suicide attempts ($n=32$), patients with a history of non-violent suicide attempts ($n=37$), control patients with affective disorders but with no history of suicidal behaviour ($n=25$), and healthy control participants ($n=82$)	N	Both suicide groups showed disadvantageous deck preference, relative to healthy control and psychiatric control groups	Y

before conscious knowledge does, and without the help of such biases, overt knowledge may be insufficient to ensure advantageous behaviour…we believe that the autonomic responses detected in our experiment (especially those evident in the pre-hunch period) are evidence for a non-conscious signalling process" (Tranel et al., 1999, p. 1055).

In other words, learning via emotion-based biasing signals is believed to precede explicit insight on the IGT. If the reward/punishment schedule can be consciously comprehended by participants prior to the development of somatic markers, this could mean that cognitive outcome expectancies rather than somatic markers could guide successful IGT performance (Turnbull et al., 2003) and seriously undermine the support the paradigm can offer for covert 'somatic marker' activation.

As discussed earlier, preliminary evidence from the Iowa laboratory appeared to indicate that participants are not fully aware of the reward/punishment schedule (Bechara et al., 1997a). Recent data from other laboratories suggest that the reward/punishment schedule used in the IGT is more cognitively penetrable than previously thought, however. Maia and McClelland (2004) argued that the broad, open-ended questions used to measure conscious awareness of the task contingencies in the Iowa laboratory studies (e.g. Bechara et al., 1997a) were not sufficiently sensitive to identify all conscious knowledge held by participants. Using more detailed, focused questions in a replication study with 20 healthy participants answering questions after each block of 20 trials, they found that advantageous performance on the task was nearly always accompanied by verbal reports of reasonably accurate quantitative and qualitative knowledge about the outcomes of the decks that was sufficient to guide behaviour. Indeed, participants tended to report accurate knowledge about the reward/punishment schedule more reliably than they selected from the advantageous decks, mirroring the VMPFC lesion patients who reached the conceptual stage in the Iowa laboratory experiments (Bechara et al., 1997a). To control the possibility that asking more detailed questions somehow helped participants to construct a clearer problem space to comprehend the task structure, another 20 participants completed the task with the original questions given by Bechara et al. (1997a) and their behavioural performance was shown to be identical. The fact that participants have earlier awareness of the reward/punishment schedule means that the anticipatory SCR signals found on the IGT could be a consequence of conscious knowledge of the situation rather than being causally involved in the decision-making process.

Bowman et al. (2005) have also reported that participants are more aware of the IGT reward/punishment schedule than previously believed, rating the 'goodness' and 'badness' of decks at above chance levels as early as after the initial 20 trials.

Maia and McClelland (2004) concluded that the IGT can be performed through access to conscious, explicit knowledge, and it is therefore inaccurate to claim that task acquisition *necessarily* requires the generation of non-conscious somatic marker signals. Of course, these findings do not rule out the possibility that participants would sometimes utilise non-conscious bodily biasing signals to perform the task. A number of features about the IGT design make it likely to promote explicit rather than implicit reasoning, however (Maia and McClelland, 2004). First, it gives participants time to deliberate over each decision if they so wish and the outcomes are presented in explicit numerical form. Second, there is little variation in the magnitude of rewards and punishments used, making it relatively easy to track deck characteristics consciously and therefore potentially promoting explicit reasoning. In particular, participants only need to pay attention to the punishment delivered on each deck to successfully acquire the task (see Section 2.7).

In many ways, these criticisms reflect a broader literature debating the validity of the claim that so-called 'implicit' learning really can take place without conscious, 'explicit' awareness (see Shanks, 2005 for a critical review). Shanks argues that empirical evidence demonstrating that learning can take place without conscious knowledge is undermined by the use of inappropriate measures of awareness. These measures often do not index all the conscious knowledge the participant has (the 'exhaustiveness' criteria; Shanks and St. John, 1994) and do not always tap the same stored knowledge that is actually controlling relevant task behaviour (the 'information' criteria; Shanks and St. John, 1994). For example, the use of forced-choice stimulus identification may be too stringent a measure of conscious awareness in associative learning paradigms where partially

identified stimulus features could shape responding and where participants adopt a conservative response criterion (Lovibond and Shanks, 2002; Shanks and Lovibond, 2002). The measures of awareness included in the original IGT cognitive penetrability studies (Bechara et al., 1997a) appear to meet neither Shanks' exhaustiveness nor information criteria.

In response to the criticisms of the cognitive impenetrability of the IGT reward/punishment schedule, in particular the points raised by Maia and McClelland (2004), a robust rebuttal was generated by the Iowa laboratory (Bechara et al., 2005). This argued that the emotion-based rather than implicit nature of biasing signals is core to the SMH, so it is not problematic if the IGT is more transparent than previously believed: "The central feature of the SMH is not that non-conscious biases accomplish decisions in the absence of conscious knowledge, but rather that emotion-related signals assist cognitive processes even when they are non-conscious" (Bechara et al., 2005, p. 159). In many ways, this seems a retreat from earlier arguments emphasising the frequently implicit nature of somatic markers (e.g. Tranel et al., 1999) and makes the SMH hard to distinguish from other accounts of decision-making.

Bechara et al. (2005) also argued that SCRs appear sufficiently early in the IGT to precede the conscious knowledge identified by other laboratories, and so may still reflect implicit learning. This assertion is hard to reconcile with the fact that some understanding of the reward/punishment schedule was found as early as the first block of 20 trials by Maia and McClelland (2004). It is also unclear whether it is valid to draw a direct comparison between insight data gathered in different laboratories using very different insight assessments (Maia and McClelland, 2005). Finally, Bechara et al. (2005) argued that Maia and McClelland's explanation could not account for why correct knowledge of a situation did not guarantee correct decisions, instead suggesting that somatic markers need to be postulated to explain this scenario. An alternative explanation, however, is that apparently non-optimal rational decision-making behaviour in the face of partial explicit knowledge reflects exploratory behaviour by the participant to further garner information about the task at hand (Maia and McClelland, 2005). A recent case report has demonstrated that a patient with hippocampal amnesia was able to acquire the IGT and actually showed improved performance over multiple administrations of the paradigm. This was interpreted as reflecting non-conscious emotional learning in the absence of awareness. It has previously been argued, however, that dissociations of performance in amnesia cannot be taken as strong evidence for multiple memory systems (Shanks & St John, 1994). Even if amnesia is associated with spared non-conscious and impaired conscious learning systems, it still cannot be assumed that these non-conscious processes are necessarily emotional in nature. Finally, there are other ways to account for acquisition of the task in hippocampal amnesia other than through recourse to the SMH. For example, hippocampal lesions in rats facilitate instrumental learning with delayed reinforcement by reducing competition from context-reinforcer associations that normally hinder the formation or expression of response-reinforcer associations (Cheung & Cardinal, 2005). Therefore, normal acquisition of the IGT in amnesia is not conclusive proof that the IGT crucially relies on non-conscious emotional learning systems.

In summary, there seems little support for the claim that the reward/punishment schedule of the IGT is fully cognitively impenetrable. This means that the assertion that the IGT in the early stages involves covert, non-conscious somatic markers to regulate decision-making (Tranel et al., 1999; Bechara et al., 1997a) can no longer be confidently endorsed. Instead, it appears that participants have at least some conscious awareness of the reinforcement contingencies used in the task. It remains unclear whether this is best conceptualised as a full rational understanding of the reward/punishment schedule used or simply that participants are able to consciously label the valence of the decks (i.e. 'this one is good', 'this one is bad') in a heuristic fashion.

The 'fully explicit' interpretation would undermine the utility of the SMH, since if somatic markers arise only after full conscious knowledge about a situation is available it is unclear why people would ever need to make use of them. It would seem more parsimonious to act on the basis of the explicit knowledge rather than waiting for the emotional consequences of the decision to be communicated via the periphery. Further, it would mean that the somatic marker signal could be interpreted as a consequence of the explicit knowledge rather than being of causal importance in the decision-making chain, making the SMH indistinguishable from other accounts of task performance described later (see section 2.9). The heuristic interpretation, while challenging the impenetrability of the IGT, is consistent with the broader claim of the SMH that emotion (in this case a conscious verbal affective label) can guide decision-making. Indeed, Damasio (1994) argues that somatic markers may act at either an overt or covert level, so some awareness of the reward/punishment schedule in the IGT is not problematic for the SMH in its weakest sense.

2.3. Interpretation of psychophysiological data on the IGT

The pivotal finding in support of the SMH is that anticipatory SCRs differentiate between the advantageous and disadvantageous decks over time. The original paper reported this effect in 15 healthy control participants and found that it was absent in VMPFC lesion patients (Bechara et al., 1996). While the IGT has been used in a wide range of studies, only a subset has included psychophysiological measures. Studies that do report psychophysiology data have generally replicated the findings of elevated anticipatory SCRs to the disadvantageous decks, relative to the

advantageous decks (for example, Bechara et al., 1999, 2002; Bechara and Damasio, 2002; Campbell et al., 2004; Jameson et al., 2004; Hinson et al., 2002; Tomb et al., 2002; Suzuki et al., 2003; Crone et al., 2004). Further, there is some evidence that anticipatory marker signals correlate with both successful performance on the task (Carter and Smith-Pasqualini, 2004; Oya et al., 2005) and individual difference measures of neuroticism (Carter and Smith-Pasqualini, 2004). This indicates that the anticipatory SCR results are reasonably reliable and may relate to meaningful individual differences in sensitivity to reward and punishment, but exactly how to interpret them is complicated by a number of factors.

First, reliable anticipatory SCR differences have been reported only in a sub-group of the best performing healthy control participants in some studies. For example, Crone et al. (2004) gave 96 students a modified version of the IGT, while recording participants' anticipatory and feedback SCR and heart rate (HR) responses. Participants were split into three equal sized groups of poor, moderate and good performers, based on the total number of selections they made from the advantageous decks during the task. Anticipatory SCRs were greater for the disadvantageous than advantageous decks for the good performance group only. Similarly, anticipatory HR slowing was greater for the disadvantageous decks, relative to the advantageous decks, in the good performance group. Crucially, the moderately performing group (who nevertheless did successfully acquire the task) did not show any such psychophysiological differentiation between the decks. These findings are potentially problematic for the SMH since they show that a number of participants can acquire the task without needing to generate anticipatory HR or SCR signals, therefore suggesting that somatic markers are not necessary or sufficient to do well on the paradigm. In partial defence of this criticism, it can always be argued that other non-measured forms of peripheral feedback (e.g. facial) helped these participants to perform the task or that there may have been some important differences between the original IGT and the modified decision-making task used in this study.

Second, there appear to be other ways to interpret the elevation of SCR to the disadvantageous decks. The SMH claims that anticipatory SCRs on the IGT reflect growing awareness of the negative, long-term consequences of the 'risky' decks. Tomb et al. (2002) investigated whether anticipatory SCR changes on the IGT are correlates of correct versus incorrect decision-making or correlates of low-magnitude reward/punishment versus high-magnitude reward/punishment decision-making. If the anticipatory SCRs found on the IGT reflect the increased variance of reward and punishment offered on the 'disadvantageous' decks rather than their profitability over time, this does not offer support for the SMH.

To examine this issue, Tomb et al. (2002) contrasted the original IGT (where the bad decks are associated with a higher magnitude of punishment and reward than the good decks) with a modified version using a different reward/punishment schedule (linking the advantageous decks with a higher magnitude of punishment and reward than the disadvantageous decks). On the original task, participants picked more from the good than the bad decks and showed higher anticipatory SCRs for the bad decks. In the altered version, volunteers picked more from the good than the bad decks but showed higher anticipatory SCRs for the good decks, which is consistent with the interpretation that increased SCRs reflect the greater variance in the rewards and punishments offered rather than their 'goodness' over time. The Tomb et al. interpretation is also consistent with a number of behavioural studies describing how participants differed in number of selections made from the low frequency/high magnitude of punishment decks and the high frequency/low magnitude of punishment decks (e.g. Wilder et al., 1998; Shurman et al., 2005).

In response to the Tomb et al. findings, Damasio et al. (2002) argued that somatic markers can serve to record the long-term positive as well as negative consequences of a particular response option (so a larger SCR to the advantageous decks in the Tomb et al. study is not inconsistent with the SMH). Further, they argued that the larger SCR to the good decks in the Tomb et al. data may still reflect a non-conscious danger signal related to the likely risk of a large penalty, but that this signal is overridden by conscious assessment of the overall goodness of the decks (but see Maia and McClelland, 2004).

Other authors have argued that the psychophysiological response to feedback rather than in anticipation of deck selection is more important for regulating task behaviour. Suzuki et al. (2003) explored the influence of anticipatory and feedback SCRs on a Japanese version of the IGT completed by a population of 40 Japanese students. Consistent with the SMH, the 'risky' decks produced a greater anticipatory SCR than the 'conservative' decks. No relationship was found between the number of 'risky' selections in each block of the task and the amplitude of anticipatory SCRs, however. Further, there was no difference in anticipatory SCRs for early and late trials, which is inconsistent with the claim that anticipatory markers are acquired through experience (Bechara et al., 1996, 1997a). Instead, it was found that feedback SCRs (referred to as appraisal responses by Bechara) may be more important for mediating task performance. Feedback SCRs were greater following punishment than reward and on selections from 'risky' rather than 'conservative' decks. Participants who showed greater feedback SCRs tended to have a greater learning curve on the task (selecting fewer times from the 'risky' decks in late versus early trials). These results suggested that feedback rather than anticipatory SCRs may be more crucial in shaping decision-making on the IGT.

Against this conclusion, Crone et al. (2004) found that anticipatory rather than feedback physiological responding was related to task performance on their variant of the IGT.

All participants showed a greater SCR and greater HR deceleration following punishment than reward (particularly on decks that had a low frequency of punishment), but this did not differentiate between good, average and poor performers on the task. Further, findings of elevated anticipatory SCRs to the disadvantageous decks in the high performing sub-group stood even when feedback on the previous trial was entered as an additional factor in the analysis. In addition, other studies have found a correlation between anticipatory SCRs and successful acquisition of the task (Carter and Smith-Pasqualini, 2004; Oya et al., 2005).

Third, the anticipatory marker signals generated on the IGT may not be directly involved in the decision-making process. A simplified but equivalent decision-making task to the IGT has been used with rhesus monkeys, finding that SCRs were associated with anticipation of reward after a decision had been made rather than before a decision had been made (Amiez et al., 2003). This was interpreted as indicating that SCRs are a correlate of anticipatory appetitive behaviour rather than reflecting the decision-making process directly. This is an important finding and is potentially extremely problematic for the SMH, since it suggests that the 'anticipatory' changes found on the IGT could relate to expectancies about reward and punishment after a deck has been selected rather than driving deck selection behaviour in the first place. In other words, these signals may not play a causal role in shaping decision-making behaviour.

At face value it is difficult to separate responses prior to response selection and prior to feedback, due to the relatively slow time course of SCR signals. One way to explore this issue, however, would be to acquire SCR data at a higher temporal resolution and apply various signal processing strategies to separate the components of the signal that relate to selection of response versus anticipation of feedback following selection (e.g. see Lim et al., 1997) or use other psychophysiological responses with a faster time course than SCRs.

