The Somatic Marker Hypothesis and the Possible Functions of the Prefrontal Cortex [and Discussion]

Author(s): Antonio R. Damasio, B. J. Everitt and D. Bishop

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The somatic marker hypothesis and the possible functions of the prefrontal cortex

ANTONIO R. DAMASIO*

Department of Neurology, Division of Behavioral Neurology and Cognitive Neuroscience, University of Iowa College of Medicine, Iowa City, Iowa, U.S.A.

SUMMARY

In this article I discuss a hypothesis, known as the somatic marker hypothesis, which I believe is relevant to the understanding of processes of human reasoning and decision making. The ventromedial sector of the prefrontal cortices is critical to the operations postulated here, but the hypothesis does not necessarily apply to prefrontal cortex as a whole and should not be seen as an attempt to unify frontal lobe functions under a single mechanism.

The key idea in the hypothesis is that 'marker' signals influence the processes of response to stimuli, at multiple levels of operation, some of which occur overtly (consciously, 'in mind') and some of which occur covertly (non-consciously, in a non-minded manner). The marker signals arise in bioregulatory processes, including those which express themselves in emotions and feelings, but are not necessarily confined to those alone. This is the reason why the markers are termed somatic: they relate to body-state structure and regulation even when they do not arise in the body proper but rather in the brain's representation of the body.

Examples of the covert action of 'marker' signals are the undeliberated inhibition of a response learned previously; the introduction of a bias in the selection of an aversive or appetitive mode of behaviour, or in the otherwise deliberate evaluation of varied option-outcome scenarios. Examples of overt action include the conscious 'qualifying' of certain option-outcome scenarios as dangerous or advantageous.

The hypothesis rejects attempts to limit human reasoning and decision making to mechanisms relying, in an exclusive and unrelated manner, on either conditioning alone or cognition alone.

1. INTRODUCTION

This text is about a hypothesis, known as the somatic marker hypothesis, which concerns the possible role of some regions of the prefrontal cortex in the processes of reasoning and decision making. The text follows closely, in form and substance, several reviews in which my colleagues and I have presented the hypothesis and its preliminary testing (see Damasio 1994, 1995*a*).

The hypothesis developed as a response to a number of intriguing observations made in neurological patients with focal damage in the frontal lobe. Briefly, patients with damage to the prefrontal region, especially when the damage is centred in ventral and medial aspects of this region, present with severe impairments in personal and social decision making, in spite of otherwise largely preserved intellectual abilities (Damasio 1979, 1994). Before the onset of brain damage the patients may be described as intelligent, creative and successful; but after damage occurs the patients develop a pattern of abnormal decision making which is most notable in personal and social matters. Specifically, patients have difficulty planning their work day; difficulty planning their future over immediate, medium and long ranges and difficulty choosing suitable friends, partners and activities. The plans they organize, the persons they elect to join, or the activities they undertake often lead to financial losses, losses in social standing and losses to family and friends. The choices these patients make are no longer personally advantageous, socially inadequate and are demonstrably different from the choices the patients were known to have made in the premorbid period.

The patients' intellect remains normal, as measured by conventional IQ tests, so does the learning and retention of factual knowledge at both unique and non-unique levels and the learning and retention of skills. The ability to use logic in the solution of problems commonly posed in neuropsychological testing is also normal, so is language. Basic attention and working memory are not affected, nor is the ability to make estimates (as tested in a paradigm developed by Shallice & Evans 1978), to perform normally in the Wisconsin Card Sorting Test and to judge recency and frequency of events (see Milner 1963, 1964; Petrides & Milner 1982; Milner *et al.* 1985). The patients' repertoire of social knowledge is still retained and can be accessed in a laboratory situation (Saver & Damasio

Address for correspondence: Department of Neurology, University of Iowa Hospitals and Clinics, Iowa City, Iowa 52242, U.S.A.

