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”Challenging the Somatic Marker Hypothesis: Are somatic markers indicators of risk?“

Verfasserin

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1. Introduction

Decision-making under uncertainty and risk has gained high attention in recent decades (Damásio, 1994; Gigerenzer, 2007; Glimcher, 2003). For example, it has been argued that indeterminate human behaviour can be at least partially explained through probabilistic tools (Glimcher, 2003). Glimcher (2003) specifically assumes that the findings obtained in the quite nascent field of neuroeconomics are able to contribute to a more thorough understanding of behavioural and neuronal processes that involve indeterminate processes, like decision-making under uncertainty and risk. Our investigation of impaired human decision-making in Bechara's Iowa Gambling Task (IGT) aims to incorporate the theoretical assumptions of Bechara's proposed neurocognitive model of human decision-making (Bechara, 2005), current empirical findings in neuroeconomics (e.g., representation of subjective reward value in human neocortex und subcortical structures), recent findings in neurobiology and psychophysiology, and behavioural experimental data into a unified picture of how decision processes under uncertainty and risk might work. Furthermore, our work aims to investigate the importance of risk aversion and risk seeking behaviour, respectively, in healthy human subjects in the IGT, since huge differences in control data imply such an involvement. The theoretical and empirical findings (from three different computational cognitive models of IGT performance) are fitted into a refined version of the Expectancy-Valence Model (Busemeyer and Stout, 2002) which aims to give a thorough and parsimonious account for the inconclusive findings in the card drawing behaviour of healthy human subjects: It still needs to be shown that healthy human subjects decide advantageously in a consistent manner in the IGT. Furthermore, the impaired performance observed in ventromedial prefrontal cortex

(VMPFC) patients is discussed. The first eight chapters deal with the empirical findings obtained so far, inconsistencies, and potential flaws in the original interpretation of IGT results. Chapter 9 proposes a solution to these problems and describes the refined version of the Expectancy–Valence Model. Finally, chapter 10 deals with the conclusions and discusses directions for future research within the IGT paradigm.

2. Experimental setup of the Iowa Gambling Task

The IGT (Bechara et al., 1994) was designed to study the role of the VMPFC (and later also the amygdala) in real-life decision-making within a laboratory setting. Individuals with lesions in this cortical area display abnormal behaviour in their social and emotional lives (e.g., ignoring of future consequences of decisions, impulsiveness, flattened emotionality), although their intellectual abilities remain intact.

In this task, participants have to choose cards from four decks named A, B, C, and D. The players are instructed to decide so as to minimize their losses and maximize their gains. The seed capital for the game is a loan of $2000 of play money. Turning a card leads to an immediate monetary gain of $100 for decks A and B, and of $50 for decks C and D. However, losses are incurred in an unforeseeable manner, which are large for decks A and B (the “bad” decks) and small for decks C and D (the “good” decks). Apart from the magnitude of the losses, another difference is that losses are more frequent for decks A and C than for decks B and D. The net gain is the same for decks A and B (minus $25 per trial), and also the same for decks C and D (plus $25 per trial). Each deck consists of 40 cards. Thus, the players have to be mindful of the possibility of running out of cards.

Table 1: Payoff schedule of the original IGT. Table adapted from Colombetti (2008, Table 1, p. 59).

	Deck A (+100)	Deck B (+100)	Deck C (+50)	Deck D (+50)
1				
2				
3	-150		-50	
4				
5	-300		-50	
6				
7	-200		-50	
8				
9	-250	-1250	-50	
10	-350		-50	-250
11				
12	-350		-25	
13			-75	
14	-250	-1250		
15	-200			
16			-25	
17	-300		-75	
18	-150			
19			-50	
20				-250
21		-1250		
22	-300			
23				
24	-350		-50	
25			-25	
26	-200		-50	
27	-250			
28	-150			
29			-75	-250
30			-50	
31	-350			
32	-200	-1250		
33	-250			
34			-25	
35			-25	-250
36				
37	-150		-75	
38	-300			
39			-50	
40			-75	

To maximize the overall monetary gain, participants have to turn cards predominantly from decks C and D. It is important to note that the players are not aware when a loss

will be incurred and are not able to calculate exactly the net gain of the four different decks. Furthermore, the players are not instructed as to how many cards they will have to turn until the game stops (the game stops after 100 cards have been turned). The sequence of cards is identical for all participants (see Table 1) and the available time for deciding which card to turn next is self-determined.

3. The Somatic Marker Hypothesis

According to Damásio and Bechara (Bechara and Damásio, 2005; Damásio, 1994) there are two possibilities how somatic markers are generated. First, somatic markers are theorized to be emotional responses of the body proper. If an emotionally arousing experience triggers a somatic state, then a pattern for it is stored in memory (i.e., generation of affective memories). Any stimulus that evokes these emotive memories connected to the experience re-enacts this pattern and produces a somatic state equivalent to that triggered by the original experience (i.e., somatic markers generated via the body loop). Second, activation of neural representations of somatic states in the insula, somatosensory cortices, and the brainstem can induce changes in neurotransmitter release which simulate somatic states "intra-cerebrally" (i.e., somatic markers generated via the as-if body loop). In these cases somatic states are not re-enacted in the body[1].

Damásio (1994) assumes that somatic markers are activated before any kind of cost-benefit analysis of the premises takes place and before problem-solving reasoning processes occur, and lead to a pleasant or unpleasant *gut feeling* (when conscious). Thus, somatic markers can bias or adapt the decision process under uncertainty and

[1] Interestingly, this argument is reminiscent of the idea of advance modelling in motor control (Wolpert and Ghahramani, 2000).

risk. Furthermore, he argues that somatic markers not only provide relevant emotional information in an experimental setup like the IGT (Bechara et al., 1994) but also can limit the generally huge range of possible alternatives in real-life decision-making. In this way, somatic markers can be seen to help constrain the search space taken into consideration. Evans proposes a similar idea in his Search Hypothesis of Emotion (Evans, 2002). Accordingly, De Sousa also states that emotion is "one of Nature's ways of dealing with the philosophers' frame problem[2]" (De Sousa, 1987, p. 195). In real-life decision-making, somatic markers generated from secondary emotions[3] can support a prediction of the outcome of emotionally similar conditions and can narrow down the range of available alternatives for (re)action. Only after the options are confined do cost-benefit analyses and deduction processes take place. Thus, the restriction of the search space can help increase the accuracy and efficiency of the decision process. *Positive* somatic markers can help overcome immediate negative events in favour of future rewards. Complementarily, *negative* somatic markers can aid avoidance of immediate positive events in order to prevent future losses.

4. Experimental results from the Iowa laboratory

To test the SMH, Bechara and colleagues (1994; 1996; 1997) compared the behaviour of healthy individuals to patients suffering from bilateral damage of the VMPFC in the IGT. They compared the behavioural performance (i.e., how many cards are selected from the "good" decks compared to the "bad" decks), the generation of

[2] The frame problem refers to the problem of constraining the beliefs that need to be updated as a consequence of action.

[3] Secondary emotions are affective reactions that have already been associated to particular situations, objects, and persons by Hebbian learning (Damásio, 1994, pp. 134-139).

anticipatory skin conductance responses (SCRs)[4], and the subjects' knowledge of the game structure. In the series of experiments conducted by Bechara and colleagues (1996; 1997), all participants showed normal SCRs *after* experiencing punishment and reward, respectively (i.e., normal reward/punishment SCRs mediated via the amygdala system; see Chapter 5.1. The impulsive system). After turning 10 to 50 cards and thus encountering some of the penalties, normal controls began to generate SCRs *before* turning cards from the "bad" decks (Bechara et al., 1997). Simultaneously, they also began to avoid turning cards from these decks and shifted their initial preference for the "bad" decks towards the "good" ones. In contrast, VMPFC patients failed to shift their initial preference for the "bad" decks towards the "good" ones. Furthermore, while VMPFC patients developed punishment/reward SCRs, they did *not* generate anticipatory SCRs *before* turning cards from the "bad" decks, which suggests that they fail to produce anticipatory somatic markers. Bechara and colleagues (1997) concluded that these *anticipatory* SCRs act as *negative* somatic markers to support the decision process under uncertainty and risk. Interestingly, VMPFC patients persisted in turning cards from the "bad" decks, although the declarative knowledge that the "good" decks were more advantageous was mostly present in the conceptual period (after drawing the 80th card) of the game[5].

[4] A skin conductance response (SCR) is an often used and well defined measure of emotional arousal. It is usually recorded together with other autonomic parameters like heart rate, eye movements, respiration rate, skin temperature, muscle tension, and blood pressure (Stern et al., 2001).

[5] The IGT can be divided into four periods: (1) The pre-punishment period before the participants encounter their first loss (cf. *exploration* phase of a game), (2) the pre-hunch period, where they still have no notion of what is going on in the game (cf. *exploitation* phase of a game), (3) the hunch period, where they express certain preferences for the four different decks, and (4) the conceptual period, where verbalized knowledge is available (Bechara et al., 1997, p. 1294).

Bechara and colleagues (1997) conclude that emotive memories associated with the actual situation trigger characteristic somatic states through the VMPFC system (see Chapter 5.2. The reflective system). These somatic states are supposed to bias subsequent cost-benefit analyses and reasoning processes. It is important to note that impairments in the IGT only arise when the *right* hemisphere is affected (Manes et al., 2002) and that VMPFC patients generally show normal intellectual abilities, including memory, attention, and other cognitive functions. To summarize, Bechara and colleagues (1997) suppose that *overt* reasoning processes — including the recall of available knowledge of a given situation and deriving behavioural strategies to handle it — are preceded by *covert* emotional biases.

5. A neurocognitive framework for the Somatic Marker Hypothesis

Damásio's SMH can be seen as a reformulation and refinement of the well-known James-Lange Theory. The American psychologist William James and the Danish psychologist Carl Lange independently proposed (James, 1884; Lange, 1885) that emotions are cognitive responses to information received from the periphery (i.e., from somatic signals). Similarly, Damásio (1994) assumes that somatic markers as measured through anticipatory SCRs are bodily signals that can bias cognitive responses. For example, they are supposed to guide the card drawing behaviour of participants in the IGT (Bechara et al., 1997). If these important somatic signals are missing, the decision process of the subjects is impaired, since calculation of the expected value is *not* easily possible in the IGT and thus participants have to rely on their gut feelings (if conscious) to decide from which deck to draw the next card (Bechara et al., 1997).

