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The Somatic Marker Hypotheses, and what the Iowa Gambling Task does and does not show

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Abstract

Damasio's Somatic Marker Hypothesis (SMH) is a prominent neuroscientific hypothesis about the mechanisms implementing decision-making. This paper argues that, since its inception, the SMH has not been clearly formulated. It is possible to identify at least two different hypotheses, which make different predictions: *SMH-G*, which claims that somatic states generally implement preferences and are needed to make a decision; and *SMH-S*, which specifically claims that somatic states assist decision-making by anticipating the long-term outcomes of available options. This paper also argues that neither hypothesis is adequately supported empirically; the task originally proposed to test SMH is not a good test for *SMH-S*, and its results do not support *SMH-G* either. In addition, it is not clear how *SMH-G* could be empirically invalidated, given its general formulation. Suggestions are made that could help provide evidence for *SMH-S*, and make *SMH-G* more specific.

1 Introduction

The Somatic Marker Hypothesis (SMH) was proposed by cognitive neuroscientist Antonio Damasio in the early nineties and has garnered a great deal of attention in cognitive science ever since. The SMH was featured prominently in Damasio's influential book *Descartes' Error: Emotion, Reason and the Human Brain* (Damasio [1994]), and has made its way into philosophical discussions of the nature of rationality and its relation to emotion (e.g. Blackburn [1998]; Churchland [1996]; DeLancey [2002]; Greenspan [2005]; Ledwig [2006]; Pizarro [2000]; Roskies [2003]; Thagard [2001]), as well as into sociology (Elster [1999]) and neuroeconomics (Bechara and Damasio [2005]).

In *Descartes' Error* Damasio argued that reason, rather than being opposed to emotion, instead requires it. This proposal was not altogether new in philosophy; notably de Sousa ([1987], p. 195) had already argued that emotion assists "pure reason" in the retrieval of relevant information, i.e. emotion is 'one of Nature's ways of dealing with the philosophers' frame problem'. Damasio ([1994]) referred in particular to studies from his laboratory suggesting that the capacity to make decisions is severely compromised in patients in which the brain mechanisms responsible for integrating reasoning and emotion are disrupted. The SMH was originally formulated to make sense of these findings, and to provide a model of how the brain organizes the interaction of emotion and decision-making. Specifically, "emotion" in this context refers to a set of changes in bodily arousal or activation—or, at least, to the neural representations of such changes. Damasio calls the latter "as-if body loops", and describes them as brain mechanisms that can misrepresent the state and activity of the body, namely trick the brain into believing that the body is in a specific state of arousal, when in fact it is not (Damasio [1994], p. 184; see also Damasio [1999], [2003]). "Somatic markers" are thus emotional bodily (or "as-if-bodily" brain) processes that, according to Damasio, interact with our ability to reason and generate options, and are necessary for making decisions.[1]

In the past few years, however, several laboratories have questioned the empirical work that Damasio and his group have done to validate the SMH, and have proposed alternative, more parsimonious interpretations of their data. Several scientists now believe that the SMH needs more empirical support (for a comprehensive review of the experimental literature related to the SMH, see Dunn *et al*. [2006]).

In this paper I will argue that the issue between Damasio and his critics is not merely an empirical one, to be settled by further experiments. There are also conceptual issues that need to be clarified. Specifically I will show that, since its inception, the SMH has not been clearly formulated. I will argue that what is usually referred to as "the SMH" amounts in fact to at least *two* distinct and logically independent hypotheses. One hypothesis (which I shall call *SMH-G*) is rather general, and claims that somatic markers are embodied preferences necessary for choosing among options. The other hypothesis (*SMH-S*) attributes a more specific role to somatic markers; it states that somatic markers are embodied preferences needed to be able to consider the *long-term* outcomes of available options. These two hypotheses, as we will see, make different predictions; in order to formulate adequate experimental tests it is thus crucial to distinguish between them.

Conceptual issues arise also in relation to the interpretation of the experimental results allegedly supporting the SMH. I will argue that neither *SMH-S* nor *SMH-G* are adequately supported empirically, albeit for different reasons. In particular, the Iowa Gambling Task (Bechara *et al*. [1994]), used by Damasio and his group to support *SMH-S*, turns out not to be a good test for it; also, it does not provide evidence for *SMH-G* either. As for *SMH-G*, it is hard to see how it could be disproved in the laboratory, given the very general definition of somatic markers it relies on.

These problems do not imply that the question of whether and how somatic-emotional processes are involved in decision-making is not interesting and not worth pursuing. The take-away message of this paper is that more still needs to be done, conceptually and empirically, in order to address that question properly. Specifically, more finely grained distinctions need to be drawn to design adequate experiments that could help make some progress in this interesting research area.

