

Two Myths About Somatic Markers*

Two Myths about Somatic Markers

Abstract

Research on patients with damage to ventromedial frontal cortices suggests a key role for emotions in practical decision making. This field of investigation is often associated with Antonio Damasio's Somatic Marker Hypothesis—a putative account of the mechanism by which autonomic tags guide decision making in typical individuals. Here we discuss two questionable assumptions—or 'myths'—surrounding the direction and interpretation of this research. First, it is often assumed that there is a single somatic marker hypothesis. As others have noted, however, Damasio's 'hypothesis' admits of multiple interpretations (Colombetti, [2008]; Dunn et al. [2006]). Our analysis builds upon this point by characterizing decision making as a multi-stage process and identifying the various potential roles for somatic markers. The second myth is that the available evidence suggests a role for somatic markers in the core stages of decision making, i.e. during the generation, deliberation or evaluation of candidate options. To the contrary, we suggest that somatic markers most likely have a peripheral role, in the recognition of decision points, or in the motivation of action. This conclusion is based on an examination of the past 25 years of research conducted by Damasio and colleagues, focusing in particular on some early experiments that have been largely neglected by the critical literature.

1. *Introduction*
2. *What is the somatic marker model?*
3. *Multiple somatic marker hypotheses*
 - 3.1 *Are somatic markers necessary for practical decision making?*
 - 3.2 *Speed, accuracy, or both?*
 - 3.3 *At which of the five stages of decision making are somatic markers engaged?*
4. *Anecdotal evidence suggests a peripheral role for somatic markers*

- 4.1 Chronic Indecisiveness*
- 4.2 Extreme impulsiveness*
- 4.3 Enhanced decision making in the lab*
- 4.4 Lack of motivation.*
- 5. *Early experiments suggest that VMF damage leaves core processes intact*
 - 5.1 The evocative images study*
 - 5.2 Five problem solving tasks*
- 6. *Recent experiments fail to discriminate among alternate versions of SMH*
- 7. *Conclusion*

1 Introduction

The Somatic Marker Hypothesis (SMH) is familiar to researchers working in a wide range of disciplines including psychology, neuroscience, economics, philosophy and AI. This hypothesis is usually taken to imply that practical decision making involves a neglected emotional component. This message has found its way into popular thinking about the relationship between emotion and reason, where the somatic marker concept has become something of a household item. We challenge two popular myths about SMH. First, contrary to conventional wisdom there is no single somatic marker hypothesis. Even within the writings of its chief proponent, Antonio Damasio, somatic markers are assigned a variety of different functions. Although other authors have critiqued SMH as vague and ambiguous (Colombetti [2008]; Dunn et al. [2006]; Evans [2002]), the range of alternative somatic marker hypotheses has not been adequately distinguished in the literature. To this end, we offer a framework for systematizing them. This framework identifies three logical dimensions along which versions of SMH vary. Since these dimensions cut across one another the space of logical possibilities is quite large. By our count, there are no fewer than 38 distinct somatic marker hypotheses. A second myth concerns the stages of practical decision

making at which somatic markers are most likely engaged. The received view is that the available evidence suggests a role for somatic markers in the ‘core stages’ of decision making, where a subject generates, evaluates and selects among alternative courses of action. We argue that Damasio’s own evidence suggests otherwise.

Somatic markers appear to be involved (if at all) in the ‘peripheral’ stages of decision making: one possibility is that they are engaged early-on, in notifying the subject that a decision-point has been reached; another possibility is that somatic markers are engaged at the terminal stage, in motivating the subject to execute a decision. These conclusions are based on our analysis of the past 25 years of Damasio and colleagues’ research—particularly their early work conducted prior to the adoption of the Iowa Gambling Task.

Recent years have seen the emergence of several philosophical critiques of SMH (Evans [2002]; Gerrans [2007]; and Colombetti [2008]). Most of these critiques focus on the failings of a specific experimental paradigm—the Iowa Gambling task (IGT)—for discriminating among alternative versions of SMH. We agree with these assessments and view our criticism as building on them in at least three respects. First, we identify and systematize the many different versions of SMH that have appeared since its inception. Although other critics note that SMH is vague and ambiguous, our analysis is the first to spell out in detail the many different versions of this ‘hypothesis’. Second, we examine the possible contribution somatic markers might make to each stage of decision making. When the stages of decision making are brought to bear on the analysis, the alleged contribution of somatic markers appears far less definite than even previous critics have supposed. Finally, much of our analysis focuses on Damasio’s early experiments that were conducted prior to the widespread adoption of the Iowa Gambling Task. To our knowledge these early

experiments have been overlooked within the critical literature. However, compared to the IGT, the experimental paradigm that eventually supplanted them, these experiments are arguably sharper instruments for testing whether somatic markers are involved in particular stages of practical decision making. Our analysis further reveals that the results of these early experiments, though inconclusive, suggest the *opposite* conclusion from the one that Damasio drew about the role of somatic markers in the core stages of decision making. It is therefore doubly puzzling that these experimental protocols have been abandoned and all but forgotten.

Our argument proceeds as follows. In section 2 we distinguish the somatic marker model from somatic marker hypotheses. The somatic marker *model* provides a general account of the functional association between autonomic tags—somatic markers—and mental representations. Somatic marker *hypotheses* implicate this model in some aspect of cognition. Section 3 identifies three dimensions along which alternative somatic marker hypotheses presented by Damasio and colleagues tend to vary. As mentioned, there are at least 38 of these. The remainder of this paper addresses the second myth that somatic markers are involved in core decision making processes according to the available evidence presented by Damasio and colleagues. Section 4 reviews the anecdotal evidence presented by Damasio and his coauthors describing the cognitive peculiarities of patients with damage to ventromedial frontal cortices. Section 5 reviews some early experimental work on these subjects, while section 6 reviews more recent experiments using the Iowa Gambling Task. We argue that all three sources of evidence suggest that these patients suffer from damage to peripheral rather than core processes.

2 What is the Somatic Marker Model?

It is important to note from the outset that somatic markers are potentially involved in other cognitive functions besides practical decision making, even though this is the context in which they are usually discussed. Other suggested roles for somatic markers range from theoretical reasoning to object recognition (Barrett and Bar [2009]; Thagard [2006]). To allow for such possibilities, we find it useful to distinguish the somatic marker model from somatic marker hypotheses. The somatic marker model, as it is outlined below, describes a putative neuro-cognitive mechanism for associating autonomic tags with mental representations. Somatic marker hypotheses, by contrast, invoke this model to explain some aspect of cognition, such as practical decision making. Our focus in this paper is on the family of somatic marker hypotheses introduced by Damasio and colleagues. The current section lays some of the necessary groundwork by outlining the somatic marker model in general terms. Since this model is not our target, most of the neurological details will be omitted from this presentation. Our modest goal in this section is to illustrate that the somatic marker model can be characterized independent of any claims about its role in decision making, or other forms of complex cognition. We take this model to consist of the following six features:

Associationism: Somatic markers are formed during ‘primary activation’ when some representation (e.g. a hypothetical or perceived state of affairs) becomes associated with an autonomic response. Once such an association is formed, reactivating a representation of the same type triggers the associated marker in a process called ‘secondary activation’.

Valence: Each somatic marker has either positive or negative valence.

Biasing: Co-activation of a representation and an associated marker biases the subject favourably towards the object of that representation (if the marker is positive), or against that object (if the marker is negative). These biasing effects can be either conscious or subconscious.

Brain regions: The somatic marker mechanism is implemented by dedicated neural hardware. Specifically, the amygdalae are considered necessary for primary activation of autonomic responses, and the ventromedial frontal cortices (VMF) are thought to be necessary for secondary association of markers with representations.

Physiology: Somatic markers are closely linked to certain autonomic nervous system changes, such that the activation of a marker can induce those changes and *vice versa*. Damasio sometimes describes these changes in terms of a ‘body-loop’ of somatic state activation.

‘As if’ activation: Sometimes, however, somatic information is processed without the accompanied physiological changes. The ‘as-if-body-loop’ involves somatic marker activation without the corresponding physiological change.