Interestingly, substance dependent individuals (SDIs) show impairments in the IGT that are highly similar to those of VMPFC patients (Bechara and Damásio, 2002; Bechara et al., 2002). They seem oblivious to the ultimate outcomes of the IGT (as well as the future consequences of drug intake in their real lives) favouring immediate rewards. Furthermore, SDIs fail to exhibit anticipatory SCRs (i.e., somatic markers) when turning cards from the "bad" decks. Complementarily, recent imaging studies (Volkow et al., 2004) show abnormal activation in the orbitofrontal cortex (including the VMPFC) in SDIs (i.e., decreased activity during drug withdrawal and increased activation during drug exposure). Thus, an altered functionality of the VMPFC in addicts (at least in cocaine addicts) seems plausible. Furthermore, other imaging studies also suggest hyperactivity in the amygdala, and associated efferent and afferent connections in cocaine addicts (Childress et al., 1999). In a modified version of the IGT with high immediate punishment and long-term gain in the "good" decks and low immediate punishment and long-term loss in the "bad" decks, SDIs (alcohol addicts (n=17), cocaine/crack addicts (n=14), and methamphetamine addicts (n=8)) showed the following responses: 64% of SDIs[6] who were impaired in the original IGT were not impaired in this alternative version (Bechara and Damásio, 2002; Bechara et al., 2002). Furthermore, this subgroup showed higher reward SCRs compared to normal controls and VMPFC patients in relation to the "good" decks. The anticipatory SCRs towards the "good" decks were not significantly different from normal controls, while VMPFC patients failed to show anticipatory SCRs towards both types of decks

[6] To our knowledge there is no empirical evidence that different kinds of addiction (i.e., alcohol, cocaine, and methamphetamine) lead to distinct behavioural and psychophysiological responses within the IGT paradigm.

(Damásio et al., 2002). These results may indicate a hypersensitivity to reward, enabling SDIs to perform advantageously on this version of the IGT.

The SMH may thus provide a neurocognitive framework to understand the processes operating in the cognition of SDIs (Bechara, 2005). These processes fail to overcome immediate gains in order to prevent negative future consequences (monetary loss in the IGT and loss of important social relationships, of social standing, and financial losses in real life). The VMPFC and the amygdala are both assumed to be key structures for the triggering of the relevant somatic states, with the amygdala responding to environmental stimuli and the VMPFC responding to already associated memories, knowledge, and cognition. The functional theory proposed in Bechara (2005) mainly distinguishes two interacting (or competing) systems (see Figure 1): First, there is an impulsive system, which is mediated by subcortical structures like the amygdala and the striatum, which trigger the somatic states of *immediate* events (positive and negative, respectively). Second, there is a reflective system, represented by the VMPFC, brainstem nuclei (e.g., nucleus parabrachialis), and somatosensory cortices (e.g., insula and somatosensory cortex), which trigger the somatic states associated with expected *future* outcomes.

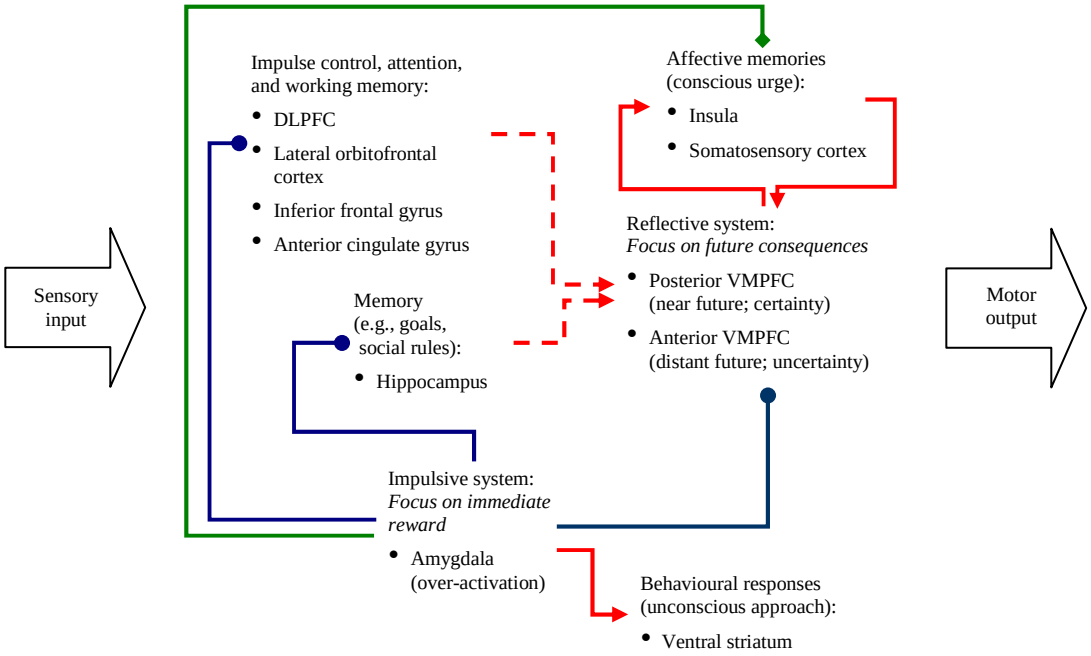

Figure 1: Derived neurocognitive model of proposed functional system interactions. An over-activation of the impulsive system can bias or outweigh the influence of the reflective system in decision-making. Red lines (no line marker) indicate excitatory connections; dashed red lines indicate modulated excitatory influences; blue lines (filled circle ● line marker) indicate inhibitory connections; green lines (diamond ◆ line marker) indicate modulatory connections.

5.1. The impulsive system

The basolateral nuclei of the amygdalae receive information from different sensory modalities. Afferents project from the basolateral nucleus, the input region of the amygdala, to the central nucleus, the major output region of the amygdala. Bidirectional pathways project from the central nucleus to the brain stem, to the dorsal medial nucleus of the thalamus, to the anterior cingulate gyrus of the cortex, and to the orbitofrontal cortex (the latter two are involved in the *conscious* perception of emotion) through the ventral amygdalofugal pathway. Furthermore, bidirectional projections connect the central nucleus with the lateral hypothalamus, the bed nucleus of the stria terminalis, and the nucleus accumbens through the stria terminalis (i.e., one of the two major dopaminergic pathways) (Kandel et al., 2000). Thus, the

amygdala might be able to serve a dual purpose, influencing both the autonomic/reflexive *and* the cognitive/reflective components of emotion.

According to the functional theory proposed in Damásio (1994) and Bechara (2005) the amygdala functions as the trigger of the impulsive system. The amygdala associates sensory stimuli with their immediate rewarding and aversive properties, respectively. Emotional responses activated by the amygdala are relatively fast and transient. The amygdala responds by activating visceral motor structures (i.e., hypothalamus and brainstem) which lead to changes in the peripheral nervous system. Furthermore, the amygdala activates action-related structures (i.e., striatum, periaqueductal grey, and brainstem) that mediate affective facial expressions and approach/avoidance behaviour. Patients with bilateral lesions of the amygdala show similar behavioural results in the IGT as VMPFC patients (Naqvi et al., 2006). They fail to produce anticipatory SCRs, which is also the case in VMPFC patients, but — in contrast to VMPFC patients — amygdala patients also fail to show SCRs in response to rewarding and punishing events. The latter indicates that the ability to form immediate emotional responses to objects, persons, or situations is also disturbed in amygdala patients.

5.2. The reflective system

The ventral amygdalofugal pathway and the mesocorticolimbic pathway (i.e., the second major dopaminergic pathway) both play important roles in consolidating the associations between sensory stimuli and their rewarding and aversive properties. According to the proposed functional theory (Bechara, 2005; Damásio, 1994), the VMPFC is supposed to trigger these emotive memories (i.e., representations of affective neuronal patterns already associated with a certain situation, object, or

person) which can be either real or imagined. Thus, the bodily states triggered by the VMPFC are emotional reactions in response to affective memories which are similar to an actual confronted situation (person or object).

Conscious gut feelings triggered through the reflective system are represented within the insula, somatosensory cortex, and the anterior cingulate gyrus (within the temporal association cortex), whereas *unconscious* responses are represented within the mesolimbic dopaminergic system (especially, the ventral striatum and the nucleus accumbens). Both can bias the decision process either with or without specific feelings of desire or aversion for the option in question (Naqvi et al., 2006). Interestingly, Pineda (2008) recently suggested that the somatosensory cortex should be regarded as part of an extended mirror neuron system because of its specific functional and anatomical characteristics. Furthermore, he assumes that this extended mirror neuron system, comprising the amygdala, insula, and the somatosensory cortex, is important for the understanding of affective facial expressions as well as for empathy in humans. Thus, the mirror neuron system might also contribute to the development and activation of emotive memories (cf. simulation theory).

5.3. Further functional systems

In addition, several other systems are involved in decision-making. They are briefly described in the following sections, and their functional role is also discussed.

5.3.1. Impulse control and response inhibition

According to Bechara's (2005) neurocognitive account of decision-making under uncertainty and risk, the ability to inhibit intruding memories and thoughts is mediated through the lateral orbitofrontal and dorsolateral prefrontal cortex (including

the inferior frontal gyrus). Perseveration errors and problems in shifting attention are cited as typical symptoms in patients with lesions in these cortical areas. The most posterior part of the VMPFC, including the anterior cingulate gyrus and the basal forebrain, are identified as critical structures in impulse control — the ability to suppress autonomic responses. Thus, these systems are taken to have to be intact in order to be able to succeed in complex behavioural tasks, like the IGT.

5.3.2. Attention and working memory

The dorsolateral prefrontal cortex and the hippocampus are essential structures for working memory and attention as well as for memory formation and consolidation (Kandel et al., 2000). The ability to attend to stimuli and to memorize the history of former events is essential for the performance of almost any cognitive or behavioural task. It could be demonstrated that decision-making in the IGT relies on an intact working memory system, but that working memory is independent of deficits in decision-making (Bechara et al., 1998).

5.3.3. Motivation and behaviour

The striatum, especially the ventral-striatal system (dopaminergic nuclei), is important for mediating the appetitive phase of acting (Panksepp, 2005). The alteration of approach/avoidance behaviour is supposed to occur at an *unconscious* level, whereas the formation or triggering of feelings by the VMPFC in the insula, the somatosensory cortex, and the anterior cingulate gyrus is assumed to be *conscious* (Bechara and Damásio, 2005; Naqvi et al., 2006).

5.4. Interaction of the functional systems in the case of addiction

The proposed functional theory provides a theoretical framework explaining the "myopia for future consequences" (Bechara, 2005, p. 1458) observed in SDIs as well as in VMPFC patients. In SDIs, the reflective system is supposed to get suspended through hyperactivation of the impulsive system. It is unclear whether the over-activation of the amygdala and its efferent and afferent connections is the cause of addictive behaviour or rather its consequence. Nevertheless, Bechara (2005) clearly assumes a causal role of a weakened reflective system (through both environmental and innate factors) in addiction.

Over-activation in the impulsive system, with the amygdala acting as the major trigger, can lead to neuropharmacological changes (especially in the release of dopamine and serotonin, respectively) within the different afferent and efferent projections (see Figure 1). First, the ventral striatum may be affected, leading to an unconscious bias in approach behaviour towards drug-related stimuli. Imaging studies already have indicated that cocaine addicts show altered activity in the amygdala (Childress et al., 1999). Second, alterations in the activity of the insula and the somatosensory cortex (especially in the right hemisphere) can lead to modulated affective neuronal representations of persons, situations, or objects. Recent results show that individuals who are suffering from insula damage show a higher success rate in remaining abstinent from smoking compared to individuals with an intact insula (Naqvi et al., 2007). Thus, the insula may be implicated in the subjective utility of drug reward or conscious urge component of addiction (at least in smoking). Third, neuropharmacological changes in the lateral orbitofrontal cortex, the inferior frontal gyrus, the dorsolateral prefrontal cortex, the hippocampus, and the anterior cingulate

gyrus can lead to difficulties in impulse control, memory (working memory and long-term memory), and attention, respectively (Bechara, 2005).