1991). The disturbance shown by this particular class of patients cannot be explained in terms of defects in (a) pertinent knowledge; (b) intellectual ability; (c) language; (d) basic working memory; or (e) basic attention. As if this challenge were not enough, the patients pose yet another. Although their impairment is obvious in everyday life, there has not been, until recently, a laboratory probe to detect it or measure it.

In the text below, I outline the somatic marker hypothesis, which is part of a framework to account for the condition, and describe new laboratory probes designed to detect and measure aspects of the condition. I do not address the condition of patients whose frontal lesions are located in anatomical sectors other than the ventromedial. Some of those patients may also have defects in reasoning or decision making, but those defects are accompanied by impairments in abilities which are preserved in patients with ventromedial lesions. The conditions with which such other patients present may or not be accountable by the somatic marker hypothesis. The hypothesis should not be seen as a general theory for how prefrontal cortices work as, in all likelihood, this large and parcellated sector of the brain accomplishes several separate albeit cooperative functions.

2. THE SOMATIC MARKER HYPOTHESIS

The idea for the somatic marker hypothesis came from the realization that, while the ventromedial patients were intact in neuropsychological laboratory tests they did have a compromised ability to express emotion and to experience feelings in situations in which emotions would normally have been expected and would presumably have been present during the premorbid period. In other words, along with normal intellect and abnormal decision making, there were abnormalities in emotion and feeling. In the absence of other cognitive impairments that might effectively account for the salient aspects of the condition, I reasoned that the defect in emotion and feeling, along with its neurobiological underpinnings, would play an important role in the pathological process; and on the basis of the pathological process I then specified a number of structures and operations to be found in the normal condition. Because I see emotion as expressing itself most importantly, though not solely, through changes in the representation of body state, and because I believe that the results of emotion are primarily represented in the brain in the form of transient changes in the activity pattern of somatosensory structures, I designated the emotional changes under the umbrella term 'somatic state.' Note that by somatic I refer to musculoskeletal, visceral, and internal milieu components of the soma rather than just to the musculoskeletal aspect; and note also that a somatic signal or process, although related to structures which represent the body and its states does not need to originate in the body in every instance (see Damasio 1994, 1995*b* for details). The summary of the proposal's background assumptions and specific structures and operations is presented below.

(*a*) *Background assumptions*

In addition to an operative self and consciousness, the basis for neither of which I will discuss, the mechanisms I envision require four main assumptions:

1. that human reasoning and decision making depend on many levels of neurobiological operation, some of which occur in mind (i.e. are conscious, overt cognitive), and some of which do not. Minded (conscious, overt cognitive) operations depend on sensory images which are based on the coordinated activity of early sensory cortices.
2. that all mind operations regardless of the content of images, depend on support processes such as attention and working memory.
3. that reasoning and decision making depend on the availability of knowledge about situations, actors, options for action and outcomes. Such knowledge is stored in 'dispositional' form throughout higher-order cortices and some subcortical nuclei. (By the term dispositional I mean coded, implicit and non-topographically organized; see Damasio 1989*a*, *b*, 1994; Damasio & Damasio 1994; for details on dispositional knowledge and convergence zone framework.) Dispositional knowledge can be made explicit in (a) motor responses of varied types and complexity (some combinations of which can constitute emotions), and in (b) images. The result of all motor responses, including those that are not generated consciously (i.e. minded), can be represented in images and become minded.
4. that knowledge can be classified as follows:
   A. innate and acquired knowledge concerning bioregulatory processes and body states and actions, including those which are made explicit as emotions.
   B. knowledge about entities, facts (e.g. relations, rules), actions and action-complexes, and stories, which are usually made explicit as images.
   C. knowledge about the linkages between B items and A items, as reflected in individual experience.
   D. knowledge resulting from the categorizations of items in A, B and C.