Two aspects of this model deserve special mention. First, an essential commitment of the model involves the role of the VMF in associating somatic markers with representations. This assumption is presupposed by much of the evidence offered in support of the somatic marker model, which draws heavily on VMF damaged patients. Second, despite the frequent suggestion that this model explains the influence of ‘emotion’ or ‘feeling’ on some aspect of cognition, this is not a core assumption of the model. It is a separate question whether somatic markers qualify as a type of emotion (cf. Prinz [2005]). With this general outline of the

somatic marker model in place, we now turn to the family of hypotheses implicating this mechanism in practical decision making.

3 Multiple Somatic Marker Hypotheses.

Throughout his writings, Damasio explains that SMH was originally formulated to account for the abnormal behaviour of a particular patient known initially as EVR and later referred to as Elliot. At age 35 Elliot underwent surgical removal of his ventromedial frontal cortices. Before the operation Elliot reportedly led a normal social life with a nuclear family and a successful accounting career. Following the operation Elliot developed ‘profoundly abnormal personality characteristics’ (Damasio et al. [1991]). He lost his job due to tardiness and disorganization. He lost his savings through an ill-conceived business partnership. His marriage soon ended. Although Elliot landed several other jobs, he was unable to hold any of them. The misfortunes suffered by Elliot are well documented and tragic (Eslinger and Damasio [1985]; Damasio et al. [1991]; Damasio [1994]). What remains less clear is the underlying cause of his personality disorder.

Damasio reports that Elliott performed normally or above average on a battery of psychometric tests including IQ, personality, memory and social comprehension (Eslinger and Damasio [1985]; Saver and Damasio [1991]). In conversation, also, ‘[h]is knowledge and comprehension of complex social issues, the economy, industry and financial matters were clearly above average’ (Eslinger and Damasio [1985], p. 1733). According to Damasio, the most salient feature of Elliot’s psychological profile was his flattened affect or ‘general lack of feeling’ (Damasio [1994]). It was this connection between Elliot’s absence of emotion, his VMF lesion, and his

apparent decision-making deficit that led Damasio to hypothesize a relationship between somatic markers and decision making (Damasio [1994]; Bechara and Damasio [2005]). Over the past 25 years this hypothesis has taken a variety of different forms.

Giovanna Colombetti ([2008]) identifies two distinct versions of SMH. The more general version claims that somatic markers encode preferences (in the form of embodied experiences) that allow decision makers to select among options. The more specific version states that somatic markers are necessary for seeing beyond the immediate consequences of an option in order to consider its long-term consequences. Colombetti argues that the available evidence fails to support the specific version of SMH, while the general version ‘is hard to disprove [...] in the laboratory, because somatic markers are *very broadly* characterized’ ([2008], p. 66, *original italics*). Our examination of the literature identifies a much larger range of somatic marker hypotheses. In particular, there are three issues on which alternate versions of SMH tend to equivocate. The first concerns the modality of this hypothesis: are somatic markers necessary for decision making, or, do they merely contribute to this process? The second question concerns the nature of their contribution: do they enhance the speed of decision making, the accuracy of this process, or perhaps both? The third question concerns the particular stage of decision making at which somatic markers are engaged. Recent literature on practical decision making subdivides this process into at least five stages. Our analysis categorizes these stages into ‘peripheral’ and ‘core’ processes, and asks what it would mean for somatic markers to be involved at each step. The remainder of this section elaborates on these three dimensions and concludes with a matrix of 38 alternative somatic marker hypotheses.

3.1 Are Somatic Markers Necessary for Practical Decision Making?

In his discussion of several VMF patients, Damasio hypothesizes that these individuals are incapable of making decisions because they lack somatic markers (see [1994], p. 193). Likewise, Bechara and Damasio assert that ‘[SMH] specifies a number of structures and operations required for the normal operation of decision making’ ([2005], p. 339). Such claims suggest that somatic markers are necessary for decision making. Yet, in other circumstances, Damasio suggests that somatic markers merely contribute to decision-making. For example, he describes VMF patients who make all sorts of decisions despite the alleged absence of somatic markers ([1994], ch. 3). Damasio further hypothesizes that, absent somatic markers, decision-makers resort to explicit cost-benefit analysis ([1996]). If this account of VMF patients is correct then somatic markers are not necessary for decision-making. Clearly, the modality assigned to SMH has important implications for the interpretation of experimental and anecdotal data.

3.2 Speed, Accuracy, or Both?

Advocates of the somatic marker model are often unclear about whether somatic markers enhance the speed of decision making, the accuracy of decision making, or both. In a ([1996]) paper, Damasio asserts that a failure in somatic marking will result in slower, more error-prone decisions (p. 1415). Similarly Bechara and Damasio write that failure in somatic marking ‘degrades the speed of deliberation [...] and also degrades the adequacy of the choice’ ([2005], p. 339). Elsewhere, discussion of speed is dropped while they assert that conscious knowledge *must* be supplemented with emotional input to guarantee advantageous decisions (Bechara et al., [2005]). This is a slight change from another article in which decision accuracy is also addressed, but

the modal force has changed: ‘knowledge and reasoning alone are *usually* not sufficient for making advantageous decisions’ (Bechara and Damasio [2005], p. 337, *our italics*). In some of the earliest accounts of SMH, however, Damasio insists that somatic markers are necessary for speedy decisions, but only *potentially* required for accurate ones ([1994], p. 172). But even this is at odds with many descriptions of VMF patients as ‘random and impulsive’ decision-makers (Damasio [1996], p. 1415), which suggest perhaps accuracy is sacrificed, but not speed.

What is missing here is a description of the relation between speed and accuracy in decision making, and how somatic markers affect this relationship. Speed and accuracy are not logically incompatible. However, it seems plausible that decision making will often involve a trade-off between the two. For example, hasty decisions are often more error prone, and rigorous deliberations may come at the expense of speed. The hypothesis that somatic markers enhance accuracy should therefore be distinguished from the hypothesis that they enhance speed, since these claims potentially have different empirical implications.

3.3 At Which of the Five Stages of Decision Making are Somatic Markers Engaged?

Psychological accounts of decision making often break this process down into component stages (e.g. Carroll and Johnson [1994]). This level of description is seldom utilized in the specification of neuropsychological theses, SMH included. Although they are not explicit about the different sub-processes involved in decision making, SMH theorists do assign somatic markers a variety of functional roles, some of which are best understood as distinct hypotheses about alternative stages of decision making. In what follows we identify five such stages in which somatic markers have been implicated.

The first stage is what we call *decision-point recognition*. This is the process by which an agent becomes alerted (either consciously or subconsciously) that a decision is called for. A simple example will illustrate the role that somatic markers might play at this initial stage. Imagine a child who is engrossed in a game of catch. Her ball escapes into the road and, without checking for traffic, she chases after it. Now suppose that her mother witnesses the event. It is likely that the girl would receive a stern scolding. On the somatic marker model, such a scolding would invoke an autonomic response and thereby ‘mark’ the daughter’s representation of the road, the ball, and other surrounding features. On subsequent occasions, the model predicts, this marker would become reactivated under similar conditions, serving as an alarm bell for diverting attention away from the game and onto the decision-processes associated with cautious road crossing.

In an early paper coauthored with Paul Eslinger ([1985]), Damasio briefly suggests that somatic markers might play such a role. These authors entertain several explanations for the discrepancy between Elliot’s laboratory and real world performance. They note that in the lab he performs well on hypothetical reasoning problems that are similar to the situations with which he struggles in real life. One explanation for this discrepancy is that, in real world scenarios, Elliot’s decision making machinery fails to become activated by the right sorts of perceptual cues:

In artificial problems, the premises are furnished verbally, ‘post analysis,’ within close temporal proximity; in real life, premises are often presented through different sensory modalities and at different times. They may not be integrated as a unit or not be appreciated in their configuration, and thus fail to evoke the cognitive programs necessary to solve the problem intelligently.

(Eslinger and Damasio [1985], p. 1737)

It is interesting that this hypothesis, which assigns somatic markers a role in the early stages of decision making, is abandoned in Damasio's later writings. In later sections we reconsider this hypothesis in light of the available evidence.