It is important to note that in decision-making (e.g., whether to use a drug) both positive and negative somatic states arise, with the stronger one biasing the final decision (cf. winner-takes-all). Consequently, if there is an over-activation in the impulsive system, this activity can become so strong that it can bias or even outweigh the activity triggered by the reflective system. Thus, ignoring probable future consequences of regular drug intake and focusing on the immediate rewarding properties of the situation can be the result.

Addicts tend to prefer smaller over larger and sooner over later rewards (Bechara, 2005). Hence, the time component in drug use seems to be important and has to be considered. Interestingly, the VMPFC can be divided into two sections, with one area (posterior) responding to near future consequences, and the other (anterior) to distant future consequences (Bechara and Damásio, 2005). Furthermore, the same two sections respond differently to the degree of certainty of options, with the posterior VMPFC getting active when the options are quite sure and the anterior part of the VMPFC responding to highly uncertain options. As indicated by imaging studies, (cocaine) addicts show abnormal activations in these neural structures dependent on the level of (un)certainty to get access to their preferred drug at the end of the experimental session (Bechara, 2005).

Finally, one should keep in mind that there are large interindividual differences in SDIs. Addicts that act similarly to VMPFC patients in the IGT show higher impairments in real-life decision-making compared to addicts (although the minority) that show IGT results lying in between normal individuals and VMPFC patients. Drug

takers that behave like normal individuals in the IGT show almost no impairments in their social and financial realms (Bechara, 2005).

6. Challenging the Somatic Marker Hypothesis

The SMH has been called into question by several different laboratories. The most important arguments against the original interpretation of IGT results are discussed and critically evaluated in the following sections.

6.1. Is the proposed bias really unconscious?

A study by Maia and McClelland (2004) shows that participants in the IGT have knowledge of the advantageous strategy before they apply this knowledge to the decision as to which card to draw next. This is in direct contrast to the original interpretation of somatic markers as covert signals, proposed by Bechara and colleagues (1997). It is unclear why the participants do not apply their knowledge immediately. According to the authors, possible answers might be that this time lag is due to exploration of the decks or risk seeking tendencies. The authors infer from their results that there is no need to consult unconscious or covert biases to explain the shift in behaviour towards the "good" decks: The participants eventually act advantageously because they *know* the correct strategy. Maia and McClelland (2004) suggest that there are several reasons to assume explicit reasoning. First, the time to decide which card to draw next is *self-paced*. Second, rewards and punishments are presented in an *explicit numerical way*, and third, the approximate characteristics of the game are relatively *easy to recognize*.

Recent reports (Bechara et al., 2005; Naqvi et al., 2006) assume that somatic markers can be either conscious or unconscious, i.e. conscious when leading to certain gut

feelings or unconscious when such a feeling component is missing. In their early studies, Bechara and colleagues (1994; 1997) supposed that the influence of somatic markers in the IGT acts at an unconscious level only, in spite of the fact that their participants were *not* asked whether they did experience a certain feeling before turning the cards or not. In this regard, it must be noted that Damásio (1994) already stated that somatic markers can be either conscious or unconscious. However, when knowing the difference between the "good" and the "bad" decks in the conceptual period of the game, VMPFC patients continue to draw cards from the "bad" decks (Bechara et al., 1997). This finding is taken to clearly suggest that an important support mechanism or an important component in decision-making (e.g., the somatic marker) is missing in these patients.

6.2. What exactly do anticipatory SCRs encode?

6.2.1. Anticipatory SCRs: Indicators of risk?

A potential objection to the original interpretation of anticipatory SCRs before drawing cards from the "bad" decks as *negative* somatic markers comes from Maia and McClelland (2004). The authors suggest that anticipatory SCRs reflect the higher amount of gains and losses in the "bad" decks. In agreement with this hypothesis, Tomb and colleagues (2002) found that in an alternative version of the IGT, in which the "good" decks are associated with higher amounts of both reward and punishment (see Table 2), the participants showed higher SCRs before turning cards from the "good" decks. Thus, it seems that anticipatory SCRs do *not* indicate the "goodness" or "badness" of the respective deck but rather the *magnitude* of gains and losses or the *riskiness* of the different decks. They assume that "card selection is driven by long-term consequences, whereas anticipatory SCRs are driven by the immediate act to be

performed, independently of the positive or negative long-term value of the decision" (Tomb et al., 2002, p. 1103).

In a reply, Damásio and colleagues (2002) argue that the study of Tomb and colleagues (2002) is conceptually flawed because the "good" decks in their alternative version provide *both* higher immediate rewards and long-term gains, whereas their "bad" decks lead to low immediate gains and long-term loss. Thus, *no conflict* between the two decks arises. Their interpretation is that "the immediate tendency to prefer the higher reward does not need to be opposed in order to achieve" (Damásio et al., 2002, p. 1104). Furthermore, they assume that somatic markers can be both positive and negative (Damásio, 1994) and that the function of somatic markers is to help *adapting* the decision process under uncertainty and risk. But if there are both positive *and* negative somatic markers, what is it that *distinguishes* them? It would be useful to include other autonomic parameters that are known to differentiate between positive and negative affective states (Vernet-Maury et al., 1999) in future studies.

Bechara and colleagues (2000; 2002) experimented with their own modified version of the IGT, which involves a reversed schedule of punishment and reward but maintains the important component of *conflict* (i.e., high immediate punishment and long-term gain in the "good" decks and low immediate punishment and long-term loss in the "bad" decks). They found SCRs before turning cards from the "good" decks in normal controls and interpret this finding as the triggering of *positive* somatic markers. VMPFC patients performed disadvantageously on this version of the IGT also.

To summarize, VMPFC patients seem to lack an important physiological signal, i.e. the somatic marker, which can help adapt the decision process under uncertainty and risk. Although it cannot be ruled out that anticipatory SCRs also represent higher

variance decisions, it seems implausible to reduce them to an indication of magnitude. The failure to produce this kind of emotive signals coincides with disadvantageous decision-making in the IGT, so a certain role in *guiding* the decision process seems likely, even if no causal role can be inferred (see Chapter 6.3. Causality).

Table 2: Payoff schedule in the original IGT (above) and in the alternative version of the IGT (below). Table adapted from Tomb and colleagues (2002, Figure 1, p. 1103).

Deck	**Type**	**Gain every 10 cards**	**Loss every 10 cards (# punishments)**
A	Bad	$1000	$1250 (5)
B	Bad	$1000	$1250 (1)
C	Good	$500	$250 (5)
D	Good	$500	$250 (1)

Deck	**Type**	**Gain every 10 cards**	**Loss every 10 cards (# punishments)**
A	Good	$2250	$1500 (5)
B	Good	$2250	$1500 (1)
C	Bad	$250	$1000 (5)
D	Bad	$250	$1000 (1)

A recent study by Fum and colleagues (2008) reports that the *frequency* of punishments can explain the card drawing behaviour in the IGT paradigm. They found a *preference* for deck B (one of the "bad" decks) and deck D (one of the "good" decks), both of which involve *fewer* but high magnitude punishments. These results are at odds with the SMH. Since they did not record any psychophysiological signals, nothing can be inferred about the role of anticipatory SCRs in their investigation. It would be interesting to study whether anticipatory SCRs would align with the participants' card drawing behaviour in their experimental setup. At the moment, it

remains largely unclear why their subjects did not draw more cards from the "good" compared to the "bad" decks. We think that either another factor is contributing to the observed disparate behaviour of their participants or an important factor inherent to the original IGT paradigm is missing in their experimental setup. The authors argue that in the original IGT the influence of punishment frequency is intertwined with the expected value (i.e., the net gain) of the respective deck. They further assume that this could explain the "prominent deck B" phenomenon[7] and the fact that some healthy individuals show decision patterns reminiscent of those produced by VMPFC patients in the IGT (Bechara et al., 2002). However, it could also be the case that punishment frequency is an important additional factor that possibly explains IGT results better than the SMH. But at the moment we doubt that this is a valid inference, especially without further experimental evidence to back up this claim. We think that punishment frequency should be regarded as a possible additional factor that might contribute to the overall perception of the *riskiness* of decks together with the magnitude of rewards and punishments involved[8]. However, Fum and colleagues (2008) further found that participants choose significantly more cards from decks B and D when the net gain is held constant between all four decks, with punishment frequency being the only distinguishing factor between the four different decks.

To summarize, it seems that at least three dissociable influences contribute to the individual performance of participants in the IGT: (1) The subjective reward value[9], (2) the magnitude of rewards and punishments involved, and (3) the frequency with

[7] It could be shown that healthy participants tend to draw more cards from the disadvantageous deck B than from the advantageous decks C or D (Toplak et al., 2005; Wilder et al., 1998).

[8] Furthermore, we assume that separate sensitivities to reward and punishment play an important role in the decision strategy of healthy human subjects as well as VMPFC patients in the IGT.

[9] The influence of subjective reward values to the decision process is discussed in detail in Chapter 8. Neural representation of subjective reward value.

which rewards and punishments appear in the IGT. Interestingly, ongoing work in our laboratory was able to show that the results of Fum and colleagues (2008) may solely depend on the altered payoff schedule they used (Rakovský, 2009, unpublished work). Rakovský (2009) found that the decision strategy in the IGT paradigm is highly sensitive to the payoff matrix. The author employs computational modelling based on a refined ACT-R model of IGT performance originally described in Fum and Stocco (2004) to back up this claim and found that the implemented card drawing algorithm behaved exactly like healthy human subjects in the original IGT and in the IGT with the altered reward/punishment schedule (see Figure 2) used by Fum and colleagues (2008).

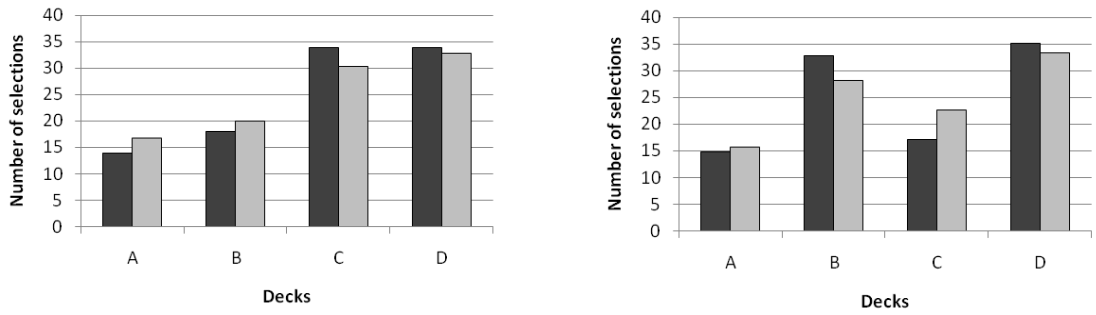

Figure 2: Reproduction of the card drawing behaviour of healthy human subjects (dark grey) in the original IGT (left panel) and the altered version of the IGT (right panel) by a computational model (light grey). Figure reproduced from Rakovský (2009, Figure 1 and 4, pp. 7-8).