(*b*) *Specific structures and operations*

(i) *Ventromedial prefrontal cortex as a repository of dispositionally recorded linkages between factual knowledge and bioregulatory states*

Structures in ventromedial prefrontal cortex provide the substrate for learning the association between certain classes of complex situation, on the one hand, and the type of bioregulatory state (including emotional state) usually associated with that class of situation in prior individual experience. The ventromedial sector would hold linkages between the facts that compose a given situation, and the emotion previously paired with it in an individual's contingent experience. The linkages are 'dispositional' in the sense that they do not hold the representation of the facts or of the emotional state explicitly, but hold rather the potential to reactivate an emotion by acting on the appropriate cortical or subcortical structures (see Damasio 1989*a*, *b*, 1994; Damasio & Damasio 1994; for discussion of

the concept of disposition and the convergence zone framework; see also Damasio 1994, 1995*b* for a discussion on the neurobiology of emotion). What I envision here is that the experience we acquire regarding a complex situation and its components – a certain configuration of actors and actions requiring a response; a set of response options; a set of immediate and long-term outcomes for each response option – is processed in sensory imagetic and motor terms and is then recorded in dispositional and categorized form. (The records are maintained in distributed form in large-scale systems which involve many cortices including those in prefrontal sectors other than the ventromedial). But the experience of some of those components, individually or in sets, has been associated with emotional responses, which were triggered from cortical and subcortical limbic sites that were dispositionally prepared to organize such a response. I propose that the ventromedial prefrontal cortex establishes a simple linkage, a memory in fact, between the disposition for a certain aspect of a situation (for instance, the long-term outcome for a type of response option), and the disposition for the type of emotion that in past experience has been associated with the situation.

(ii) *The reactivation of signals related to previous individual contingencies*

When a situation arises for which some factual aspect has been previously categorized, related dispositions are activated in higher-order association cortices (including in good likelihood some prefrontal cortices). This leads to the recall of pertinently associated facts which will be experienced in imagetic form. Simultaneously, or nearly so, the related ventromedial prefrontal linkages are also activated, and as a consequence, the emotional disposition apparatus is activated too (e.g. in the amygdala). The result of these combined activations is the approximate reconstruction of a previously learned factual-emotional set. In short, when a situation of a given class recurs, factual knowledge pertaining to the situation – possible options of action, outcomes of such actions immediately and at longer term – is evoked in sensory images based on the appropriate sensory cortices. But depending on previous individual contingencies, signals related to some or even many of those images, or even the entire situation, act on the ventromedial prefrontal cortex (which has previously acquired the link between the situation or its components and the class of somatic state), and trigger the re-activation of the somatosensory pattern that describes the appropriate emotion.

The re-activation described above can be carried out in one of two ways: via a 'body loop', in which the soma actually changes in response to the activation and the ensuing changes are relayed to somatosensory cortices; or via an 'as if body loop', in which the re-activation signals are conveyed to the somatosensory cortices which then adopt the appropriate pattern, the body being bypassed. From both evolutionary and ontogenetic perspectives I believe that the 'body loop' is the original mechanism but has been superseded by the 'as if' body loop and is possibly used less frequently than the 'as if' loop. The results of either 'body loop' or 'as if body loop' may become overt (conscious) or remain covert (non-conscious).

(iii) *A marker role for signals related to previous emotional state contingencies*

The establishment of a somatosensory pattern appropriate to the situation, via the 'body loop' or via the 'as if' loop, either overtly or covertly, is co-displayed with factual evocations pertinent to the situation and, qualifies those factual evocations. In doing so, it operates to constrain the process of reasoning over multiple options and multiple future outcomes. For instance, when the somatosensory image which defines a certain emotional response is juxtaposed to the images which describe a related scenario of future outcome, and which triggered the emotional response via the ventromedial linkage, the somatosensory pattern marks the scenario as good or bad. In other words, the images of the scenario are 'judged' and marked by the juxtaposed images of the somatic state.

When this process is overt, the somatic state operates as an alarm signal or an incentive signal. The somatic state is alerting you to the goodness or badness of a certain option-outcome pair. The device produces its result at the openly cognitive level. When the process is covert the somatic state constitutes a biasing signal. Using an indirect and non-conscious influence, for instance through a non-specific neurotransmitter system such as dopamine, the device influences cognitive processing.