The second stage of decision making involves generating candidate options. For example, imagine arriving at a party where the friend who invited you is absent among a room full of strangers. At this point you might begin to consider some options: stand near the punch bowl, ask around for your friend, attempt to join a conversation, shrink into the background, etc. We refer to this process as *option generation*. Damasio occasionally suggests that somatic markers are necessary for this process. For instance, in *Descartes Error* he chastises the 'high reason' model of (emotionless) decision making because, 'nothing is ever heard about the mechanism by which a diverse repertoire of options is generated for selection' ([1994], p. 166). A few pages later he argues that option generation involves two other processes: basic attention and working memory. These processes, he claims, are influenced by somatic markers:

In the full somatic marker hypothesis, I propose that a somatic state, negative or positive, caused by the appearance of a given representation, operates not only as *a marker for the value of what is represented, but also as a booster for continued working memory and attention.* ([1994], p. 198, *original italics*)

If option generation is one of the functions performed by somatic markers, as Damasio suggests in this passage, then one might expect a detailed analysis of how damage to this mechanism might impact decision making. To the contrary, we are unaware of any further research aimed at probing this function. As we discuss in what follows, this version of SMH receives scant attention in later SMH writings.

The third stage of decision making that we call *deliberation* has been characterized in a variety of ways. Some authors identify a general category of ‘information search’ (Carroll and Johnson [1994]), which includes everything from scanning the immediate environment to drawing on memory and imagination as a means of gathering information pertaining to a decision. Others characterize deliberation more narrowly, as the anticipation of consequences associated with options (Evans [2002]). Deliberation, as we define it here, involves the identification of ‘factors’ (e.g. anticipated benefits, moral implications, or even the ease of attainment) associated with one or more options. It is important not to confuse this process with option generation—the two stages are logically distinct. For instance, in some decision-making scenarios the options are externally fixed (e.g. tea or coffee?) but the range of factors over which one might deliberate are open ended (which drink contains more caffeine? Which will go better with this meal? Which drink does this restaurant specialize in? etc.) It is an empirical question whether these processes are psychologically dissociable, but for the purposes of formulating hypotheses it is expedient to distinguish them.

There are two versions of the hypothesis that somatic markers are involved in deliberation. The first proposal states that somatic markers are involved in the identification of factors that are *relevant* to some decision. This hypothesis stems from the observation that deliberation can go wildly off in all directions. As Damasio notes,

The sheer amount of possible response options [i.e. factors] makes it likely that an assistance device is required to sort out the responses that are more likely to be relevant for the overall goals of the organism in both the short and long term. (Damasio et al. [1991], p. 221)

It is perhaps noteworthy that relevance is a fuzzy notion. Some factors appear clearly irrelevant to a decision. Suppose that an airline host offers you the lasagna or the beef. Clearly irrelevant considerations include whether one is sitting in an odd or even row, the number of km above sea level, or the server's horoscope. Other factors are potentially relevant. For example, does it matter which meal makes one appear more attractive? Should the dietary preferences of neighbouring passengers be considered? Often, the relevance of some factor to a given decision is determined by the agent's goals. Setting aside the issue of what determines relevance, the claim that somatic markers are involved in the identification of factors relevant to some decision shall be referred to as the 'Relevance Hypothesis' in what follows.

A second respect in which somatic markers might influence deliberation is by limiting the time or energy one invests in this process. This hypothesis stems from the observation that deliberation can go on indefinitely. Some of the anecdotes describing Elliot's indecision seem to illustrate this condition. For example, Eslinger and Damasio ([1985]) report that he would agonize over decisions about where to dine, spending hours deliberating over the seating plan, menu options and atmosphere. Notice that these factors are all relevant to the decision of where to dine, the problem was that there was no end in sight. The philosopher Dylan Evans ([2002]) dubs this 'Hamlet's problem'. Likewise, the proposal that emotions enable one to avoid such lengthy bouts of deliberation is referred to by Evans as the 'Search Hypothesis' of emotion. Although Damasio does not explicitly distinguish these two aspects of deliberation, relevance and termination are conceptually distinct. Failure in one of these tasks will result in different sorts of deficits from failure in the other. We therefore consider it important to distinguish the Relevance and Search hypotheses as alternative versions of SMH.

A fourth component of decision making involves value assignment and ranking. According to Damasio, when a given option or factor is represented this sometimes triggers a somatic marker which serves as an emotional ‘vote’ in favour or against it. We call this process *value assignment*. An important theoretical point is that value assignment, on its own, is not sufficient for evaluating a set of options. In order to arrive at a decision, one also requires some criterion for tabulating the votes associated with each option or factor. Damasio refers to this as the ‘ordering problem’ and his solution invokes somatic markers. He argues thusly:

(1) If order is to be created among available possibilities, then they must be ranked. (2) If they are to be ranked, then criteria are needed (values or preferences are equivalent terms). (3) Criteria are provided by somatic markers, which express, at any given time, the cumulative preference we have both received and acquired. ([1994], p. 199)

Note that Damasio here assigns to somatic markers both a role in the assignment of value to particular options and also a role in ranking. It is unclear whether he regards these as conceptually distinct steps in decision making. However, it seems clear that there are two distinct roles which somatic markers might play: one role involves the assignment of valence to individual options or factors, the second role involves the tabulation of those assignments. Therefore it is prudent to distinguish these as alternative hypotheses.

Following successful evaluation, the fifth and final stage of decision making is the *execution* of some action arising from the decision. Here, too, Eslinger and Damasio ([1985]) see a possible role for somatic markers. They hypothesize that an impaired individual might be capable of forming choices but lack the ability to execute those decisions, lacking somatic markers to assist in ‘translating plans into

action'. Dunn et al. outline a similar version of this deficit which they call 'apathy', in which, '[VMF] patients and other impaired groups may simply not care enough about negative outcomes to avoid them' ([2006], p. 257). These authors conclude that such a syndrome deserves more careful attention. This proposal also accords with the long tradition in philosophy and psychology that views emotions as necessary for inspiring action. We discuss the possibility that somatic markers are involved in decision execution in greater detail below.

Note that in distinguishing these five stages we do not mean to suggest that decision making is a serial process: presumably some of these processes occur in parallel. It should also be noted that the hypotheses are not mutually exclusive. Somatic markers might play different roles at various stages of decision making. However, differentiating among these alternatives is important for both clarifying SMH and interpreting the available evidence.

To summarise, the general hypothesis that somatic markers are involved in decision making leaves open many important questions. We identify three dimensions along which alternate versions of this hypothesis tend to vary: the modality of the hypothesis (necessary for decision making or not), the contribution somatic markers make at a given stage of decision making (speed, accuracy or both), and the five general stages of decision making, two of which can be further subdivided. All three dimensions cut across one another with the exception of the execution of a decisionⁱ. In total, this renders a matrix of no fewer than 38 alternate somatic marker hypotheses (see figure 1)ⁱⁱ.

In what follows we consider whether the available anecdotal and experimental evidence favours some of these alternative hypotheses over others. For the sake of simplicity we distinguish 'core' subprocesses (option generation, deliberation and

evaluation) from ‘peripheral’ subprocesses (decision-point recognition and execution). This distinction provides an easy way of organizing alternate somatic marker hypotheses.

(Place Figure 1 about here)

4 Anecdotal Evidence Suggests Peripheral Roles for Somatic Markers.

The somatic marker ‘hypothesis’ has been popularized by several books containing vivid anecdotal accounts of VMF patients. These anecdotes have influenced popular understanding of this impairment while suggesting specific roles for somatic markers in ‘normal’ individuals. Interestingly, however, the anecdotal evidence does not speak with a single voice. Some anecdotes describe behaviours that could plausibly stem from damage to either core or peripheral subprocesses. Others suggest damage to peripheral processes only. It is clear that Damasio favours interpretations that emphasize core processes: ambiguous anecdotes are frequently interpreted as evidence for damage to core processes and those suggesting damage specifically to peripheral processes are given less emphasis. In this section we distinguish four types of anecdote that have been identified as salient by Damasio and colleagues. The balance of this evidence, we argue, fails to support Damasio’s preferred versions of SMH.

4.1 Chronic Indecisiveness

In the most popular type of anecdote, VMF patients are described as becoming easily lost in an endless loop of indecision. The following example will sound familiar to Damasio’s readers.