Finally, another potential problem with the psychophysiology findings on the IGT is that investigators typically only report SCRs (see Crone et al., 2004 for an exception to this). Work from other laboratories has generally shown that the SCR is not particularly sensitive in discriminating between positive and negative valence (e.g. Bradley et al., 2001a,b), so this measure may not be the best candidate to index an underlying emotion-based marker 'signal' that indicates if a decision is 'good' or 'bad'. There are a variety of other sources of bodily feedback that could be measured, including facial muscle activity with electromyography (EMG) or heart rate with electrocardiogram (ECG). Given the uncertain nature of the interpretation of SCRs, stronger support for the SMH would be provided by evidence of anticipatory signals emerging across a range of bodily systems. Further, it is increasingly realised in the psychophysiology literature that bodily responses to particular environmental demands cannot be reliably modelled on a uni-dimensional arousal spectrum and instead are better conceptualised as a complex of related responses (see Lacey, 1967 for a critical review of activation theory). For example, attentional orienting, rather than leading to a simple increase or decrease in bodily response, is characterised by decreased motor activity, increased SCR, delayed respiration, and heart rate deceleration (Graham, 1979). Therefore, the anticipatory SCR changes seen in the IGT may form part of a broader response complex, which may not be valid to interpret as a simple increase or decrease in bodily response.

In summary, there has been limited external replication of the key anticipatory SCR data on the IGT. Further, when the pattern of findings is successfully replicated, it remains unclear exactly what these SCRs represent. They may be a response to feedback, an indicator of risk, a marker of post-decision emotion state, or a signal of how good or bad a particular response option is. Additional clarification of the mechanism by which these anticipatory marker signals act to bias decision-making is necessary if these data are to be used to support the SMH. We will return to this point later in the manuscript (section 4.2 on 'Specification of the somatic marker mechanism'). To more fully support the SMH measurement of other bodily responses in addition to SCR is needed to better model the complex bodily activation typically seen in response to environmental stimuli. Further, causal designs that manipulate somatic state and look at the effects on IGT performance are also required (see Section 2.6)

2.4. Variability in control participant performance on the IGT

It is increasingly apparent that substantial minorities of control participants do not learn to select from the advantageous decks over time on the IGT. Bechara and Damasio (2002) reported that not all control participants perform advantageously on the IGT and this did not clearly relate to the development of anticipatory SCRs. They found that around 20% of normal adults performed disadvantageously on the IGT, selecting more from bad decks than good decks. Within this poorly performing sub-group, the variance in anticipatory SCRs was high, with some showing normal SCRs and some showing a profile similar to VMPFC lesion patients. Those participants with impaired behavioural performance but normal anticipatory SCRs were characterised post hoc as 'high-risk takers', choosing to override 'somatic marker' information with conscious deliberation.

Similarly, a variety of studies have reported that there are sub-groups of healthy control participants who do not show a preference for the advantageous decks at the end of the task (e.g. Crone et al., 2004; Adinoff et al., 2003) or an overall reduction in the number of selections made from advantageous decks in healthy populations (Lehto and Elorinne, 2003: see Table 1). While these findings are

perhaps not directly problematic for the SMH (since it does not claim to be a truly nomothetic model of decision-making), they do make interpretation of IGT data complex. Where no differences between a patient/lesion group and a control group are found, does this genuinely reflect an absence of a deficit in the experimental group, or rather that a poorly performing group of control participants was selected? This variability in control data suggests that performance on the IGT should perhaps be compared to objective criteria (for example, selecting from advantageous decks at greater than chance levels), as well as relative to the performance of a comparison group. Further, the variability in behavioural performance of control participants raises doubts about the ecological validity of the paradigm. If a group of control participants can perform extremely poorly on the task and yet presumably does not always show gross difficulties in day to day decision-making, it is then unclear to what extent the psychological mechanisms measured by the paradigm bear any relevance to everyday, real-world decision-making. Of course, it remains possible that control participants could exhibit impaired decision-making in everyday life, so perhaps measures of quality of everyday decision-making should be included in studies to check for this possibility.

2.5. Specificity and reliability of the IGT in clinical populations

Another issue with the IGT is that performance deficits do not appear to be specific to particular neurological or psychiatric conditions, since a majority of patient groups show impairments on the measure. This partially undermines its utility in understanding decision-making difficulties across different disorders. Moreover, there have been some failures to replicate findings within a particular clinical group, suggesting that findings may not be that reliable. For example, of six studies examining schizophrenia, one found no deficit (Wilder et al., 1998), one found a disadvantageous deck preference (Ritter et al., 2004), one found deficits were dependent on medication type (Beninger et al., 2003), one found preference for the infrequent punishment decks (Shurman et al., 2005), and two found only sub-types of schizophrenic patients were impaired on the task (Bark et al., 2005; Turnbull et al., in press).

2.6. The causal relationship between body-state feedback and IGT performance

Another potential issue with the IGT is that the psychophysiology data implicating 'somatic marker' generation with successful performance are correlational only. This means that no causal conclusions about the role of body-state feedback on decision-making can be reliably drawn. Although healthy volunteers seem to show anticipatory SCR activity when performing well on the task, this may reflect the end product of the decision-making process rather than being a key feature in its development. For example, the SCR changes may reflect anticipation of reward and punishment to be delivered after a deck has been selected (Amiez et al., 2003). Further, it may be the case that some other mechanism regulates both behavioural selection and somatic marker generation on the IGT. It could also be argued that the most parsimonious account of the IGT data is that greater somatic marker activation actually leads to impaired behavioural performance (since greatest somatic marker activation actually temporally precedes selection from the disadvantageous decks). Causal methodologies are needed to refute these criticisms.

A number of attempts have been made to demonstrate a causal link between body-state feedback and IGT performance, but have so far provided mixed support for the SMH. The primary methodology has been to look at groups with impaired feedback from the body and see if this leads to sub-optimal performance on the IGT. Feedback from the body can arise from multiple routes (e.g. Bechara, 2004; Damasio, 2004; for a more detailed review see Craig, 2002), including spinal cord, vagus nerve, endocrine system, feedback from facial muscles, and from the physiochemical environment of the brain (for example, temperature).

Preliminary work found that 20 individuals suffering from peripheral neuropathy, a condition that reduces afferent feedback to the brain, were mildly impaired on the IGT compared to control volunteers (Bechara et al., 1998a). This study has so far only been published as conference abstract without peer review, so a full analysis of its methodological merits is not possible. This means it should not yet be viewed as strong supporting evidence for the SMH.

Subsequently, IGT performance has been examined in six patients with pure autonomic failure (PAF), a condition that leads to a peripheral denervation of autonomic neurons and therefore an absence of peripheral autonomic responses (Heims et al., 2004). Contrary to prediction, PAF patients performed better than a comparable control group on the IGT (i.e. selected from the advantageous decks to a greater extent). The fact that PAF patients were significantly superior on the IGT relative to controls means that the findings cannot be explained away as a lack of statistical power due to a small sample size. Further, it has been demonstrated that long standing PAF also leads to changes in the morphology of brain regions involved in the representation and regulation of body state (Critchley et al., 2003). Voxel-based morphometry on the structural MRI scans of 15 PAF patients revealed decreases in grey matter volume and concentration in both the anterior cingulate and insula cortices, perhaps as a result of loss of afferent input to brain regions involved in autonomic representation. This is significant for the SMH because it means that PAF patients have both body-state feedback and regions of the 'as-if' loop compromised, so some kind of

impairment would have been expected. Therefore, the finding that PAF patients are not impaired on the IGT is difficult for the SMH to explain. Heims et al. (2004) argue, however, that their data do not disprove the SMH since other sources of peripheral feedback are still intact in PAF patients, in particular somatic systems.

There is some tentative evidence that the vagus nerve, a major feedback pathway for signals from the peripheral nervous system to the brain, is important in the generation of somatic markers (Martin et al., 2004a). Patients who had vagus nerve stimulators implanted to treat epilepsy performed two repeat versions of the IGT, one when vagus nerve stimulation was covertly delivered and one without stimulation. The stimulated group initially selected more from the disadvantageous decks and then moved to the advantageous decks, whereas the group without stimulation initially selected more from the advantageous decks and then moved away from them as the task progressed. This improvement in performance over time in the stimulated relative to the un-stimulated condition was interpreted as evidence that the vagus nerve influences decision-making (Martin et al., 2004a). This cannot be viewed as strong support for the SMH, however, since there was no overall group difference.

The study of patients with spinal cord damage has failed to generate support for the SMH. Patients with spinal cord sections at the sixth cervical vertebrae, thus blocking somatic feedback, displayed no deficit on the IGT relative to healthy controls (North and O'Carroll, 2001). This is despite the fact that spinal cord injury has been sometimes shown to reduce the intensity of emotion experience (Hohmann, 1966; Chwalisz et al., 1988; Montoya and Schandry, 1994; although see Cobos et al., 2002 for a recent exception). North and O'Carroll (2001) suggested that their data can be reconciled with Damasio's model if it is assumed that feedback from the hormonal route and nerves outside the spinal cord (e.g. facial muscles) is more important than the afferent feedback sent via the spinal cord. It is also possible that people who have lived with long-term spinal damage have adapted to the loss of peripheral feedback and make more extensive use of the 'as-if' loop (see Fig. 1). Thus, a deficit may have been revealed if the patients were studied immediately after injury. Imaging studies of such patients could examine whether activity in the 'as-if' loop mediates intact performance on the IGT, as predicted by the SMH.

An important development for SMH will be to examine the conditions under which the body loop or 'as-if' loop are involved in decision-making. Bechara (2004) suggests that the body loop will be engaged in decision-making under uncertainty, whereas decision-making under certainty (i.e. where the outcome is explicit and predictable) will engage the 'as-if' loop, although it is currently unspecified as to why this should be the case.

In summary, studies of IGT performance in patients with altered feedback from the body have not provided strong support for the SMH, so the causal status of the model remains unclear. Interpretations of these experiments perhaps raise issues about the testability of the SMH. Nearly all of the negative findings can be explained away through some aspects of peripheral feedback remaining intact in the patient groups studied (for example, the somatic system in PAF, endocrine feedback following spinal injury) or through recourse to the 'as-if' loop. It seems virtually impossible to test the theory in a scenario where all peripheral feedback routes are disturbed and the 'as-if' loop cannot be utilised, therefore making it difficult to disprove.

2.7. Task design issues

A number of methodological features of the task design and psychophysiology analysis of the IGT also complicate interpretation of the data it generates. First, in the psychophysiology analysis the deck that participants eventually select is used to designate each anticipatory 'somatic marker'. In the deck selection phase, however, people are free to shift their attentional focus across all of the decks before settling on one. This means that the physiological marker generated may not reflect attention to a single deck but a shifting attentional focus across all decks before arriving at a choice.

Second, the magnitude of rewards given in the task is predictable (either 50 or 100 points), meaning that to do well on the tasks participants simply have to attend to the punishment component of the schedule (see Fig. 2). This possibility is consistent with the finding that neuroticism positively correlates with successful IGT performance (Carter and Smith-Pasqualini, 2004), given that neuroticism is characterised by a sensitivity to punishment. This issue introduces two complications to the interpretation of the data: the reward/punishment schedule may be easier to comprehend consciously than previously claimed and the task only measures variance in response to punishment rather than reinforcement more generally. This possibly explains why recent findings indicate that the task reinforcement schedule is easier to consciously grasp than originally assumed (e.g. Maia and McClelland, 2004).

Third, deck position is not counterbalanced in the task, meaning preferential selection from good or bad decks could reflect a location bias rather than a genuine decision-making deficit.

Fourth, the way in which the decks are classified as advantageous or disadvantageous is problematic (Maia and McClelland, 2004). At any point in the game, participants have nothing but their prior experience to decide whether the decks are good or bad. In the early trials, the bad decks actually are the most advantageous and it is only later that they become disadvantageous. Therefore, analysis of each trial should standardly classify each deck based on net outcomes up until that point in the game, as is typical in the decision-making literature, rather than on eventual reinforcement, as currently done in the Iowa laboratory.

Such an approach has recently been taken in a reinforcement learning model of the IGT (Oya et al., 2005). In this light, early selection from the disadvantageous decks can be interpreted as 'rational' exploratory behaviour rather than impaired decision-making. Another way to control for this issue is to exclude the first block of 20 trials from the analysis and see if a similar behavioural pattern emerges.

Fifth, participants can only select 80 times from each deck. This raises the possibility that behaviour in the last 20 trials, a point in the game where some participants will run out of cards in their preferred advantageous deck, may deteriorate due to this confound. This reduces sensitivity to detect impairment, since it effectively 'penalises' individuals who learn early in the game. One way round this possibility would be allow participants to make 100 selections from each deck.

Sixth, there are various statistical issues that undermine the IGT data. In the behavioural analysis, learning on the task is indexed in a relatively insensitive fashion by looking at the number of advantageous deck selections in each block of 20 trials, which could perhaps mask successful acquisition in patient populations. A more sensitive analysis would be to carry out some kind of trend analysis (e.g. a linear contrast). Further, rate of learning on the task is always compared relative to the performance of the control group. It is possible that patient groups are still learning the task but just at a slower rate than control volunteers. To explore this possibility it is necessary to look at deck selection relative to chance as well as relative to control participant performance.

In the psychophysiological analysis, there is a problem of an unequal number of means in each cell of the analysis of variance (ANOVA). In particular, participants make a large number of selections from the advantageous decks and only a handful of selections from the disadvantageous decks. This means that the differences in SCRs to the advantageous and disadvantageous decks may be partially an artefact of the statistical assumptions of ANOVA being violated. Moreover, the elevations in response to the disadvantageous decks (which are typically only selected early on in the task) may be an artefact of novelty/habituation. To control for this possibility, it would be necessary to devise a variant task where the different decks are equally sampled from across different periods of time.

To better support the SMH, it needs to be seen whether a similar pattern of findings emerges when these methodological issues are addressed.

2.8. Alternative mechanisms potentially underlying IGT task performance

The SMH purports that impaired performance on the IGT relates to an inability to link past emotional response to punishing stimuli with various future response options via somatic marker biasing signals, leading to a 'myopia for the future' (Damasio, 1994; Bechara et al., 1996). This account is derived largely from observation of patients with VMPFC damage and interpretation of the behavioural deficits they display on the IGT. Consistent with this interpretation, it has recently been shown using an 'expectancy-valence' model that the choices of VMPFC patients on the IGT are guided predominantly by the most recent outcome rather than by outcomes on all past trials (Busemeyer and Stout, 2002).

Damasio's account of the deficits displayed by patients with VMPFC damage has recently been questioned (MacMillan, 2000), however, and alternative accounts to explain the deficits displayed by VMPFC lesion cases have been put forward (e.g. Gomez-Beldarrain et al., 2004; Camille et al., 2004). Moreover, there is evidence that some VMPFC patients show impaired performance on the IGT even when their emotional reactions are in the normal range. Naccache et al. (2005) report the results of detailed investigation on patient RMB, who had suffered damage to left mesio-frontal cortex (encompassing the ACC, the genu of the corpus callosum, and part of the orbitofrontal cortex). RMB showed impaired performance on the IGT, replicating results from the Iowa laboratory (e.g. Bechara et al., 1994). Problematically for the SMH, RMB showed impaired performance on the IGT even though the affective response system of RMB seemed to be largely intact (e.g. a normal self-report and physiological response to emotion inducing pictures was found). This suggests that there can be deficits on the IGT in the absence of deficits in somatic marker generation, indicating these emotion-based biasing signals are neither necessary or sufficient to complete the task.

Given that Damasio's interpretation of the function of the VMPFC has come into question, it seems sensible to examine whether alternative functional explanations may also be able to account for impaired performance on the IGT. If other mechanisms provide an equally valid account of why behavioural deficits may be shown on the IGT, this undermines support it can offer the SMH. A range of evidence that suggests other mechanisms could account for the findings on the IGT will be evaluated below.

2.8.1. Working memory

The realisation that the reward/punishment schedule on the IGT is less impenetrable than previously thought (Maia and McClelland, 2004) raises the possibility that explicit learning mechanisms are more important for task acquisition than previously believed. In particular, a number of studies have examined whether intact working memory is necessary to do well on the IGT.