2 Two hypotheses: Somatic markers as embodied preferences, and as a source of farsightedness

The formulation of the SMH was preceded by the observation and study of the neuropsychological patient known as EVR ("Elliot" in Damasio [1994]). As Eslinger and Damasio ([1985]) tell us, at the age of 35 EVR was diagnosed with a brain tumour and underwent bilateral surgical excision of ventromedial prefrontal cortices (VMPFC). Before the operation, EVR was leading a successful life and was described by his relatives as an

excellent student, a "role model" and a "natural leader". At 25 he was employed as a staff-accountant in a home-building firm, and at 32 he was promoted chief accountant and became comptroller; he married at 25 and at the time of the operation he had two children. The operation is described as uneventful, and EVR was discharged two weeks later. He returned to work after a three-month recovery period, and soon became involved in a business partnership with a man who had been fired earlier from the company, apparently because of his questionable reputation. Despite warnings from relatives, EVR invested all his money in this partnership, and eventually declared bankruptcy. He had shorter jobs after that, and was fired from each one because of disorganization and tardiness. His wife left with the children after seventeen years of marriage, and just one month after the divorce EVR remarried (against the advice of his relatives), and divorced again two years later.

Two years after the operation and before being referred to Damasio's unit, EVR was re-evaluated and no tumour was detected. He scored well and above average in IQ, memory and personality tests. Yet his employment problems continued, and we are told that:

> [h]e needed about 2 hours to get ready for work in the morning, and some days were consumed entirely by shaving and hair-washing. Deciding where to dine might take hours, as he discussed each restaurant's seating plan, particulars of menu, atmosphere, and management. […] Purchasing small items required in-depth consideration of brands, prices, and the best method of purchase. He clung to outdated and useless possessions, refusing to part with dead houseplants, old phone books, six broken fans, five broken television sets, three bags of empty orange juice concentrate cans, 15 cigarette lighters, and countless stacks of old newspapers. (Eslinger and Damasio [1985], p. 1732)

Once in Damasio's lab, EVR performed a series of psychometric tests, neurological and personality evaluations; his performance in all of them resulted normal, or well above average. During interviews, EVR showed superior knowledge of complex economical and financial matters; in addition, when presented with problems requiring social and moral judgments, EVR's replies were reasonable and analogous to those of healthy subjects (see also Saver and Damasio [1991]).

Note that this characterization presents EVR's impairment in dual terms. On the one hand, EVR is portrayed as generally not spontaneously motivated for action: 'he rarely acted

on impulse, spending instead an inordinate amount of time reviewing detailed and not necessarily pertinent aspects of a proposition without keeping the whole problem in perspective' (Eslinger and Damasio [1985], p. 1739). EVR has difficulties making and implementing decisions, and tends to procrastinate. When he acts, we are told, it is mainly in reaction to the advice of friends and relatives, and under strong environmental pressure. On this account, in short, EVR is indecisive and inactive: when faced with options, he becomes lost in thought, wavers continuously between possibilities, and cannot implement any decision.

On the other hand, EVR is also described as making decisions, for example as taking actions against his relatives' and friends' advice. In these cases he does not procrastinate, and he appears motivated; he certainly *does* things, yet things that turn out to have infelicitous consequences for himself in the long term. On this second account, EVR is then a bad decision-maker not because he does not make up his mind and does not take action; quite the opposite, because he acts impulsively, namely without consideration for the long-term consequences of his decisions.

The SMH was originally advanced to make sense of EVR's impairment, and is in fact characterized by the same duality. In the paper that first formulated the hypothesis, Damasio *et al*. ([1990], p. 83) proposed that EVR's problem is that he 'cannot choose an advantageous course of action', and speculated that this incapacity is due to a

> failure to activate the specific *somatic states*, both visceral and skeletal, that were part of the previous learning of a given social situation. […] During learning, somatic states, positive or negative, […] appose a *marker* to entities and events. When somatic states are reactivated, we believe that they also […] mark the appropriately recalled set. They mark unambiguously not only the value of current perceptions, but most importantly, the value of *certain outcomes* to given courses of action […] Somatic states provide an automated way for the brain to select […] among response options. (*ibid*., emphasis in original)

On the one hand, this passage characterizes somatic markers as bodily states that indicate ("mark") the value of possible options. Somatic markers develop through experience as various bodily states become associated with positive and negative events. When one contemplates possible options before making a decision, somatic signals are re-enacted,

which "mark" such options as either positive or negative and thus allow one to choose. Somatic markers thus appear to be "embodied preferences", that is, bodily signals that carry value and that allow the reasoning system to weigh options and ultimately make a decision.