He needed about two 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 each menu, atmosphere and management. He would drive to each restaurant to see how busy it was, but even then he could not finally decide which to choose. Purchasing small items required in-depth consideration of brands, prices, and the best method of purchase. (Eslinger and Damasio [1985], p. 1732)

From this type of anecdote Damasio infers that Elliot's impairment lies in the core stages of decision making. Sometimes the problem is thought to emerge at the termination stage of deliberation, or what Evans ([2002]) calls Hamlet's Problem. Elsewhere, Damasio suggests that Elliot's indecision results from a defect in evaluation (value assignment or ranking). However, it is easy to imagine how chronic indecisiveness might also arise from damage to peripheral processes.

Suppose that Elliot suffers from a defect in his decision-point recognition mechanism. One possible effect of damage to this mechanism is that decision-point recognition shuts down altogether. This outcome is inconsistent with chronic indecisiveness. However, there is another way in which damage to this mechanism might manifest itself. Another possibility is that this mechanism becomes hyper sensitive. Consider the analogy of a loose wire that has come slightly dislodged from its contact point. Sometimes the wire will fail to transmit current when it should; on other occasions it will transmit a spark inappropriately. Perhaps the problem with Elliott is that his VMF is like a loose wire. Sometimes he fails to recognize decision points when he should; other times there are *too many* cues that send him into decision making mode. Such a deficit potentially explains Elliot's chronic

indecisiveness. This hypothesis also explains why Elliot is afflicted with chronic indecisiveness in some situations but not others.

A second possibility is that Elliot suffers from a faulty option generator. Once again, the analogy to the loose wire is instructive. On occasions when his VMF fails to make a ‘connection’, Elliot is unable to identify alternative options as such. On other occasions when Elliot’s option generator misfires, this sends him into a hyperactive mode. In the latter scenario, Elliot’s deliberation and evaluation systems might become overwhelmed with input, such that he ends up deliberating about a much wider range of options than a normal individual would consider.

A third possibility is that Elliot suffers from a lack of motivation to follow through with a decision, and that this re-initiates the decision making process. On this interpretation, Elliot is able to decide on a restaurant in the sense that his deliberation process terminates and the options are adequately evaluated. However, when it comes to getting out of the car and going through the door, Elliot cannot stick to his guns. This leads him to try again at another restaurant where the motivation, hopefully, will be a little stronger.

Our point in raising these possibilities is that the most popular type of anecdote for conveying the effects of VMF damage—chronic indecisiveness—can potentially be explained in terms of deficits to any one of the five decision-making stages. This contrasts with the interpretation typically favoured by somatic marker theorists, in which chronic indecisiveness is taken to suggest damage to core processes.

4.2 Extreme Impulsiveness

Perhaps the second most common anecdote associated with VMF patients is that they are unable to delay gratification. When portrayed in these terms Elliot is described as impulsive, dismissive, and uncalculating. Supposedly this renders him unable to resist actions with short-term appeal but disastrous and foreseeable consequences. In an early paper Damasio et al. ([1990]) classified this condition as a form of ‘acquired sociopathy’. In accord with this characterization the authors proposed that the function of the VMF, in normal individuals, is to process ‘the somatic state generated to the deferred outcome rather than the alternative at hand or the immediate consequence [of the action]’ ([1990], p. 83). In subsequent writings, Damasio further emphasizes Elliot’s impulsiveness by describing him as a ‘modern day Phineas Gage’ ([1994]).

This hypothesis, that the VMF processes information about future reward and punishment, has been a running theme throughout Damasio’s writings (Colombetti [2008]). A striking thing about this type of anecdote is that it stands in sharp contrast to the aforementioned picture of Elliot as chronically indecisive. Indeed, these two conditions appear to lie at opposite ends of the decision-making spectrum. Giovanna Colombetti ([2008]), who first drew attention to this tension, suggests that more care must be taken in describing what it means to be a ‘bad’ decision maker. Our matrix of alternative somatic marker hypotheses provides a framework making such claims more explicit.

As with the previous anecdote, Elliot’s impulsiveness admits of multiple interpretations. Suppose, again, that Elliot’s VMF functions like a loose wire. If the VMF is involved in decision-point recognition, then there will be some occasions when the mechanism fails to identify a decision point and Elliot acts impulsively, like a child chasing her ball into the road. It is also possible that Elliot’s impulsiveness

arises out of problems that emerge at the terminal stages, in the Execution of a decision. On this proposal, Elliot's higher control centres are unable to override his more basic impulses. Thus, as in the case of his chronic indecisiveness, this second category of anecdote is potentially explainable in terms peripheral processes.

4.3 Enhanced Decision Making in the Lab

In many of his early papers, Damasio supplements experimental results with suggestive anecdotes about how VMF patients responded to the laboratory setting. Of particular interest is the way that Elliot responds to artificial decision making tasks, since these mirror the types of problems with which he struggles in real life. Interestingly, some of these anecdotes portray Elliot as a rather adept decision maker. Eslinger and Damasio ([1985]) illustrate the sort of moral dilemma on which Elliot performs well:

Two people were shipwrecked on a desert island and ran out of food. The one person killed the other for food and survived. After rescue, he had recurring nightmares and went to a psychiatrist for treatment. The psychiatrist refused to treat the patient. Was the psychiatrist right or wrong? (p. 1733)

To convey Elliot's degree of competency in solving these tasks, the authors recount part of his response:

'Wrong [...] because the psychiatrist's duty is to treat a patient for whatever his mental ills may be, not to judge whether he should have or shouldn't have treatment'.

Eslinger and Damasio add that,

On several other examples of this sort, he always answered *without hesitation* and with responses that examiners judged as sensible. (ibid, *our italics*)

In our opinion, this type of anecdote suggests that the core components of Elliot's decision making machinery are intact. If Elliot suffered from Hamlet's Problem or a deficit in value assignment or ranking, his performance on these reasoning tasks would not be above average. As we noted earlier, Eslinger and Damasio attribute the discrepancy between his laboratory and real world performance to a defect in what we call decision-point recognition. Though viable, this hypothesis disappears from Damasio's later writings.

4.4 Lack of Motivation

Finally, these authors identify a fourth type of anecdote which, in our opinion, is extremely suggestive of the nature Elliot's impairment. They note that,

In general, [Elliot] was not spontaneously motivated for action. He seemed not to have available, automatically, programs of action capable of driving him to motion. As he awoke, there was no evidence that an internal, automatic program was ready to propel him into routine daily activities of self-care and feeding, let alone those of traveling to a job and discharging the assignments of a given day. It was as if he 'forgot to remember' short- and intermediate-term goals. (ibid, p. 1738)

Such behaviour suggests that Elliot's problem is motivational in nature. As Eslinger and Damasio put it, he might suffer from a deficit in 'translating plans and decisions into action' (ibid, p. 1738). Oddly, these authors dismiss this possibility despite supportive anecdotal evidence and inconclusive experimental results.

Taken together, these anecdotes paint a picture of Elliot's impairment that is quite unlike the one that Damasio usually advocates. Elliot exhibits cognitive or behaviour deficits in three areas: extreme indecisiveness, impulsivity, and lack of

motivation. All three of these are consistent with defects in the ‘peripheral’ stages of decision making. Neither his impulsivity nor his lack of motivation are consistent with a defect in ‘core’ processes. This interpretation is supported by Elliot’s strong performance in artificial reasoning tasks, which suggest that his peripheral processes are damaged while his core processes are functioning normally.

One might object to this argument on the grounds that it is based on anecdotal reports drawn primarily from one individual. Indeed, we agree that these reports are at most suggestive. However, in the case of Damasio’s work, perhaps more than usual for hypotheses in cognitive neuroscience, these anecdotes have been influential in the communication and uptake of SMH. Anecdotes feature prominently in Damasio’s writings, and they are frequently repeated by proponents of this work. At the very least it is rhetorically significant that some anecdotes are in conflict and that they admit of multiple interpretations.

