

Mental time travel, somatic markers and “myopia for the future”

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Abstract Patients with damage to the ventromedial prefrontal cortex (VMPFC) are often described as having impaired ability for planning and decision making despite retaining intact capacities for explicit reasoning. The somatic marker hypothesis is that the VMPFC associates implicitly represented affective information with explicit representations of actions or outcomes. Consequently, when the VMPFC is damaged explicit reasoning is no longer scaffolded by affective information, leading to characteristic deficits. These deficits are exemplified in performance on the Iowa Gambling Task (IGT) in which subjects with VMPFC perform significantly worse than neurotypicals in a task which requires them learn from rewarding and punishing experience to make decisions. The somatic marker theory adopts a canonical theory of emotion, in which emotions function as part of a valencing system, to explain the role of affective processes. The first part of the paper argues against this canonical account. The second part provides a different account of the role of the VMPFC in decision-making which does not depend on the canonical account of emotion. Together the first and second parts of the paper provide the basis for a different interpretation of results on the Iowa Gambling Task (IGT). In fact the IGT may be probing a deficit in what has been called mental time travel: the ability to access and use information from previous experience and imaginatively rehearse future experiences as part of the process of deliberation.

Keywords Emotion · Somatic markers · Mental time travel · Ventromedial prefrontal cortex · Iowa gambling task

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1 Introduction

The somatic marker hypothesis associated with the work of Antonio Damasio and collaborators is an extremely influential attempt to account for the influence of emotions on decision-making in terms of neural architecture (Damasio et al. 1991; Damasio 1994; Bechara et al. 1999). Very briefly the SMH has four important aspects. The first is that emotions have their basis in interoception, that is bodily and visceral states (the “body” loop) or the representation of those states (the “as if” loop), hence the name somatic markers. The second is that somatic markers signal the valence (positive or negative value linked to approach or avoidance behaviour) of contingencies for an organism. The third is that the brain has evolved specialised structures, of which the ventromedial prefrontal cortex is the most important, that associate somatic markers with information relevant to decision making. For example information which is unmarked or weakly marked will be discounted or ignored while information which is strongly marked will be accentuated in decision making processes. Consequently, somatic markers play an essential role in planning and deliberation. The fourth aspect is that this biasing role of valence information is often covert or tacit, inaccessible to consciousness and unable to be explicitly verbalised.

These four aspects combine to support an account of implicit emotional memory which integrates some recent important findings in cognitive neuroscience with canonical psychological theories of emotion. The first piece of evidence is that elevated skin conductance responses (SCRs) can be evoked by emotionally salient memories even when the subject has no explicit memory of the event in question (Tranel and Damasio 1988). A subject for example who has no explicit memory of a stimulus (such as a visually presented face) nonetheless exhibits SCRs to that stimulus (Young and Burton 1999; Schweinberger and Burton 2003). This concurs with decades of psychological research showing that behaviour can be elicited by a stimulus, without explicit representation of that stimulus (Graf and Schacter 1985; Gabrieli et al. 1997; Faulkner and Foster 2002). The somatic marker hypothesis is that SCRs in these cases are evidence of automatic activation in systems which associate tacit representations with valence. The second strand of evidence is provided by the fact that subjects with absent SCRs for recall or anticipation of emotionally significant experience often have significant deficits in decision-making, despite intact ability for explicit means-end reasoning. Relatedly, they can be reckless, callous, impulsive, oddly flattened in affect, unmoved by the disastrous consequences of their actions or exhibit a combination of these states (Damasio et al. 1990; Damasio 1994; Levine et al. 1999). The somatic marker hypothesis is that damage to the systems on which SCR depends means that subjects cannot access or integrate, either tacitly or explicitly, information about emotional valence and hence cannot properly evaluate the consequences of personal decisions.

The somatic marker hypothesis explains the role of SCRs in these cases using a canonical account of emotions. Canonical accounts of emotions treat them as processes which motivate an organism according to the value, positive or negative, for an organism of the object which elicits them. Thus emotions such as fear motivate flight or withdrawal and are described as having negative valence. For canonical accounts, the characteristic experience of emotions is a way of focusing cognition on the value of the eliciting object, thereby making it *salient to cognition*. Fear, for example, makes

dangerous objects salient because the experience of fear is a way of being aware of the negative value of dangerous things. In effect the standard model assumes that salience (the ability to command attention and focus cognitive resource allocation) and motivation can be explained in terms of the *valence* (positive or negative) of the affective process which produces an emotion. For a review of the massive literature on the valencing role of emotions see (Colombetti 2005).