(iv) *Somatic markers participate in process as well as content*

Certain emotion-related somatosensory patterns also act as boosters in the processes of attention and working memory. In addition to assisting with the process of specific experiential contents (e.g. certain combinations of facts and emotions), I believe they may also assist with response inhibition.

(v) *Somatic markers facilitate logical reasoning*

The operation of logical reasoning is facilitated by steps (iii) and (iv). Certain option-outcome pairs can be rapidly rejected or endorsed and, pertinent facts can be more effectively processed. The hypothesis thus suggests that somatic markers normally help constrain the decision-making space by making that space manageable for logic-based, cost-benefit analyses. In situations in which there is remarkable uncertainty about the future and in which the decision should be influenced by previous individual experience, such constraints permit the organism to decide efficiently within short time intervals.

In the absence of a somatic marker, options and outcomes become virtually equalized and the process of choosing will depend entirely on logic operations over many option-outcome pairs. The strategy is necessarily slower and may fail to take into account previous experience. This is the pattern of slow and error-prone decision behaviour we often see in ventromedial frontal lobe patients. Random and impulsive decision making is a related pattern.

Whether body states are real or vicarious (what I term 'as if'), the corresponding neural pattern can be made conscious and constitute a feeling. However, although many important choices involve feelings, a number of our daily decisions undoubtedly proceed without feelings. That does not mean that the evaluation that normally leads to a body state has not taken place, or that the body state or its surrogate has not been engaged, or that the dispositional machinery underlying the process has not been activated. It simply means that the body state or its surrogate have not been attended. Without attention, neither will be part of consciousness, although either can be part of a covert action on the mechanisms that govern, without willful control, our appetitive (approach) or aversive (withdrawal) attitudes toward the world. While the hidden machinery underneath has been activated, we may never know it.

There is yet another mechanism for covert action: it consists of triggering activity in certain neurotransmitter nuclei (e.g. dopamine), which is part of the 'emotional response', a physiological step which will subsequently bias cognitive processes, thus influencing the mode of reasoning and decision making.

3. A NEURAL NETWORK FOR SOMATIC MARKERS

Why is it that ventral and medial prefrontal cortices are ideally situated to establish the kind of linkages outlined above? These cortices, judging from what is known of nonhuman primate neuroanatomy, receive projections from all sensory modalities, directly or indirectly (Pandya & Kuypers 1969; Jones & Powell 1970; Chavis & Pandya 1976; Potter & Nauta 1979; Petrides & Pandya 1995; Pandya & Yeterian 1996 and this volume). In turn, they are the only known source of projections from frontal regions toward central autonomic control structures (Nauta 1971), and such projections have a demonstrated physiological influence on visceral control (Hall *et al.* 1977). The ventromedial cortices have extensive bidirectional connections with the hippocampus and amygdala (Van Hoesen *et al.* 1972; Van Hoesen *et al.* 1975; Porrino *et al.* 1981; Amaral & Price 1984; Goldman-Rakic *et al.* 1984;). Moreover, as shown by Rolls and colleagues (see this volume), nearby cortices in the orbitofrontal region contain the secondary association areas for taste and olfaction, receive other sensory inputs, namely visual, and are clearly involved in the signalling of reward to perceived stimuli.

This anatomical design is quite compatible with the idea that the ventromedial cortices contain convergence zones which hold a record of temporal conjunctions of activity in other neural units (e.g. varied sensory, limbic structures) hailing from both external and internal stimuli. This would be a record of signals from regions that were active simultaneously and which, as a set, defined a given situation or salient aspects of it. As noted, when parts of certain exteroceptive-interoceptive conjunctions are re-processed, consciously or not, their activation is signalled to ventromedial cortices, which in turn activate somatic effectors in amygdala, hypothalamus, and brainstem nuclei, or activate somatosensory structures directly. One might describe this process as an attempt to reconstitute the kind of somatic state that belonged to the conjunction in the first place.