On the other hand, the text quoted above also mentions 'certain outcomes' and continues, '[b]y forcing attention on a conflict, a pertinent somatic marker would signal the *ultimately deleterious consequences* that might arise from a response that might nonetheless bring immediate reward' (Damasio *et al*. [1990], p. 83, my emphasis). Damasio *et al*. ([1991], p. 220) similarly claimed that somatic signals mark the "future"—in the sense of the "ultimate" or "long-term"—consequences of a decision: 'normal individuals can be assisted in the complex decision-making process by *the appearance of a somatic signal that marks the ultimate consequences of the response option*' (emphasis in original); a certain response option, 'regardless of its predictable immediate reward, can evoke a future scenario that is potentially threatening to the individual, and is marked by a negative somatic state' (*ibid.*). On this account, somatic markers provide *farsightedness*, namely they induce the decision-maker to consider future, long-term or "ultimate" consequences of available options instead of focussing exclusively on immediate or short-term consequences, and they tell her which of them is preferable.[2]

This dual specification of the function of somatic markers corresponds to the previously noted dual characterization of EVR as indecisive and inactive, and yet also as impulsive, and pervades subsequent works on the SMH. Damasio ([1994]), for example, often describes EVR as unable to choose among options, as cool-blooded, and with a "flat" decision-making landscape (*ibid*., pp. 50-1); also, EVR formulates plans but does not implement them (p. 55). In line with this description, Damasio ([1994)] draws several analogies between EVR and other patients who are unable to make decisions in the personal and social domain, who show no willingness to cooperate with the therapist and no motivation to get better (see the description of anosognosics on p. 67); he also notes similarities between EVR and patients who manifest a pervasive impairment of drives (p. 73). These and other passages (pp. 193-4) are all consistent with the account of EVR as inactive. In developing this characterization, Damasio ([1994]) also criticizes "models of rational decision-making" which, on his view, assume that decisions are made merely on the basis of logical reasoning. According to him, engaging in complex calculations of costs and benefits when faced with several options is not a good strategy to make decisions:

> if this strategy is the *only* one you have available, rationality […] is not going to work. At best, your decision will take an inordinately long time, far more than acceptable if you are to get anything else done that day. At worst, you may not even end up with a decision at all because you will get lost in the byways of your calculation. (Damasio [1994], p. 172, emphasis in original)

Damasio's proposal, in sum, appears here to be that somatic markers assist logical reasoning by embodying preferences; somatic markers are needed to avoid becoming lost in thought and consequently incapable of taking action.[3]

In other contexts however Damasio ([1994]) gives a characterization of the function of somatic markers that is consistent with the account of EVR as impulsive rather than indecisive and inactive. According to this second characterization, somatic markers are necessary 'to learn to be guided by future prospects rather than by immediate outcomes' (Damasio [1994], p. 218). Absence of somatic markers accordingly induces the inability to 'generate automated predictions of the significance of a future outcome' (*ibid*., p. 219).

The same duality reappears in more recent formulations of the SMH:

> In the absence of somatic markers, response options and outcomes become more or less equalized. Subjects may then resort to a strategy of deciding on an option based on extremely slow and laborious logic operations over many potential alternatives, themselves lacking somatic markers, and hence fail to be timely, accurate, and propitious. Another possible consequence of missing somatic markers is that decision making may become random or impulsive. (Tranel *et al*. [2000], p. 1049)

Bechara and Damasio ([2005], p. 339) write that the lack of somatic markers due to VMPFC damage 'degrades the speed of deliberation (e.g. choosing between two brands of cereal may take a patient a very long time because of endless reasoned analyses of the pros and cons of each brand), *and also* degrades the adequacy of the choice, i.e. patients may choose disadvantageously' (my emphasis). In a recent review, Dunn *et al*. ([2006], p. 242) say that, according to the SMH, lack of somatic markers leads to 'a marked decision-making impairment characterised by *either* extreme procrastination *or* the selection of inappropriate response options' (my emphasis).

To sum up, there appear to be at least *two* somatic marker hypotheses, which are logically independent and which make different predictions:

(1) The first hypothesis is rather general and I shall thus indicate it with "*SMH-G*". This hypothesis claims that somatic markers embody preferences which allow decision-makers to choose among options. It predicts that lack of somatic markers will induce inability to choose, procrastination and inactivity; the decision-maker does not have preferences, is caught in endless reasoning, and cannot make any decision.