5 Early Experiments Suggest that VMF Damage Leaves Core Processes Intact

This section reviews two sets of experiments published in the early 1990s, before the adoption of the Iowa Gambling Task as the dominant tool for probing decision making disorders. These experiments are not often cited in contemporary discussions of SMH. We focus on them here for two reasons. First, these articles reveal some important theoretical refinements to SMH made during this period. In what follows we document a narrowing in the range of hypotheses put forward to explain Elliot-like impairments. Specifically, the possibility of damage to peripheral processes was dropped from consideration, while focus was placed on hypotheses positing damage to core processes. This trend is arguably not supported by the results published in the

same papers where these refinements occur. Rather, to the extent that they address SMH whatsoever, data suggest that VMF damage impacts peripheral rather than core processes. This brings us to the second reason for focusing on these early publications. Although they were not explicitly recognized for doing so at the time, some of these experiments (i.e. the second set of tasks described in section 5.2) test for ability in particular stages of decision making. Hence they distinguish experimentally among alternate versions of SMH that lie along this dimension. It is our view, therefore, that during this early period of SMH research some pioneering research was conducted on SMH, yet these results had little impact on the direction taken by somatic marker theorists.

5.1 The Evocative Images Study

The Evocative Images Study (as we are calling it) was first published in *Behavioural Brain Research* (Damasio et al. [1990]) and again a year later in an edited volume (Damasio et al. [1991]). This experiment measured skin conductance responses (SCRs) to evocative images, such as pictures of car crashes and nudes. VMF patients were exposed to these images under a ‘passive’ condition in which they simply observed the images in silence, and also under an ‘active’ condition in which image contents were verbalized. In the passive condition VMF patients differed from control subjects in exhibiting no SCRs. However, under the active condition when image contents were verbalized, VMF patients exhibited the same sorts of elevated SCRs as controls.

The theoretical significance placed on these experiments is interesting. Although they claim to be testing for VMF patients’ deliberative or evaluative

abilities, neither of these subprocesses are engaged by the experimental protocol.

Damasio et al. interpret their findings as follows:

The finding of defective skin conductance responses to socially significant stimuli suggests that the Bifrontal subjects failed to generate somatic states to the implications of those stimuli at the most basic level. This defect is probably a correlate of the failure to experience a somatic state appropriate to the consequences of social situations. ([1990], p. 90)

We are puzzled by this interpretation. There is no obvious reason to conclude that normal subjects were responding to the *implications* or *consequences* associated with the evocative images rather than to the images themselves. If anything, the modality-specific nature of these SCRs suggest a perceptual deficit. Perhaps this has something to do with the perceptual mechanism involved in decision-point recognition. At the very least, one might expect further investigation of this possibility. However, these questions have not to our knowledge been pursued by Damasio's group.

Of further interest is the range of alternative hypotheses put forward to explain the relationship between VMF damage and impaired decision making. Like most of Damasio's publications, these articles begin with a discussion of Elliot and his decision making impairment. However, unlike earlier publications (e.g. Eslinger and Damasio [1985]), damage to peripheral processes are no longer raised as viable hypotheses. Instead Damasio et al. ([1990]) mention just two hypotheses, both of them appealing to deficits in core processes.

The first hypothesis posits that EVR-type patients do not conjure up enough information to define the implications of social situations and consequently cannot act appropriately. ([1990], p. 82)

The process that these authors describe as ‘conjuring up of the implications of social situations’ corresponds to what we identified earlier as factor generation. Hence it is fairly clear that the authors are proposing Elliot’s decision making deficit is caused by an impaired deliberative capacity. Apparently, the idea is that Elliot chooses self destructive behaviours because he is incapable of formulating, and therefore anticipating their implications. The authors go on to contrast this hypothesis with a second possibility.

‘The second [hypothesis] states that although able to conjure up the myriad of implications of a social situation (e.g. response options, consequences), EVR-type patients are unable to *mark* those implications with a signal that would automatically distinguish advantageous from pernicious actions, in the perspective of social rules and current contingencies. (original emphasis, *ibid*)

Here the authors are referring to the process we call value assignment, the component of evaluation wherein factors are associated with autonomic tags and thereby biased as either favourable or aversive according to their valences. On this proposal, Elliot can formulate options but he lacks a preference for any one of them.

It is surprising that the field of candidates has been narrowed to just these two alternatives. There is no explicit acknowledgement or consideration of the potential roles for decision-point recognition, option generation or execution. Even more striking, the authors rule out the first hypothesis that they mention (factor generation) even before presenting the results of the Affective Images Study. Rather, the authors reject the possibility of a deficit in factor generation by appealing to the anecdotal observation that,

E.V.R. remains capable of conjuring up many complex implied meaning scenarios in laboratory paradigms. Such evidence in turn suggests that the

problem may reside with choosing an advantageous course of action, *or with implementing it.* (Damasio et al. [1990], p. 83, *our italics*)

Although the authors cite no data to support of this claim, it is true that Elliot's strong performance on certain problem solving tasks (see below) suggests that his deliberative capacities are intact. However, it does not follow that his deficit is therefore evaluative in nature. As we have argued, there are several alternative hypotheses which have not been excluded. In fact, careful readers will note that the italicized portion of this sentence alludes to damage in the final stage of decision making (Execution) as a remaining possibility once factor generation has been discarded. However, this alternative is no longer being identified as a distinct hypothesis that receives its own consideration. Instead, the possibility of damage to this peripheral process is being lumped together with the second (favoured) hypothesis, that Elliot suffers from a defect in evaluation. It is as if these hypotheses were not distinct.

5.2 Five Problem Solving Tasks

During the same period as the Affective Images Study, Damasio published a second round of experiments in an article coauthored with Jeffrey Saver ([1991]). This set of experiments exposed one VMF patient (Elliot) to five problem solving tasks. Unlike other experimental paradigms, these tasks probe particular stages of the decision making process. Thus, despite the lack of an adequate sample these experiments are unusual in providing data that discriminates among several alternative somatic marker hypotheses.

The first experiment, called the The Optional Thinking Test, measured Elliot's ability to generate candidate solutions to hypothetical social dilemmas. On this test

Elliot performed like a star, generating significantly more options than controls. To illustrate, Saver and Damasio share a portion of Elliot's response to the question, How would you convince a friend to watch a different television program?

‘Just change the channel to his program. Go watch another TV set in another room. Tell the friend to leave so that he can watch whatever he wants to.

Convince the friend that the program he wants to watch [Sic] is the best one.

Make an agreement with the friend that they can alternate their times and have a compromise on the programs that they each watch at different times so that they can both be satisfied between themselves with a compromise as to what they would do. One of those ought to work; if not, they can just get rid of the TV set and not watch anymore’. ([1991], p. 1244)

Elliot's strong performance in this task suggests that his VMF lesion does not impact his capacity for option generation. Insofar as the VMF is involved in the production of somatic markers, this finding suggests that somatic markers are not necessary for option generation.

The Awareness of Consequences Test presented Elliot with four hypothetical predicaments that present a temptation to transgress certain social norms. For example, subjects imagine receiving an over-payment from a bank teller, and are asked to explain the sorts of considerations they would take into account when deciding what to do. The dependent variable is the number of consequences that subjects spontaneously take into account. Interestingly, Elliot generated significantly more consequences than controls, suggesting that his capacity for deliberation (both factor generation and termination) is also intact.

The Means Ends Problem-Solving Test is a story completion task requiring participants to generate a step-by-step plan for achieving some social objective, for

example, the goal of making friends in a new neighbourhood. Scoring is based on the number of relevant versus irrelevant factors that participants generate. Hence this task is particularly well suited for testing the Relevance Hypothesis, i.e. the hypothesis that somatic markers contribute to the generation of factors that are relevant to some set of options. Once again Elliot's relevance scores were significantly above normal, suggesting that he does not suffer from this defect in deliberative capacity.

The fourth, Cartoon Predictions Test is a non-verbal task in which participants are presented with pictorial representations of social scenarios, and required to select among three possible outcomes—also presented as cartoon pictures—as the most likely. In our opinion this test does not probe for any particular stage of practical decision making. Although this test was directed at the visual modality, it falls short of testing for a deficiency in decision-point recognition because participants were not required to self-initiate a decision making process. Similarly, this test does not test for evaluation because subjects are asked to predict an outcome, not to express a preference. Nor does this test probe the Execution stages of decision making.

A more controversial question is whether the fifth task, the Moral Judgment Interview, can be legitimately construed as a probe for damage to core processes. This task is identical to an experiment reported by Eslinger and Damasio ([1985]) in which participants are presented with a moral dilemma and asked how they would respond. Saver and Damasio argue that this task is the only one in which participants were actually required to arrive at a choice.