The somatic marker hypothesis is that emotions are typically elicited rapidly and automatically. For example seeing a shark in the water in which one is swimming or a friendly face in a hostile audience are situations with negative or positive valence. Representations of sharks or friendly faces are associated, rapidly, automatically and implicitly with their characteristic somatic marker. These implicit emotional memories help solve a frame problem (a role postulated for emotions by a number of theorists) by ensuring that cognition is appropriately focused on *salient* information, that is information which is relevant to the goals of the organism. Somatic markers also implicitly rank salient contingencies and bias responses towards them (Dunn et al. 2006).

These implicit processes interact with explicit conscious processing in approximately the same way as implicit and explicit motor cognitive processes. In effect implicit processes do most of the work automatically processing routine situations and “referring” non-standard or intractable problems to explicit executive cognition. Explicit cognition exploits controlled voluntary processing to solve problems (should you cremate or bury a relative? Invest in biotech or IT shares?) which exceed the capacities of the implicit systems. The systems are interdependent, however, because a decision made by explicit cognition still needs to be associated with a valencing response before it can be translated into action. Otherwise it remains motivationally inert.

Although interdependent, the systems do dissociate. Both in everyday life, as when best-laid plans reached by logically impeccable reasoning are frustrated by impulsive or deeply ingrained behaviour prompted by somatic markers, and in pathological cases. The pathological cases follow damage to structures which allow explicit conclusions to be somatically marked. The crucial structure here is the circuit linking ventromedial prefrontal cortex to the amygdala. “The VMPFC is believed to be the crucial area of the brain that integrates actual or predicted bioregulatory state representations with potential response options, so is central to the generation of somatic markers” (Dunn et al. 2006, p. 242). It follows that subjects with damage to the VMPFC may be able to reason explicitly about actions but be unable to act on the basis of these conclusions, and remain emotionally and motivationally detached from those conclusions, because they cannot associate an implicit somatic marker with an explicitly-generated conclusion (Bechara et al. 1999; Maia and McClelland 2004; Dunn et al. 2006).

The Iowa Gambling Task (IGT) was developed to examine the interaction between somatic markers and explicit cognition. The IGT requires participants to select from four decks of 100 cards, each of which is associated with a different regime of financial rewards and punishments. Participants learn through trial and error which decks are advantageous and, typically, after repeated trials, come to favour those decks. Crucially (i) the development of SCRs is associated with learning (ii) participants’ selections reflect this learning *before* they can explicitly articulate the reward/punishment schedule for each deck, an example of implicit learning, (iii) patients with

VMPFC or amygdala damage failed to develop “anticipatory” SCRs, a failure which correlates with impaired task performance relative to normals.

The conclusion of Bechara et al. was that these VPMFC patients had a form of “myopia for the future” based on inability to use somatic markers derived from previous experience to guide implicit *or* explicit decision making. When such patients are faced with a range of options they have no implicit somatic information signalling which decks are favourable and which are unfavourable. Consequently their selections don’t reflect lessons from previous experience. This idea generalises as an explanation of the indifference to consequences and inability to plan of VPMFC patients.

Both the somatic marker hypothesis and the interpretation of the IGT have been criticised on a number of fronts (Tomb et al. 2002; Maia and McClelland 2004; Dunn et al. 2006). For example, there are empirical and conceptual reasons to reject the identification of emotions with representations of body state.

For the remainder of this paper I will concentrate on the consequences for a theory of the role of emotions in decision making of findings which are not in dispute. VMPFC patients *do* seem to have a form of myopia for the future *correlated* with failure to develop anticipatory SCRs. Furthermore anticipatory SCRs index amygdala activation. The amygdala is a structure whose cognitive complexity may have been underestimated, but there is general agreement that it is implicated in processing indexed by SCR. VMPFC-amygdala circuitry is crucial to the ability to produce anticipatory SCRs, either because the VMPFC stores information about valence initially derived from the amygdala or because it enables top-down reactivation of amygdala as part of the process of retrieving affective information (The somatic marker hypothesis adopts the latter interpretation).

It is possible, in principle, to retain both the spirit of the somatic marker hypothesis and the canonical view of emotions as mechanisms which make contingencies salient and motivate behaviour in virtue of the fact that they represent valence. Such a revised account would abandon the idea that representations of body state are essential to emotion but retain the idea *that distributed circuitry involving the amygdala and VMPFC represents valence and thereby confers salience and motivation*. If patients with impairments to VMPFC lack a mechanism which provides access to information which confers salience, valence and motivation on representations of potential actions or outcomes their deficits remain explicable. This revised account would still explain the correlation between “myopia for the future” and absent SCR in terms of the role of the VMPFC in associating valencing information, which depends initially on amygdala activation, with explicit representations.