(2) The second hypothesis attributes a more specific role to somatic markers, and I shall call it "*SMH-S*". *SMH-S* states that somatic markers provide *farsightedness* i.e. they are necessary to consider and evaluate the long-term consequences of an option. Unlike *SMH-G*, this hypothesis predicts that lack of somatic markers will induce impulsive decisions, namely decisions driven only by a concern for immediate or short-term outcomes; the decision-maker cannot value the long-term consequences of his choices and makes decisions that provide only short-term advantages.[4]

3 Lack of evidence for somatic farsightedness

What evidence is there in support of either hypothesis? Consider first *SMH-S*. The empirical evidence that Damasio and colleagues have relied most heavily on to support the SMH refers to the results of what is now known as the Iowa Gambling Task (IGT). This task was first proposed by Bechara *et al*. ([1994], p. 8) in the attempt to simulate 'in real time, personal real-life decision-making relative to the way it factors uncertainty of premises and outcomes, as well as reward and punishment'.

At the beginning of the IGT, the subject is in front of four decks of cards and is given a $2000 loan of play money. The subject is told that the experiment involves selecting one card at a time from any deck, for an indefinite amount of time until told to stop. The subject is also told that, after every card turn, she will win some money; sometimes, however, she will also lose some money. The amount of the reward (or of the reward and of the penalty), will be announced after each card turn. Finally, the subject is told that the goal of the task is to maximize profit on the initial loan of money.

Unbeknownst to the subject, the schedule of rewards and punishments is pre-programmed as follows (see table 1): turning a card from the first two decks (A and B) always yields $100, whereas turning a card from the other two decks (C and D) always yields $50.

All decks, however, also yield occasional losses; specifically, 10 card selections from either A or B yield a total loss of $1250, and 10 selections from either C or D yield a total loss of $250. In sum, turning 10 cards from either A or B produces a net loss of $250, whereas turning 10 cards from either C or D produces a net gain of $250; A and B are therefore called "bad decks", and C and D "good decks". Note that the distribution of penalties varies from deck to deck. In A, the $1250 loss that occurs every 10 cards is distributed over 5 cards (penalties range from $150 to $350); in B, it occurs all at once with one card turn. Similarly, in C the $250 loss that occurs every 10 cards is distributed over 5 (with penalties ranging from $25 to $75), whereas in D it occurs all at once.

table 1 about here

Bechara *et al*. ([1994]) showed that, in this task, patients with damage to VMPFC (like EVR) behave differently from healthy subjects. The latter initially sampled all decks and showed a preference for A and B in the early trials; as the experiment continued, however, they came to prefer C and D (on average between the 20th and 40th card selection) and kept selecting more cards from these decks until the end of the experiment (namely, after 100 card selections). As a result, they ended the task with a net gain. VMPFC subjects behaved like healthy subjects in the early trials, but then failed to develop a preference for C and D. Overall, they picked more cards from A and B and thus ended the task with a net loss.

In a later study, Bechara *et al*. ([1996]) also measured the somatic markers of IGT participants by measuring skin conductance responses[5] (SCRs). Three types of response were distinguished: (1) *reward* SCRs, generated after a card selection that yields a gain; (2) *punishment* SCRs, generated after a card selection that yields a loss; (3) *anticipatory* SCRs (***a***SCRs henceforth) generated prior to any card selection (i.e. during the time lapse between completion of the money exchange and the next card selection). Results showed that VMPFC subjects generated SCRs only *in response* to card selection, whereas controls (healthy and other brain damaged subjects) also generated ***a***SCRs. In particular, they generated stronger ***a***SCRs to the bad decks than the good ones.

Bechara *et al*. ([1994]) interpreted their results as showing that VMPFC subjects have a "myopia for the future"—in other words, as I shall put it here, that they are *shortsighted*. On this interpretation, VMPFC subjects prefer A and B throughout the task because they are interested only in the high *immediate* rewards of these two decks (compared to the lower

immediate rewards of C and D), and are blind to the future or *long-term* disadvantageous consequences of this strategy.[6] In line with this interpretation, Bechara *et al*. ([1996], pp. 215-6, p. 223) suggested that VMPFC subjects' failure to generate ***a***SCRs is a failure to consider the future or *long-term* outcomes of their card selections. In their view, VMPFC subjects choose A and B because they lack somatic markers informing them of the long-term disadvantageous character of these decks.

Yet importantly the interpretation of VMPFC subjects as shortsighted is unwarranted, because maximizing profit on the IGT *does not require farsightedness*. To see this, it is useful to imagine how a subject who was interested only in maximizing expected gain *on the next card* would behave. Such a subject, using past rewards and penalties as a guide to what to expect on the next card, might very well favour A and B at first, and, once A and B start to yield more losses than gains, reverse this preference, exactly as normal subjects do (as tables 2 and 3 show, in the original IGT the "bad decks" are objectively *advantageous* in the early trials; deck B, in particular, yields the first loss only when subjects hit card 9). There is no reason to think that a subject concerned with maximizing long-term gain, and a subject concerned with maximizing expected gain on the next card only, would behave differently.