A role for working memory on performance of the IGT has been shown by Hinson et al. (2002). They asked healthy control participants to perform a modified, more difficult variant of the IGT (where only one deck was profitable and the differences in profitability between the four decks were less extreme) whilst simultaneously completing a secondary task with or without a working-memory load. The working-memory condition (holding a string of digits in memory),

compared to the no working-memory condition (repeating digits flashed up on the screen), led to poorer behavioural performance and impaired acquisition of anticipatory somatic markers on the modified IGT. The authors concluded that working-memory processes therefore contribute to the development of somatic markers.

In a follow-up study, Jameson et al. (2004) explored whether secondary task performance influenced working-memory capacity by reducing central executive capacity or interefering with short-term capacity in the phonological loop. They achieved this by comparing the effects of articulatory suppression (rehearsing the word 'the' over and over again to occupy the phonological loop) to the two conditions used in the original experiment in a within-subjects design using 20 healthy control participants. More 'good' choices were made during the keypad (no working-memory load) and articulatory suppression conditions, compared to the digit maintenance task (working-memory load). Moreover, performance improved over time and there was a difference between anticipatory SCRs between the 'good' and 'neutral/bad' options on all conditions except the digit maintenance task. The authors concluded that secondary tasks interfere with IGT performance because of the demands they place on central executive function rather than via blocking the phonological loop. This is reconciled with the finding that VMPFC patients with normal working memory show impaired IGT performance by stating that central executive resources are necessary but not sufficient for the development of somatic markers.

The Hinson et al. (2002) and Jameson et al. (2004) studies can be criticised on the grounds that they did not use the original IGT paradigm, however, and it is possible that the mechanism through which their variant task is learned differs in some important way. A recent study looking at dual-task effects on the original IGT has found a less clear role for working-memory. Turnbull et al. (2005) tested the contribution of working-memory-dependent and working-memory-independent processes to performance on the IGT using a dual-task methodology. Seventy-five healthy control participants were randomly allocated to one of three experimental conditions: IGT with no secondary task, IGT with a non-executive secondary task (articulatory suppression), and IGT with an executive secondary task (random number generation). Results found that the rate of learning in the three groups was not significantly different, with all three groups successfully acquiring the task. This was interpreted as offering support for the claim that IGT performance is relatively independent of working memory. Further, both secondary tasks presumably involved some activation of working memory, so the fact that they did not impair performance supports the claim that the IGT is largely independent of working-memory capacity. There was a trend for the no secondary task group to show superior performance, however, which perhaps partially undermines this interpretation.

Other evidence consistent with a working-memory account of IGT impairment are findings that task performance relates to regions of prefrontal cortex believed to regulate working memory and general intelligence, such as dorsolateral prefrontal cortex (DLPFC). Bechara et al. (1998a) compared patients with damage to DLPFC and VMPFC on the IGT and two tests of working memory. It was shown that working memory did not depend on the intactness of decision-making; participants could have normal working memory in the presence or absence of decision-making impairment. On the other hand, decision-making was affected by working memory. Performance on the IGT tended to be worse in those with working-memory problems. In summary, an asymmetrical relationship between working memory and decision-making was found. VMPFC damage led to decision-making impairments, which were exacerbated by working-memory problems in those with more extensive lesions. Patients with right-sided DLPFC damage showed impaired working-memory, which also led to low-normal results in the decision-making task. Damage to broader regions of the prefrontal cortex including DLPFC has also been found to impair IGT performance in studies from other laboratories (e.g. Manes et al., 2002). A similar asymmetric dependence between working memory and decision-making has been found in individuals with substance abuse (Bechara and Martin, 2004). Moreover, IGT acquisition has been found to correlate with resting state activity in DLPFC (Adinoff et al., 2003).

In summary, while dual-task methodologies have provided mixed support for the role of working memory on the IGT, lesion and neuroimaging findings suggest that more dorsal regions of the frontal lobes known to regulate working-memory do appear to be important for successful task performance. It is debatable to what extent these findings are problematic for the SMH, since Damasio (1994) explicitly states that one function of somatic markers is to indicate which options should have working memory and attentional resources allocated to them. In other words, successful acquisition of the IGT may involve first the development of somatic markers to distinguish between good and bad options and second the use of these signals to devote system resources to the better options. In this light, there is no need to posit independence between these two systems.

2.8.2. Reversal learning/inhibition

Another candidate mechanism that could underlie impaired performance on the IGT is a difficulty in reversal learning. A crucial aspect of the IGT is that participants have to perform a response reversal: they have to shift their preference away from the decks that are initially rewarding in the first few trials following subsequent punishment. A number of studies have found impaired reversal learning in VMPFC patients, indirectly suggesting this may account for their deficit on the IGT. Patients with ventral PFC damage

were shown to have difficulty in simple reversal learning (Rolls et al., 1994), although it has been argued that the patients studied had lesions extending more laterally in orbitofrontal cortex than those studied by the Iowa laboratory (Clark et al., 2003). A later study has now shown that lesions restricted to VMPFC allow normal acquisition but impaired reversal on simple reversal learning tasks (Fellows and Farah, 2003). Similarly, Hornak et al. (2004) reported that bilateral lesions to OFC but not DLPFC reliably impair reversal learning on a task previously found to activate OFC in earlier fMRI studies of healthy control participants. There is also some evidence that reversal learning impairments correlate with real life decision-making problems in patients with VMPFC damage (e.g. Lawrence et al., 1999).

To directly test the possibility that a reversal deficit explains impaired IGT performance, Fellows and Farah (2005a) rearranged the initial reward/punishment schedule on the task such that the two disadvantageous decks no longer had an initial advantage in the opening trials. Following this shuffling of the opening trials, the performance of VMPFC patients was the same as that of control volunteers, suggesting that it is a difficulty in reversing early learning that is underpinning the behavioural profile of VMPFC patients on the IGT.

This inability to show reversal learning can be understood as a deficit in response inhibition (Rescorla, 1996). A common response to initial feedback is to continue a response when it is rewarded and to change a behaviour when it is punished (referred to as win-stay/lose-shift behaviour in the animal decision-making literature, Restle, 1958; Gaffan, 1979; Reid and Morris, 1992). There is some evidence to suggest that over time animals are able to override this pattern to maintain long-term goal-directed behaviour in the face of short-term setbacks (e.g. Killcross and Coutureau, 2003). One way to understand successful acquisition of the IGT is that participants have to be able to learn over time to inhibit the lose-shift pattern of responding to the advantageous decks following punishment. Patients with VMPFC lesions may be unable to show this inhibition, meaning their responses are driven by feedback in the moment rather than longer-term profitability. The details of such inhibitory mechanisms have been well specified and may represent a genuine alternative to the SMH to model impaired performance on the task (Rescorla, 1996).

In defence of the reversal critique, Bechara et al. (2005) argued that, while the IGT does contain elements of contingency-reversal, there are other aspects of the task that need to be learned to explain successful performance. Consistent with this defence are results from a recent study examining performance on a variant of the IGT that builds in a more marked reversal learning component (Turnbull et al., in press). After completing 100 trials of the standard task, participants then completed a further series of trials where the original contingencies are modified (the decks that are good and bad are systematically changed) over three shift phases. Schizophrenic patients with marked negative symptoms showed no deficit on acquisition of the original task, but showed chance performance on the shift trials. This preliminarily suggests that the original IGT measures some aspects of decision-making that are dissociable from simple reversal learning (i.e. the task can be acquired despite the presence of a deficit in reversal learning). It could be the case, however, that schizophrenic patients have a milder reversal learning deficit and that this problem builds up over time on the task. Further work more closely examining control participant and VMPFC lesion patient performance on this modified task is warranted to support this conclusion.

Bechara et al. (2005) further refuted a simple reversal explanation by asserting that it cannot explain why some patients perform disadvantageously on the complex reversal on the IGT, even when they consciously conceptualise the reward/punishment schedule on the task towards the end of the experiment. Models of reversal learning (e.g. Rolls, 1999) posit more than one output system, perhaps operating at different levels of consciousness, however, which can therefore account for this pattern. Bechara et al. (2005) also argued that for reversal to take place requires a 'stop signal' to be acquired, which could take the form of an emotion-based biasing signal. In other words, the acquisition of somatic markers may underpin successful reversal learning. Against this defence, it has been demonstrated that reversal learning does not depend on an intact amygdala (Izquierdo et al., 2004), suggesting reversal is independent of emotion processing systems. Reversal accounts therefore offer an explanation of IGT performance distinct from the SMH.

2.8.3. Risk-taking

Another explanation of impaired performance on the IGT following VMPFC damage is that risk-taking behaviour is changed. Alterations in deck selection behaviour may simply reflect individual differences in preference for risk rather than 'good' or 'bad' decision-making behaviour. For example, sensation-seeking individuals may prefer decks that generate the most arousal or interest (i.e. 'risky' cards) rather than are the most profitable (i.e. 'safe' cards) (Zuckerman, 1994). Thus, selection from the 'disadvantageous' decks can be perfectly rational depending on the individuals' preference structure. This may be particularly likely to be the case given that play rather than real money is used. Differences in preference structure, as opposed to underlying decision-making, could underlie the variation found in control participant performance on the task and also account for impaired performance in patient groups. Indeed, the 'impaired' decision-making style shown by VMPFC lesion patients may in some circumstances be more adaptive than that shown by healthy control participants. For example, Shiv et al. (2005) found that VMPFC lesion patients showed superior performance on a financial investment paradigm, as their decisions were less affected by previous reward and punishment delivered than were those of healthy control participants.

One paradigm that has been used to investigate risk-taking is the Cambridge Gambling Task (CGT: Rogers et al., 1999). On each trial of the CGT an array of 10 red and blue boxes is presented, behind one of which a token is randomly hidden. Participants are asked to judge which colour box the token will be hidden behind and then bet some of their points total on the outcome. The numbers of each colour of box are systematically varied across trials, making it more or less likely the token will be located behind a particular colour. Therefore, the task measures accuracy of decision-making via a relatively simple probabilistic judgement and then measures risk-taking via analysis of betting patterns. Healthy control subjects have been found to select the more likely outcome and to conservatively adjust their betting according to the ratio of red and blue boxes, for example betting more when the colour ratio is 9:1 rather than 6:4.

Patient studies have generally found a pattern of normal but slowed accuracy of decision-making accompanied with increased, riskier betting behaviour on the CGT in conditions that damage ventral portions of the prefrontal cortex (for a review, see Clark and Manes, 2004). This profile has been shown by patients with frontal variant Fronto-Temporal Dementia (Rahman et al., 1999), and large prefrontal lesions including VMPFC (Manes et al., 2002). These findings are consistent with the hypothesis that ventral PFC damage leads to a riskier, more impulsive decision-making style. Against this conclusion, however, a study that looked at patients with VMPFC lesions found impaired decision-making accuracy and decreased betting (Rogers et al., 1999). One way to reconcile these findings is to propose that risky behaviour may be reduced in those with a severe deficit in decision-making as a compensatory strategy (Clark and Manes, 2004).

Similarly, Sanfey et al. (2003) looked at VMPFC lesion performance on another variant of the IGT to test the risk hypothesis. Participants were asked to select from four decks of cards that differed in their variance of reward and punishment over time but not in their overall profitability. Therefore, participants' risk preference can be indexed in isolation from the accuracy of their decision-making. Control participants showed a marked avoidance of risk, picking from the more secure, low variance decks. The VMPFC lesion group could be split into those who were also risk-averse and those who showed marked risk-taking behaviour (selecting mostly from the high variance decks). Intriguingly, these two sub-groups did not differ in any clear way in terms of lesion location, although the risk-taking group tended to have lesions extending to the DLPFC. This finding contrasts to the IGT data, where VMPFC lesion patients were insensitive to risk on both the advantageous and disadvantageous decks. It is important to note, however, that a number of frameworks have conceptualised risks as feelings to guide decision-making (e.g. Loewenstein et al., 2001), so the risk explanation may still be broadly compatible with the weak form of the SMH.

2.8.4. Insensitivity to rewarding and punishing outcomes

As discussed in the earlier psychophysiology section, yet another possibility is that it is the response to reward and punishment, rather than anticipatory change in the body, that is regulating performance on the task. Differences in SCRs to winning and losing on the IGT have indeed been described (see Bechara, 2000b) and may support this interpretation. Further, differential responses to positive and negative feedback may explain impaired performance on the IGT in various patient groups. For example, Dalgleish et al. (2004) found that an insensitivity to punishing outcomes on the IGT characterised the performance of patients who had recovered from severe depression following psychosurgery in comparison to healthy control volunteers, depressed patients who had not recovered following the surgery, and depressed patients who had recovered with medication. Successful performance on the IGT has also been found to correlate with self-report of increased sensitivity to reward (Franken and Muris, 2005).

The Suzuki et al. (2003) data on the IGT discussed earlier are consistent with a feedback hypothesis, since feedback SCRs were more clearly related to behaviour performance on the task than anticipatory SCRs. An alteration in the response to rewarding and punishing outcomes would be consistent with a number of other models of decision-making. For example, decision affect theory (see Mellers et al., 1997) suggests that the emotions people experience after a decision depend on a comparison between what the consequences actually were and the consequences that would have come about if another response option was chosen. These emotions then determine how likely it is that a given response option will be repeated or changed (i.e. an option will not be repeated if it induced regret).

Some data are inconsistent with an insensitivity to rewarding and punishing outcomes, however. Crone et al. (2004) showed that anticipatory physiological changes differed between the advantageous and disadvantageous decks even when feedback on the previous trial was controlled for, which undermines the plausibility of response to, rather than anticipation of, feedback as the central mechanism for learning the task. Similarly, performance on the variant IGT task where reward and punishment were reversed also undermines this feedback explanation (Bechara et al., 2000b).

2.8.5. Apathy

Another mechanism that could explain impaired performance on the IGT is apathy (lack of motivation). Rather than being unable to make the correct decision, VMPFC patients and other impaired groups may simply not care enough about the negative outcomes to actively avoid them. Consistent with an apathy deficit, Barrash et al. (2000) report that apathy is a symptom exhibited by VMPFC lesion patients from the Iowa laboratory and the deficit in emotional response to affective images in VMPFC lesion patients can be ameliorated when they are directed to look

carefully (Damasio et al., 1991). Apathy could produce both the behavioural deficit and the failure to generate anticipatory SCRs seen on the IGT in VMPFC lesion patients and it is possible that IGT performance would similarly improve if engagement levels were raised. Also consistent with the apathy argument is a recent study drawing a distinction between different aspects of future-directed thinking (Fellows and Farah, 2005b). VMPFC damage was found to leave temporal discounting (the subjective devaluation of reward as a function of delay) intact but to impair future time perspective (a measure of the length of an individual's self-defined future). Crucially, this deficit in future time perspective was found to correlate with symptoms of apathy rather than impulsivity, suggesting that the syndrome of apathy may deserve more attention in understanding impaired future thinking and decision-making following frontal lobe damage.

2.8.6. Overview of alternative mechanisms accounting for IGT deficits

In summary, there is evidence that a range of mechanisms other than a 'myopia for the future' due to an inability to acquire somatic markers may explain impaired performance on the IGT.

One way to measure which of these competing explanations best explain deficits on the IGT is to correlate performance within groups on the IGT and measures of the other related constructs. For example, Monteresso et al. (2001) compared performance of 32 cocaine-dependent patients on the IGT, the CGT, and a delayed discounting procedure (where participants chose between smaller-sooner and larger-later rewards). These data allowed preliminary examination of whether IGT deficits reflect altered risk-taking behaviour (in which case there should be a correlation with the CGT) or myopia for the future (in which case there should be a correlation with the delayed discounting procedure). There were significant correlations between IGT performance and delayed discounting procedure performance. The link between the IGT and the CGT was less clear-cut, with reaction times but not behavioural choices on the CGT correlating with IGT performance. This suggests that there is some commonality in the construct that the different measures are indexing, with IGT impairment perhaps being more clearly linked with a difficulty in considering future outcomes than increased risk-taking. These data only speak to what impairs IGT performance in this specific group of cocaine-dependent patients, however, and different relationships may exist in a healthy population. This approach could usefully be extended to a larger sample of healthy control participants to test this possibility.