However this revision would be an unfortunate theoretical epicycle. In fact the canonical account of emotions requires revision, and once this is provided a different explanation of both the role of the VMPFC and the IGT is required. The rest of this paper is devoted firstly, to revising the canonical account of the relationship between salience, valence and motivation and secondly to applying this revised account to the explanation of “myopia for the future”.

2 Emotion and motivation

In effect valencing systems have multiple roles within the somatic marker account. They make objects salient and motivate appropriate action as well as assigning valence. In this respect the somatic marker account preserves the main feature of canonical accounts in which salience and motivation are explained in terms of valence. For example, the canonical account explains preferential attention to disgusting, rather than neutral, objects *and* aversive reaction to disgusting objects as a consequence of the activation of processes which represent the negative value of contamination.

This why, on the canonical account, VMPFC patients who have lost the ability to access valencing information cannot choose among options or behave appropriately to emotionally relevant stimuli. Intuitively this is an attractive idea, nicely dramatised by the IGT. Faced with four options of different valence the VMPFC patient does not appear to focus preferentially on any option or to be motivated to pursue reward or avoid punishment.

However recent research, given a conceptual underpinning by formal modelling shows that the mechanisms which make objects salient and motivate behaviour, are *independent, neurally and cognitively*, from those which determine valence.

This is clearly shown in cases of addiction or compulsion, where people continue to pursue unpleasurable or distasteful contingencies (Schultz et al. 1997; Egelman et al. 1998; Berridge and Robinson 2003; Smith et al. 2006). Somatic marker accounts have difficulty accounting for such phenomena since they tie approach behaviour to positive valence. Consequently, they treat such cases as instances in which higher cognition or abstract reasoning is unable to override motivation produced by the positive somatic marking. The positive affect implicitly associated with representations of drug taking by the somatic marker system overrides the cold-blooded calculation of cost benefits by the explicit reasoning system. Thus the addict might endorse the abstract proposition that methamphetamines are bad for her but the anticipated positive affect associated with the representation of the drug drives behaviour.

This type of explanation generalises to the explanation of any dissociation between explicit cognition and motivation. Just as explicit cognition without somatic markers is motivationally inert, explicit cognition which recommends an action contrary to that associated with a somatic marker is always liable to be overridden by an automated somatically-marked response.

However, recent studies show that the addict continues to be motivated because her *dopamine system* continues to make narcotic drugs salient, irrespective of their valence. Organisms will continue to act on a salient contingency even when *valence and qualitative systems are lesioned*. Conversely lesion to the dopamine systems does not affect either the valence or the qualitative character of an affective experience. For example even when the ability to experience a pleasant taste is removed, artificially or by lesion, rats (and humans) will still pursue the food. This is described by Berridge and Robinson as a dissociation between liking and wanting. Subjects want things they don't like because the wanting (motivational) system can function independently of the liking system. Thus subjects will pursue things they don't like provided that the contingency remains *salient* suggesting that salience is the real mechanism of motivation.

Dopamine systems produce salience by enhancing activation in neural systems that represent contingencies. A representation which is the target of dopaminergic innervation motivates characteristic behavior. These effects are explained by experimental studies of the dopamine system combined with some elegant computational modelling. The idea which comes from this work is that the dopamine system makes contingencies salient by *predicting reward* (Schultz et al. 1997; Egelman et al. 1998; Berridge and Robinson 2003; Smith et al. 2006). Once a contingency becomes salient in this way it becomes a motivational target irrespective of affective/valencing associations. This explains why in pathologies or subclinical forms of dysfunction subjects behave inappropriately. Salience has become unmoored from valence.

Note that it could be said, as versions of the canonical account do, that action in these cases is driven by the *anticipation* of an experience with positive or negative valence, perhaps as a result of conditioning. And in fact the somatic marker hypothesis endorses this view. The invocation of “anticipatory” SCRs (hypothesised to result from the activation of valencing systems) to explain choices in the IGT is a case of adapting this canonical view of emotion. However this does not explain how such anticipation occurs (O’Reilly and Munakata 2000; Smith et al. 2006) and why it should motivate. The canonical theory and the somatic marker hypothesis suggest that when a subject considers an action she tacitly experiences the consequent emotion. She samples the affective consequences of her action. And is thereby motivated.