Tables 2 and 3 about here

In other words, the IGT is not a task that allows discriminating between the decisions made by shortsighted subjects who are only concerned with maximizing expected benefit from the next card, and the decisions of farsighted subjects striving to maximize long-term benefit. The upshot is that to interpret the behaviour of VMPFC subjects in the IGT as due to shortsightedness is unwarranted. It might be that VMPFC are shortsighted, but the IGT is not a test that can establish this. As a consequence, the IGT cannot provide evidence in support of *SMH-S*. This hypothesis predicts that lack of somatic markers should induce indifference to long-term outcomes and impulsivity, but the IGT cannot reveal whether decisions are made on the basis of considerations of long-term or immediate outcomes. The IGT is thus not a good task to test *SMH-S.*

The idea that the IGT requires farsightedness has been endorsed for quite some time by both critics and supporters of the SMH. An exception is Maia and McClelland ([2004]), who have shown that healthy IGT participants can identify the good decks without considering the long-term outcomes of their decisions. Maia and McClelland proposed a

variant of the original IGT where subjects were interrupted every 10 card selections starting from the 20th, and were presented with a detailed questionnaire aimed at assessing their conscious knowledge of the reward/punishment schedule of each deck. Results showed that from card selection 20 already, healthy IGT participants can calculate the outcomes obtained from a given deck and accordingly accurately estimate the outcome of the next card selection. As Maia and McClelland ([2004], p. 16080) rightly remarked, '[t]here is no sense in which a selection done at a certain point in time is associated with a loss that only becomes apparent later in the game'. It is intrinsic to the structure of the IGT that participants need not take into consideration delayed outcomes to maximize profit.[7]

In addition, alternative explanations of the role of somatic responses generated during the IGT by healthy subjects are now available, which do not appeal to farsightedness. Tomb *et al*. ([2002]) have pointed out that, in the original task, the magnitude of the gains and losses that follow selecting any card from the bad decks (A and B) is much higher than the magnitude of the gains and losses that follow selecting any card from the good decks (C and D). Assuming that the magnitude of SCRs produced in response to gains and losses is directly proportional to the amount of money involved (for example, a gain or loss of $300 yields stronger SCRs than a gain or loss of $50), Tomb *et al*. explained the development of stronger ***a***SCRs to the bad decks as merely reflecting the participants' covert expectation of the *immediate* outcome of those decks. Simply put, on this account ***a***SCRs are higher before card selections from the bad decks because participants expect a higher-magnitude outcome, and not because ***a***SCRs somehow mediate considerations of long-term outcomes.

To test this interpretation, Tomb *et al*. designed a variant of the original IGT in which decks A and B are still disadvantageous yet associated with lower amounts of money—both for rewards and penalties—than the advantageous decks C and D (see table 4). Every 10 cards, the good decks yield a gain of $2250 and a loss of $1500, and the bad decks yield a gain of $250 and a loss of $1000. In this task, normal subjects generated higher ***a***SCRs to the advantageous decks, consistent with the hypothesis that ***a***SCRs only reflect the expectation of the *immediate* outcome of the deck that is reached for at any given trial.[8]

Table 4 about here

The upshot is that another experiment is needed to test *SMH-S* appropriately—an experiment that can unambiguously show when decisions are made on the basis of

consideration of immediate outcomes only, and when they are made by considering long-term consequences.

Looking at work on delay discounting could be useful here. Delay discounting tasks show that, under specific conditions, subjects choose options with smaller yet immediate outcomes, as opposed to bigger yet delayed ones (see e.g. Bickel *et al*. [1999]). One could try to use similar experimental designs to discriminate decisions that are made considering the long-term future, from decisions that are made on the basis of short-term outcomes only. Evidence supporting *SMH-S* would then consist for example in finding out that, in healthy subjects, ***a***SCRs precede farsighted decisions but not shortsighted ones.

Relevant results could also come from comparing autonomic activity in characteristically "shortsighted patients" (such as drug abusers) and healthy subjects sensitive to long-term outcomes. It would be interesting also to track other measures of autonomic activity in participants, i.e. not only SCRs but also heart rate, blood pressure, pupil dilation, etc., and see whether shortsighted and farsighted decisions are preceded by different bodily signals respectively (see also the discussion below on how to measure somatic markers).

4 Does making decisions require somatic markers, and can it be shown in the laboratory?

SMH-S (the hypothesis that somatic responses mark long-term outcomes) thus lacks empirical evidence. What about *SMH-G*—the hypothesis that somatic responses are embodied preferences needed to make decisions?