The systems network necessary for somatic markers to operate thus includes the following essential structures: (1) ventromedial frontal cortices which contain convergence zones that record links between (a) the dispositions that represent categorizations of certain complex situations and their components, and (b) the dispositions that represent the somatic states that have been prevalently associated with the situations referred above; (2) central autonomic effectors, for example the amygdala, which can activate somatic responses in viscera, vascular bed, endocrine system and nonspecific neurotransmitter systems; (3) somatosensory cortices (namely insula, SII, and SI) and their interlocking projections (especially in the non-dominant hemisphere), which can receive signals from the soma (or signals from ventromedial cortices prescribing an 'as if' somatic pattern).

It is possible that structures in basal ganglia are also part of this network and can mediate responses from ventromedial cortices by acting on somatomotor structures (Tranel & Damasio 1993).

It is important to note that the evocation of a somatic marker for stimuli that are unconditioned and basic, for instance, a startling noise or a flash of light, uses a different and simpler network, that is a network that can cope with behaviourally relevant stimuli that do not need the complex informational processing that social configurations do. The alternate network would bypass the cerebral cortex altogether and activate autonomic centres (e.g. amygdala and others) directly from thalamus (Clugnet *et al.* 1988; Farb *et al.* 1988). My formulation predicts a dissociation between responses to complex stimuli which require cortical processing, and to basic stimuli which do not.

4. THE NATURE OF THE MARKER

Why should somatic signals be so critical to the process of reasoning and decision making? My answer is that certain classes of situation, namely those that concern personal and social matters, are frequently linked to punishment and reward and thus to pain, pleasure, and the regulation of homeostatic states, including the part of the regulation that is expressed by emotion and feeling. The inevitability of somatic participation comes from the fact that all of these bioregulatory phenomena, including emotion, are represented via the somatosensory system.

One may also ask why a signal external to the representations over which one reasons is needed at all. The answer, as suggested above, has to do with the uncertainty of outcomes, the dimension of the logical operations required by deciding under uncertainty, and the advantage of constraining the decision-making space.

The realm of basic survival behaviour provides the right setting to explain the possible origin of somatic markers. Let us assume that the brain has long had

available, in evolution, a means to select good responses rather than bad ones in terms of survival. I suspect that the mechanism has been co-opted for behavioural guidance outside the realm of basic survival. Nature would have evolved a highly successful mechanism of guidance to cope with basic problems whose answer might maximize survival. But a very large range of other problems, including those which pertain to the social realm, are indirectly linked to precisely the same framework of survival versus danger, of advantage versus disadvantage, of gain and balance versus loss and disequilibrium. It is plausible that a system geared to produce markers and signposts to guide basic survival, would have been pre-adapted to assist with 'intellectual' decision making. The somatic markers would not necessarily be perceived in the form of 'feelings'. They could act covertly to highlight, in the form of an attentional mechanism, certain components over others, and to direct, in effect, the go, stop, and turn signals necessary for much decision making and planning on even the most abstract of topics. Shallice & Burgess (1993), have also proposed that some form of marker is needed in decision making, although they have not specified the neurobiological nature of the marker and it may be different from what I propose here. Nonetheless my proposal and theirs do share this trait.

In conclusion, in normal individuals, certain situations require high-order composite memories formed by 'facts' and by the 'body states' which usually accompany those facts in an individual's experience. The 'fact' memories are held in dispositional form in the appropriate association cortices. The 'body state' memories do not need to be held permanently, as body states can be re-enacted on demand. Only the memory of the linkage between certain classes of situation and certain body states must be held permanently, and I believe the system necessary for such memories is in ventral and medial prefrontal cortices.

Patients with ventromedial frontal lobe damage fail to evoke part of the composite memory, for a class of situation; the part that describes the association between the class of situation and the somatosensory state linked to the situation. The factual knowledge component of the composite memory can still be evoked, but somatic states cannot be re-enacted, overtly or covertly, relative to those facts. This limitation poses no problem for situations that have minimal somatic state associations in previous experience, but is catastrophic for situations that do.