The sampling account misconstrues the problem of motivation, which is to get an organism to perform an instrumental action, which is not intrinsically rewarding, for the sake of a distant reward which is. Canonical accounts suggests that the problem of motivating instrumental action can be solved by associating a current instrumental action with the affective consequences of its future outcome. Many people enjoy eating but dislike cooking and shopping for the ingredients. The canonical account suggests that such people go shopping (or animals go foraging) because shopping is associated with positive affect by the process of anticipating the positive affect produced by eating. In other words, if you can learn to attach the enjoyment of food to shopping and cooking you will be motivated.

The reward prediction framework gives a more satisfying explanation. The basic idea of the reward prediction framework is that an adaptive organism needs to allocate expensive cognitive resources to outcomes which are rewarding such as avoiding danger or predation or securing nutrition or reproduction. In the brain, signals from brainstem dopamine systems enhance activation in neural assemblies which represent potentially rewarding contingencies or control rewarding behaviour. Thus the dopamine system makes motivationally relevant contingencies salient to organisms prior to any experience of their actual affective valence. It *predicts* reward. Note that the concept of reward here applies equally to contingencies with positive and negative valence. It is rewarding to flee predation or pursue nutrition although the affective/qualitative states involved have opposite valence (fear and gustatory pleasure respectively).

The great breakthrough of computational modelling in this area is to show how a neurotransmitter system (the dopamine system) which influences almost all brain function through its diverse projections can be understood as performing the crucial cognitive role of reward prediction for the organism as a whole (Grace 1991; Egelman et al. 1998; Braver et al. 1999; Braver and Cohen 1999; Moore and Grace 1999;

Smith et al. 2006). In effect it co-ordinates the integrated functioning of distinct systems, such as attention, executive function and action-control, as well as short and long term memory by ensuring that they are jointly focused on salient contingencies. In effect mechanisms of salience ensure that representations of relevant contingencies dominate the global workspace. Initially this allows learning of the connections between a stimulus and the reward to be obtained by pursuing or avoiding it. *Once the association has been learnt* the organism will be motivated to pursue, cognitively or behaviourally, a salient contingency. The cognitive case is the allocation of cognitive resources such as attention and memory. The behavioural case is the initiation and continuation of appropriate action. Both these cases have pathological variations. Cognitive examples are inability to switch attention from a particular experience or thought so that it becomes the object of obsessive or compulsive patterns of thinking. The behavioural case is addiction, compulsive or obsessive behaviour.

The canonical explanation of pathological cases is that affective states, understood as valencing mechanisms, *bias* cognition or action tendencies by focusing them on a particular contingency and motivating an appropriate response. In other words valence explains salience and motivation. The correct aspect of the canonical account is that a well calibrated systems needs to focus on rewarding contingencies and install appropriate behavioural tendencies. The somatic marker hypothesis imports this feature of the canonical account, adding the idea that the mechanisms involved often function automatically, prioritising and ranking potential actions, before we are explicitly evaluate them.

However, the somatic marker hypothesis, like the canonical account, inverts the causal relationship between valence and salience and motivation. As pathological cases show salience is prior to valence and it is salience not valence which explains motivation. Mechanisms of salience which focus attention and executive function motivate cognitive and behavioural resource allocation.

The point is not that valence is irrelevant or not necessary to learning. It is very important that an organism allocates cognitive resources to contingencies with positive or negative consequences for the organism rather than those which are neutral or insignificant. Thus salience should track valence (positive or negative) *in the learning phase*. Valence is insufficient for learning, and, once a contingency is learnt, not necessary for retrieval of representations of salient contingencies or the association of those representations with motivational force. The installation and reinforcement of motivation are accomplished by the dopamine system. This is why, once a behavioural pattern has been installed, it is hard to overcome even when a contingency is no longer rewarding. Reversal learning involves more than reversing the valence of a contingency, it also requires the installation of a new pattern of motivation towards that contingency accomplished by the dopamine system.

One might say that these niceties do not unseat the somatic marker account precisely because the IGT focuses on a learning period in which valence can play a role in focusing cognition, enabling the dopamine systems to help install motivation toward appropriate contingencies. Thus we might expect inability to experience reward and punishment (evidenced by absent SCRs in the IGT) to lead to a failure to *learn* on the assumption that SCRs are indices of valence consequent on amygdala activation.

A system which experienced no patterned rewards would not install any patterned motivational tendencies.

However it would not follow that motivation, once installed, would require *reactivation* of those same systems. Representations of salient contingencies can be retrieved and play a motivational role irrespective of their valence. As we saw above salience and valence dissociate after the learning phase.

The somatic marker account is forced to deny this dissociation between salience and valence in retrieval and decision because it assumes that the interaction between salience and valence is the identical in both learning and retrieval and decision. This denial depends on the adoption of the canonical account of the relationship between salience and valence which in fact applies only as a general constraint on learning.