It is important to observe that these explanations of IGT deficits are not necessarily mutually exclusive. It is possible that a variety of different mechanisms are involved in successful task acquisition and that damage to any one of these can impair task performance. Further, different mechanisms may be adopted at different points in the task and across different individuals. It may also be the case that other cognitive functions form integral parts of the somatic marker apparatus. For example, the fact that working memory appears to be needed to perform the task well can be interpreted either as a factor that confounds the tasks or as a integral part of the mechanism underlying decision-making via somatic markers (Clark and Manes, 2004). In many ways, a deficit in somatic marker activation could underlie a majority of the other explanations put forward to account for the data (e.g. 'risks as feelings'; Loewenstein et al., 2001). What is needed to tease apart these mechanisms is the development of experimental designs where the different mechanisms make competing predictions, plus a more detailed specification of the SMH to allow its predictions to be differentiated from those of other theories of decision-making.

2.9. Current status of the IGT data

The previous sections have described how the IGT reward/punishment schedule has been found to be less opaque than previously believed; how a variety of other mechanisms other than somatic markers could equally well explain task behavioural performance and psychophysiology results; that there is an absence of causal evidence linking disturbed feedback from the body to impaired performance; that there are a number of task design issues that complicate interpretation; and that control participant performance on the task is more variable than previously believed. For these reasons, it is argued that the IGT is no longer sufficient to be a major source of evidence for the SMH and additional empirical support should be sought.

3. Evaluation of the proposed neural substrate of the SMH

A considerable strength of the SMH is that the neural substrate considered to mediate such markers has been specified in some detail (for an overview, see Damasio, 1994, 2004; Bechara and Damasio, 2005). Damasio draws a distinction between two different kinds of stimuli that require a decision-making response, each of which is believed to be regulated by different regions of the brain. 'Primary inducers' are innate or learned stimuli that generate pleasurable or aversive states, whereas 'secondary inducers' are the thoughts and memories induced by the recall or imagination of an emotional event.

As noted earlier, the pivotal area in the putative SMH network is the portion of the frontal lobes above the eye sockets, referred to as the VMPFC by the Iowa laboratory. The VMPFC is believed to be the structure that encodes an association between secondary inducers and the bioregulatory state linked with that situation in the past experience of the individual (including the bodily aspects of emotional

response). It is therefore centrally involved in the generation of somatic markers when contemplating future response options. These associations are 'dispositional' only, meaning that they do not hold a representation of the situation or bioregulatory state directly but can reactivate it through triggering appropriate other regions of the brain. These include regions of the brain that effect changes in the body (hypothalamus, autonomic centres, and periaqueductal grey) and represent change in the body (somatosensory cortex, insula, cingulate, basal ganglia and brainstem sensory/neurotransmitter nuclei). The amygdala is believed to serve a similar function to the VMPFC for primary rather than secondary inducers (Bechara et al., 2003).

The biasing action of somatic states on response selection is proposed to be regulated by neurotransmitter systems (including dopamine [DA], serotonin [5-HT], noradrenaline [NA], and acetylcholine [Ach]) in the brainstem. When the 'as-if' loop rather than the body loop is being utilised, feedback from the body is short-circuited by direct interactions between brainstem areas and regions representing body-state (e.g. somatosensory cortex) [Fig. 1].

In more recent accounts, a distinction has been drawn between posterior and anterior portions of the VMPFC. The posterior region is believed to hold representations of tangible, future events close in time that are more certain to occur, whereas the anterior region is believed to hold representations of abstract, future events further away in time that are less likely to occur (Bechara and Damasio, 2005), although there is little evidence to support this fractionation at present. Fig. 1 displays a simplified representation of the neural systems believed to be involved in the body loop and 'as-if' loop proposed by Damasio (Damasio, 1994; Bechara and Damasio, 2005). For a more detailed account of regions of the brain that represent and regulate the periphery, see Craig (2002), Saper (2002), Critchley (2002, 2004), and Bernston et al. (2003).

The extent to which there is support for this neural substrate will now be evaluated, focusing on lesion, neuroimaging, and psychopharmacology data in turn.

3.1. Lesion data

If the SMH is correct, damage to the areas that regulate and represent body-state change, in particular the VMPFC, should impair performance on the IGT and related paradigms. It is useful to spend some time describing the anatomical classification of the VMPFC before reviewing available lesion data, as there is some controversy about how to define it and this may explain why different studies have generated a different pattern of findings. Damasio's classification of VMPFC includes the entirety of the medial and aspects of the lateral orbitofrontal cortex (OFC) and overlaps with Brodmann's areas 10, 11,12 (medial aspects), lower 24, 25, and 32 (Bechara et al., 2000a; Bechara, 2004). Other researchers use the term orbitofrontal cortex (OFC) damage to refer to similar lesion cases that also extend to the lateral orbital surface (Rogers et al., 1999; Manes et al., 2002). The VMPFC/OFC is an anatomically heterogeneous structure that communicates with a wide variety of other brain areas. Öngur and Price (2000) argue that a distinction can be drawn between an orbital and a medial network within this broad region that serve slightly different functions. The orbital network is proposed to be a system for sensory integration, crucially involving Brodmann's areas 11, 13 and 12/47. The medial network is proposed to be a visceromotor system crucially involving Brodmann's areas 9, 10, 11, 13, 14, 24, 25, and 32. For the remainder of this article, the term VMPFC will be adopted, since this is the one used by Damasio and colleagues, but we note the lack of specificity of this term.

Lesion work from the Iowa laboratory has generally supported the neural substrate proposed in the SMH, although some revisions to the original framework have been made. As discussed earlier, it has been demonstrated in a number of studies that lesions encompassing VMPFC do reliably impair performance on the IGT (for example, see Bechara et al., 1994, 1996, 1997a, 2000). Interestingly, it appears that the earlier the age of VMPFC damage, the more severe the deficits in behaviour that emerge. Anderson et al. (1999) studied two adults who had suffered prefrontal cortex lesions occurring before age 16 months. They showed the expected pattern of impaired decision-making despite otherwise intact cognitive function. Additionally, deficient social and moral reasoning was found, suggesting that the acquisition of complex social conventions and moral rules had been impaired. This resulted in a syndrome resembling sociopathy.

Also consistent with the SMH, damage to the amygdala has been shown to alter performance on the task, but in a subtly different way to VMPFC ablation (Bechara et al., 1999). As well as being unable to form anticipatory markers while pondering deck choice, the amygdala group also failed to show any SCRs in response to winning and losing. In a classical conditioning paradigm, the amygdala group failed to generate SCRs to a visual stimulus that reliably predicted the onset of a loud noise, whereas the VMPFC group was unimpaired (Tranel et al., 1996). Bechara et al. (1999) proposed that the amygdala is involved in linking stimuli to effective attributes, whereas the VMPFC is crucial for deciding amongst a variety of response options in terms of the different affective responses they generate.

Consistent with the SMH, there is also preliminary evidence showing that the insula and somatosensory cortex need to be intact to acquire the IGT. In a conference abstract Bechara et al. (1997b) describe performance of patients with right-sided ($n=12$) and left-sided lesions ($n=6$) to the somatosensory/insula cortex, in comparison to age- and education-matched healthy control participants ($n=13$). The right-sided but not left-sided lesion group were impaired on the IGT relative to control participants, showing a behavioural preference for the disadvantageous decks.

A partial revision to the original neural substrate put forward in the SMH is that only the right hemisphere VMPFC appears to be implicated in IGT performance. To explore the contribution of laterality, Tranel et al. (2002) contrasted patients with bilateral, left only, and right only VMPFC lesions. The bilateral and right only VMPFC lesion group showed impaired everyday decision-making (assessed by clinical interview) and a marked deficit in performance on the IGT. The left only VMPFC lesion group was not severely impaired in everyday decision-making and their performance on the IGT fell in the low-normal range. These data imply that the type of decision-making measured by the IGT crucially depends on the right hemisphere VMPFC, although this conclusion can only be preliminary due to the small sample size used.

One explanation for this finding is that the right hemisphere has been associated with sensitivity to withdrawal and punishment learning, whereas the left hemisphere has been associated with sensitivity to approach and reward learning by some authors (e.g. Davidson and Irwin, 1999). As discussed earlier, the reward/punishment schedule on the IGT only varies in terms of punishment. It is plausible that if reward rather than punishment was key to the task, then left-sided VMPFC lesions would cause the greatest impairment. Consistent with this interpretation, in recent reviews from the Iowa laboratory, it has been suggested that the positive somatic states are represented in left-hemisphere VMPFC and negative somatic states are represented in right-hemisphere VMPFC (Bechara and Damasio, 2005). Also fitting with a punishment account is the finding that successful IGT performance positively correlates with self-reported neuroticism (Carter and Smith-Pasqualini, 2004). A slightly different interpretation is provided by Crucian et al. (2000), who suggest that the central role of the right hemisphere is to integrate cognitive interpretation of emotional information and perceived arousal.

Another change to the neural substrate of the SMH is that additional regions of the PFC appear to influence performance on the IGT. As discussed earlier, performance on the IGT is often worse in those with damage extending into the working memory/attentional control system of the DLPFC (Bechara et al., 1998b; Bechara and Martin, 2004). This finding is not particularly problematic for the SMH, since one way through which 'somatic markers' are believed to operate is by allocating processing resources via interactions with the attentional control circuitry of the DLPFC. It does make the predictions of the SMH difficult to distinguish from other, non-somatic theories, however.

In summary, a consistent performance deficit has been found on the IGT following damage to the VMPFC, particularly in the right hemisphere. Further, the contribution of this region to decision-making can be fractionated from the role of related structures such as the amygdala and DLPFC. Lesion work from other laboratories, however, has provided more mixed support for the neural substrate of the SMH.

When evaluating the lesion data on the IGT, it is important to bear in mind that the nature of brain damage means that lesions described in these studies are very rarely confined to clearly defined cerebral areas. This means that most of the patients with VMPFC damage studied on the IGT (and other decision-making paradigms) often have lesions extending into other portions of the frontal lobes and basal forebrain (Clark and Manes, 2004). It is therefore possible that these other areas, instead of or in addition to VMPFC, are the crucial neural substrate regulating task performance.

A range of evidence suggests that other regions of the PFC may be crucially involved in IGT performance. Manes et al. (2002) divided patients into categories of discrete OFC lesions (including VMPFC), discrete dorsolateral PFC lesions, discrete dorsomedial PFC lesions, and larger lesions affecting both dorsal and ventral portions of PFC. Patients with OFC lesions performed in the normal range on the IGT and two other measures of decision-making, except that they took longer to choose response options. The group with large PFC lesions was impaired on the IGT (selecting more from the disadvantageous decks) and on the two other measures of decision-making. This would appear to suggest that the VMPFC is not critical to performance on the IGT as asserted in the SMH. These results may have been biased by laterality differences in the lesions, however, since the OFC group had predominantly left-sided lesions whereas the large lesion group had predominantly right-sided lesions.

In a later study controlling for laterality confounds, Clark et al. (2003) found that right frontal lesions ($n=21$) severely impaired IGT performance, whereas left frontal lesions ($n=20$) only led to a slight attenuation in performance relative to controls ($n=20$). Importantly, it was found that the extent of the performance deficit on the IGT positively correlated with right-lesion but not left-lesion size. In particular, region of interest analysis from MRI scans revealed that damage to the right middle frontal gyrus, right superior frontal gyrus, and right medial prefrontal cortex was linked to task performance, indicating a prefrontal contribution to the IGT extending beyond the VMPFC as defined by Damasio.

3.2. Neuroimaging data

Functional neuroimaging provides an additional source of evidence to evaluate the neural substrate of the SMH. In particular, it can be usefully applied to study the activation of the 'as-if' loop proposed by Damasio. A number of studies have looked at neural activation while participants perform the IGT. Ernst et al. (2002) asked participants to perform a modified version of the IGT while in a positron emission tomography (PET) scanner, with the blood flow tracer injection roughly coinciding with the second block of 20 trials on the task. The subtraction task used asked participants to select cards from four decks in a specified

order, thereby isolating the active decision-making component of the task. A predominantly right-sided network of prefrontal and posterior cortical regions was activated, including OFC, anterior cingulate/medial PFC, dorsolateral PFC, insula, and inferior parietal cortex. Task performance was found to correlate with regional cerebral blood flow in right ventrolateral PFC and right anterior insula.

A subsequent PET study using a similar protocol in cocaine users (Bolla et al., 2003) allowed preliminary investigation of how this network is altered in groups who are impaired at behavioural acquisition of the IGT. Cocaine use was associated with increased activation in right OFC and decreased activation in right dorsolateral PFC, relative to control participant performance. Superior performance on the task correlated with right OFC activation in both groups.

Adinoff et al. (2003) explored the relationship between IGT performance and resting state blood flow measured using PET in healthy control participants ($n=15$) and abstinent cocaine-dependent individuals ($n=13$). In contrast to the findings discussed above, there was no relationship between resting state activity in OFC and task performance in either group. This was not due to a lack of variability in task performance, since the cocaine-dependent group showed considerable variance in the extent to which they selected from the advantageous decks. Instead, IGT performance correlated with resting state levels in anterior cingulate and left DLPFC. This study was repeated with patients in the more acute phase of cocaine abstinence (Tucker et al., 2004), which found IGT performance negatively correlated with activity in anterior cingulate gyrus, middle frontal gyrus, medial frontal gyrus, and superior frontal gyrus.

Similarly, Bolla et al. (2005) looked at neural activation in abstinent marijuana users as they performed the IGT. The marijuana users, particularly those who had a history of heavy use, showed greater activation in the left cerebellum, less activation in the right lateral OFC and right DLPFC, while performing the IGT, relative to a control group with no drug use history.

Ernst et al. (2003b) looked at neural network recruited by the IGT in adults with attentional deficit hyperactivity disorder (ADHD), relative to control participants. In both groups the task-activated ventral and dorsal PFC and the insula. Despite no group differences in behavioural performance, activation in ADHD participants was less extensive and did not include anterior cingulate and hippocampus. Further, the ADHD group showed decreased activation in the insula and increased activity in caudal portion of the right anterior cingulate.

Fukui et al. (2005) have recently examined the neural substrate of the IGT in 15 healthy control volunteers using event-related functional magnetic resonance imaging (fMRI). The superior temporal resolution of fMRI makes it possible to focus on the risk anticipation period of the task only. Subtracting selections from the advantageous decks from the disadvantageous decks in an event-related fMRI design, they found that risk anticipation exclusively activated the medial frontal gyrus. Further, this activation correlated with the accuracy of behavioural performance during the task. Surprisingly, the subtraction did not highlight differences in orbitofrontal regions.

In summary, neuroimaging studies using the IGT provide mixed support for the SMH. While parts of the neural network put forward by Damasio for both the body-state loop and 'as-if' loop have been linked to task performance, there is mixed support for the central role of VMPFC/OFC. It is important to note that the failure to reliably activate this area may be partially due to methodological issues associated with neuroimaging. PET studies have relatively poor temporal resolution, meaning it is not possible to focus solely on the risk anticipation period of each trial. MRI data are know to have a number of areas of signal dropout (including OFC) due to distortion artefacts (see Cusack et al., 2005). A role for DLPFC in IGT performance is again suggested from the neuroimaging data, which can be integrated with the SMH by the notion that somatic markers partially influence behaviour by boosting cognitive resources allocated to viable response options.

3.3. Psychopharmacology data

Bechara and Damasio (2005) propose that the biasing action of somatic states on response selection is mediated by the release of neurotransmitters such as dopamine (DA), serotonin (5HT) and norepinephrine (NE). For example, one mechanism through which 'somatic markers' could aid decision-making is through boosting cognitive resources allocated to advantageous response options via neurotransmitter modulation of attentional control systems.