5. TESTING THE SOMATIC MARKER HYPOTHESIS

We have begun a series of experiments aimed at providing a possible physiopathological explanation for the defect. Some pertinent results are described below.

(*a*) *Somatic responses to emotionally charged stimuli*

In these experiments we tested the hypothesis that patients with bilateral damage in the ventromedial prefrontal cortices would not generate somatic states in response to emotionally charged stimuli. The basic idea was that the processing of stimuli with emotional significance would be affected by the previous experiences the subjects had with those stimuli, and that the ventromedial prefrontal cortex would be pivotal to reactivate the somatic states that had been usually engendered when those stimuli were experienced.

In order to assess the presence or absence of a change in somatic state we decided to measure a standard autonomic index, the skin conductance response (SCR). We studied 3 groups of subjects. The first was constituted by normal controls, without neurological or psychiatric illness. The second comprised subjects with lesions located outside the frontal cortices. The third comprised subjects with lesions in the ventromedial frontal cortex. All subjects in the third group had both bilateral damage in the target region and the index condition, that is, acquired defects in decision making in their real life, real time behaviour.

The experimental condition called for the subjects to view two types of visual image. One type was emotionally neutral, for example landscapes or abstract patterns. The other was emotionally charged, for example scenes of social catastrophe, or body mutilation.

The state of responsivity of the autonomic nervous system was assessed in all three groups of subjects by their SCRs to startling stimuli such as loud noises, or to the behaviours that reliably elicit SCRs, for example deep breath. All three groups had normal SCRs in that condition. In the experimental condition, however, while both normal controls and nonfrontal brain damaged groups exhibited standard SCR responses to the emotionally charged stimuli and little or no response to the neutral stimuli, the subjects with ventromedial frontal damage failed to react to the emotionally charged stimuli (Damasio *et al.* 1990; Damasio *et al.* 1991; Tranel 1994; Tranel *et al.* 1995). The findings suggest that patients with bilateral ventromedial frontal damage and decision-making defects in personal and social domain, no longer have a normal ability to generate somatic responses to stimuli with an emotional component.

(*b*) *The gambling experiments*

Another approach to the testing of the somatic marker hypothesis relied on a novel card gambling task (Bechara *et al.* 1994). The task is an attempt to create in the laboratory a realistic situation in which subjects gradually learn how to play a card game, to their best advantage, in situations of limited knowledge about the contingencies, and under the control of rewards and penalties. As described in our original publication, the task operates as follows: the subjects sit in front of four decks of cards equal in appearance and size, and are given a $2000 loan of play money (facsimile US dollar bills). They are told that the game requires a series of card selections, one card at a time, from any of the four decks, until they are told to stop. The subjects are also told that (1) the goal of the task is to maximize profit on the loan of play money, (2) they are free to

switch from any deck to another, at any time, and as often as wished; but (3) they are not told in advance how many card selections must be made. The task is stopped after 100 card selections. After each card turning, the subjects receive some money. The amount is announced after the turning and varies with the deck. Turning any card from decks A or B yields $100; turning any card from decks C or D yields $50. After turning some cards of any deck, however, the subjects are both given money and asked to pay a penalty. Again the amount is announced after the card is turned and varies with the deck and the position in the deck according to a schedule unknown to the subjects. The ultimate yield of each deck varies because the penalty amounts are higher in the high-paying decks (A and B), and lower in the low-paying decks (C and D). For example, after turning ten cards from deck A, the subjects have earned $1000, but they have also encountered five unexpected punishments bringing their total cost to $1250, and incurring a net loss of $250. They encounter the same problem on deck B. On the other hand, after turning ten cards from decks C or D, the subjects earn $500, but their unpredicted punishments only amount to $250, that is subjects incur a net gain of $250. In short, decks A and B are equivalent in terms of overall net loss over the trials. The difference is that in deck A, the punishment is more frequent, but of smaller magnitude, whereas in deck B, the punishment is less frequent but of higher magnitude. Decks C and D are also equivalent in terms of overall net loss. In deck C, the punishment is more frequent and of smaller magnitude, while in deck D the punishment is less frequent but of higher magnitude. Decks A and B are 'disadvantageous' because they cost the most in the long run, while decks C and D are 'advantageous' because they result in an overall gain in the long run.