3 Learning from experience and ‘myopia for the future’

The somatic marker account suggests that when a subject is faced with a decision her VMPFC will swing into action to retrieve an implicit representation of the valence of the alternatives.

When subjects decide to select from a specific deck the neural activity pertaining to this information is signalled to the VMF cortices which in turn activate the amygdala (Damasio et al. 1991). This latter activity would reconstitute a state that integrates numerous conflicting instances of reward and punishment encountered with individual draws from that deck. If in the end negative somatic states outweigh the positive ones, an overall negative state is enacted and is indexed by the anticipatory SCRs we observed before the selection of cards from the disadvantageous decks. In turn, this influences the decision to avoid the deck under consideration (Bechara et al. 1999).

The nature of conscious awareness and its role on decision making is not something on which the somatic marker account concentrates but a full account of the role of prefrontal systems in explicit decision making suggests a different and larger role for the VMPFC in normal cognition and a different interpretation of the nature of the deficit introduced by damage.

We can see this by reflecting on the nature of the problem experienced by Bechara’s patients. It looks as if they are unable to *learn from experience* which decks are associated with reward and punishment. Whereas the normal subject gradually learns the punishment schedule, first implicitly then explicitly, both amygdala and VMF patients never do so. The amygdala patients because they cannot generate negative somatic markers, the VMF patients because they cannot retrieve and/or manipulate them.

This raises the interesting question of the way in which normal people apply the lessons of bitter experience when confronted with a decision. The most obvious case is to consciously remember the previous experience and assess it for information episodic or semantic relevant to the current decision. It is of course part of the somatic marker hypothesis that this is not what occurs in the IGT, but it is also part of the somatic marker hypothesis that both neurotypical subjects and VMPFC patients can, ultimately, give explicit explanations of the punishment schedules (50% of Bechara’s

patients reached the “conceptual” period but still performed disadvantageously on the task). So explicit semantic memory is presumed to be intact. The somatic marker hypothesis depends on the further assumption that implicit emotional memories are cognitively impenetrable. That is to say that knowledge gained through the explicit memory systems cannot alter the valence assignments accomplished by the amygdala, insula and somatosensory systems. Somatic markers bias explicit cognition not the other way around.

A competing hypothesis is that when explicit knowledge, both semantic and episodic is tested for in different ways it may be present. In the episodic case one recalls an experience, including its affective aspects. In the semantic case one knows facts without being able to retrieve the relevant experience. The distinction is important in the study of memory since the systems involved are dissociable (Keane et al. 1995). McLelland and Maia found *some* explicit knowledge of the punishment schedule was present within the first twenty rounds and suggested that poor selections reflected exploratory behaviour. Thus McLelland and Maia claimed that information about the decks is consciously available to subjects even though they may not be able to *fully* explain the patterns of reward and punishment (Maia and McClelland 2004, 2005). For example they might be able to say that some decks are better than others, and use that knowledge to make decisions, without being able to fully describe the punishment schedules.

A study by Tomb et al. divided 200 participants in the IGT into high, moderate and low performing groups based on results. Interestingly only in the high performing group were anticipatory SCRs as predicted by the somatic marker hypothesis. This suggests that some subjects (the moderate group) can learn the schedule *without developing anticipatory SCR*. It also suggests that the SCR in the high performing groups may be a *consequence not a cause of explicit knowledge*. It is also consistent with the idea that the high performers may have been quicker to develop the relevant explicit knowledge (Tomb et al. 2002). Of course the results do not disprove the somatic marker hypothesis precisely because what we are observing in case of anticipatory SCRs and good performance in the IGT is a *correlation whose causal structure remains a matter of hypothesis*.

In response Bechara et al. claimed that “the central feature of the SMH is not that non-conscious biases accomplish decisions in the absence of conscious knowledge, but rather that emotion-related signals assist cognitive processes even if they are non-conscious” (Bechara and Damasio 2005) Note that this represents a retreat from the claim that implicit learning scaffolded by valencing mechanism explains neurotypical performance on the IGT.

If in fact the somatic marker system functions hand in hand with explicit memory then the role of the VPMFC in decision making needs come reinterpretation. Apparently it functions not just to associate emotional information with implicit memory but also explicit memory. This makes sense of the intuitive case we described above. Someone making a decision typically recalls previous experience, including how she felt on that occasion, as a guide. The VMPFC might play a role here in helping to retrieve and link affective states to explicit memories. Note that explicitness might come in degrees of qualitative richness (Tulving 2002).