However, the interpretation of these results is difficult at the moment. The changes in card drawing behaviour might be explained by the following different facts: (1) The decks are not restricted to 40 cards; participants can draw as many cards as they want from any single deck. (2) The punishments in decks B and D arise much earlier (card number 4) than in the original IGT (cards 9 and 10). (3) The rewards vary in

magnitude[10] and thus require more cognitive capacity to memorize; it could therefore be argued that the participants experience more difficulties in developing a hunch about the overall benefit or riskiness of the four different decks. It would be quite interesting to determine how the subjects react psychophysiologically (i.e., measurement of anticipatory SCRs) in the altered version of the IGT proposed by Fum and colleagues (2008). However, if seemingly minor changes to the reward/punishment schedule produce dramatically different behavioural results, what consequences for the SMH and in particular the IGT paradigm should be inferred? We assume that the frequency of rewards and punishments together with their magnitude contribute to the perceived *riskiness* of the four different decks and that risk perception could be captured by the individual's separate sensitivities to reward and punishment (see Chapter 7.3. Altered sensitivity to rewarding and punishing events).

6.2.2. Response to feedback

Another objection to the original interpretation of IGT results comes from a study by Suzuki and colleagues (2003). They found that participants who show higher feedback SCRs tend to have a better learning curve on the IGT, which means they choose the "bad" decks (i.e., decks A and B) less frequently in late trials than in early trials (Suzuki et al., 2003). Furthermore, feedback SCRs are larger following punishments and following selections from "bad" versus "good" decks (Suzuki et al., 2003). Thus, it could be inferred that mastering the task is related to experienced feedback rather than to emotional farsightedness as endorsed by Damásio (1994).

[10] The rewards vary between $80 and $100 in the disadvantageous decks and between $40 and $60 in the advantageous decks. Furthermore, the punishments in deck B vary between $1210 and $1290.

Moreover, Crone and colleagues (2004) found larger SCRs and larger heart rate (HR) deceleration following punishment compared to reward, especially with decks that involve a low frequency of punishment (and thus large penalties). However, these differences could not be used to distinguish between good, average, and poor performers (healthy subjects) in their variant of the IGT. Anticipatory heart rate slowing and skin conductance levels were higher before choosing cards from the disadvantageous decks, but only for good performers. This latter finding provides some support for the SMH. However, the empirical results obtained in different laboratories reveal rather inconsistent results.

6.2.3. A marker of post-decision emotional state

It can be argued that anticipatory SCRs may reflect expectancies about reward and punishment *after* a decision has been made. Amiez and colleagues (2003) report empirical evidence for such an interpretation. They trained rhesus monkeys[11] to perform a target-selection task in which the animal had to choose between two differently rewarded targets on a touch screen. They found SCRs occurring after the monkey touched a target, and conclude that SCRs do not contribute to cognitive information processing but indicate a post-decision emotional state. This is in direct contrast to the assumptions of the SMH, which states that anticipatory SCRs bias or guide the decision process by emotional signals (Damásio, 1994). However, the task developed by Amiez and colleagues (2003) involves reward contingencies that differ inherently from the reward/punishment schedule used in the IGT paradigm. First, the rhesus monkey was always rewarded — although with varying probability, which led

[11] It has to be noted that it might be problematic to draw inferences from animal studies without knowledge about the behaviour of humans in such tasks.

to minimization of risk in the task. Thus, if risk perception contributes to the development of biasing emotion-related markers, such signals should be attenuated in their experimental setup. Second, the task is not novel for the monkeys. Therefore, effects of familiarity and training might contribute to the observed time-line of SCRs in the monkeys. Even so, it remains an open question whether anticipatory emotional signals in humans reflect *post*-decision expectations about reward and punishment or *biasing* signals. Further studies are needed to clarify whether an interpretation of anticipatory SCRs as pre- or post-decisional in humans is warranted.

6.3. Causality

Maia and McClelland (2004) found that advantageous decision-making in the IGT is nearly always accompanied or preceded by conscious knowledge of which decks are "good" and which decks are "bad". Thus, according to the authors there is no need to infer a causal role for anticipatory SCRs in the decision process of healthy human subjects: Their behavioural and psychophysiological results can be interpreted as a consequence of the already available conscious knowledge. But without further experimental evidence such a conclusion is not yet warranted: VMPFC patients also report knowledge about the reward/punishment schedule of the IGT and still show impaired decision-making and absent anticipatory SCRs. Furthermore, the finding of Maia and McClelland (2004) does not rule out the possibility that healthy subjects at least sometimes rely on unconscious emotional signals in complex situations requiring a decision. Whatever the case, causal evidence linking peripheral feedback to IGT performance is scarce and thus a conclusive answer to the question whether anticipatory SCRs act as a biasing signal in the decision process is not possible at the moment. Damásio (1994) originally stated that somatic markers can act either

unconsciously or consciously: If the emotion-related signal reaches consciousness, it is experienced as a gut feeling.

Another potentially problematic result for a causal interpretation of anticipatory SCRs is that a group of healthy individuals did well on the IGT without generating somatic markers (Crone et al., 2004). It may be inferred that anticipatory SCRs are neither necessary nor sufficient for successful IGT performance. The results of Amiez and colleagues (2003) described in the preceding section further undermine an interpretation of anticipatory SCRs as causally involved in guiding the decision process. In summary, we can assert that it is rather difficult to produce direct support for a causal involvement of anticipatory SCRs in the decision process under uncertainty and risk. Ingenuous and probably more sophisticated experimental designs are needed to shed more light on this highly interesting question.

6.4. VMPFC lesions: Impairments in affective shifting?

Maia and McClelland (2004) propose a different explanation for the behaviour of VMPFC patients in the IGT. They assume that it is difficult for these patients to overcome an acquired response tendency towards the “bad” decks. The authors predominantly base this hypothesis on a study by Rolls and colleagues (1994) in which VMPFC patients were unable to adapt their behaviour in a simple reversal task. Unfortunately, these VMPFC patients showed lesions extending to the dorsolateral prefrontal cortex (DLPFC) which is an important functional area for attention and working memory and therefore necessary to accomplish nearly every kind of cognitive task: Lesions in the DLPFC should lead to impairments in IGT performance irrespective of somatic markers (see Chapter 7.1. Working memory impairment). But further evidence for this impairment theory comes from another study by Fellows and

Farah (2005). They tried to differentiate the functions of the VMPFC and the DLPFC in the IGT. Their results provide evidence for reversal learning impairments in patients with VMPFC lesions. Furthermore, they show that VMPFC patients are able to manage a modified version of the IGT with no need to overcome a certain response tendency. Their interpretation is that the neural correlate of associative stimulus-reinforcement learning (Rolls, 2007) is disturbed in these subjects (see Chapter 7.4. Difficulties in reversal learning: Impaired inhibition of learned responses). However, Bechara and colleagues (2005) report that VMPFC patients show different degrees of impairment in reversal learning, depending on the actual task to be performed. For example, the majority of VMPFC patients show good performance on the Wisconsin Card Sorting Test which requires contingency reversal learning. Furthermore, they report that their patients switched decks right after encountering a punishment in the IGT. Thus, their *immediate* reactions were similar or equal to those observed in healthy participants. But in contrast to normal controls, VMPFC patients got back to the disadvantageous decks sooner and more often. Therefore, a complete lack of reversal learning abilities seems implausible. Busemeyer and Stout (2002) report that the choices of patients do not seem to be guided by the average outcome of the decks but rather the most recent outcome of past trials providing further empirical evidence for such an interpretation.

It is of no surprise that reversal learning deficits sometimes (co)occur with impairments in the IGT, since the VMPFC is an essential cortical region for affective shifting, even though this function is located in more posterior orbital areas (Rolls, 2004). However, reversal learning requires the ability to inhibit immediate (behavioural) impulses (see Chapter 5.3.1. Impulse control and response inhibition). Here, a negative somatic marker may function as a “stopping” signal needed to inhibit

a certain response tendency that is no longer rewarding. Accordingly, the inhibition can be seen as a "decision" in itself (Bechara et al., 2005). In a reply to this hypothesis, Maia and McClelland (2005) argue that there are direct projections from the VMPFC to the striatum both of which are important structures for reversal learning. Lesions to either the VMPFC or the striatum can lead to severe impairments in this ability. Therefore, these "projections could directly guide action selection (…). It would be noisy and inefficient for action selection to rely on markers that are generated in VMPFC, go through the body, and are then read back by the brain" (Maia and McClelland, 2005, p.163). They are also critical about the performance of VMPFC patient in the Wisconsin Card Sorting Test because here the switching from one sensory category (e.g., colour) to another is required — a function that involves predominantly the lateral prefrontal cortex and not the VMPFC (Dias et al., 1996).

We argue that the results of Fellows and Farah (2005) and their interpretation are not necessarily in conflict with the SMH (Damásio, 1994). Damásio states that VMPFC patients are not able to overcome immediate rewarding stimuli in the favour of future gains. In accordance with difficulties in reversal learning, the initially learned association between turning a card from the "bad" decks and the immediate reward cannot be revised because a necessary support mechanism is not longer available, i.e. the somatic marker. Furthermore, Damásio (1994) and Bechara (2005) explicitly distinguish between the body loop and the as-if body loop. Thus, the objection stated by Maia and McClelland (2005) that somatic markers might be inherently inefficient for action selection is not necessarily warranted at least in regard to the proposed as-if body loop. However, as pointed out by Fellows and Farah (2005), the IGT is a rather complex task that involves not only stimulus-reinforcement learning and affective shifting, but also "the ability to attend to, synthesize, and remember complex

reinforcement histories [as indicated by the performance of DLPFC patients; note from the author] and to resolve the approach avoidance conflicts" (Fellows and Farah, 2005, p. 58). Thus, it is necessary to keep in mind that other functional abilities, besides the proposed reflective processes, have to be intact (e.g., working memory and attention) in order to do well on the IGT and in real-life decision-making.

7. Other psychological mechanisms

Since Damásio's (1994) SMH has been called into question by recent research, it is important to evaluate the explanatory power of other psychological mechanisms within the IGT paradigm. Thus, the following sections in this chapter deal with their proposed contribution to decision-making under uncertainty and risk and qualify their validity with respect to IGT performance.