The performance of a group of normal control subjects (21 women and 23 men) in this task was compared to those of ventromedial prefrontal subjects (4 men and 2 women). The age range of normal controls was from 20 to 79 years; for ventromedial subjects it was from 43 to 84 years. About half the number of subjects in each group had a high school education, and the other half had a college education.

The results were clear cut. Normals and patients without frontal damage sample from all decks for a while and gradually begin playing more frequently from the good decks than from the bad. About half way through the game they finally adopt this strategy and never abandon it. As a result they come out ahead. Ventromedial frontal lobe patients, on the contrary, continue to play predominantly from the bad decks, in spite of repeated losses. As a result they lose all of their loan and need to borrow money.

Although the gambling task involves a long series of gains and losses, it is not possible for subjects to perform an exact calculation of the net gains or losses generated from each deck as they play. (A group of normal control subjects with superior memory and IQ, whom we asked to think aloud while performing the task and keep track of the magnitudes and frequencies of the various punishments, could not provide figures for the net gains or losses from each deck). The subjects must rely on their ability 'to sense', overtly or not, which decks are risky and which are profitable. The performance profile of ventromedial patients is comparable to their real-life inability to decide advantageously, especially in personal and social matters, a domain for which in life, as in the task, an exact calculation of the future outcomes is not possible and choices must be based on approximations. This task offers, for the first time, the possibility of detecting these patients' elusive impairment in the laboratory.

My colleagues and I have considered several possibilities for why the target patients make choices that have high immediate reward but severe delayed punishment. The first is that patients are so sensitive to reward that the prospect of future (delayed) punishment is outweighed by that of immediate gain. The second is that they are insensitive to punishment, and thus the prospect of reward always prevails, even if they are not abnormally sensitive to reward. The third is that they are generally insensitive to future consequences, positive or negative, and thus their behaviour is mostly guided by immediate prospects. We also considered mechanisms behind these three possibilities. For instance, an apparent sensitivity to immediate reward might be caused by defective response inhibition. This would assume that previous learning in comparable situations would have led to a systematic suppression of a prior, more basic drive to reach for reward. There is much in both animal and human studies to support this idea (see, for instance, Diamond 1990 and this volume; Dias *et al.* 1996). But the complexity of the task, the wealth of knowledge available to the minds of the players, and the length of time over which the result continues to be consistently obtained, makes this mechanism implausible as the sole source of the defect. A change in the task's design (placing punishment up front and using unpredictable reward schedules as the unexpected variable) reveals that the patients continue to behave the same way, which goes against both the possibility of hypersensitivity to reward and insensitivity to punishment (Anderson *et al.* 1996).

Our preferred account, given evidence from other studies to indicate that these patients retain and access the knowledge necessary to conjure up options of actions and scenarios of future outcomes and yet fail to act on such knowledge, is that a lack of both covert as well as overt markers for scenarios of future outcome, fails to provide helpful 'positive' signals to guide the performance and thus cannot counteract the influence of 'negative' signals (Bechara *et al.* 1994). It is also possible that the somatic marker failure weakens support processes such as attention and working memory thus rendering unstable the representations of future outcomes that these patients evoke. In other words, the representations would not be held in working memory long enough for attention to enhance them and for reasoning strategies to operate on them. This mechanism invokes a defect along the lines proposed for behavioural domains dependent on dorsolateral prefrontal cortex networks (Goldman-Rakic 1987), also invoked in other accounts of frontal

lobe defect (see Fuster 1989; Posner & Petersen 1990; Baddeley 1995).