The effects of systemic drug manipulations of these systems on IGT performance have recently been examined, finding that dopamine and serotonin are related to IGT performance but noradrenaline appears not to be. Bechara et al. (2001a) reported preliminary findings in a conference abstract that blockade of dopamine impairs and stimulation of dopamine improves performance on the early part of the IGT in healthy control participants ($n-9$), whereas blockade of serotonin impairs and stimulation of serotonin improves performance only on the latter part of the task in healthy control participants ($n=9$). They concluded therefore that dopamine might be more important for covert decision-making, whereas serotonin is more important for overt decision-making. This interpretation crucially depends on the cognitive penetrability of the IGT reward/punishment schedule at different stages of the game, however, and, as discussed earlier, it now appears that participants are consciously aware of the good and bad decks far earlier than previously assumed (Maia and McClelland, 2004). Bechara et al. (2001a) findings are also equally consistent with the idea that dopamine mediates

exploratory behaviour (Montague et al., 2004), so do not offer exclusive support for the SMH.

O'Carroll and Papps (2003) compared IGT performance in 30 healthy adult participants either administered a placebo, 4 mg of reboxetine (a selective nor-adrenergic reuptake inhibitor that boosts central noradrenergic [NA] activity), and 8 mg of reboxetine. There were no differences in performance between the three groups, suggesting that NA is not crucially involved in the proposed biasing action of somatic states on response selection, contra to the SMH.

3.4. Overview of neural substrate underpinning the SMH

Lesion, neuroimaging and psychopharamcology data to date have to some extent supported the neural substrate put forward in the SMH (see Fig. 1). Lesions to VMPFC, amygdala, insula and somatosensory cortex have all been found to impair performance on the IGT and are activated in many neuroimaging studies of decision-making and body-state representation. A slight revision is needed to take into account the fact that the right-hemisphere seems to be implicated to a greater extent than the left-hemisphere and that other regions of the PFC are also involved in the acquisition of the IGT.

4. Further conceptual issues with the SMH

Some additional potential problems with the SMH at a theoretical level will now be considered.

4.1. Novelty of the SMH: a historical overview

The novelty of the SMH has been challenged by some authors (e.g. McGinn, 2003), based on the fact that there is a long tradition of theory arguing that emotion-related feedback from the body can mediate adaptive behaviour, which predate the SMH. Further, a number of other fields have been developing the notion that emotion can bias decision-making in parallel to Damasio. This section reviews historical and contemporary parallels to the SMH and then evaluates the extent to which the SMH advances the field beyond these other accounts.

As discussed in the introduction, the SMH builds on earlier peripheral feedback theories of emotion that argue that emotion experience is based on the perception of changes activity in the body (e.g. James, 1884, 1894; Lange, 1885). Similarly, in the early behavioural literature, two-factor learning theory (Mowrer, 1947) linked conditioned visceral activity to the mediation of instrumental responding. It was observed that when a neutral stimulus is paired with an unconditionally aversive event, the neutral stimulus soon produced a conditioned visceral response. The aversive emotion state associated with visceral activation (for example, anxiety) was believed to motivate behaviour in the organism to reduce it (for example, avoidance). Empirical support for this position was mixed, however (Mowrer, 1960). A poor correlation was generally found between directly measurable autonomic responses and the maintenance of instrumental responding. In addition, autonomic responses were not always reliably found prior to the instrumental response, undermining their causal status. Further, dogs immobilised with the poison Curare (who therefore cannot modify proprioceptive feedback to the brain) were shown to be able to still acquire discriminative avoidance responses on the basis of classical conditioning, suggesting that peripheral feedback is not essential to this learning mechanism (Solomon and Turner, 1962). It was therefore concluded that the instrumental response and visceral response do not directly influence one another but instead are both mediated by some other central nervous system state that follows the rules of Pavlovian conditioning (Rescorla and Solomon, 1967). Interestingly, the lack of a causal relationship between visceral activity and instrumental responding parallels the findings discussed earlier that disturbance of feedback from the periphery does not reliably alter IGT performance.

Damasio accounts for these findings by arguing that the 'as-if' loop rather than the body loop can be activated to guide decision-making (e.g. see Damasio, 2004). Again, the notion of the 'as-if' loop is also present in earlier accounts (James, 1884, note 4; Marston, 1928). For example, Marston wrote: "Though any given emotion, experimentally tested, can be shown not to depend upon sensation, may not the emotion have been built up, originally, by compounding of sensations containing minute differences from other major emotional compounds, and subsequently remembered in connection with that type of stimulus" (Marston, 1928, p. 56). Damasio extends and more clearly specifies how the 'as-if' loop could operate than these earlier accounts, however.

False feedback studies, where participants are given inaccurate information about responses taking place in their body, also illustrate that body-state feedback had been thought to influence decision-making prior to the conception of the SMH. For example, Dienstbier and colleagues produced a series of studies looking at the consequences of false physiological feedback when contemplating acting immorally (for review see Dienstbeir, 1978). Building on the seminal attribution of arousal work by Schachter and Singer (1962), they found that college students were more likely to cheat if they had been informed that a vitamin pill they had taken would produce increased physiological arousal rather than reduced physiological arousal. In a position very similar to the SMH, Dienstbeir (1978) concluded that one of the ways in which we make decisions is by assessing the emotional arousal that follows thinking about each behavioural option. Batson et al. (1999) gave people false physiological feedback while listening to scenarios threatening to personal freedom and equality values. When asked to make a decision implicating these values, decisions favoured whichever value had received

stronger feedback while hearing the original situations. Feedback did not, however, affect judgements about the relative importance of the two values in general, which were believed to derive from cognitive retrieval from memory of a stored value hierarchy. These results are broadly consistent with the SMH.

Perhaps the most similar framework to the SMH was proposed by Nauta (1971), who argued the frontal lobes integrate adaptive behaviour using feedback from the periphery. Nauta came to this conclusion on the basis of the strong reciprocal anatomical connections of the frontal lobes with sensory processing regions and the limbic system as described by MacLean (1949, 1975), speculating that a key role of the frontal lobes was to integrate feedback from the senses and use this to guide adaptive behaviour. Damage to the frontal lobes would impair the ability to integrate internal and external sensory information: "part at least of the behavioural effects of frontal-lobe destruction could be seen as the consequence of an 'interoceptive agnosia', i.e. an impairment of the subject's ability to integrate certain informations from his internal milieu with the environmental reports provided by neocortical processing mechanisms" (p. 182). Further, this could then result in an inability to regulate behaviour advantageously: "the reciprocal fronto-limbic relationship could be centrally involved in the phenomenon of behavioural anticipation, and elucidate the 'loss of foresight' that has so long been recognised as one of the most disabling consequences of massive frontal-lobe lesions…..The normal individual decides upon a particular course of action by a thought process in which …strategic alternatives are compared….The comparison in the final analysis is one between the affective responses evoked by each of the various alternatives….It is entirely conceivable that this anticipatory selection process is severely impaired in the absence of the frontal cortex" (p. 183). Finally, part of the affective response for each alternative is based on feedback from internal sensory information: "a pre-setting of …..interoceptive information…..could be thought to establish a temporal sequence of affective reference points serving as 'navigational markers". (p. 183). This account, predating the SMH by over 20 years, seems to have come to remarkably similar conclusions. There were even some attempts to experimentally test this framework through disconnection studies in animal models (e.g. Divac et al., 1975), although these generally offered little support for the model. Similarly, Pribram (1970) suggested that feelings, or sensations arising from lower brain regions and the body, could serve as 'monitors' to co-ordinate behaviour. It should be noted, however, that the SMH clearly extends these frameworks by specifying much more clearly how this system could be implemented in the brain.

There is also a large normative literature looking at the impact of emotion on decision-making, which has evolved in parallel to the SMH. In a review of this work, Loewenstein and Lerner (2003) argued that a consideration of emotional factors, both in terms of an effect experienced at the time of the decision (immediate emotions) and the affect an individual anticipates they will feel following a particular response option (expected emotions), can help to better model human decision-making than purely rational accounts of choice selection. One illustration of a normative model of decision-making where emotion is central is 'affect as information' theory (Clore, 1992), which proposes that people monitor how they feel at the present time to help them evaluate a situation. Therefore, if current feelings happen to be positive, then the evaluation of a specific decision-making option being contemplated is likely to be positive. This appears to be particularly true for consideration of unfamiliar choices where affect is likely to be relevant (for example, choosing which movie to see). Similarly, decision affect theory (see Mellers et al., 1997) claims that the emotions that people experience after a decision depend on a comparison between what the consequences actually were and the consequences that would have come about if another response option was chosen. People are disappointed if the option they chose produced a less beneficial outcome than other alternatives could have done but are pleased if their response led to a superior outcome.

The notion that decision-making may be particularly influenced by emotion at a more covert level is also present in the cognitive literature, where a series of theorists have converged on the notion that at least two parallel but interacting routes to information processing may exist. One route may use 'high reason' to cognitively appraise situations at a more explicit level (a 'cognitive' loop) and the other route may bias behaviour at a more implicit, automatic level (an 'automatic' loop). As a representative example, cognitive-experiential self-theory (CEST; Epstein, 1991) will be outlined. CEST draws a distinction between rational and experiential systems for processing information. The experiential system is influenced largely by affect and emotion (or 'what feels good') and is believed to be the 'natural' or 'default' mode of responding to situations (Epstein et al., 1996). It produces rapid, reflexive responses but is relatively slow to change and acts primarily at an unconscious level. Its outputs are similar to the heuristics, or cognitive short-cuts, postulated by Kahneman and Tversky (1972) to simplify complex decision-making. The rational system works at a conscious level and functions using established rules and principles of inference. It is relatively slow to respond but quick to change, generating complex, dispassionate analysis. It has evolved more recently than the experiential system and requires justification via logic and evidence. These two systems act in parallel, but interact with each other regularly. The experiential system is believed to be more important in the regulation of everyday behaviour, automatically encoding, interpreting, and organising experience, and directing behaviour. In other words, information exists at both a conscious, verbal level and a pre-conscious, experiential level. Similar accounts of

the interface between cognition and emotion have been put forward in other multiple representation frameworks, including Interactive Cognitive Subsystems (ICS; Teasdale and Barnard, 1993), the Schematic Propositional Analogical and Associative Representation System (SPAARS; Power and Dalgleish, 1997), and Lambie and Marcel's (2002) account of emotion experience. It is important to observe, however, that at present many of these models are less well specified than the SMH, particularly at the neural level.

The critical question that this historical synopsis raises is what is the unique contribution that the SMH makes to the literature? The notion that emotion, partially via peripheral mechanisms, influences decision-making is reflected in a variety of earlier and parallel models (Mowrer, 1960; Pribram, 1979; Nauta, 1971). Further, the notion that emotion-based influences may act at a more implicit, covert level of decision-making (Epstein, 1991) and that a representation of expected body-state change in the brain is sufficient to shape higher level processing without waiting for actual bodily responses (for example, see James, 1884 note 4; Marston, 1928) are also present in earlier accounts.

The resonance of the SMH with earlier work is both a strength and weakness; it gives the model strong concurrent validity with over a hundred years of psychological theory but at the same time it challenges the novelty of the framework. Perhaps the value of the SMH is the way it integrates these components from different models into a unitary framework, although these earlier influences are not always explicitly apparent in Damasio's writings. Clear advances in the SMH are that it expands the range of bodily states underlying emotions beyond visceral feedback (e.g. including hormonal feedback through the bloodstream) and that it puts forward a more elaborate psychological mechanism and a clearer neural substrate to regulate how emotion and peripheral feedback shape decision-making. Further, Damasio extends and more clearly specifies the role of the 'as-if' loop compared to earlier accounts, and discusses more fully how feedback from the body is integrated with cognitive appraisal to mediate both emotion experience and decision-making (see Damasio, 1994, p. 139). Perhaps most importantly, Damasio outlines how emotion-based biasing signals can act at a variety of levels, both conscious and non-conscious. These advances make the SMH more robust in the face of the criticisms usually voiced against Jamesian models (e.g. Cannon, 1927), in particular allowing the system to be rapid, flexible, and to subtly differentiate between complex emotion states. Further, the 'as-if' loop can explain why surgical isolation of the periphery does not always impair emotion experience or decision-making. Finally, through the development of novel experimental tools and the study of lesion groups, Damasio has provided ways to directly empirically test these ideas. Therefore, the SMH does appear to be making a novel and valuable contribution to the literature.

4.2. Specification of the somatic marker mechanism

Perhaps more problematic is the fact that the 'somatic marker' mechanism is currently poorly specified. According to SMH, a large number of somatic signals, for example from the viscera, vascular bed, skeletomotor system and endocrine system need to be integrated into a pattern image that marks outcomes as either 'good' or 'bad'. It is unspecified in the SMH how this complex data reduction is computed. However, a number of physiological plausibe data reduction algorithms (such as principal components analysis) could be used to reduce the high-dimensional body-state pattern into a low-dimensional 'somatic marker'. Work in other fields has begun to apply these data reduction approaches to understand other complex problems. For example, Bar-Gad et al. (2003) propose a model of how the basal ganglia compresses cortical information, effectively acting as a central dimensionality reduction system which is modulated by a reinforcement signal. The mechanism by which this could be achieved in the SMH is currently underspecified and would benefit from more detailed examination. Other researchers have begun to map out in detail how signals from the body are relayed to the brain (e.g. the lamina I spinothalamocortical system; Craig, 2002), which could aid this process.

4.3. Parsimony of the SMH

Another criticism that has been voiced against the SMH is that the theory does not provide the most parsimonious explanation of the phenomenon of interest (e.g. Rolls, 1996). Damasio's theory neglects to explain what generates specific patterns of body-state change in the first place and presumably some kind of central, cognitive appraisal must be taking place (i.e. the system must 'know' that a 'bad' decision is about to be made to generate an SCR). Given that this is occurring, it would seem a more parsimonious solution for this system to communicate directly with higher levels of representation, rather than taking the rather circuitous route through the body. Rolls (1996) argued that it would be very inefficient for the execution of behaviour following the appraisal of the value of a stimulus to have to go through the periphery or a central representation of the periphery. Instead, it would be much more adaptive to have a direct connection to output motor centres to implement the target behaviour.

In fact, rather early on proponents of the SMH anticipated the parsimony criticism (Damasio et al., 1991). They essentially argued that the 'somatic marker' system is evolutionarily ancient, and has proved to be highly effective. Further, they argued that powerful 'somatic markers' are necessary to effectively reduce complex decision spaces. However, a number of computational algorithms, in particular in computational reinforcement learning, have been explicitly designed for decision-making under uncertainty, essentially by using approximations for

the value of future actions in order to guide action selection (Montague et al., 2004). These types of algorithm can be used to solve n-armed bandit problems, of which the IGT is a variant, and are also well specified at an anatomical level. Therefore, the neural instantiation of such decision-making mechanisms does not seem to necessitate the use of the complex somatic and emotion-related machinery described by Damasio. Interestingly, recent work from the Iowa laboratory utilises a reinforcement learning algorithm to model IGT data without making any reference to the SMH (Oya et al., 2005). A number of other non-somatic mechanisms for constraining decision-making (e.g. cognitive heuristics; Kahneman and Tversky, 1972) have also been proposed. One way to defend this issue would be to argue that these reinforcement learning algorithms are simply the inbstantiation of the central components of the 'somatic marker' system, rather than a genuinely alternative account (see Damasio, 1994).