7.1. Working memory impairment

Recent studies have suggested that conscious awareness of the reward/punishment schedule used in the IGT paradigm contributes to the emergence of anticipatory SCRs and characteristic behavioural results in the IGT (Maia and McClelland, 2004). It therefore seems necessary to evaluate the potential contributions of explicit learning mechanisms more thoroughly, in particular the contribution of human working memory on successful IGT performance. Bechara and colleagues (1998) argue that decision-making relies on intact working memory function [12] but that the functionality

[12] In contrast, Turnbull and colleagues (2005) report that performance on the IGT is not altered in conditions that involve a working-memory dependent secondary task (i.e., random number generation) or another secondary task (i.e., articulatory suppression). The performance of participants in both tasks was indistinguishable to a control group without a secondary task. Thus, it might be inferred from these results that IGT performance is *independent* of working memory load. Nevertheless, another study

of human working memory is *in*dependent of decision-making. In particular, they assume that participants with intact working memory function can show both impaired and non-impaired performance on the IGT and that participants' performance is significantly impaired if they show compromised working memory functionality. Working memory function can be correlated with activity in the dorsolateral prefrontal cortex (DLPFC). Thus, decision-making impairments in the IGT paradigm are expected if there are lesions either extending to or limited to the DLPFC (Bechara et al., 1998). Another study from a different laboratory was able to confirm this hypothesis and showed that DLPFC damage leads to impaired decision-making in the IGT (Manes et al., 2002). Accordingly, Damásio (1994) states that somatic markers inform the decision maker about the "goodness" and "badness" of the options in question and *mark* these for subsequent cognitive information processing (e.g., attention and working memory). Thus, even though the empirical results about the necessity of an intact working memory system are still inconclusive neither finding would necessarily pose serious problems for the SMH.

7.2. Risk seeking behaviour

Several independent laboratories report experimental evidence for the involvement of altered risk seeking behaviour in the decision-making process of healthy subjects in the IGT (Lerner and Keltner, 2000; Loewenstein et al., 2001; Raghunathan and Pham, 1999). For example, Lerner and Keltner (2000) report that fear and anxiety leads to cautious risk-averse decision-making in human subjects. Furthermore, Raghunathan

(Jameson et al., 2004) reports differences between these groups in a comparable experimental setup concluding that working memory function is *necessary* but *not sufficient* to do well on the IGT. Thus, the available experimental data is inconclusive about the necessity of an intact working memory system for successful IGT performance.

and Pham (1999) argue that induced anxiety leads to risk aversion and a preference for low reward options, whereas induced sadness leads to a preference for high risk and high reward options. Interestingly, Loewenstein and colleagues (2001) report that depression is associated with choosing options that did not involve taking an action independent of risk, whereas trait anxiety is associated with a preference for low risk options independent of action taking.

Thus, depression and anxiety can alter behavioural responses to perceived risks. Furthermore, it can be argued that these psychological characteristics vary in healthy human subjects without reaching a pathological level. Several studies could confirm that anxiety and especially neuroticism (Carter and Smith-Pasqualini, 2004) is associated with risk averse behaviour in the IGT, suggesting a potential role of differences in risk aversion in this experimental paradigm (see also next section). To summarize, it could be argued that variability in risk perception cause or at least contribute to the observed inconsistencies in the behavioural results of healthy human subjects. Furthermore, since risks can be characterized as feelings that aid the decision process (Loewenstein et al., 2001), an interpretation of somatic markers as risk related-signals is not necessarily at odds with the SMH.

7.3. Altered sensitivity to rewarding and punishing events

Another possibility, which is directly related to risk perception, is that altered sensitivities to reward and punishment are able to explain the behavioural and psychophysiological results obtained within the IGT paradigm. Such altered sensitivities are generally observable in highly anxious individuals as well as in subjects that show high scores in neuroticism. Thus, a thorough investigation of these individuals within the IGT paradigm could lead to interesting results and also might

aid the interpretation of the inconsistent experimental results obtained in healthy human subjects. For example, Schmauk (1970) was able to show that high levels of anxiety are correlated with increased avoidance learning in male sociopaths. Carter and Smith-Pasqualini (2004) demonstrated that good performance on the IGT is positively correlated with high levels of neuroticism, as operationalized with the Eysenck Personality Questionnaire (EPQ-R) (Amelang and Bartussek, 1997). Since neuroticism is conceptualized as positively associated with anxiety, these more recent findings are in accordance with the early findings of Schmauk (1970). Furthermore, Carter and Smith-Pasqualini (2004) confirmed that the *strength* of SCRs before choosing a card from the disadvantageous decks is positively correlated with IGT performance. Thus, it seems that Damásio's (1994) neural framework of the SMH and Eysenck's concept of the "visceral brain" as the neural substrate of neuroticism (Amelang and Bartussek, 1997) are congruent with respect to IGT performance. However, Miu and colleagues (2008) reported that anxiety impairs accurate decision-making in the IGT which is at odds with the other empirical findings and needs further investigation.

Interestingly, neuroticism correlates with *both* high sensitivity to punishment and high sensitivity to reward as measured with the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) (Torrubia et al., 2001). This finding can possibly account for the mixed results obtained with anxious (Miu et al., 2008; Suhr and Tsanadis, 2007) and neurotic (Carter and Smith-Pasqualini, 2004) individuals in the IGT, given that as mentioned above neuroticism is positively correlated with anxiety. However, anticipatory SCRs were correlated with both successful performance on the IGT and individual differences in neuroticism (Carter and Smith-Pasqualini, 2004).

Thus, anticipatory SCRs may relate to varieties in disparate sensitivities to reward and punishment.

High sensitivity to reward may lead to impairments in IGT performance as demonstrated by the behavioural results of SDIs (Bechara and Damásio, 2002; Bechara et al., 2002) and individuals with high scores in sensation and fun seeking (Suhr and Tsanadis, 2007). On the other side, neuroticism, which is associated with both high sensitivity to punishment and high sensitivity to reward, leads to superior performance in the IGT. However, *sensitivity to reward* seems to produce robust results with respect to impaired decision-making in the IGT and thus its contribution to the decision process under uncertainty and risk should be investigated in more detail in future studies. Similarly, high sensitivity to punishment could correspond to risk avoidance behaviour and could therefore also contribute to the observed variability in performance among healthy subjects in the IGT. It would be highly interesting to investigate whether neurotic individuals ever show the "prominent deck B" phenomenon (see Chapter 6.2.1. Anticipatory SCRs: Indicators of risk). We would assume that this effect is absent in neurotic participants because of their enhanced sensitivity to punishment.

Finally, it seems that when people are sensitive to both, reward *and* punishment, a superior decision strategy can emerge. This finding should be investigated in future studies including variants of the original IGT and other risk sensitive decision-making tasks (e.g., the Rogers Decision-Making Task[13]). However, as pointed out before (end of Chapter 6.3. Causality), task design again seems to be a major factor with respect to the question whether a certain decision strategy is successful: High sensitivity to reward, as it is usually observed in SDIs, impairs decision-making in the IGT but can

[13] In this task, subjects have to choose between higher and lower probability gambles.

lead to superior performance when risk taking is rewarded (Shiv, Loewenstein, Bechara et al., 2005): In a recent study, Shiv and colleagues (2005) were able to show that VMPFC patients and SDIs make significantly more advantageous decisions in an investment task compared to healthy control subjects. In this task, each participant is endowed with $20 of play money[14]. The subjects participate in several rounds of investment decisions in which they have to decide either to invest $1 or not invest $1. In case they decide to keep their $1 bill, the next round starts immediately. If they decide to invest their money in the current trial, they hand a $1 bill over to the experimenter. After the investment is paid, the experimenter tosses a coin. They lose their invested money if the outcome of the toss is heads (50% chance) and win $2.50 if the outcome is tails (50% chance). The next trial starts immediately after the participants pays or receives the respective amount of money. The whole investment task consists of 20 trials. The expected value on each trial is higher for investing money ($1.25) than for declining the offer to invest ($1). If one invests on each trial, there is a chance of 87% to end up with a higher amount of money at the end of the task than if one simply keeps the $20. The authors anticipated that healthy subjects would invest in fewer trials and thus behave less advantageously or sub-optimally in comparison to VMPFC patients and SDIs. Indeed, Shiv and colleagues (2005) found that healthy subjects showed pronounced risk avoidance behaviour in this investment task when compared to the other two groups. Interestingly, they were more likely to invest (i.e., act less risk aversely) in trials that followed previous wins than losses (see Table 3).

[14] At the end of the study the participants get a gift certificate for the amount of money they have won in the investment task.

Table 3: Healthy human subjects showed more risk averse behaviour after losing and winning in previous trials than SDIs and lesion patients. The numbers indicate the mean (median) percentage of decisions to invest. Table adapted from Shiv and colleagues (2005, Table 3, p. 88).

	Lesion patients	**SDIs**	**Normal controls**
Decision to invest – overall	83.3% (90.0%)	80.9% (95.0%)	57.6% (50.0%)
Invested and lost on previous round	85.4% (95.5%)	81.8% (100.0%)	40.5% (33.3%)
Invested and won on previous round	84.2% (100.0%)	84.6% (100.0%)	61.7% (66.7%)

The authors conclude that the outcomes of preceding trials influenced the subsequent decisions of normal control subjects more and thus led to the observed pronounced risk avoidance behaviour of these participants in the investment task. In contrast, VMPFC patients and SDIs seemed to be not influenced by affective reactions to preceding outcomes leading to the observed risk seeking behaviour in these subjects. However, it is important to note that no direct inferences about the relevance of emotion-related signals in this task are warranted, since they did not include any psychophysiological measures of affective reactions (e.g., anticipatory SCRs) in their study.

Shiv and colleagues (2005) further hypothesize that SDIs and VMPFC patients would still invest in the majority of trials if the expected value of each trial turned negative (instead of the positive value in the current experimental setup). Thus, the two patient groups would behave disadvantageously in a modified version of the investment task. The authors base their prediction on previous results from a subgroup of SDIs who showed advantageous decision-making in a variant of the original IGT. In this altered

version SDIs drew more cards from decks that involved larger and more frequent punishments and a larger subsequent reward (Bechara et al., 2002).

Their current findings suggest that certain impairments in the processing of emotion-related signals can lead to attenuated levels of risk aversion. In tasks that are conceptualized like the investment task (i.e., where risk-taking behaviour is rewarded) such impairments can lead to advantageous decision-making behaviour.[15]

7.4. Difficulties in reversal learning: Impaired inhibition of learned responses

The initial advantage of the "bad" decks in the IGT can cause a problem for the interpretation of IGT results in terms of the SMH, since difficulties in reversal learning can account equally well for these results (see Chapter 6.4. VMPFC lesions: Impairments in affective shifting?). Impairments in reversal learning or response inhibition occur when the participants are not able to suppress learned response behaviour to a given task. To evaluate the possibility that such mechanisms are involved in successful and impaired IGT performance, Fellows and Farah (2005) compared the behaviour of orbitofrontal patients (VMPFC and DLPFC patients) with the behaviour of healthy subjects (i.e., the control group) in the original IGT and in a modified version of the IGT in which the initial advantage of the "bad" decks is side-stepped.