(*c*) *The psychophysiological dimension of the gambling experiments*

In a further test to the somatic marker test hypothesis we undertook a continuous monitoring of SCRs, while normal subjects and patients were engaged in the gambling task (Bechara *et al.* 1996). The most salient result of this study was the finding that, in normal subjects, during the time window that precedes the selection of a card from a given deck – an interval of about four seconds – normal subjects begin to respond with high amplitude skin conductance responses whenever they are about to make a selection from a bad deck. They show no comparable responses when they are about to make a selection from a good deck. As the task unfolds, the SCRs to the bad decks continue to appear systematically and they rise in amplitude. This does not happen for the responses associated with the good decks. Quite remarkably, no such anticipatory responses are seen in the patients with ventromedial frontal damage, who do show, nonetheless, normal SCRs to actual loss of money, that is SCRs to punishment.

One possible interpretation is that the SCR is part of a very early and automated alarm signal, which is triggered, as proposed in the somatic marker hypothesis, from the ventromedial region. The signal affects further processing of the factual knowledge connected with the situation by marking a particular option-outcome pair with a negative bias. Incidentally, this interpretation holds whether the signal is overt and fully appreciated in consciousness, or covert and entirely operated at an unconscious level.

An alternative interpretation is that normal subjects reason, early on, that certain decks are bad and certain decks are good, and that on the basis of their cognition of 'badness' and 'goodness', they generate a somatic response which is indexed by the SCR. I find the latter interpretation less plausible for reasons that have to do with my perspective on the evolutionary biology and adaptive value of an automated somatic marker device. Moreover, recent studies in our laboratory suggest that normal subjects begin producing their SCRs to bad decks long before they have, according to their testimony, any notion whatsoever of the good or evil nature of each deck and of the design of the game they are playing.

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Discussion

B. J. Everitt (*Department of Experimental Psychology, University of Cambridge, Downing Street, Cambridge CB2 3EB, U.K.*). To what extent do the peripheral somatic nervous system changes contribute to the risk-taking behaviour that you describe in your gambling test. For example, if you experimentally manipulate these peripheral changes would you expect to see changes in behaviour?

A. R. Damasio. The role of peripheral signals in current behaviour guidance varies as I see it, between developmental and adult phases of an individual. The somatic marker hypothesis suggests that during development and in non-average situations, body states are actively engaged and very much a part of the emotion/feeling loop. However, in most circumstances an 'as if' body loop is engaged instead, and the body, that is the periphery, is bypassed entirely. Only neural structures which represent states of the body are then modified. Thus, in the more frequently operational mode, changes in the periphery ought to have relatively little impact in central functions related to emotion/feeling and reasoning/decision making. The final answer on this issue, will however, require further study. For instance, evidence from the study of both cord damage as well as peripheral neuropathy suggests that, in spite of the obligate incompleteness of such peripheral lesions, there may be some effect of the periphery on central processes.

D. Bishop (*MRC Applied Psychology Unit, 15 Chaucer Road, Cambridge CB2 2EF, U.K.*). In your gambling task, subjects did not simply have to learn a particular response to a particular stimulus. They also had to learn to change that response as all subjects at the start of the test chose decks A and B in preference to decks C and D, as the former decks initially provided larger rewards. Thus, was the impaired performance of the patients with frontal damage on this gambling task due to a failure to inhibit an earlier association rather than simply a failure to form associations? For examples, would you a) predict, or b) have you found that such patients are impaired on forming conditioned associations using a standard classical conditioning paradigm?

A. R. Damasio. It is conceivable that the performance of patients with ventromedial damage in the gambling task reveals an inability to inhibit a previously acquired response to a reward. Nonetheless, the most intriguing aspect of the experiment is the finding that such a failure would not be compensated by the patients' realization that their strategy has led them to losses and will continue to do so. The point is, thus, that reasoning unaided by something as simple as the learning required by our task, does not seem to operate normally, or, if it does, does not prevail in guiding the behaviour of the individual. On the matter of conditioning: we have evidence that most patients who fail the gambling task acquire classical conditioning normally. This is especially interesting because it implies that there are dissociable aspects of 'conditioning' relative to the nature of the task.