As a general explanation of decision making the somatic marker hypothesis suggests that decisions can be based on *either* explicit declarative reasoning such as expected utility calculation, automated emotional processes, or, in a well-calibrated mind on emotionally-reinforced declarative reasoning. However there is another alternative. Recent work on memory has concentrated on the role in decision-making of episodic memory. Episodic memories are those in which a subject recreates the experience of a previous episode in her life including its emotional associations. Episodic memories are qualitatively rich. Sensory perceptual and emotional details combine with a sense of self, of being present in the episode (known as autoneoesis) (Wheeler et al. 1997; Eichenbaum 1998; Levine et al. 1999; Klein and Loftus et al. 2002; Tulving 2002; Levine et al. 2004).

Recent accounts of episodic memory have explained it in terms of its contribution to global executive functions such as planning. The cognitive costs of a system which effectively recreates experience are high. Most animals make do with a combination of semantic memory (for facts, tested in humans by the ability to verbalise past information) and procedural memory (of habits or skills). In this respect most animals are like human infants who rely on a combination of semantic and procedural memory until late toddlerhood (Wheeler et al. 1997). There is in fact a hot debate about the extent to which other animals possess episodic memory. Scrub jays and chimpanzees have competencies for the use of stored information (such as returning to food caches) which have led some researchers to conclude that, not only are they retrieving information from previous experience, but are using episodic memories to do so (Clayton and Bussey et al. 2003; Zentall 2006).

From our point of view it does not matter whether the human capacity for episodic memory is unique. Our interest is the way humans integrate that capacity with other metacognitive capacities to control their behaviour. It is not a coincidence that the arrival of episodic memory coincides with the development of frontal cortex and more extensive connections between frontal and posterior areas. The greater executive abilities provided by frontal development include the ability to recreate experiences in the absence of the stimulus which caused them. This allows the toddler to begin the process of learning from experience and integrating memories of past events with linguistic and other forms of representation to make inferences about the world around her. In combination with the ability to inhibit automated responses, also conferred by prefrontal systems, access to episodic memory in effect frees the toddler from the stimulus-bound present. Development of the frontal cortex confers the ability to selectively inhibit and activate and sustained patterns of neural activity required for goal directed action (Knight 1999). The importance for cognitive control, planning and decision making cannot be overstated. Rather than actually undertake an action the child can inhibit an automatic action tendency and recall the consequences of previous efforts. In effect she can project herself backwards in time to plan for the future. (Suddendorf and Corballis 1997; Suddendorf 1997; Suddendorf and Busby 2003, 2005; Suddendorf and Corballis in press).

Thus the arrival of episodic memory is the ontogenetic marker of a phylogenetically recent capacity which while it may not distinguish humans from other animals contributes to our uniquely developed capacity for metacognitive control of behaviour.

However once we think of episodic memory this way, as part of a system for offline rehearsal of experiences, it seems that that the encoding and retrieval of *previous* experience is only part of the story. Because planning and decision making also involve information about the *future* as well as the past. And, as with memory, this information can be declarative or episodic. As well as factual knowledge about the future we can imagine ourselves living out future scenarios, rehearsing different possibilities. This form of imaginative rehearsal is known as *prospection*: the future- directed analogue of episodic memory (Suddendorf and Corballis 1997; Suddendorf 2006).

And indeed it seems that planning involves the seamless integration of both forms of projection of oneself in time, episodic memory and *prospection*. For this reason the ability to retrieve past episodes and imagine future ones and integrate the results with other forms of knowledge as part of planning have been baptised *mental time travel*. (Suddendorf and Busby 2003; Bayley et al. 2006; D'Argembeau and Van der Linden 2006; Steinvorh et al. 2006; Suddendorf and Corballis in press).

There is an emerging body of evidence that mental time travel does not exploit different systems, for episodic memory and imagination. Rather both memory and imagination involve the activation of relevant perceptual sensory and emotional systems in the absence of an environmental stimulus (Klein and Loftus et al. 2002; Wallis and Miller 2003). Thus the essential feature of mental time travel is the ability to create and recreate these experiences under voluntary control rather than via the presentation of an eliciting situation or object. The consequences for planning are enormous. Mental time travel gives humans an enormous database of situations and responses to them which can be safely rehearsed offline.