[15] As already stated by Damásio (1994, p. 174) "somatic markers are a special instance of feelings generated from secondary emotions. Those emotions and feelings have been connected by learning to predicted future outcomes of certain scenarios. When a negative somatic marker is juxtaposed to a particular future outcome the combination functions as an alarm bell. When a positive somatic marker is juxtaposed instead, it becomes a bacon of incentive".

Table 4: Payoff schedule of the shuffled IGT. Table adapted from Fellows and Farah (2005, pp. 58-63).

	Deck A (+100)	Deck B (+100)	Deck C (+50)	Deck D (+50)
1	-250	-1250	-50	
2	-350		-50	-250
3		-1250		
4	-350		-25	
5			-75	
6	-250			
7	-200			
8			-25	
9	-300		-75	
10	-150			
11			-50	
12				-250
13		-1250		
14	-300			
15				
16	-350		-50	
17			-25	
18	-200		-50	
19	-250			
20	-150			
21			-75	-250
22			-50	
23	-350			
24	-200	-1250		
25	-250			
26			-25	
27			-25	-250
28				
29	-150		-75	
30	-300			
31			-50	
32			-75	
33				
34				
35	-150		-50	
36				
37	-300		-50	
38				
39	-200		-50	
40				

To this end, they reorganized the sequence[16] of the cards (see Table 4) to ensure that the punishments are experienced on the first trials, thus, eliminating the need for reversal learning in the IGT. Fellows and Farah (2005) found that healthy human subjects chose more cards from the "good" decks than from the "bad" decks, whereas participants with orbitofrontal cortex damage (VMPFC and DLPFC patients) were significantly impaired in their card drawing behaviour in the original version of the IGT (replicating the behavioural results usually obtained). Furthermore, they were able to show that VMPFC (but not DLPFC) patients draw significantly more cards from the advantageous decks in the shuffled version of the task compared to their performance in the original IGT. Their behaviour was statistically indistinguishable from normal controls in this shuffled version (see Figure 3). Interestingly, DLPFC patients did not do well, on *neither* the original IGT *nor* the shuffled version. Thus, their behavioural impairments seem not to be due to difficulties in reversal learning, but might reveal further evidence for the assumption that an intact working memory is necessary to do well on the IGT (see Chapter 7.1. Working memory impairment). Nevertheless, further studies are needed to corroborate this interpretation. However, converging empirical evidence seems to indicate that VMPFC (but not DLPFC) damage significantly impairs reversal learning in humans and animals (Dias et al., 1996; Fellows and Farah, 2003; Fellows and Farah, 2005).

[16] In the shuffled version of the IGT each deck begins with card 9 by moving cards 1–8 to the bottom of the corresponding deck. Additionally, the cards number 11 and 14 are switched in the disadvantageous deck B.

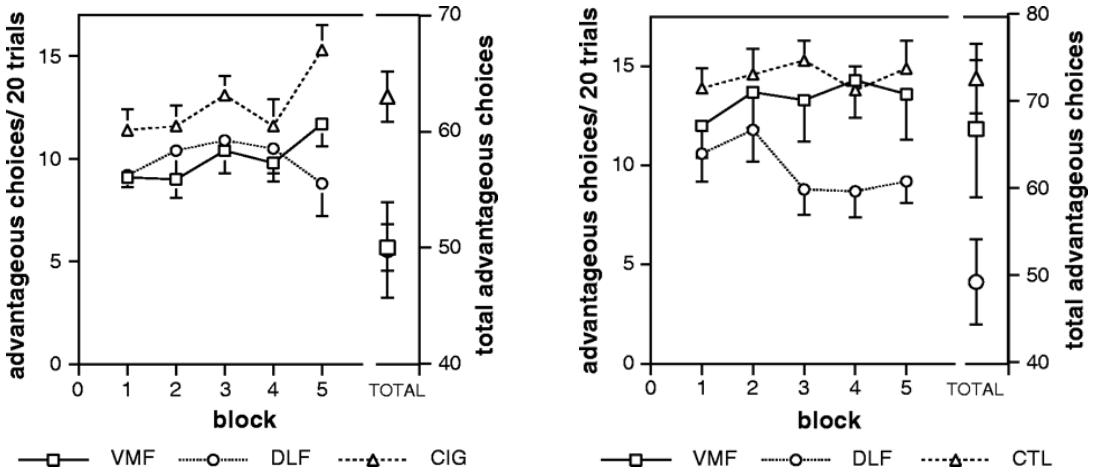

Figure 3: The behavioural results for ventromedial prefrontal cortex (VMF) patients, dorsolateral prefrontal cortex (DLF) patients, and normal control subjects (CIG and CTL, respectively) in both the original version of the IGT (left panel) and the shuffled analogue (right panel) are shown. Figures reproduced from Fellows and Farah (2005, Figure 2 and 3, p. 60).

We argue that reversal learning is an essential pre-requirement to do well on the IGT. Without the ability to change learned reward/punishment contingencies, one is necessarily impaired in decision-making, regardless in which paradigm it is tested. However, we assume that the IGT captures more than an inability in reversal learning, since the behavioural results are nicely correlated with psychophysiological measurements (i.e., anticipatory SCRs) that have not been recorded in the studies conducted by Fellows and Farah (2003; 2005). Thus, it is still possible that the inability to produce somatic makers is associated with impaired decision-making in the IGT. We agree that problems with reversal learning can account for the performance of VMPFC patients, or at least a subgroup of these patients, but we doubt that difficulties in reversal learning can easily explain the high variability in IGT performance in healthy subjects and the difficulties experienced by SDIs. Since IGT performance is also impaired when the amygdala is damaged, it seems warranted

to infer an involvement of emotion-related signals in the IGT[17]. Furthermore, since the sequence of cards is altered in the shuffled version of the IGT, it is possible that the important component of conflict between the "good" and "bad" decks is less pronounced and that this fact also contributes to the observed behavioural results in this shuffled version.

8. Neural representation of subjective reward value

The following sections aim to discuss recent research concerning the neural correlates of subjective reward value and related concepts. We assume that the empirical findings of such studies are highly relevant for the interpretation of IGT results, because they discuss among other things how immediate and future reward values are encoded cerebrally as well as the neurobiological basis of impulsiveness and the emergence of risk-taking behaviour.

8.1. Interaction of an impulsive and a patient system?

McClure and colleagues (2004) assume that the often observed discrepancy between immediate (*short-run*) and future (*long-run*) preferences in intertemporal choice reflects the differential activation of disparate neuronal systems. They argue that *short-run impulsivity* is driven by the limbic system, which responds preferentially to immediate rewards and is less sensitive to future consequences. This interpretation is in accordance with the neurocognitive framework proposed by Bechara (2005) and Damásio's (1994) SMH. Both, sensitivity to the value of *distant* rewards as well as *long-run patience* in temporal discounting tasks are assumed to be mediated by the

[17] However, it should be noted here that at least one study reports that reversal learning operates independently from emotional information processing (Izquierdo et al., 2004).

lateral prefrontal cortex and associated areas. These cortical structures evaluate trade-offs between distinct reward options, ranging from immediate to distant future rewards. Moreover, McClure and colleagues (2004) argue that human behaviour is shaped through the competing influences of limbic and paralimbic structures that trigger automatic processes and cortical structures that enable abstract domain-general reasoning and future planning. They assume that human idiosyncrasies in decision-making and subjective reward value reflect the interaction of these two *competing* systems, both relevant in adapting and guiding human behaviour. Their argumentation is consistent with the SMH and the neurocognitive model proposed by Bechara but is challenged by a quite recent study conducted by Kable and Glimcher (2007) presented in the subsequent section.

8.2. Subjective preference functions in humans: Medial prefrontal cortex, ventral striatum, and posterior cingulate gyrus

According to Kable and Glimcher (2007) subjective values or preference functions, as they are called in economics, are encoded in the human brain. Temporal discounting tasks (Frederick et al., 2002) have shown that subjective reward values are not correlated linearly with the absolute value of a reward. For example, a person that would trade a $20 check that could be cashed in one week for an immediate monetary gain of $18, would trade the same $20 check for an immediate monetary reward of $15 dollars if the check could be cashed in one month. This decline in subjective reward value with increasing time delay varies significantly across subjects (*patient* versus *impulsive* discounters). Thus, the objective value together with the time difference cannot predict how people decide in temporal discounting tasks. Kable and Glimcher (2007) suggest that an idiosyncratic function (i.e., a person-specific

function) is necessary to relate subjective reward value accurately to disparate time delays. To investigate how subjective reward value is represented in the human brain they measured neuronal activity in healthy human subjects using functional magnetic resonance imaging (fMRI) while these subjects were deciding between different immediate and delayed monetary rewards. They found a significant correlation between neuronal activity in the ventral striatum, medial prefrontal cortex, and posterior cingulate cortex on the one hand and subjective reward value as measured by psychometric tests (i.e., individual preference curves that show how subjective reward value varies as a function of delay and monetary gain) on the other (see Figure 4). It is important to note that they only included subjects with stable preference functions over a six month period in their fMRI measurements (i.e., ten out of twelve subjects).

The neuronal activity in the ventral striatum, medial prefrontal cortex, and posterior cingulate cortex was correlated with subjective reward value on the individual level: Every subject's idiosyncratic pattern of neuronal activity was predicted by that subject's subjective preference function. Moreover, they could show that time delay had stronger effects on subjective reward value in impulsive than in patient discounters: Impulsive decision-makers showed steeper decreases whereas patient subjects showed a gradual decrease in their preference functions. Furthermore, neuronal activity in the ventral striatum, medial prefrontal cortex, and posterior cingulate cortex increased as the objective amount of the reward value increased and decreased as the delay to reward increased. The neural tradeoffs across subjects and between amount and delay, as described by the individual discount functions, correlated with the behavioural tradeoffs between these variables indicating that

choosing between immediate and delayed monetary rewards involves comparing neuronally represented subjective values (see Figure 4).