4.4. Emphasis on the periphery in the SMH

The SMH has also been criticised for being 'somatocentric' (Panksepp, 2003). Panksepp suggested that Damasio has taken the peripheral feedback theories to an extreme by saying that most mental states are made up purely of different types of bodily awareness. Instead, Panksepp suggests that a more moderate conclusion is warranted, whereby most cognitive states include some kind of emotional aspect, made up partially of somatosensory feedback but also other components. For example, consideration of the different 'action apparatus' of the brain that has evolved to regulate different emotion states could help to clarify how relatively crude peripheral biasing signals contribute to distinct, refined emotion states. Craig (2002) also suggests that the SMH has focused on the representation of body-state at the cost of other aspects of emotion processing, in particular neglecting how these representations of body-state can then motivate behavioural action. Similar points in relation to the SMH have been made by McGinn (2003).

These criticisms seem a slight distortion of the SMH, however, since Damasio (1999) explicitly includes a mental evaluative component that interprets emotional sensations of bodily change to lead to more complex emotion experience. In fact, some kind of specification of how body-state interacts with central mental evaluation seems a positive advance in the SMH compared to other Jamesian theories (see also Prinz's 'somatic appraisal theory', 2004).

5. Conclusion: current status of the somatic marker hypothesis

The SMH (Damasio, 1994) represents an intriguing model of how feedback from the body may contribute to successful decision-making in situations of complexity and uncertainty. This builds on earlier work linking activity in the body to emotion experience (e.g. James, 1884, 1894; Lange, 1885) and decision-making (e.g. Pribram, 1970; Nauta, 1971). Key support for this theory has been largely drawn from data on the IGT, a decision-making task that has been claimed to rely on emotion-related feedback from the body to guide accurate performance (Bechara et al., 1996). While the IGT has proved to be a sensitive, ecologically valid measure of decision-making impairment that has generated a large body of empirical research, three key assumptions that need to be held for it to offer support for the SMH may not be tenable. First, the claim that the task measures implicit learning as the reward/punishment schedule is cognitively impenetrable (although see Bechara et al., 2005) is inconsistent with data showing accurate knowledge of the task contingencies (Maia and McClelland, 2004) and that mechanisms such as working-memory or cognitive outcome expectancies appear to exert a strong influence on task performance. Second, the assertion that this learning takes place via anticipatory marker signals arising from the body is not supported by competing explanations of the psychophysiology profile generated on the task (Tomb et al., 2002), the failure to establish a clear causal relationship between disturbed feedback from the periphery and impaired decision-making (e.g. Heims et al., 2004), and the possibility that the 'anticipatory' changes may actually reflect expectancies about reward and punishment generated after a decision has been made (Amiez et al., 2003). Third, the assumption that the task impairment is due to a 'myopia for the future' is undermined by the existence of a number of other perhaps better specified and more plausible psychological mechanisms explaining deficits on the task (including reversal learning, risk-taking, and working-memory deficits). In addition, there may be greater variability in control performance on the task than previously believed (complicating interpretation of empirical findings).

While the psychological mechanism underpinning the SMH therefore seems to require some revision, the neural substrate that Damasio has proposed has been reasonably well supported by the findings to date. Lesion and neuroimaging studies have found that VMPFC, amygdala, somatosensory cortex, insula and related areas are involved in decision-making processes as suggested in the framework. This is a clear advance on earlier models of decision-making. The neural substrate of the SMH still needs further clarification in the light of ongoing advances in knowledge about VMPFC anatomy, however (e.g. Öngur and Price, 2000).

Therefore, the SMH seems to have accurately identified some of the brain regions involved in decision-making, emotion, and body-state representation, but exactly how they interact at a psychological level is still somewhat unclear.

Of course, none of these reservations falsify the SMH; they just suggest that other sources of evidence need to be

gathered to support it. Indeed, we remain open to the idea that feedback from the periphery can influence higher-level cognitive and emotional processes. Bechara et al. (2005) have recently proposed a series of unanswered questions about the SMH that could be usefully explored in future research. These include assessing if different kinds of decision-making (e.g. under certainty or uncertainty) recruit different neural networks, identifying when emotions are unhelpful as well as helpful when making decisions (e.g. Shiv et al., 2005), further specifying the nature of biasing signals that guide decision-making, fractionating the underlying cognitive processing involved in complex decision-making tasks, and exploring individual differences in decision-making. We wholeheartedly agree that consideration of these issues would improve the literature on both the SMH and broader decision-making processes. Here we suggest some specific future studies.

A variety of potential designs could be used to further test the SMH. One approach is to develop variants of the IGT that control some of the methodological issues and task interpretation ambiguities raised. This could include removing the reversal learning confound and making the task less easy to consciously comprehend (for example, using a variant artificial grammar learning paradigm). Another approach would be to more aggressively pursue causal tests of the SMH in a wider range of populations with altered peripheral feedback (e.g. patients with facial paralysis, Keillor et al., 2002). As well as looking at patient groups with impaired somatosensory feedback, pharmacological challenge studies in healthy control volunteers could be run. For example, the impact of the peripherally acting beta-blocker nadalol on IGT performance could be measured. Moreover, different causal methodologies could be combined to minimise the amount of feedback from the body reaching the brain (for example, looking at the impact of peripheral blockade in patients with PAF). An alternative body of existing literature that could speak to the validity of the SMH is false feedback experiments (e.g. see Crucian et al., 2000). It may be possible to use false feedback while patient groups with disturbed bodily feedback perform the IGT (e.g. Linton and Hirt, 1979) to further test the framework. Finally, an individual differences approach could be adopted, for example looking at how variance in interoceptive ability relates to IGT performance (e.g. see Katkin et al., 2001). As well as looking at behavioural measures in these studies, it is important for future studies to simultaneously record a wider range of peripheral variables (e.g. facial electromyography, heart rate) and central variables (e.g. fMRI and event-related potentials) wherever possible (e.g. Oya et al., 2005).

Until a broader range of empirical approaches are used to test the SMH, the current status of the framework would appear to be that it is an intriguing idea in need of some clearer specification and some more supporting evidence. Despite these limitations, the SMH and the IGT have made a valuable contribution to the literature. They have helped to reintroduce the idea that emotion can be a benefit as well as a hindrance when making a decision to the wider neuroscience community; they have outlined a plausible neural substrate that could underpin this mechanism, and they have developed an innovative experimental paradigm to test the framework. Through the development of the 'as-if' loop, the SMH also addresses a number of weaknesses in other Jamesian models and can better account for the speed and complexity of emotion responses that are typically observed. The IGT has been an extremely generative paradigm, applied to a wide range of neurological and psychiatric conditions and used in numerous experimental studies. Further, the SMH has importantly extended MacLean's pioneering limbic system framework in order to better understand how emotion is represented in the brain and to more clearly model the influence emotion can have on other cognitive processes.

Acknowledgements

This review was supported by the Medical Research Council of the United Kingdom. Address reprint requests to Dr Barnaby Dunn, MRC CBU, 15 Chaucer Road, Cambridge, CB2 2EF, UK; barney.dunn@mrc-cbu.cam.ac.uk (e-mail)

References

Adinoff, B., Devous Sr., M.D., Cooper, D.B., Best, S.E., Chandler, P., Harris, T., et al., 2003. Resting regional cerebral blood flow and gambling task performance in cocaine-dependent subjects and healthy comparison subjects. American Journal of Psychiatry 160 (10), 1892–1894.

Amiez, C., Procyk, E., Honore, J., Sequeira, H., Joseph, J.-P., 2003. Reward anticipation, cognition, and electrodermal activity in the conditioned monkey. Experimental Brain Research 149, 267–275.

Anderson, S.W., Bechara, A., Damasio, H., Tranel, D., Damasio, A.R., 1999. Impairment of social and moral behaviour related to early damage in human prefrontal cortex. Nature Neuroscience 2 (11), 1032–1037.

Apkarian, A.V., Sosa, Y., Krauss, B.R., Thomas, P.S., Fredrickson, B.E., Levy, R.E., et al., 2004. Chronic pain patients are impaired on an emotional decision-making task. Pain 108 (1–2), 129–136.

Bar-Gad, I., Morris, G., Bergman, H., 2003. Information processing, dimensionality reduction and reinforcement learning in the basal ganglia. Progress in Neurobiology 71, 439–473.

Bark, R., Dieckmann, S., Bogerts, B., Northoff, G., 2005. Deficit in decision making in catatonic schizophrenia: an exploratory study. Psychiatry Research 134, 131–141.

Barrash, J., Tranel, D., Anderson, S.W., 2000. Acquired personality distrubances associated with bilateral damage to the ventromedial prefrontal region. Developmental Neuropsychology 18 (3), 355–381.

Batson, C.D., Engel, C.L., Fridell, S.R., 1999. Value judgements: testing the somatic-marker hypothesis using false physiological feedback. Personality and Social Psychology Bulletin 25 (8), 1021–1032.

Bechara, A., 2004. The role of emotion in decision-making: evidence from neurological patients with orbitofrontal damage. Brain and Cognition 55 (1), 30–40.

Bechara, A., Damasio, H., 2002. Decision-making and addiction (part I): impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. Neuropsychologia 40 (10), 1675–1689.

Bechara, A., Damasio, A.R., 2005. The somatic marker hypothesis: a neural theory of economic decision. Games and Economic Behaviour 52(2), 336–372.

Bechara, A., Martin, E.M., 2004. Impaired decision making related to working memory deficits in individuals with substance addictions. Neuropsychology 18 (1), 152–162.

Bechara, A., Damasio, A., Damasio, H., Anderson, S., 1994. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 50, 7–15.

Bechara, A., Tranel, D., Damasio, H., Damasio, A.R., 1996. Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. Cerebral Cortex 6 (2), 215–225.

Bechara, A., Damasio, H., Tranel, D., Damasio, A.R., 1997a. Deciding advantageously before knowing the advantageous strategy. Science 275, 1293–1295.

Bechara, A., Tranel, D., Damasio, H., Damasio, A.R., 1997b. An anatomical system subserving decision-making. Society for Neuroscience Abstracts 23, 495.

Bechara, A., Damasio, H., Tranel, D., Anderson, S.W., 1998a. Dissociation of working memory from decision-making within the human prefrontal cortex. The Journal of Neuroscience 18 (1), 428–437.

Bechara, A., Tranel, D., Wilson, J., Heberlein, A.S., Ross, M., Damasio, A.R., 1998b. Impaired decision-making in peripheral neuropathy. Society for Neuroscience Abstracts 24, 1176.

Bechara, A., Damasio, H., Damasio, A.R., Lee, G.P., 1999. Different contributions of the human amygdala and the ventromedial prefrontal cortex to decision-making. Journal of Neuroscience 19 (13), 5473–5481.

Bechara, A., Damasio, H., Damasio, A.R., 2000a. Emotion, decision making and the orbitofrontal cortex. Cerebral Cortex 10, 295–307.

Bechara, A., Tranel, D., Damasio, H., 2000b. Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. Brain 123, 2189–2202.

Bechara, A., Damasio, H., Damasio, A.R., 2001a. Manipulation of dopamine and serotonin causes different effects on covert and overt decision-making. Society for Neuroscience Abstracts 27, 126.

Bechara, A., Dolan, S., Denburg, N., Hindes, A., Anderson, S.W., Nathan, P.E., 2001b. Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. Neuropsychologia 39 (4), 376–389.

Bechara, A., Dolan, S., Hindes, A., 2002. Decision-making and addiction (part II): myopia for the future or hypersensitivity to reward? Neuropsychologia 40 (10), 1690–1705.

Bechara, A., Damasio, H., Damasio, A.R., 2003. Role of the amygdala in decision-making. Annals of the New York Academy of Science 985, 356–369.

Bechara, A., Damasio, H., Tranel, D., Damasio, A.R., 2005. The Iowa Gambling Task and the somatic marker hypothesis: some questions and answers. Trends in Cogntive Sciences 9 (4), 159–162.

Beninger, R.J., Wasserman, J., Zanibbi, K., Charbonneau, D., Mangels, J., Beninger, B.V., 2003. Typical and atypical antipsychotic medications differentially affect two nondeclarative memory tasks in schizophrenic patients: a double dissociation. Schizophrenia Research 61, 281–292.

Bernston, G.G., Sarter, M., Cacioppo, J.T., 2003. Ascending visceral regulation of cortical affective information processing. European Journal of Neuroscience 18, 2103–2109.

Best, M., Williams, J.M., Coccaro, E.F., 2002. Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. Proceedings of the National Academy of Science U S A 99 (12), 8448–8453.

Bolla, K.I., Eldreth, D.A., London, E.D., Kiehl, K.A., Mouratidis, M., Contoreggi, C., et al., 2003. Orbitofrontal cortex dysfunction in abstinent cocaine abusers performing a decision-making task. Neuroimage 19 (3), 1085–1094.

Bolla, K.I., Eldreth, D.A., Matochik, J.A., Cadet, J.L., 2004. Sex-related differences in a gambling task and its neurological correlates. Cerebral Cortex 14 (11), 1226–1232.

Bolla, K.I., Eldreth, D.A., Matochik, J.A., Cadet, J.L., 2005. Neural substrates of faulty decision-making in abstinent marijuana users. Neuroimage 26, 480–492.

Bowman, C.H., Turnbull, O.H., 2003. Real versus facsimile reinforcers on the Iowa Gambling Task. Brain and Cognition 53 (2), 207–210.

Bowman, C.H., Evans, C.E., Turnbull, O.H., 2005. Artificial time constraints on the Iowa Gambling Task: the effects on behavioural performance and subjective experience. Brain and Cognition 57 (1), 21–25.

Bradley, M.M., Codispoti, M., Cuthbert, B.N., Lang, P.J., 2001a. Emotion and motivation I: defensive and appetitive reactions in picture processing. Emotion 1 (3), 276–298.

Bradley, M.M., Codispoti, M., Sabatinelli, D., Lang, P.J., 2001b. Emotion and motivation II: sex differences in picture processing. Emotion 1 (3), 300–319.

Busemeyer, J.R., Stout, J.C., 2002. A contribution of cognitive decision models to clinical assessment: decomposing performance on the Bechara gambling task. Psychological Assessment 14, 253–262.

Camille, N., Coricelli, G., Sallet, J., Pradat-Diehl, P., Duhamel, J.R., Sirigu, A., 2004. The involvement of the orbitofrontal cortex in the experience of regret. Science 304, 1167–1170.

Campbell, M.C., Stout, J.C., Finn, P.R., 2004. Reduced autonomic responsiveness to gambling task losses in Huntington's disease. Journal of the International Neuropsychological Society 10, 239–245.

Cannon, W.B., 1927. The James-Lange theory of emotions: a critical examination and an alternative theory. American Journal of Psychology 39, 106–124.

Carter, S., Smith-Pasqualini, M.C., 2004. Stronger autonomic response accompanies better learning: a test of Damasio's somatic marker hypothesis. Cognition and Emotion 18 (7), 901–911.

Cavedini, P., Riboldi, G., D'Annucci, A., Belotti, P., Cisima, M., Bellodi, L., 2002a. Decision-making heterogeneity in obsessive compulsive disorder: ventromedial prefrontal cortex function predicts different treatment outcomes. Neuropsychologia 40, 205–211.

Cavedini, P., Riboldi, G., Keller, R., D'Annucci, A., Bellodi, L., 2002b. Frontal lobe dysfunction in pathological gambling patients. Biological Psychiatry 51 (4), 334–341.

Cavedini, P., Bassi, T., Ubbiali, A., Casolari, A., Giordani, S., Zorzi, C., 2004a. Neuropsychological investigation of decision-making in anorexia nervosa. Psychiatry Research 127, 259–266.

Cavedini, P., Bassi, T., Zorzi, C., Bellodi, L., 2004b. The advantages of choosing antiobsessive therapy according to decision-making functioning. Journal of Clinical Psychopharmacology 24 (6), 628–631.

Cheung, T.H., Cardinal, R.N., 2005. Hippocampal lesions facilitate instrumental learning with delayed reinforcement but induce impulsive choice in rates. BMC Neurosci 6(1), 36.