Suppose that Elliot displays chronic indecisiveness in responding to these dilemmas. This would suggest that he suffers from Hamlet's Problem, a defect in deliberation. In addition, Elliot's inability to resolve moral dilemmas might suggest a defect in evaluation, perhaps because he sees all options as neutral or cannot rank

them. By contrast, competency in this task would suggest that his problem lies elsewhere, perhaps in decision-point recognition or execution. Elliot's performance on this task was normal in the sense that he showed the same level of sophistication in his capacity to resolve moral dilemmas as typical adults, indicating perhaps that his deficit lies outside of the two core areas being probed.

To summarise the findings of these five experiments, we have argued that four of them test for ability in core stages of decision making. On all of these tasks however, Elliot performed at or above normal levels. This suggests that his decision making deficit does not stem from damage to core processes. Before considering Saver and Damasio's contrasting interpretation of these findings, it is important to consider the way in which SMH is formulated in this paper.

Recall that in the other two publications mentioned earlier in this section, the spectrum of alternative SMHs was reduced to just deliberation and value assignment. In this publication, Saver and Damasio also present two potential explanations for Elliot's defect. However, these two hypotheses differ substantially in content from those which were mentioned earlier.

There are several possible mechanisms for [Elliot's] defect. In one, these patients may no longer possess or be able to access enough knowledge to define the implied meanings of social situations, or to conjure up possible response options and their future consequences. (Saver and Damasio [1991], p. 1241)

This hypothesis combines option generation and deliberation into a single 'mechanism', even though these are distinguished as distinct processes elsewhere in Damasio's writings. The second hypothesis makes a similar move, combining at least three distinct processes.

In another possible mechanism, although able to conjure up a myriad of implications, response options, and consequences, patients fail to choose an option, or choose a disadvantageous option, or fail to implement the choice.

(ibid)

Three processes are lumped into this ‘mechanism’: (1) ‘failing to choose an option’ describes a deficit in the Termination stage of deliberation (associated with Hamlet’s Problem); (2) ‘choosing a disadvantageous option’ apparently refers to a defect in the accuracy of evaluation; and (3) ‘failing to implement a choice’ apparently describes a motivational defect. The authors offer no argument for why these distinct hypotheses should be clustered into just two mechanisms. It is also surprising that Saver and Damasio should opt for such a course-grained presentation of these alternatives, since the actual experiments described in this paper investigate decision making at a more refined level. Another peculiar feature of this presentation is that a deficit in value assignment receives little mention as a likely explanation for Elliot’s condition. In fact, this publication is unusual in that the term ‘somatic marker’ receives only a passing mention in the discussion. We assume that these differences in emphasis are a reflection of the different co-authors with whom Damasio was working during this period.

We have argued that Elliot’s strong performance on the tasks that test for ability in core processes suggest that his decision making deficits lie elsewhere. However, Saver and Damasio argue nonetheless that the evidence suggests a deficit in the ‘choice component’ of decision making, a conclusion that does not appear to be borne out by the testing data. Their argument is based largely on an anecdote describing the way in which Elliot responded to moral dilemmas. Saver and Damasio report that, ‘In the one task that required a choice (Moral Reasoning) E.V.R. was

reluctant to choose and needed to be prompted to do so' ([1991], p. 1247). The suggestion is that Elliot was unable to solve moral dilemmas on his own and that, therefore, he suffers from damage to his core decision making processes—either Hamlet's problem or an evaluation deficit.

We find this argument highly problematic. For one thing, the authors present no data quantifying Elliot's hesitation. This strikes us as a rather obvious avenue for investigation (nor to our knowledge has it been pursued subsequently). Without such data it is impossible to determine whether Elliot's hesitation was significantly abnormal. Moreover, this anecdote flatly *contradicts* earlier anecdotes in which Elliot is portrayed as an adept decision maker. In particular, in Elsinger and Damasio ([1985]) Elliot is described as responding to moral dilemmas 'always without hesitation' (p. 1256). Perhaps this discrepancy failed to capture the attention of the original authors.

To summarise our argument in this section, we have focused on two early experiments that tend to receive little attention in SMH literature. However, these studies are insightful on several fronts. First, in all three articles one finds a narrowing of the range of candidate somatic marker hypotheses. The particular set of hypotheses that get excluded or lumped together on a given occasion varies across papers without obvious justification. There has been a trend in Damasio's theorizing away from considering peripheral process in favour of core processes as an explanation for VMF patients' abnormal behaviour. This shift conflicts with the balance of the anecdotal and experimental evidence available at the time. Second, although the Evocative Images Study does not probe for decision making, this does not prevent Damasio from interpreting these data as evidence in favour of a deficit in core processes. We have argued that, at most, this study suggests a perceptual deficit, perhaps having to

do with decision-point recognition. It is very surprising to us that this avenue has not been explored in subsequent work. Third, some of the problem solving tasks devised by Saver and Damasio are particularly effective in probing certain core stages of decision making. The fact that Elliot performs at or above average on these tasks reveals competency in these stages. Likewise, these results suggest that his deficiency stems from damage to peripheral stages, in accordance with the balance of the anecdotal evidence mentioned in the previous section. Of course, one must be cautious about deriving any such conclusions given that these experiments have been performed on only one VMF patient. It is also surprising to us that these experiments have not, to our knowledge, been replicated given their effectiveness as stage-specific probes.

It is perhaps unfortunate that the trend in this field has been to adopt the Iowa Gambling Task as the primary research tool instead of refining the techniques pioneered by Saver and Damasio ([1991]). In the following section we briefly review this body of research, arguing that this tool is a rather blunt instrument for discriminating among alternate versions of SMH.

6 Recent Experiments Fail to Discriminate Among Alternate Versions of SMH

More recent attempts to test SMH employ an experimental paradigm that purports to reflect real-world decision making. The Iowa Gambling Task (IGT) was devised by Damasio's postdoctoral student, Antoine Bechara, who pitched it as 'a neuropsychological task which simulates, in real time, personal real-life decision making relative to the way it factors uncertainty of premises and outcomes, as well as reward punishment' (Bechara et al. [1994], p. 8). The IGT is a gambling task in which

participants are presented with four decks of cards, 'A', 'B', 'C', and 'D'. Participants begin with \$2000 in facsimile money. They are told that the task requires a long series of card selections and that the goal is to maximize profit. On the reverse of each card is a dollar figure, either gained or lost. Decks 'A' and 'B' generally yield gains of \$100 with occasional large losses. Overall, 'A' and 'B' are net loss decks, averaging a net loss of \$250 every 10 cards. Decks 'C' and 'D', generally yield small gains of only \$50 but with smaller losses. Decks 'C' and 'D' are net gain decks, averaging a net gain of \$250 every 10 cards. Participants are permitted to switch freely between decks at any time, but are not told when the task will terminate.

The first iteration of the IGT found that the VMF patients performed drastically worse than controls, selecting more cards from the bad decks ('A' and 'B') than from the good ones ('C' and 'D') (Bechara et al. [1994]). The researchers developed two hypotheses to explain these results. The first attributed the performance of VMF patients to an inability to focus attention on the bad decks, and the second to a failure to associate valence with the options. Following the initial IGT publication, the former hypothesis was inexplicably dropped. We are puzzled by this move.

In an effort to detect somatic activation, and thus confirm the latter hypothesis, Bechara and colleagues turned to skin conductance responses (SCR). All three groups exhibited 'reward' and 'punishment' SCRs after selecting high value cardsⁱⁱⁱ. Yet these responses also tend to emerge in non-VMF patients in anticipation of selections from bad decks, which were usually followed by a shift to the good decks. Hence anticipatory responses, which can occur without awareness and are unique to non-VMF patients, are interpreted as evidence that somatic markers influence decision making in these individuals. The authors took this result as confirmation of the

somatic marker interpretation of the earlier ([1994]) IGT study (Bechara et al. [1996], p. 223).