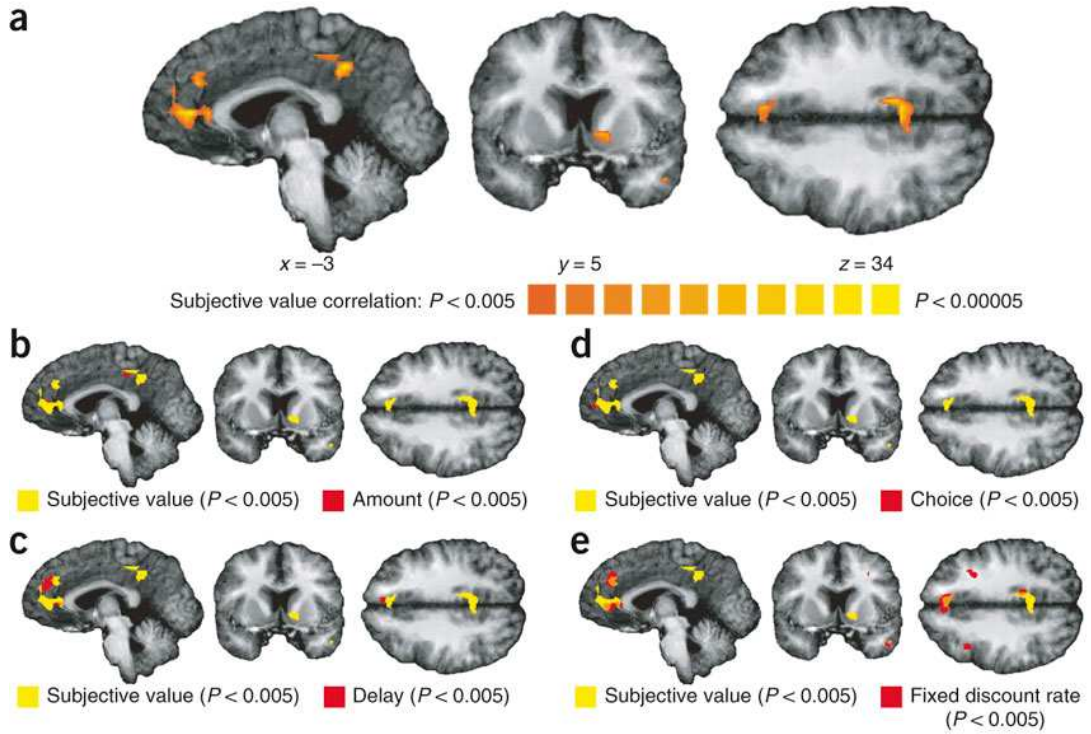

Figure 4: (a) Cortical areas (medial prefrontal cortex, posterior cingulate cortex, and ventral striatum) that show correlated neural activity with subjective reward value. Neural activity in the ventral striatum, medial prefrontal cortex, and posterior cingulate cortex was better correlated with subjective value (yellow) than with (b) the objective amount of the delayed reward (red), (c) the inverted delay of the delayed reward (red), (d) the choice of the subject (red), or (e) the value of the delayed reward (red). Figure reproduced from Kable and Glimcher (2007, Figure 3, p. 1627).

What are the implications of these findings for the behaviour of healthy and impaired subjects in Bechara's IGT? Bechara (2005) proposes that an overactive impulsive system in SDIs is responsible for their impaired performance in the IGT. The results of Kable and Glimcher (2007) are at odds with such an interpretation, since they do not assume two interacting systems. However, they provide evidence that differences in decision-making are related to the impulsiveness of the decision-maker: Impulsive discounters are more likely to trade a check for less money with increasing time

delays than patient subjects. Since immediate rewards seem to be more attractive than delayed monetary rewards for impulsive decision-makers as reflected in the steeper discount functions in these subjects, it would be interesting to know whether impulsive discounters show altered activations in the VMPFC compared to patient discounters. If there were significant differences in neuronal activity, one could hypothesize that the VMPFC is directly related to the degree of impulsiveness with respect to choices that involve immediate and delayed monetary rewards. Furthermore, it would be highly interesting to measure the activity of the amygdala in this task and to correlate its activation to the impulsiveness of the participants as well as to the activation of the VMPFC.

It seems that subjective valuations of delayed reward play an important role in delayed discounting tasks and thus may be very likely involved in adapting one's decision strategy in situations that involve disparate immediate and delayed monetary gains, like the IGT. The investigation by Kable and Glimcher (2007) did not involve a pronounced component of uncertainty and risk while making a decision. Thus, it is not likely that differences in risk aversion directly contributed to the decision strategy in their subjects, but did contribute to the overall performance of subjects in the IGT. However, it seems unequivocal that the medial prefrontal cortex together with the ventral striatum and posterior cingulate cortex are involved in the subjective valuation of monetary outcomes during decision-making and that their activity is highly correlated with the individual preference functions of the participants.

It is also unclear whether their results are at odds with the refined version of the Expectancy–Valence Model discussed later on (see Chapter 10.5. Refinement of the Expectancy–Valence Model). Since this model assumes that the idiosyncrasies

observed in healthy subjects are related to differences in risk perception[18], further studies are needed to investigate the role of subjective reward functions in situations that involve uncertainty and risk before any inferences can be drawn.

8.3. Reward expectation, prediction error, and risk perception

It has been shown that dopaminergic neurons respond to the probability of a reward and to the difference between an actual reward and the conditional expectation[19] of this reward (i.e., the reward prediction error) (Fiorillo et al., 2003). Fiorillo and colleagues (2003) recorded the activity of dopaminergic neurons in the ventral midbrain of alert monkeys and discovered that the spike trains of target neurons varied with respect to reward probability (see Figure 5). The conditioned stimuli were visual cues that the monkey had previously learned to associate with certain probabilities (ranging form 0 to 1) to get a reward (i.e., squirts of fruit juice). As can be seen in Figure 5, the recorded neuronal activity of the dopaminergic neurons was correlated with the reward prediction error: If the learned probability to get a squirt of fruit juice after presentation of an associated visual stimulus was P = 0.25, then the response at the time of the reward (i.e., the US) was three times as large as at the time of the CS.

[18] Risk perception is modelled through disparate sensitivities to reward and punishment.

[19] In Pavlovian conditioning an animal learns to associate a neutral stimulus — the conditional stimulus (CS) — with another stimulus — the unconditional stimulus (US). In the present experiment the US is a certain amount of fruit juice delivered subsequently to CS-onset (i.e., different visual cues). After learning the association between CS and US the CS-stimulus still remains intrinsically non-rewarding but has acquired the function of a predictor for the US. If the US has stochastic properties, for example in terms of magnitude and occurrence, the experimental setup resembles a *gamble*. Since different associations can be learned simultaneously (through various different CS-UC pairings) such an experimental design allows the investigation of the effect of different reward magnitudes and probabilities.

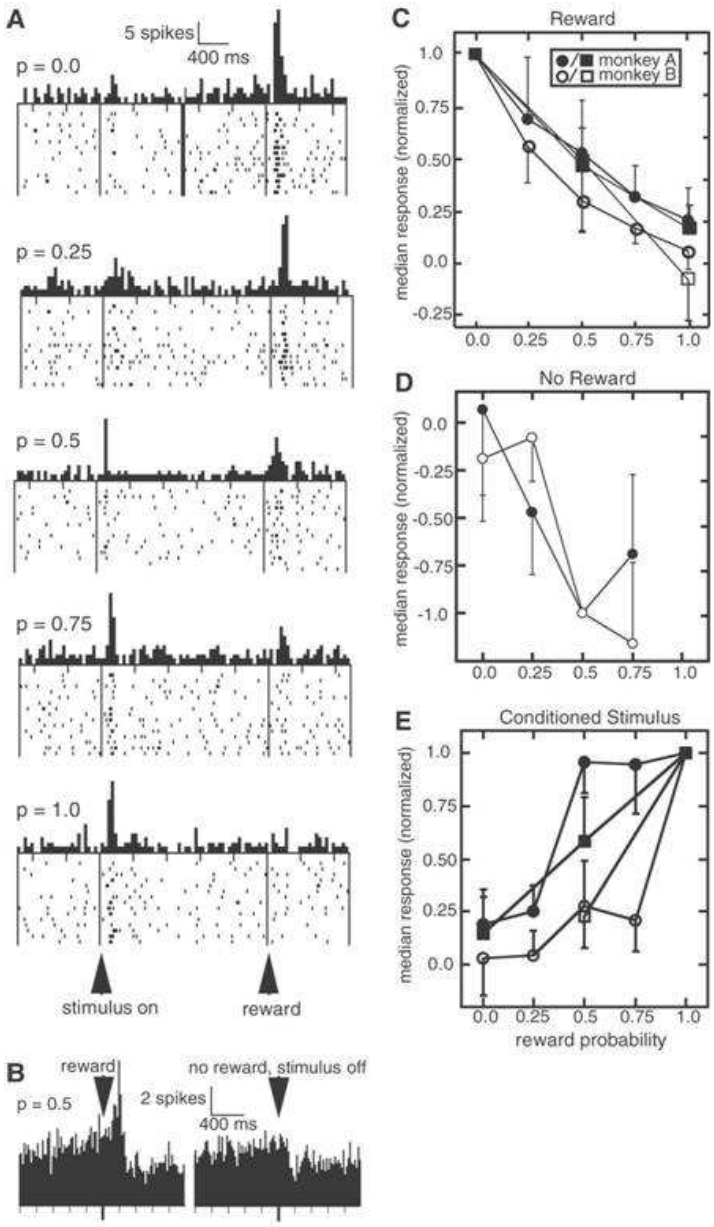

Figure 5: (A) Spike trains of dopaminergic neurons in the ventral midbrain of monkeys. "CS" indicates the time at which the visual cue was presented, and "reward" indicates the time of the fruit juice reward delivery. Each sequence of vertical dashes in the raster below the horizontal line shows the spike train of an individual neuron, whereas the single cell histograms are shown above the horizontal line. (B) Population histograms of rewarded and unrewarded trials at maximal uncertainty (P = 0.5). (C) The magnitude of the reward responses increased as reward probability decreased (i.e., coding of prediction error). (D) Trials in which reward was predicted but did not occur, showed increasingly suppressed neuronal activity with increasing reward probability. (E) Conditioned stimuli (i.e., squirts of fruit juice) triggered increasing phasic activations with increasing reward probability. Figure reproduced from Fiorillo and colleagues (2003, Figure 2, p. 1899).

Accordingly, if the probability was P = 0.75, then the neuronal activity at the time of the reward was only one third as large as at the time of the presentation of the conditioned visual cue. Moreover, using fMRI Knutson and colleagues (2003) were able to show that neuronal activity in the area of the ventral striatum and the nucleus accumbens increases with reward expectation in human subjects as well.

Fiorillo and colleagues (2003) further showed that dopaminergic neurons also respond to risk perception, even though with a temporal delay. In their experiment the amount of juice squirts was fixed, whereas the probability to get a fruit juice reward was varied across trials (P = 0, P = 0.25, P = 0.5, P = 0.75, and P = 1). Risk perception[20] was maximal when the trial had a probability of P = 0.5 and minimal when the probability was P = 0 or P = 1. They found that dopaminergic neurons in the ventral midbrain of alert monkeys responded with a sustained increase in activity that grew from the onset of the conditioned stimulus to the expected time of reward delivery (see Figure 6). The response was maximal when risk was maximal, i.e. at a probability of P = 0.5, less pronounced at P = 0.25 and P = 0.75, and absent at P = 0 and P = 1. Statistical analysis revealed a significant effect of uncertainty on neuronal activity, indicating that uncertainty is encoded by this sustained neuronal response (see Figure 6). Since phasic and sustained activations differed in timing and relation to reward probability, as well as in their occurrence in single neurons, it is assumed that both occur *independently*. Fiorillo and colleagues (2003) further demonstrated that the observed sustained neuronal activation is specifically related to uncertainty about *motivationally relevant* stimuli: (1) Sustained activity was correlated with reward magnitude and (2) was not observable when the CS was another visual cue replacing the subsequent fruit juice reward.

[20] Risk is measured by variance.