Chwalisz, K., Diener, E., Gallagher, D., 1988. Autonomic arousal feedback and emotional experience: evidence from the spinal cord injured. Journal of Personality and Social Psychology 54, 820–828.

Clark, L., Manes, F., 2004. Social and emotional decision-making following frontal lobe injury. Neurocase 10 (5), 398–403.

Clark, L., Iversen, S.D., Goodwin, G.M., 2001. A neuropsychological investigation of prefrontal cortex involvement in acute mania. American Journal of Psychiatry 158 (10), 1605–1611.

Clark, L., Manes, F., Antoun, N., Sahakian, B.J., Robbins, T.W., 2003. The contributions of lesion laterality and lesion volume to decision-making impairment following frontal lobe damage. Neuropsychologia 41 (11), 1474–1483.

Clore, G.L., 1992. Cognitive phenomenology: feelings and the construction of judgement. In: Martin, L.L., Tesser, A. (Eds.), The Construction of Social Judgements. Erlbaum, Hillsdale, NJ.

Cobos, P., Sanchez, M., Garcia, C., Vera, M., Vila, J., 2002. Revisiting the James versus Cannon debate on emotion: startle and autonomic modulation in patients with spinal cord injuries. Biological Psychology 61, 251–269.

Craig, A.D., 2002. How do you feel? Interoception: the sense of the physiological condition of the body. Nature Reviews Neuroscience 3, 655–666.

Critchley, H.D., 2002. Electrodermal Responses: what happens in the brain. The Neuroscientist 8 (2), 132–142.

Critchley, H.D., Good, C.D., Ashburner, J., Frackowiak, R.S., Mathias, C.J., Dolan, R.J., 2003. Changes in cerebral morphology consequent to peripheral autonomic denervation. Neuroimage 18, 908–916.

Critchley, H.D., Wiens, S., Rotshtein, P., Ohman, A., Dolan R.J., 2004. Neural systems supporting interoceptive awareness. Nature Neuroscience. 7, 189–195.

Crone, E.A., van der Molen, M.W., 2004. Developmental changes in real life decision making: performance on a gambling task previously shown to depend on the ventromedial prefrontal cortex. Developmental Neuropsychology 25 (3), 251–279.

Crone, E.A., Somsen, R.J., Van Beek, B., Van Der Molen, M.W., 2004. Heart rate and skin conductance analysis of antecendents and consequences of decision making. Psychophysiology 41 (4), 531–540.

Crucian, G.P., Hughes, J.D., Barrett, A.M., Williamson, D.J.G., Bauer, R.M., Bowers, D., 2000. Emotional and physiological responses to false feedback. Cortex 36, 623–647.

Cusack, R., Russell, B., Cox, S.M.L., De Panfilis, C., Schwarzbauer, C., Ansorge, R., 2005. An evaluation of the use of passive shimming to improve frontal sensitivity in fMRI. Neuroimage 24, 82–91.

Dalgleish, T., 2004. The emotional brain. Nature Neuroscience Reviews 5 (7), 583–589.

Dalgleish, T., Yiend, J., Bramham, J., Teasdale, J.D., Ogilvie, A.D., Malhi, G., et al., 2004. Neuropsychological processing associated with recovery from depression after stereotactic subcaudate tractotomy. American Journal of Psychiatry 161 (10), 1913–1916.

Damasio, A.R., 1979. The frontal lobes. In: Heilman, K.M., Valenstein, E. (Eds.), Clinical Neuropsychology. Oxford University Press, New York.

Damasio, A.R., 1994. Descartes Error: Emotion, Reason and the Human Brain. Avon, New York.

Damasio, A.R., 1996. The somatic marker hypothesis and the possible functions of the prefrontal cortex. Philosophical Transactions of the Royal Society of London (series B) 351 (1346), 1413–1420.

Damasio, A.R., 1998. Emotion in the perspective of an integrated nervous system. Brain Research Reviews 26, 83–86.

Damasio, A.R., 1999. The Feeling of What Happens: Body and Emotion in the Making of Consciousness. Harcourt, New York.

Damasio, A.R., 2004. William James and the modern neurobiology of emotion. In: Evans, D., Cruse, P. (Eds.), Emotion, Evolution and Rationality. Oxford University Press, Oxford.

Damasio, A.R., Tranel, D., Damasio, H., 1990. Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. Behaviour Brain Research 41 (2), 81–94.

Damasio, A.R., Tranel, D., Damasio, H., 1991. Somatic markers and the guidance of behaviour: theory and preliminary testing. In: Levin, H.S., Eisenberg, H.M., Benton, A.L. (Eds.), Frontal Lobe Function and Dysfunction. Oxford University Press, New York, pp. 217–229.

Damasio, H.T., Grabowski, R., Frank, A.M., Galaburda, A.M., Damasio, A.R., 1994. The return of Phineas Gage: the skull of a famous patient yields clues about the brain. Science 264, 1102–1105.

Damasio, H., Bechara, A., Damasio, A., et al., 2002. Nature Neuroscience 5 (11), 1104 (Reply to Tomb et al.).

Damasio, A.R., Adolphs, R., Damasio, H., 2003. The contributions of the lesion method to the functional neuroanatomy of emotion. In: Davidson, R.J., Scherer, K.R., Goldsmith, H.H. (Eds.), Handbook of Affective Sciences. Oxford University Press, Oxford, pp. 66–92.

Davidson, R.J., Irwin, W., 1999. The functional neuroanatomy of emotion and affective style. Trends in Cognitive Science 3 (1), 11–21.

Davis, C., Levitan, R.D., Muglia, P., Bewell, C., Kennedy, J.L., 2004. Decision-making deficits and overeating: a risk model for obesity. Obesity Research 12 (6), 929–935.

Denburg, N.L., Tranel, D., Bechara, A., Damasio, A.R., 2001. Normal ageing may compromise the ability to decide advantageously. Brain and Cogntion 47, 156–159.

Denburg, N.L., Tranel, D., Bechara, A., 2005. The ability to decide advantageosuly declines prematurely in some normal older persons. Neuropsychologia 43 (7), 1099–1106.

Dienstbeir, R.A., 1978. In: Harvey, J.H., Ickes, W.J., Kidds, R.F. (Eds.), Attribution, Socialization, and Moral Decision-Making New Directions in Attribution Research, vol. 2. Lawrence Erlbaum, Hillsdale NJ, pp. 181–206.

Dimitrov, M., Phipps, M., Zahn, T., Grafman, J., 1999. A thoroughly modern Gage. Neurocase 5, 345–354.

Divac, I., Gade, A., Wikmark R.G., 1975. Taste aversion in rats with lesions in the frontal lobes: No evidence for intoreceptive agnosia. Physiological Psychology 3, 43–46.

Ellsworth, P.C., 1994. William James and Emotion: is a century of fame worth a century of misunderstanding. Psychological Review 101 (2), 222–229.

Epstein, S., 1991. Cognitive-experiential self-theory: an integrative theory of personality. In: Curtis, R. (Ed.), The Self with Others: Convergences in Psychoanalytic, Social and Personality Psychology. Guilford, New York.

Epstein, S., Pacini, R., Denes-Raj, V., Heier, H., 1996. Individual differences in intuitive–experiential and analytical–rational thinking styles. Journal of Personality and Social Psychology 71, 390–405.

Ernst, M., Bolla, K., Mouratidis, M., Contoreggi, C., Matochik, J.A., Kurian, V., 2002. Decision-making in a risk-taking task: a PET study. Neuropsychopharmacology 26, 682–691.

Ernst, M., Kimes, A.S., London, E.D., Matochik, J.A., Eldreth, D., Tata, S., 2003a. Neural substrates of decision making in adults with Attentional Deficit Hyperactivity Disorder. American Journal of Psychiatry 160 (6), 1061–1070.

Ernst, M., Grant, S.J., London, E.D., Contoreggi, C., Kimes, A.S., Spurgeon, L., 2003b. Decision making in adolescents with behaviour disorders and adults with substance abuse. American Journal of Psychiatry 160 (1), 33–40.

Eslinger, P.J., Damasio, A.R., 1985. Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. Neurology 35, 1731–1741.

Evans, C.E., Kemish, K., Turnbull, O.H., 2004. Paradoxical effects of education on the Iowa Gambling Task. Brain and Cognition 54 (3), 240–244.

Fein, G., Klein, L., Finn, P., 2004. Impairment on simulated gambling task in long term long-term abstinent alcoholics. Alcoholism: Clinical and Experimental Research 28 (10), 1–5.

Fellows, L.K., Farah, M.J., 2003. Ventromedial frontal cortex mediates affective shifting in humans: evidence from a reversal learning paradigm. Brain 126 (Pt 8), 1830–1837.

Fellows, L.K., Farah, M.J., 2005a. Different underlying impairments in decision-making following ventromedial and dorsolateral frontal lobe damage in humans. Cerebral Cortex 15 (1), 58–63.

Fellows, L.K., Farah, M.J., 2005b. Dissociable elements of human foresight: a role for the ventromedial frontal lobes in framing the future, but not in discounting future rewards. Neuropsychologia 43, 1214–1221.

Fishbein, D., Eldreth, D., Matochik, J., Isenberg, I., Hyde, C., London, E., 2005. Cognitive performance and autonomic reactivity in abstinent drug abusers and nonusers. Experimental and Clinical Psychopharmacology 13 (1), 25–40.

Franken, I.H.A., Muris, P., 2005. Individual differences in decision-making. Personality and Individual Differences 39, 991–998.

Fukui, H., Murai, T., Fukuyama, H., Hayashi, T., Hanakawa, T., 2005. Functional activity related to risk anticipation during performance of the Iowa Gambling Task. Neuroimage 24 (1), 253–259.

Gaffan, D., 1979. Acquisition and forgetting in monkeys' memory of informational object-reward associations. Learning and Motivation 10, 419–444.

Garon, N., Moore, C., 2004. Complex decision-making in early childhood. Brain and Cognition 55, 158–170.

Gomez-Beldarrain, M., Harries, C., Garcia-Monco, J.C., Ballus, E., Grafman, J., 2004. Patients with right frontal lesions are unable to assess and use advice to make predictive judgements. Journal of Cognitive Neuroscience 16 (1), 74–89.

Gonzalez, R., Vassileva, J., Bechara, A., Grbesic, S., Sworowski, L., Novak, R.M., 2005. The influence of executive functions, sensation seeking, and HIV serostatus on the risky sexual practices of substance-dependent individuals. Journal of the International Neuropsychological Society 11, 121–131.

Goudriaan, A.E., Oosterlaan, J., de Beurs, E., van den Brink, W., 2005. Decision making in pathological gambling: a comparison between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls. Cognitive Brain Research 23, 137–151.

Graham, F.K., 1979. Distinguishing among orienting, defense and startle reflexes. In: Kimmel, H.D., Olst, E.H.V., Orlebeke, J.F. (Eds.), The Orienting Reflex in Humans. Erlbaum, Hillsadale, NJ.

Harlow, J.M., 1868. Recovery from the passage of an iron bar through the head. Publications of the Massachusetts Medical Society 2, 327–347 (also in booklet form published in Boston in 1869 by D.H. Clapp).

Harmsen, H., in press. The relationship between impaired decision making, sensation seeking and readiness to change in cigarette smokers. Addictive Behaviours (2005; Jun 24 [Epub ahead of print]).

Heims, H.C., Critchley, H.D., Dolan, R., Mathias, C.J., Cipolotti, L., 2004. Social and motivational functioning is not critically dependent on feedback of autonomic responses: neuropsychological evidence from patients with pure autonomic failure. Neuropsychologia 42, 1979–1988.

Hinson, J.M., Jameson, T.L., Whitney, P., 2002. Somatic markers, working memory, and decision making. Cognitive Behavioural and Affective Neuroscience 2 (4), 341–353.

Hohmann, G.W., 1966. Some effects of spinal cord lesions on experienced emotional feelings. Psychophysiology 3, 143–156.

Hooper, C.J., Luciana, M., Conklin, H.M., Yarger, R.S., 2004. Adolescents performance on the Iowa Gambling Task: implications for the development of decision making and ventromedial prefrontal cortex. Developmental Psychology 40 (6), 1148–1158.

Hornak, J., O'Doherty, J., Bramham, J., Rolls, E.T., Morris, R.G., Bullock, P.R., 2004. Reward related reversal learning after surgical excisions in orbito-frontal or dorsolateral prefrontal cortex in humans. Journal of Cognitive Neuroscience 16 (3), 463–478.

Izard, C.E., 1990. The substrates and functions of emotion feelings: William James and current emotion theory. Personality and Social Psychology Bulletin 16, 626–635.

Izquierdo, A., Suda, R.K., Murray, E.A., 2004. Bilateral orbital prefrontal cortex lesions in rhesus monkeys disrupt choices guided by both reward value and reward contingency. The Journal of Neuroscience 24 (34), 7540–7548.

James, W., 1884. What is an emotion? Mind 19, 188–205.

James, W., 1894. The physical basis of emotion. Psychological Review 1, 516–529.

Jameson, T.L., Hinson, J.M., Whitney, P., 2004. Components of working memory and somatic markers in decision making. Psychonomic Bulletin and Review 11 (3), 515–520.

Jollant, F., Bellivier, F., Leboyer, M., Astruc, B., Torres, S., Verdier, R., 2005. Impaired decision making in suicide attempters. American Journal of Psychiatry 162 (2), 304–310.

Kahneman, D., Tversky, A., 1972. Judgement under uncertainty: Heuristics and biases. Psychological Review 80, 237–251.

Katkin, E.S., Wiens, S., Öhman, A., 2001. Nonconscious fear conditioning: Visceral perception and the development of gut feelings. Psychological Science 12 (5), 366–370.

Keillor, J.M., Barret, A.M., Crucian, G.P., Kortenkamp, S., Heilman, K.M., 2002. Emotional experience and perception in the absence of facial feedback. Journal of the International Neuropsychological Society 8, 130–135.

Kerr, A., Zelazo, P.D., 2004. Development of 'hot' executive function: the children's gambling task. Brain and Cognition 55 (1), 148–157.

Killcross, S., Coutureau, E., 2003. Coordination of actions and habits in the medial prefrontal cortex of rats. Cerebral Cortex 13, 400–408.

Kleeberg, J., Bruggimann, M.A., Annoni, J., Melle, G., Bougousslavsky, M.D., Schluep, M., 2004. Altered decision-making in multiple sclerosis: a sign of impaired emotional reactivity. Annals of Neurology 56 (6), 787–795.

Lacey, J.L., 1967. Somatic response patterning and stress: some revisions of activation theory. In: Appley, M.H., Trumbull, R. (Eds.), Psychological Stress. Appleton Century Crofts, New York.

Laird, J., Bresler, C., 1990. William James and the mechanisms of emotional experience. Personality and Social Psychology Bulletin 16, 630–651.

Lamar, M., Resnick, S.M., 2004. Aging and prefrontal functions: dissociating orbitofrontal and dorsolateral abilities. Neurobiology of Aging 25, 553–558.

Lambie, J.A., Marcel, A.J., 2002. Consciousness and the varieties of emotion experience: A theoretical framework. Psychological Review 109 (2), 219–259.

Lang, P.J., 1994. The varieties of emotional experience: a meditation on James-Lange theory. Psychological Review 101 (2).

Lange, C., 1885. The mechanism of the emotions. In: Dunlap, E. (Ed.), The Emotions. Williams & Wilkins, Baltimore, Maryland.

Lawrence, A.D., Sahakian, B.J., Rogers, R.D., Hodges, J.R., Robbins, T.W., 1999. Discrimination, reversal, and shift learning in Huntington's disease: mechanisms of impaired response selection. Neuropsychologia 37, 1359–1374.

Lehto, J.E., Elorinne, E., 2003. Gambling as an executive function task. Applied Neuropsychology 10 (4), 234–238.