Note first that the behaviour of VMPFC subjects during the IGT does not lend support to the consideration that they lack preferences and are inactive.[9] While engaged in the task, these subjects do appear to have preferences and to act on them by reliably choosing the "bad decks". Lack of ***a***SCRs thus does not seem to induce a total lack of preferences. It could perhaps be replied that punishment and reward SCRs are the somatic signals that implement VMPFC subjects' preferences (recall that only ***a***SCRs are impaired in these patients). It would still remain unexplained, however, how exactly somatic responses elicited *after* selecting a card causally contribute to the patients' decisions (they would seem to be mere effects of losing or winning money). Whatever their function (if any), they do not appear to count as "markers" in the sense of somatic signals that covertly anticipate decisions, and it is not clear how they can influence subsequent choices.

Moreover, Bechara *et al*. ([1999]) themselves have shown that patients with bilateral damage to the amygdala, who lack not only ***a***SCRs but also punishment and reward SCRs, behave similarly to VMPFC subjects on the IGT, namely they choose more cards from the "bad decks". In sum, performance of both VMPCF and amygdala patients during the IGT does not seem to provide evidence for *SMH-G*; if anything, it suggests that somatic markers are not necessary to choose among options and to implement a decision.

In addition, some of the studies that looked at the decision-making capacities of subjects with various impairments of bodily feedback appear to provide evidence against *SMH-G*, or at least against the claim that activation of the non-neural body is necessary for making decisions. In particular Heims *et al*. ([2004]) showed that patients with pure autonomic failure[10] perform even better than healthy subjects on the IGT; and North and O'Carroll ([2001]) showed that patients with spinal cord lesions perform like healthy subjects on the IGT (see Dunn *et al*. [2006] for a comprehensive review).

What recurrently emerges from this experimental literature however is that it is hard to disprove *SMH-G* in the laboratory, because somatic markers are *very broadly* characterized. Damasio ([1994], p. 206) for example writes: 'the idea of the somatic marker encompasses an integral change of body state, which includes modifications in both the viscera and the musculoskeletal system, induced by both neural signals and chemical signals'. In addition, as we saw, Damasio counts as "somatic markers" also those brain mechanisms (the "as-if body loops") that represent the state and activity of the body, and that can also misrepresent the latter by being activated when the body is not (e.g. Damasio [1994], p. 184). This characterization implies that there are many ways to detect somatic markers. Changes in processes controlled by the autonomic nervous system, such as skin conductance, but also heart rate, blood pressure, pupillary size and endocrine gland activity (to mention only some) would all be indicative of somatic markers; to which we ought to add changes in processes independent of autonomic nervous control, such as eyeblink, muscle activity, tension and joint position; and, finally, neural representations of those bodily changes, which can also be misrepresentations.

It follows from this broad characterization of somatic markers that if a subject turns out to be able to make decisions despite abnormalities in some measures of somatic markers, it is still possible that some other, undetected source of somatic markers might implement the subjects' preferences. The large and vaguely specified number of such sources makes it virtually impossible to disprove *SMH-G*. In fact, significantly, both Heims *et al*. ([2004], p.

1986) and North and O'Carroll ([2001], p. 523) remarked that their results do not allow drawing definitive conclusions about the role of somatic markers in decision-making, because sources of bodily feedback *other than* those impaired could have guided appropriate performance on the IGT;[11] also, as-if body loops could have been in place to support appropriate behaviour.

It seems then that a more specific definition of somatic markers is needed for *SMH-G* to become a more precise and testable hypothesis about the place of bodily processes in decision-making.

In particular, it would seem to be important to distinguish various dimensions of bodily and as-if bodily arousal, and to understand which ones, if any, are implicated in different aspects of decision-making. Research in the physiology of emotion now employs several measures of autonomic arousal, i.e. SCRs but also blood pressure, heart rate, skin temperature, etc. (see e.g. Christie and Friedman [2004]), and brain imaging techniques could also be used to measure activity in "as-if body" loops during various decision-making tasks. One could then identify various possible sources of "somatic markers" and, for each of them, try to understand whether they are in fact necessary for making decisions as well as good decisions.

More precisely, one could try to understand which dimensions of arousal (bodily and as-if), if any, are necessary for specific aspects of decision-making. Making decisions in fact requires not only having preferences, but also generating options, keeping those options in mind, weighing possible outcomes, being motivated to choose one option among several ones, and eventually implementing the decision i.e. carrying out real-world actions. Damasio does not clearly pull these different aspects apart, and what I have identified as "*SMH-G*" remains ambiguous as to how somatic markers allow decision-makers to choose among options. Do somatic markers for example specifically embody preferences so that the decision-maker can retrieve what is relevant and what is not, or do somatic markers also (or perhaps only) provide the drive to carry out the decision and realize those preferences?