The accuracy of these findings has been hotly disputed, and it remains controversial whether the Iowa Gambling Task is an adequate test for the relationship between somatic markers and decision making (see Maia and McClelland [2004]; Dunn et al. [2006]; Gerrans [2007]; and Colombetti [2008] for insightful discussions). Dunn et al. ([2006]) suggest that much of this controversy stems from the fact that the somatic marker hypothesis is so poorly specified. Colombetti ([2008]) echoes this point, providing a detailed analysis of the IGT. She concludes that the experimental structure is incapable of testing the specific version of the hypothesis she identifies, in spite of claims to the contrary. Importantly, we should also be wary of the use of SCRs as a metric for detecting somatic activation. Colombetti demonstrates that the notion of somatic activation is too broadly characterized by SMH theorists to justifiably interpret SCRs as indications of somatic markers; her recommendations for rectifying this issue have not, to our knowledge, been adopted. We agree with these assessments and view this as another place where our matrix of alternative hypotheses permits further exploration. In the remainder of this section we build on these findings by walking through the five main stages of practical decision making and considering whether the IGT is capable of identifying deficiencies in any of these processes. Even if Colombetti's excellent suggestions for a non-contentious notion of somatic activation were taken up by SMH theorists, it is unlikely that the IGT can test any of the somatic marker hypotheses identified above.

First consider whether the IGT can test whether somatic markers are involved in decision-point recognition. In its current form, this appears to be impossible because decision points are too artificially delineated by the experimental setup.

Participants are not required to generate or recall their own decision points. Instead this task is artificially structured by the card selection protocol. However, it might be possible to modify this experimental design so that decision points are less structured. For example, participants could engage in a focused task that periodically calls for self-generated bouts of reflection. If these bouts are always preceded by autonomic activity, as measured by SCR, this would suggest that somatic markers are involved in decision-point recognition. By comparing the decision points and the latency periods of VMF patients and controls, such an experiment could also test whether somatic markers contribute to the speed, the accuracy, or both dimensions of this process. Given the balance of the anecdotal and experimental evidence suggesting that Elliot might suffer from a defect in this early stage of decision making, we think that devising such an experimental paradigm would be a worthwhile pursuit.

It is less likely that any version of the IGT can be employed to test for a relationship between somatic markers and option generation. The four available options (decks of cards) are limited and clearly defined. Instead, some of the problem solving tasks devised by Saver and Damasio are much better suited to this question.

Recall that successful deliberation requires both the generation of factors that are relevant to the decision at hand as well as a rule for determining when factor generation should terminate. The IGT is poorly suited for testing the Relevance Hypothesis. Participants in this experiment are required to consider just one factor—expected profit—when deliberating about the four decks. Such a decision hardly invites factor generation and hence there are minimal opportunities for one's deliberation to go off track. In this respect the experimental design fails to mirror real-world decision making.

Nor can the IGT test whether VMF patients suffer from Hamlet's Problem, because this task provides no way of measuring the amount or nature of deliberation associated with card selection. One option is to use as a proxy for deliberation the latency between card selections. In which case, the Selection Hypothesis predicts that the inter-card interval is greater for VMF patients than for controls. Interestingly, a recent study by Fellows and Farrah ([2005]) found no such latency among these groups. Assuming that the VMF is involved in somatic marker activation, this evidence suggests that somatic markers are not necessary for solving Hamlet's problem. Of course, one must be cautious about this inference given the artificial nature of the IGT.

A more difficult question is whether the IGT probes for evaluation (either value assignment or ranking). This hypothesis predicts that negatively valenced markers bias a subject away from an associated option, whereas positive markers render the option more favourable. If the IGT is to probe the evaluative capacities of participants, it is crucial that the reward schedule of the decks is cognitively opaque. Participants must not be able to consciously decipher the schedule of reward and loss. Otherwise, it is possible that an explicit reasoning process, not somatic markers, explains improved performance on the task. Damasio et al. laud the cognitive opacity of the task,

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 the VMPFC fail this task and they fail it precisely because they are unable to represent choice bias in the form of an 'emotional hunch'. (as cited in Dunn et al. [2006], p. 245)

Bechara et al. ([1997]) claim to have confirmed this assumption by querying participants' knowledge about the respective payoffs while they are engaged in this task. However, a more recent study challenges their results:

We show that, in fact, players have extensive conscious knowledge about the game, as indicated in verbal reports obtained with a more sensitive questionnaire than that used by Bechara et al. (Maia and McClelland [2004], p. 16075)

Maia and McClelland ([2004]) ran an iteration of the IGT in which participants were stopped every 10 rounds and asked to respond to a structured questionnaire. The purpose of the questionnaire, which they deem more discriminating than that used by Bechara et al. ([1997]), is to gauge participants' knowledge of the task and the reward schedule.

[W]hen [participants] behave advantageously, they nearly always report knowledge about the outcomes of the decks that would be sufficient to guide such advantageous behaviour. Thus, our data provide no reason to posit that no conscious biases guide advantageous behaviour in this task before knowledge that is consciously accessible does. (ibid; *see* Bechara et al. [2005] for reply)

Absent some method to determine that somatic markers, not conscious knowledge, solve the ranking problem, the IGT appears insufficiently sensitive to test the role of somatic marking in this stage of decision making.

A final question is whether the IGT can determine whether somatic markers are involved in execution. As we have noted, several lines of evidence suggest that VMF damage impacts motivation. This would potentially also explain the discrepancy between Elliot's performance in the lab, where decisions are externally imposed, and real life where actions often require self motivation. Unfortunately the IGT is a poor

test of this hypothesis. For one thing, card selections are forced. Certainly a participant could refuse to choose a card altogether, thus failing to participate in the task, but to our knowledge this has not occurred. It is plausible that participants select cards simply because they have been instructed to do so, not because they are internally motivated to execute a decision. An adequate test for execution might involve a more open-ended task in which participants first select among candidate options but are then provided with the opportunity to opt out of execution. A second shortcoming of the IGT is that it does not permit participants to engage in a range of different types of behaviour—either they select from a deck or they don't. This point is important if one assumes that somatic markers merely guide execution, as opposed to being necessary for any action to occur. Evidence that somatic markers guide execution requires concordance between a decision outcome and the ensuing behaviour. Not just any behaviour following on the heels of a decision reveals that the decision is in fact being executed. Testing for concordance requires some independent determination of the outcome of the decision process to see whether it accords with the participant's behaviour. The IGT in its current form fails to satisfy this requirement.

We have already mentioned a number of critiques of the IGT; taken together with our finding that the task cannot adequately discriminate between different stages of decision making, it is puzzling that the IGT supplanted the tests pioneered by Saver and Damasio as the tool through which SMH is assessed. In its present incarnation, the IGT is incapable of testing for decision-point recognition, option generation, and execution. It is also poorly suited for assessments of either deliberative function, and it is highly controversial whether the IGT is an adequate test of evaluation. This is highly problematic, not only for SMH, but also because the IGT has subsequently

been adopted in many other studies. Dunn et al. found that by July 2005, there were 40 studies published with IGT data, each probing decision making deficits in some target population ([2006]), no data is available indicating how much use of the task has grown since then. We suggest that if the task is retained as a test for decision making deficits, that hypotheses concerning which features of decision making are being probed are clearly formulated, and that the test is modified, where necessary, to properly test those features. Likewise, until more careful delineations are made between the different versions of SMH, it will remain unclear precisely what is indicated by either success or failure on the task.

6 Conclusion

It is commonly assumed that there is such a thing as ‘the’ somatic marker hypothesis and that the evidence in its favour suggests a role for somatic markers in the core stages of decision making. The first of these two myths has recently come under scrutiny. As Dunn et al. ([2006]) argue, the Iowa Gambling Task fails to discriminate among a range of different somatic marker mechanisms. Likewise, Colombetti ([2008]) identifies an ambiguity in Damasio’s writing among two versions of SMH: a general version (SMH-G) positing a role for autonomic signals in practical decision making, and a specific version (SMH-S) proposing that somatic markers are required for considering long-term outcomes of available actions. She argues that SMH-G is too vague to admit of experimental refutation, while SMH-S is not adequately probed by the IGT. Colombetti concludes with the suggestion that, ‘[d]istinguishing 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’ ([2008], p. 69). Our three-dimensional

matrix of somatic marker hypotheses offers a first step in this direction. One advantage of this framework is that it helps to sharpen previous critiques of SMH. For instance, Colombetti's SMH-S decomposes on our analysis into three more specific hypotheses (1) that somatic markers are involved in identifying options, (2) that somatic markers guide deliberation towards potential future consequences of each option, or (3) that somatic markers facilitate the evaluation of those options and factors. In fact, our analysis calls for an even further refinement of SMH-S by demanding clarification on whether somatic markers contribute to the speed or accuracy of each sub-process, and whether their contribution is required or merely conducive at each stage. A further advantage of this framework is that it provides a foundation for generating more precise hypotheses about the various potential roles for somatic markers at particular stages in decision making. Of particular interest are the potential roles (largely unexplored) for somatic markers in what we call decision-point recognition and execution.