Levine, B., Black, S.E., Cheung, G., Campbell, A., O'Toole, C., Schwartz, M.L., 2005. Gambling task performance in traumatic brain injury. Cognitive and Behavioural Neurology 18 (1), 45–54.

Lim, C.L., Rennie, C., Barry, R.J., Bahramali, H., Lazzaro, H., Manor, B., 1997. Decomposing skin conductance into tonic and phasic components. International Journal of Psychophysiology 25, 97–109.

Linton, J.C., Hirt, M., 1979. A comparison of predictions from peripheral and central theories of emotion. British Journal of Medical Psychology 52, 11–15.

Loewenstein, G., Lerner, J.S., 2003. The role of affect in decision-making. In: Davidson, R.J., Scherer, K.R., Goldsmith, H.H. (Eds.), Handbook of Affective Sciences. Oxford University Press, Oxford, pp. 619–642.

Loewenstein, G.F., Weber, E.U., Hsee, C.K., Welch, N., 2001. Risk as Feelings. Psychological Bulletin 127 (2), 267–286.

Lovibond, P.F., Shanks, D.R., 2002. The role of awareness in Pavlovian conditioning: Empirical evidence and theoretical interpretations. Journal of Experimental Psychology: Animal Behaviour Processes 28, 3–26.

MacLean, P., 1949. Psychosomatic disease and the visceral brain; recent developments bearing on the Papez theory of emotion. Psychosomatic Medicine 11 (6), 338–353.

MacLean, P., 1975. Sensory and perceptive factors in emotional functions of the triune brain. In: Levi, L. (Ed.), Emotions—Their Parameters and Measurement. Raven Press, New York, pp. 71–92.

MacMillan, M., 2000. An Odd Kind of Fame: Stories of Phineas Gage. MIT Press, Cambridge, Massachusetts.

Maia, T.V., McClelland, J.L., 2004. A reexamination of the evidence for the somatic marker hypothesis: what participants really know in the Iowa gambling task. Proceedings of the National Academy for Science U S A 101 (45), 16075–16080.

Maia, T.V., McClelland, J.L., 2005. The somatic marker hypothesis: still many questions but no answers. Trends in Cogntive Science 9 (4), 162–164.

Mandler, G., 1990. William James and the construction of emotion. Psychological Science 1, 179–180.

Manes, F., Sahakian, B., Clark, L., Rogers, R., Antoun, N., Aitken, M., et al., 2002. Decision-making processes following damage to the prefrontal cortex. Brain 125 (Pt 3), 624–639.

Marston, W.M., 1928. Emotions of Normal People. Harcourt, Brace, New York.

Martin, C.O., Denburg, N.L., Tranel, D., Granner, M.A., Bechara, A., 2004a. The effects of vagus nerve stimulation on decision-making. Cortex 40 (4–5), 605–612.

Martin, E.M., Pitrak, D.L., Weddington, W., Rains, N.A., Nunnally, G., Nixon, H., 2004b. Cognitive impulsivity and HIV serostatus in substance dependent males. Journal of the International Neuropsychological Society 10, 931–938.

McGinn, C., 2003. Fear factor: a review of looking for Spinoza by Antonio Damasio. New York Times Book Review, 2011.

Mellers, B.A., Schwartz, A., Ho, K., Ritov, I., 1997. Decision affect theory: emotional reactions to the outcomes of risky options. Psychological Science 8 (6), 423–429.

Mongini, F., Keller, R., Deregibus, A., Barbalonga, E., Mongini, M., 2005. Frontal lobe dysfunction in patients with chronic migraine: a clinical-neuropsychological study. Psychiatry Research 133, 101–106.

Montague, P.R., Hyman, S.E., Cohen, J.D., 2004. Computational roles for dopamine in behavioural control. Nature 431, 760–767.

Monteresso, J., Ehrman, R., Napier, K.L., O'Brien, C.P., Childress, A.R., 2001. Three decision-making tasks in cocaine-dependent patients: do they measure the same construct? Addiction 96, 1825–1837.

Montoya, P., Schandry, R., 1994. Emotional experience and heartbeat perception in patients with spinal cord injury and control subjects. Journal of Psychophysiology 8, 289–296.

Mowrer, O.H., 1947. On the dual nature of learning—a reinterpretation of 'conditioning' and 'problem solving'. Harvard Educational Review 17, 102–148.

Mowrer, O.H., 1960. Learning Theory and Behaviour. Wiley, New York.

Naccache, L., Dehaene, S., Cohen, L., Habert, M., Guichart-Gomez, E., Galanaud, D., et al., 2005. Effortless control: executive attention and conscious feeling of mental effort are dissociable. Neuropsychologia 43, 1318–1328.

Nauta, W.J.H., 1971. The problem of the frontal lobe: a reinterpretation. Journal of Psychiatric Research 8, 167–187.

Nielen, M.M.A., Veltman, D.J., de Jong, R., Mulder, G., den Boer, J.A., 2002. Decision making performance in obsessive compulsive disorder. Journal of Affective Disorders 257, 260.

North, N.T., O'Carroll, R.E., 2001. Decision making in patients with spinal cord damage: afferent feedback and the somatic marker hypothesis. Neuropsychologia 39 (5), 521–524.

O'Carroll, R.E., Papps, B.P., 2003. Decision making in humans: the effect of manipulating the central noradrenergic system. Journal of Neurology, Neurosurgery and Psychiatry 74, 376–378.

Öngur, D., Price, J.L., 2000. The organisation of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. Cerebral Cortex 10, 206–209.

Overman, W.H., 2004. Sex differences in early childhood, adolescence, and adulthood on cognitive tasks that rely on orbital prefrontal cortex. Brain and Cognition 55 (1), 134–147.

Overman, W.H., Frassrand, K., Ansel, S., Trawalter, S., Bies, B., Redmond, A., 2004. Performance on the IOWA card task by adolescents and adults. Neuropsychologia 42 (13), 1838–1851.

Oya, H., Adolphs, R., Kawasaki, H., Bechara, A., Damasio, A.R., 2005. Electrophysiological correlates of reward prediction error recorded in the human prefrontal cortex. Proceedings of the National Academy of Sciences USA 102 (23), 8351–8356.

Panksepp, J., 2003. Review article: "Looking for Spinoza: Joy, Sorrow, and the Feeling Brain" by A. Damasio. Consciousness & Emotion 4(1), 111–134.

Power, M., Dalgleish, T., 1997. Cognition and Emotion: From Order to Disorder. Psychology Press, Hove.

Pribram, K.H., 1970. Feelings as monitors. In: Arnold, M.B. (Ed.), Feelings and Emotions: The Loyola Symposium. American Psychiatric Press, New York.

Prinz, J.J., 2004. Gut Reactions. Oxford University Press, Oxford.

Rahman, S., Sahakian, B.J., Hodges, J.R., Rogers, R.D., Robbins, T.W., 1999. Specific cognitive deficits in mild frontal variant frontotemporal dementia. Brain 122 (Pt 8), 1469–1493.

Reavis, R., Overman, W.H., 2001. Adult sex differences on a decision-making task previously shown to depend on the orbital prefrontal cortex. Behavioural Neuroscience 115 (1), 196–206.

Reid, I.C., Morris, R.G.M., 1992. Smells are no surer: rapid improvements in olfactory discrimination is not due to a learning set. Proceedings of the Royal Society of London, Series B 247, 137–143.

Reisenzein, R., Meyer, W.U., Schutzwohl, A., 1995. James and the physical basis of emotion: a comment on Ellsworth. Psychological Review 102, 757–761.

Rescorla, R.A., 1996. Response inhibition in extinction. Quarterly Journal of Experimental Psychology 50, 238–252.

Rescorla, R., Solomon, R.C., 1967. Two-process learning theory: relationships between Pavlovian conditioning and instrumental conditioning. Psychological Review 74, 151–182.

Restle, F., 1958. Toward a quantitative description of learning set data. Psychological Review 65, 77–91.

Ritter, L.M., Meador-Woodruff, J.H., Dalack, G.W., 2004. Neurocognitive measures of prefrontal cortical dysfunction in schizophrenia. Schizophrenia Research 68, 65–73.

Rogers, R., Everitt, B.J., Baldacchino, A., Blackshaw, A.J., Swainson, R., Wynne, K., Baker, N.B., Hunter, J., Carthy, T., Booker, E., London, M., Deakin, J.F., Sahakian, B.J., Robbins, T.W., 1999. Dissociable deficits in the decision-making cognition of chronic amphetamine abusers, opiate abusers, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. Neuropsychopharmacology 20 (4), 322–339.

Rolls, E.T., 1996. The orbitofrontal cortex. Philosophical transactions of the Royal Society of London (Series B) 351, 1433–1444.

Rolls, E.T., 1999. The Brain and Emotion. Oxford University Press, Oxford.

Rolls, E.T., Hornak, J., Wade, D., McGrath, J., 1994. Emotion-related learning in patients with social and emotional changes associated with frontal lobe damage. Journal of Neurology Neurosurgery and Psychiatry 57 (12), 1518–1524.

Rotheram-Fuller, E., Shoptaw, S., Berman, S.M., London, E.D., 2004. Impaired performance in a test of decision-making by opiate-dependent tobacco smokers. Drug and Alcohol Dependence 73 (1), 79–86.

Sanfey, A.G., Hastie, R., Colvin, M.K., Grafman, J., 2003. Phineas gauged: decision-making and the human prefrontal cortex. Neuropsychologia 41 (9), 1218–1229.

Saper, C.B., 2002. The central autonomic nervous system: conscious visceral perception and autonomic pattern generation. Annual Review of Neuroscience 25, 433–469.

Saver, J.L., Damasio, A.R., 1991. Preserved access and processing of social knowledge in a patient with acquired sociopathy due to ventromedial frontal damage. Neuropsychologia 29, 1241–1249.

Schachter, S., Singer, J.E., 1962. Cognitive, social, and physiological determinants of emotional state. Psychological Review 69, 379–399.

Schmitt, W.A., Brinkley, C.A., Newman, J.P., 1999. Testing Damasio's somatic marker hypothesis with psychopathic individuals: risk takers or risk averse? Journal of Abnormal Psychology 108 (3), 538–543.

Shanks, D.R., 2005. Implicit learning. In: Lamberts, K., Goldstone, R. (Eds.), Handbook of Cognition. Sage, London, pp. 202–220.

Shanks, D.R., Lovibond, P.F., 2002. Autonomic and eyeblink conditioning are closely related to contingency awareness. Journal of Experimental Psychology: Animal Behaviour Processes 28 (1), 38–42 (Reply to Wiens and Ohman (2002) and Manns et al. (2002)).

Shanks, D.R., St. John, M.F., 1994. Charcteristics of dissociable learning systems. Behavioural and Brain Sciences 17, 367–447.

Shiv, B., Loewenstein, G., Bechara, A., 2005. The dark side of emotions in decision-making: when individuals with decreased emotional reactions make more advantageous decisions. Cognitive Brain Research 23, 85–92.

Shurman, B., Horan, W.P., Nuechterlein, K.H., 2005. Schizophrenia patients demonstrate a distinctive pattern of decision-making impairment on the Iowa Gambling Task. Schizophrenia Research 72, 215–224.

Solomon, R.L., Turner, L.H., 1962. Discriminative classical conditioning in dogs paralysed by Curare can later control discriminative avoidance responses in the normal state. Psychological Review 69 (3), 202–219.

Suzuki, A., Hirota, A., Takasawa, N., Shigemasu, K., 2003. Application of the somatic marker hypothesis to individual differences in decision making. Biological Psychology 65 (1), 81–88.

Teasdale, J.D., Barnard, P.J., 1993. Affect, Cognition and Change. Lawrence Erlbaum Associates, Hove.

Tomb, I., Hauser, M., Deldin, P., Caramazza, A., 2002. Do somatic markers mediate decisions on the gambling task? Nature Neuroscience 5 (11), 1103–1104 (author reply 1104).

Toplak, M.E., Jain, U., Tannock, R., 2005. Executive and motivational processes in adolescents with attention-deficit-hyperactivity disorder (ADHD). Behaviour and Brain Functions 27 (1).

Tranel, D., 1994. In: Fowles, D.C., Sutker, P., Goodman, S.H. (Eds.), Acquired Sociopathy: The Development of Sociopathic Behaviour Following Focal Brain Damage Progress in Experimental Psychology and Psychopathology Research, vol. 17, pp. 258–311.

Tranel, D., Bechara, A., Damasio, H., Damasio, A.R., 1996. Fear conditioning after ventromedial frontal lobe damage in humans. Society for Neuroscience Abstracts 22, 1108.

Tranel, D., Bechara, A., Damasio, A.R., 1999. Decision making and the somatic marker hypothesis. In: Gazzaniga, M.S. (Ed.), The New Cognitive Neurosciences. MIT Press, Cambridge, Massachusetts, pp. 1047–1061.

Tranel, D., Bechara, A., Denburg, N.L., 2002. Asymmetric functional roles of right and left ventromedial prefrontal cortices in social conduct, decision-making, and emotional processing. Cortex 38 (4), 589–612.

Tucker, K.A., Potenza, M.N., Beauvals, J.E., Browndyke, J.N., Gottschalk, P.G., Kosten, T.R., 2004. Perfusion abnormalities and decision making in cocaine dependence. Biological Psychiatry 56, 527–530.

Turnbull, O.H., Berry, H., Bowman, C.H., 2003. Direct versus indirect emotional consequences on the Iowa Gambling Task. Brain and Cognition 53 (2), 389–392.

Turnbull, O.H., Evans, C.Y., Bunce, A., Carzolio, B., O'Connor, J., 2005. Emotion based learning and central executive resources: An investigation of intuition and the Iowa Gambling Task. Brain and Cognition 57 (3), 244–247.

Turnbull, O.H., Evans, C.Y., Kemish, K., Park, S., Bowman, C.H., in press. A novel 'set-shifting' modification of the Iowa Gambling Task: Flexible emotion-based learning in schizophrenia. Neuropsychology.

Turnbull O.H., Evans C.E., 2005. Preserved complex emotion-based learning in amnesia. Neuropsychologia [Epub ahead of print].

van Honk, J., Hermans, E.J., Putman, P., Montagne, B., Schutter, D.J., 2002. Defective somatic markers in sub-clinical psychopathy. Neuroreport 13 (8), 1025–1027.

van Honk, J., Schutter, D.J., Hermans, E.J., Putman, P., Tuiten, A., Koppeschaar, H., 2004. Testosterone shifts the balance between sensitivity for punishment and reward in healthy young women. Psychoneuroendocrinology 29 (7), 937–943.

Verdejo, A., Aguilar de Arcos, F., Perez Garcia, M., 2004. Alterations in the decision making processes linked to the ventromedial prefrontal cortex in drug-abusing patients. Review of Neurology 38 (7), 601–606.

Whitlow, C.T., Liguori, A., Livengood, L.B., Hart, S.L., Mussat-Whitlow, B.J., Lamborn, C.M., 2004. Long-term heavy marijuana users make costly decisions on a gambling task. Drug and Alcohol Dependence 76, 107–111.

Whitney, K.A., Fastenau, P.S., Evans, J.D., Lysaker, P.H., 2004. Comparative neuropsychological function in obsessive-compulsive disorder and schizophrenia with and without obsessive-compulsive symptoms. Schizophrenia Research 69, 75–83.

Wilder, K.E., Weinberger, D.R., Goldberg, T.E., 1998. Operant conditioning and the orbitofrontal cortex in schizophrenic patients: unexpected evidence for intact functioning. Schizophrenia Research 30, 169–174.

Wolpert, D.M., Ghahramani, Z., 2000. Computational principles of movement neuroscience. Nature Neuroscience 3, 1212–1217.

Zuckerman, M.M., 1994. Behavioural Expressions and Bisocial Bases of Sensation Seeking. Cambridge University Press, Cambridge.