It would be helpful here to specify the nature of the decision-making impairment of VMPFC subjects in more detail. On the one hand, as mentioned, Damasio sometimes claims that VMPFC subjects are able to formulate reasonable plans and to explain what ought to be done in various circumstances; and, as we saw, these subjects are not completely indifferent. What exactly then is the nature of their indecisiveness and inactivity? Perhaps they lack the drive to implement their preferences, rather than preferences altogether. Dunn *et al*. ([2006],

pp. 257-8) indeed discuss this possibility and conclude that "apathy" or "lack of motivation" requires further attention and could largely explain the nature of decision-making impairments in VMPFC subjects. Perhaps also a distinction could be introduced here between the capacity to formulate third- and first-person "oughts". Whereas VMPFC subjects might be able to formulate reasonable plans about what ought to be done—their learning capacities are intact—,they may lack personal engagement and drives that would allow them to apply those oughts to themselves (for some potentially relevant recent work here, see Koenigs *et al*. [2007]). Finally, it might be useful to distinguish between various degrees of "inactivity"; some depressive subjects, for example, are totally unmotivated, and they differ from other depressive subjects who attempt or even commit suicide. In other words, motivation appears to come in degrees, and it may be interesting to investigate whether such degrees correlate with different levels of arousal (bodily and as-if); and, if so, whether different dimensions of arousal are somehow implicated in different degrees of motivation.

5 Conclusion

Damasio and colleagues' work has generated much interest and enthusiasm in those who want to emphasize that practical deliberation is implemented by a system in which reasoning and somatic-emotional activity are integrated and mutually supportive. However, this enthusiasm seems to be premature; more needs to be done to understand the role of arousal (bodily as well as "as-if" bodily) in decision-making. As I have emphasized, the issues to be addressed are not only experimental, but also conceptual. One can be "a bad decision-maker" in various ways—for example in the sense that one always procrastinates (i.e. fails to make decisions), or in the sense that one fails to consider the long-term outcomes of the options available (i.e. makes impulsive decisions). These senses must be clearly distinguished in order to formulate unambiguous predictions and to design appropriate experiments that can test them. As discussed, formulations of the "somatic marker hypothesis" have failed clearly to distinguish between these two senses, and have conflated what I called *SMH-S* and *SMH-G*.

In addition and more specifically, I have shown that there are logical-conceptual problems in how Damasio and colleagues have tried to provide support to their hypotheses. First, their main source of evidence, the IGT, cannot unambiguously show whether participants make their choices by considering long-term outcomes, and therefore it is not a good test for the hypothesis *SMH-S* according to which somatic markers are specifically

required to make wise farsighted decisions. As such, the IGT should not be used to seek evidence for *SMH-S*; another experiment is needed that can unambiguously distinguish between shortsighted and farsighted decisions.

A further conceptual difficulty that undermines attempts to track the role of the body in decision-making depends on the very broad characterization of "somatic markers", which turn out to include all changes under the control of the autonomic and somatic nervous system, as well as neural representations thereof. Distinguishing various dimensions of neural and bodily arousal, and tracking their respective roles in various aspects of decision-making, would seem to be a step forward towards a more specific and more empirically tractable hypothesis.

As we saw, there are plenty of other questions that still need to be addressed, to which we can add the following ones: What counts as an advantageous decision in real life? Is it possible to reproduce the features of real-life decision-making in the laboratory? Is it possible reliably to track different dimensions of bodily arousal during it? More also needs to be done to understand the nature of the deficit of VMPFC patients, and its alleged relation to impaired SCRs. Finally, there is the question of whether considering the body a vehicle of cognition provides explanatory advantages. The current trend toward an interdisciplinary approach to decision-making has the potential to throw light on some of these issues. Hopefully, the debate on the role of the body in decision-making will continue, with new experimental designs that can reproduce the structure of real-life decision-making, and with increased attention to definitional and conceptual issues.

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Endnotes

[1] Damasio sometimes mentions that somatic markers can also be "feelings" (see e.g. the discussion in Damasio [1994], pp. 184-7) and in more recent work he extensively discusses the relationship between bodily states, emotions and feelings (Damasio [1999], [2003]). These issues relate to his broader account of emotion and consciousness, and I shall not address them here. The focus of this paper is specifically the SMH as a neuroscientific hypothesis as developed and debated in the experimental literature. The experiments I am concerned with, as we will see, take somatic markers to be bodily signals that *covertly* guide decision-making, and do not examine feelings.