It is this voluntary, executive, aspect of mental time travel which is so important and which makes it dependent on maturation of the frontal systems. Episodic memory and imagination produce chains of associated imagery in daydreaming for example but daydreaming is not an executive capacity: the relevant imagery is produced automatically in patterns of ungoverned association rather than under voluntary control in the service of goal directed activity. The mind is a system of hierarchically ordered neural networks with activation spreading ceaselessly back and forth as information from the periphery is combined with centrally stored information to enable successful action. The role of the frontal systems is to regulate this activation, selectively maintaining some patterns of activity while inhibiting others to ensure that information relevant to currently represented goals dominates the global workspace (Knight 1999; Waltz et al. 1999; Miller and Cohen 2001; Wood and Grafman 2003). Thus experience is the result of a succession of transient activation patterns, selectively enhanced and maintained, distributed across prefrontal and posterior systems (Braver and Cohen 1999). Maturing frontal systems confer behavioural flexibility by allowing a creature to selectively inhibit activate and maintain patterns of neural activity which represent information episodic and semantic relevant to immediate and longer-term goals.

Of course this metaphor of central control need not suggest an homunculus. It is better to think of frontal systems as allocating resources which allow some activation patterns to be sustained against competition from other patterns. The winners in the competition dominate the global workspace. The representations they implement are become the focus of attention, working memory, planning and deliberation.

These facts tell us why the frontal systems are important to mental time travel. Other animals have memory systems which encode retrievable information about the past. Only humans appear to be able to retrieve that information and to deploy extremely flexibly it in novel combinations to create and inhabit future scenarios under high degree of *voluntary control*. And it is this voluntary, executive, aspect which is the essence of mental time travel (Kapur et al. 1995; Frith 1996; Knight 1999; Waltz et al. 1999; Miller and Cohen 2001; Wood and Grafman 2003).

The crucial point is not that frontal systems encode the relevant episodic information. Rather they enable its retrieval as part of a project which requires executive control. In fact episodic information is encoded in a distributed system. We might say that there is an episodic database encoded by specialised systems which can be accessed and manipulated by frontal systems involved in mental time travel. This is why “Patients with damage restricted to the frontal cortex are impaired when mentally required to re-experience a study episode in sufficient detail to recollect contextual information about that episode even though they can often report about the factual contents of the same episode.” (Wheeler et al. 1997, p. 342). It is not a coincidence that amnesic patients such as Alzheimer’s cases are deficient in planning and executive function. Their problem is not just loss of memory, but the consequences for executive action of a data-base restricted to stereotyped stimulus- driven routines.

If this is the case then mental time travel could be compromised at different levels. Damage to the episodic database *or* damage to the frontal systems which access and manipulate the data in executive processes. Classic cases of amnesia correspond to the former and frontal damage which leads to failures of executive control to the latter.

4 The ventromedial cortex and the Iowa gambling task

The somatic marker hypothesis was developed to explain the deficits of people with VMPFC damage in social deliberation and planning for the future, despite a preserved capacity for explicit *declarative* reasoning. In particular the failure to develop SCRs was seen as a crucial evidence of the failure to associate emotionally significant information with social or personal contingencies which results in “myopia for the future”.

One impressive result from research deriving from the somatic marker literature is confirmation of the involvement of neural systems identified by the somatic marker hypothesis as crucial. The VMPFC, amygdala, insula, somatosensory cortices, brainstem systems and hippocampal structures all contribute to decision making, and clearly the VMPFC has a supervisory role.

However, despite considerable agreement about the neural architecture implicated in myopia for the future the hypothesis itself remains controversial. There is far less agreement on a cognitive model which explains exactly what this distributed neural architecture is doing in the neurotypical case. Without that model a cognitive explanation of the way VMPFC damage produces “myopia for the future” is elusive.

It is for this reason that recent work on the role of frontal systems in mental time travel is suggestive. It seems likely that the normal case of decision-making involves mental time travel. That is to say the subject remembers what happened last time and

uses that information to create and inhabit a future scenario. She imagines what would happen in the future based on her past experience. Thus, in the IGT, faced with the choice from deck A she recalls previous experiences with that deck. In so doing of course she activates the relevant emotional associations which then become available for imaginative rehearsal of the consequences of choices i.e. she *imagines* choosing deck A, losing a large amount of money and generates an aversive affective response either in the process or as a consequence of the process of constructing the relevant implicit or explicit imagery.

Damasio's characterisation of the deficits of VMPFC patients as resulting from failure to link an implicit emotional response as with an explicit representations under-specifies the nature of explicit representation involved in decision-making. Explicit/declarative representation can be semantic, as when subjects perform cost benefit analysis by manipulating probabilities expressed as propositions. Or it can be episodic as when we remember or imagine an experience. Part of the problem such patients have with decision making may not just be the lack of emotional response but the inability to use past experience to genuinely inhabit the future. The VMPFC patient does not imagine or recall the consequences of her actions, in other word she cannot perform mental time travel.