The second myth, that Damasio's evidence suggests a role for somatic markers in the core stages of decision making, is perhaps even more widely entrenched. Like previous critics, we do not think that the Iowa Gambling Task can discriminate among alternative versions of SMH. However, our analysis suggests other potential experiments that might do so. Some of these experiments were pioneered in Damasio's early work and it is ironic that they have been all but forgotten. We have argued that when one revisits these early results, the balance of evidence favours a role for somatic markers in peripheral rather than core stages of decision making. Specifically, our suggestion is that somatic markers most likely contribute to either the speed or the accuracy of decision-point recognition, or, they play a motivational role in decision-execution. Currently, this proposal is based on an analysis of

Damasio and colleagues' early work. However, we think that these 'peripheral' somatic marker hypotheses suggest promising directions for future research.

Stefan Linnquist
Department of Philosophy
University of Guelph
50 Stone Road E.
Guelph, Ontario,
N1G2W1
Canada
linquist@uoguelph.ca

Jordan Bartol
Department of Philosophy
University of Leeds
Leeds, LS2 9JT
phjnb@leeds.ac.uk

Acknowledgements

We thank Paul Griffiths, Don Dedrick, and two anonymous reviewers for their helpful comments and suggestions.

References

- Bechara, A. [2004]: ‘The role of emotion in decision-making: Evidence from neurological patients with orbitofrontal damage’, *Brain and Cognition*, **55**(1), pp. 30-40.
- Bechara, A. and Damasio, A. R. [2005]: ‘The somatic marker hypothesis: A neural theory of economic decision’, *Games and Economic Behaviour*, **52**, pp. 336-72.
- Bechara, A., Damasio, A. R., Damasio, H. and Anderson, S. W. [1994]: ‘Insensitivity to future consequences following damage to human prefrontal cortex’, *Cognition*, **50**, pp. 7-15.
- Bechara, A., Damasio, H. and Damasio, A. R. [2003]: ‘Role of the amygdala in decision-making’, *Annals of the New York Academy of Sciences*, **985**, pp. 356-69.
- Bechara, A., Damasio, H., Damasio, A. R. and Lee, G. P. [1999]: ‘Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making’, *Journal of Neuroscience*, **19**(13), pp. 5473-81.
- Bechara, A., Damasio, H., Tranel, D. and Damasio, A. R. [1997]: ‘Deciding advantageously before knowing the advantageous strategy’, *Science*, **275**, pp. 1293–5.
- Bechara, A., Damasio, H., Tranel, D. and Damasio, A. R. [2005]: ‘The Iowa gambling task and the somatic marker hypothesis: Some questions and answers’, *TRENDS in Cognitive Sciences*, **9**(4), pp. 159-62.

Bechara, A., Tranel, D., Damasio, H. and Damasio, A. R. [1996]: 'Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex', *Cerebral Cortex*, **6**, pp. 215–25.

Barrett, L. F. and Bar, M. [2009]: 'Seeing it with feeling: Affective predictions during object perception', *Philosophical Transactions of the Royal Society B*, **364**, pp. 1325-34.

Carroll, J. S. and Johnson, E. J. [1994]: *Decision Research: A Field Guide*, Newberry CA: Sage.

Colombetti, G. [2008]: 'The somatic marker hypothesis, and what the Iowa gambling task does and does not show', *The British Journal for the Philosophy of Science*, **59**(1), pp. 51-71.

Damasio, A. R. [1994]: *Descartes' Error: Emotion, Reason, and the Human Brain*, New York: Putnam.

Damasio, A. R. [1996]: 'The somatic marker hypothesis and the possible functions of the prefrontal cortex', *Philosophical Transactions of the Royal Society of London*, **351**, pp. 1413-20.

Damasio, A. R., Tranel, D. and Damasio, H. [1990]: 'Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli', *Behavioral Brain Research*, **41**, pp. 81-94.

Damasio, A. R., Tranel, D. and Damasio, H. [1991]: 'Somatic markers and the guidance of behavior: Theory and preliminary testing'. In H. S. Levin, H. M. Eisenberg and A. L. Bendon (eds.), *Frontal Lobe Function and Dysfunction*, Oxford, UK: Oxford University Press.

- Dunn, B. D., Dalgleish, T. and Lawrence, A. D. [2006]: 'The somatic marker hypothesis: A critical evaluation', *Neuroscience and Biobehavioral Reviews*, **30**, pp. 239-71.
- Eslinger, P. J. and Damasio, A.R. [1985]: 'Severe disturbance of higher cognition after bilateral frontal lobe ablation: Patient EVR', *Neurology*, **35**, pp. 1731-41.
- Evans, D. [2002]: 'The search hypothesis of emotion', *British Journal of Philosophy of Science*, **53**, pp. 497-509.
- Fellows, L. K. and Farrah, M. J. [2005]: 'Different underlying impairments in decision-making following ventromedial and dorsolateral frontal lobe damage in humans', *Cerebral Cortex*, **15**, pp. 58-63.
- Gerrans, P. [2007]: 'Mental time travel, somatic markers and 'myopia for the future'', *Synthese*, **159**, pp. 459-74.
- Prinz, J. [2005]: *Gut Reactions: A Perceptual Theory of Emotion*, Oxford University Press.
- Maia, T. and 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 of Sciences*, **101**, pp. 16075-80.
- Saver, J. L. and Damasio, A. R. [1991]: 'Preserved access and processing of social knowledge in a patient with acquired sociopathy due to ventromedial frontal damage', *Neuropsychologia*, **29**, pp. 1241-49.
- Thagard, P. [2006]: *Hot Thought: Mechanisms and Applications of Emotional Cognition*, Cambridge, MA.: MIT Press.

Figure 1: Matrix of 38 alternate somatic marker hypotheses, with columns representing the five stages (and two sub-stages) of practical decision making, rows representing the modality of the hypothesis, and inner boxes representing the type of contribution made at a particular stage.

[Figure 1 – Two Myths About Somatic Markers.docx]

Footnotes

* **Address for correspondence:** S. Linquist; Department of Philosophy, University of Guelph; 50 Stone Road E.; Guelph, Ontario, Canada; N1G2W1.
Email: linquist@uoguelph.ca

ⁱ It is not clear that execution can be distinguished in terms of speed and accuracy. Although it is conceivable that a given action could be executed more or less quickly, or with greater or lesser accuracy, to our knowledge the somatic marker mechanism has not been implicated in these sorts of motor functions.

ⁱⁱ Interestingly, an anonymous reviewer of this paper was surprised by the array of somatic marker hypotheses generated by our analysis, noting that a slightly different understanding of the decision-making process could result in a much larger or more complex space of possibilities. Undoubtedly one could expand on this framework, for example, by further decomposing each of the subprocesses that we identify. Alternately, one might offer a very different taxonomy of decision making, perhaps by breaking this process down into the respective roles of short and long term memory, attention, perception, etc. A taxonomy focusing on neuro-anatomical structures (in addition to VMF) might render yet another spectrum of hypotheses about the potential roles for autonomic signals. Our reasons for settling on this particular three-dimensional framework are twofold. First, our aim has been to work within the general contours outlined by Damasio and colleagues—the primary advocates of somatic marker hypotheses. As we have argued, many of the distinctions that we identify are found, albeit implicitly, in these authors’ own writings. Our strategy for clarifying SMH has been to render these hypotheses explicit and to identify their logical structure. Second, we have sought to dovetail our framework with cognitive research on “real world” decision making. Hence, our taxonomy of five decision-making stages is inspired by such authors as Carroll and Johnson (1994).

ⁱⁱⁱ Note that this finding stands in apparent conflict with Damasio et al.’s previous ([1990]) results in which VMC damaged patients failed to generate SCRs responses to evocative stimuli. This discrepancy seems to suggest that the two experimental paradigms probe different psychological abilities. To our knowledge this issue remains unexplored.