[2] Damasio and colleagues use the expressions "long-term", "future" and "ultimate" outcomes interchangeably, as well as "immediate" and "short-term" outcomes, and I shall do the same here. In real life, the boundary between the immediate, short- and long-term consequences of one's decisions is of course fuzzy, and it is not clear that there are "ultimate" consequences of any decision. As we will see, the meaning of all these terms is clearest in the context of the Iowa Gambling Task. The contrast between short- and farsighted decisions still makes sense however also in real-life contexts—compare for example the decision to buy cheap fancy yet uncomfortable shoes, versus investing money in an expensive yet durable and comfortable pair.

[3] This formulation leaves it ambiguous whether somatic markers provide preferences, or specifically the drive to implement those preferences into real actions. I shall come back to this issue at the end of section 4.

[4] It is worth pointing out that additional versions of the SMH can be found that have subsequently dropped out from the debate without discussion. The existence of these other versions is further evidence of the difficulty that Damasio and collaborators had in coming up with one clear hypothesis. Damasio *et al*. ([1990]) for example suggested that somatic markers signal a conflict between the value of the immediate and the delayed outcome of a given option; and Damasio *et al*. ([1990], [1991]) claimed that somatic markers allow conscious and unconscious inhibition of one's response to immediate punishment, and that they thus help resolve the perceived conflict between the value of short-term and long-term consequences. Damasio ([1994]) also often maintains that somatic markers constrain the space of options before logical reasoning is applied.

[5] Changes in skin conductance are detected via electrodes placed on two fingers and connected to a polygraph; when electrical current is passed between the electrodes and the skin's sweat glands secrete fluid, conductivity is increased. Measuring SCRs thus means measuring the change in the amount of current conducted by the skin between the electrodes. Because sweat gland activity is controlled by sympathetic nerve activity, measures of SCRs are traditionally used to monitor the autonomic nervous system.

[6] See the following claims in (Bechara *et al*. [1994]): 'we find that prefrontal patients, unlike controls, are oblivious to the future consequences of their actions, and seem to be guided by immediate prospects only' (p. 7); '[t]he subjects must rely on their ability to develop an estimate of which decks are risky and which are profitable in the long run' (p. 13); 'these subjects are unresponsive to future consequences, whatever they are, and are thus more controlled by immediate prospects' (p. 14).

[7] In a later work, Bechara and colleagues presented a variant of the IGT in which, they claim, they 'reversed the order of reward and punishment, i.e. the punishment became immediate and the reward became delayed' (Bechara *et al*. [2000], p. 2190). Again, this characterization is misleading. In this variant, the advantageous decks always yield a loss of $100, and occasional gains of $150, $200, $250, $300, $350, or $1250; the disadvantageous decks always yield a loss of $50, and occasional gains of $25, $50, $75, or $250 (see table in Bechara *et al*. [2000], p. 2193, for detailed distribution). There is no sense in which a specific card selection is associated with a gain that becomes apparent only later in the game. The "overall gain (or loss)" (namely the advantageous or disadvantageous character of each deck) is the sum of the gains and losses acquired with each card, and is apparent only after the relevant selections have been made. For this reason, the variant proposed by Bechara *et al*. ([2000]) cannot be used to provide evidence for their claim that VMPFC subjects are insensitive to future consequences and are primarily guided by immediate prospects.

[8] Note that although Tomb *et al*. ([2002]) ruled out the idea that anticipatory somatic signals mark long-term outcomes, they still endorsed Bechara and colleagues' interpretation that the IGT requires farsightedness. They concluded in fact that 'across both experiments, *card selection is driven by long-term consequences*, whereas anticipatory SCRs are driven by the immediate act to be performed' (Tomb *et al*. [2002], p. 1104, my emphasis). Yet not only is card selection in the IGT not driven by long-term consequences; it does not seem that Tomb *et al*. can draw such a conclusion for their study either. In their variant task, selecting from the

good decks always yields a gain of $225, whereas selecting from the bad decks always yields a much lower gain of $25. Participants are thus likely to figure out very soon—perhaps they never have any doubt—that the good (bad, respectively) decks are actually advantageous (disadvantageous), without having to reverse their initial preferences; and, in particular, without considering any long-term consequence.

[9] See Bechara *et al*. ([1994], p. 14): 'these patients possess and can access the requisite knowledge to conjure up options of actions and scenarios of future outcomes […] Their defect seems to be at the level of acting on such knowledge'.

[10] Pure autonomic failure involves a peripheral degeneration of sympathetic and parasympathetic autonomic neurons, and results in an inability to modulate bodily state via the autonomic nervous system. Patients affected by pure autonomic failure do not generate skin responses.

[11] Note however that, taken together, the studies of Heims *et al*. ([2004]) and North and O'Carroll ([2001]) rule out many sources of bodily feedback as potential candidates for "somatic markers" causally involved in appropriate decision-making on the IGT.