This hypothesis while it implicates the same neural machinery does make some different predictions to the somatic marker hypothesis. Firstly it predicts that patients with VMPFC damage would perform poorly on a mental time travel task. This should be the case even if the patients are not amnesic, since such patients would have an intact experiential database but no ability to retrieve or manipulate it. Unfortunately the performance of selective VMPFC patients in mental time travel tasks has not been directly tested, although to the extent that the IGT or other decision-making tasks depend on mental time travel the results are consistent with the prediction. Nonetheless Levine et. al. report a case of a patient, M.L. with amnesia and VMPFC damage with deficient mental time travel, as one would predict, *and* decision making deficits characteristic of Self Regulation Disorder (Levine et al. 1999).

A second prediction is that patients with a deficit in mental time travel would perform poorly on the IGT irrespective of their ability to generate SCRs to disadvantageous decks.

Gutbrod et al. tested the performance of amnesic patients on the IGT. Clearly such patients are disabled with respect to mental time travel since they lack the experiential database. Interestingly, 9 of 11 patients performed at chance and did not show differential anticipatory SCRs to advantageous and disadvantageous decks. Furthermore the magnitude of anticipatory SCRs did not correlate with behavioural performance, leading to the conclusion that “acquisition of a behavioural preference—be it for advantageous or disadvantageous choices—depends on the memory of previous reinforcements encountered in the task, a capacity requiring explicit memory.” (Gutbrod et al. 2006, p. 1315). Of course the explicit memory involved may be semantic rather than as I am suggesting episodic, but that explicit memory proves necessary for learning is troubling for the somatic marker hypothesis.

The fact that amnesics with an intact capacity for SCR perform poorly in the IGT is a contribution to the debate over whether or not explicit or implicit learning is involved in IGT performance. It is also evidence in favour of a richer interpretation of the nature of

the deficits in VMPFC cases because it suggests that a deficit in mental time travel can lead to a deficit in decision-making. Of course the clearest supporting evidence would be a cases with a deficit in mental time travel and the ability to generate anticipatory SCRs who failed learn from experience in the IGT and/or patients with intact mental time travel but absent SCR who successfully learnt the IGT. Patients with Peripheral Autonomic Failure may be such a group since they do not generate SCR (Recall that the somatic marker hypothesis predicts that people unable to generate SCR will not be able to learn the IGT since they will be unable to associate explicit knowledge with implicit emotional markers). Heims et al. tested a group of such patients on the IGT and found that although they did not generate SCR they were able to learn the punishment schedules. They concluded that “social and emotional functioning is not critically tied to on going experience of autonomic arousal state” (Heims et al. 2004).

5 Conclusion

I have suggested that the somatic marker hypothesis is deficient in two ways. Firstly it uses a canonical account of emotions which explains salience and motivation in terms of valence. Within this framework SCRs are interpreted as evidence of the role in decision making of valencing systems whose activation is required to produce suitable motivation. If, in fact, valence and salience/motivation dissociate this interpretation of the role of valencing systems is not persuasive. It suggests that SCRs, insofar as they are evidence of the activation of valencing systems need not be essential to performance in tasks such as the IGT which require a subject to represent future alternatives in order to *motivate* goal-directed behaviour. Of course if that is the case we need an explanation of the fact that (i) anticipatory SCRs are typically correlated with learning (ii) absence of SCR following VMF damage is correlated with “myopia for the future” The correlation is explained by the nature of the association between salience and valence in the learning period, but the correlation between SCR and decision in the IGT is not causal. The mental time travel hypothesis is an alternative hypothesis which might explain “myopia for the future”.

Of course the somatic marker hypothesis does not rest on the interpretation of the SCR data alone but on a wider theory of the role of the VMPFC in decision making: namely that its role is to associate implicit emotional memories with explicit representations of future outcomes. I have endorsed the general idea that the VMPFC does play a role in coordinating the retrieval and manipulation of information, including affective information, required for decision-making however I gave an alternative picture of prospection and hence of “myopia for the future”.

On the account I proposed normal prospection involves mental time travel. VMPFC patients cannot travel in time, not because they have no neural basis for the encoding of experiential memory, but because the VMPFC is required to access and manipulate it imaginatively in the service of goal directed behaviour. This is why they can perform logical cost benefit analysis of situations but not be motivated to behave appropriately: they cannot re-create and occupy a scenario which embodies the relevant personal information.

In contrast the somatic marker hypothesis suggests that VMPFC patients cannot associate options produced by cost benefit analysis with implicit somatic markers. The picture of cognition limits the role of experience to the input of information subsequently encoded and retrieved in semantic form.

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