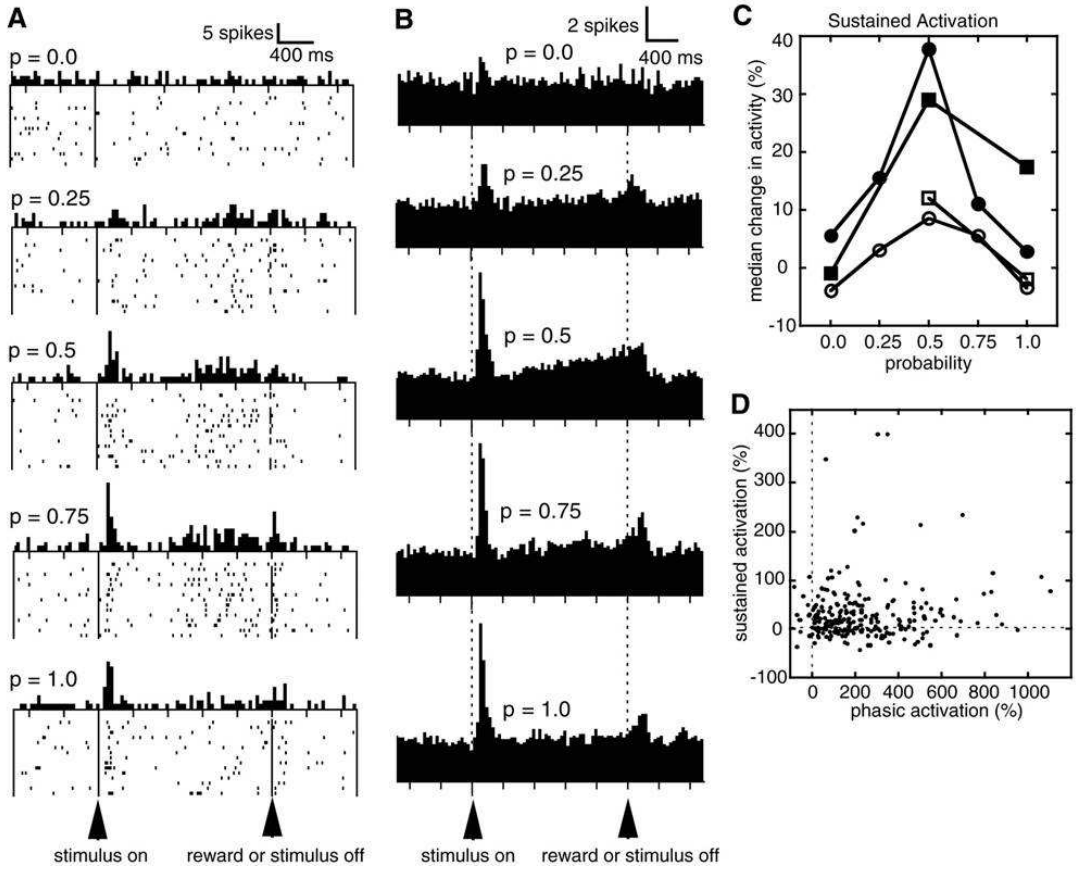

Figure 6: (A) Sustained activation of dopamine neurons precedes potential reward at all three intermediate probabilities (P = 0.25, P = 0.5, and P = 0.75). "CS" indicates the time at which the visual cue was presented, and "reward" indicates the time of the fruit juice reward delivery. Each sequence of vertical dashes in the raster below the horizontal line shows the spike train of an individual neuron, whereas the single cell histograms are shown above the horizontal line. (B) Population histograms of rewarded and unrewarded trials at reward probabilities ranging from P = 0 to P = 1. (C) Median sustained activation of dopamine neurons as a function of reward probability. (D) Magnitude of phasic activation plotted against sustained activation for each neuron. Figure reproduced from Fiorillo and colleagues (2003, Figure 3, p. 1900).

To summarize, Fiorillo and colleagues (2003) showed that dopaminergic neurons change their spiking activity in accordance to the type (i.e., magnitude and probability of subsequent reward) of the conditioned stimulus. Primarily, they found that dopaminergic neurons show two dissociable response patterns: (1) Short, phasic neuronal activity patterns that adapt monotonically to increasing reward probability, and (2) slow, sustained responses that increased with increasing reward uncertainty.

The latter may help explain the behavioural results of normal controls and SDIs in the IGT, since the sustained, uncertainty-induced increase in dopamine can be interpreted as acting to reinforce risk-taking behaviour[21], suggesting a possible explanatory role of dopaminergic activity for the inconsistencies found in these subjects. Differences in risk-taking behaviour (learned and established through dopaminergic activity) could help explain the inconsistent behavioural findings in healthy human subjects in the IGT. Thus, their contribution to the decision process in the IGT should be investigated in detail in future studies. For example, it could be studied whether ventral midbrain areas are differentially activated in healthy subjects who can be behaviourally distinguished with respect to their card drawing behaviour in the IGT and psychophysiologically discriminated by the strength of anticipatory SCRs and other autonomic parameters (e.g., heart rate deceleration).

9. Empirical results from different variants of the IGT

The SMH assumes that decision-making impairments in patients with VMPFC lesions can by explained through a deficit to produce negative or positive somatic markers that adapt the decision process in complex situations with uncertain reward/punishment contingencies. The impairments of these individuals are

[21] Fiorillo and colleagues (2003) argue that dopaminergic neurons are encoding the prediction error across the full range of probabilities and thus could provide a teaching signal for learning. Subjective uncertainty is assumed to indicate that one lacks an appropriate predictor (Fiorillo et al., 2003). Dopamine (beside its various other functionalities) might act as a non-selective attention signal enabling the learning of the accuracy of predictive stimuli and actions. If risk-taking behaviour is necessary for the learning of accurate reward predictors, then "the sustained, uncertainty-induced increase in dopamine could act to reinforce risk-taking behaviour and its consequent reward information, whereas the phasic response after prediction error could mediate the more dominant reinforcement of reward itself" (Fiorillo et al., 2003, p. 1901).

characterized by either extreme procrastination or by choosing disadvantageous response options. The latter is also apparent when declarative knowledge about correct outcome expectancies is available. Damásio (1994) and Bechara (2005) call this phenomenon “myopia for the future”. The results of different variants (Bechara, Tranel et al., 2000; Fellows and Farah, 2005; Sanfey et al., 2003) of the original IGT either support this interpretation or suggest that other psychological mechanisms may provide a more *parsimonious* and more *accurate* account for the observed behavioural differences in the acquisition of this task (see Chapter 7. Other psychological mechanisms).

Bechara and colleagues (2000) found that VMPFC patients remain impaired when the advantageous and disadvantageous decks are more clearly polarized (see Table 5) involving higher immediate punishment and larger delayed reward (i.e., advantageous decks) as well as lower immediate punishment and lower future reward (i.e., disadvantageous decks). They interpret their findings together with the results obtained in the original version of the IGT and argue that it seems unlikely that the mechanism underpinning the decision-making deficit of VMPFC patients is a loss of sensitivity to either punishment or reward. In another variant they increased the adverse future consequences associated with the “risky” decks through increasing delayed punishment and decreasing delayed reward. VMPFC patients were impaired relative to normal controls in this task, too. Bechara and colleagues (2000) interpret this finding as myopia for future consequences rather than altered sensitivity to reward and punishment in VMPFC patients. However, Fellows and Farah (2005) were able to show that VMPFC patients can do well on a shuffled version of the IGT (see Chapter 7.4. Difficulties in reversal learning: Impaired inhibition of learned responses).

Table 5: Payoff schedule of the altered IGT. Table adapted from Bechara and colleagues (2000, Figure 1, p. 2193).

	Deck E (-100)	Deck F (-50)	Deck G (-100)	Deck H (-50)
1				
2			+350	
3	+1250			
4		+25	+250	
5		+50		
6			+300	
7			+200	
8		+75		+250
9		+25	+150	
10		+75		
11	+1250	+50		
12				
13		+25	+350	
14				
15			+250	
16		+25		
17		+75	+200	
18			+150	
19				
20		+75	+300	+250
21	+1250			
22			+300	
23				
24		+25	+350	
25		+75		
26		+50	+150	
27			+200	
28			+250	
29		+75		
30		+25		+250
31			+150	
32			+200	
33	+1250		+350	
34		+50		+250
35		+50		
36				
37		+25	+200	
38			+350	
39		+75		
40		+50		

Furthermore, it could be shown that the card drawing behaviour of healthy individuals is highly sensitive to the actual payoff schedule (Fum et al., 2008; Rakovský, 2009).

Thus, the findings with different variants of the IGT led to rather inconclusive results and need further investigations.

Sanfey and colleagues (2003) tested the hypothesis that VMPFC patients may perform disadvantageously on the original version of the IGT because of a decreased tendency to avoid risky decisions when rewards are involved. The task they designed deviates from the original IGT in its focus on *risk preference* of VMPFC patients, patients with other neural lesion sites, and normal controls. Participants were asked to select cards from five decks that differed in their variance of rewards and punishments over time but had identical expected values (see Figure 7). Thus, participants' risk seeking behaviour could be investigated independently of the accuracy of their decision-making abilities.

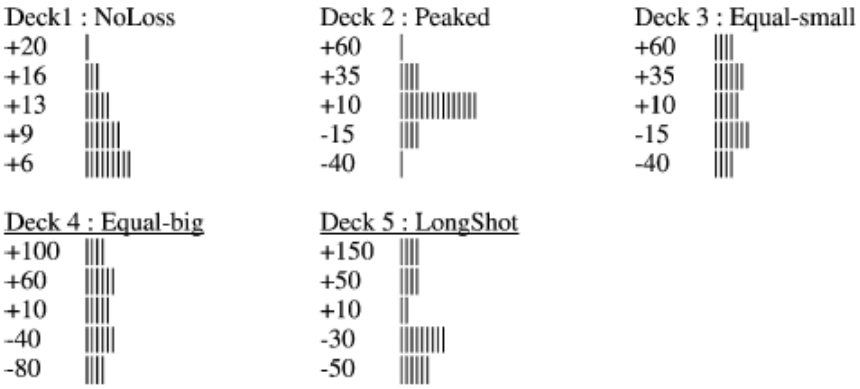

Figure 7: Each vertical line represents a single outcome when selecting a card from the five different decks. In sum, each deck had 25 outcomes. Figure reproduced from Sanfey and colleagues (2003, Figure 2, p. 1223).

Normal controls showed risk-averse behaviour in this task and selected significantly more cards from the *safe*, low variance decks (decks 1 and 2) than from two of the three *riskier*, high variance decks (decks 3 and 5[22]). In contrast, VMPFC patients

[22] They were neutral towards deck 4.

could be split into two sub-groups with those who were also risk averse and those who displayed risk-taking behaviour. The latter demonstrated a preference for decks 4 and 5 — two of the three riskier, high variance decks — were neutral towards deck 3, and avoided decks 1 and 2. Interestingly, the risk-taking subgroup tended to have lesions extending to the DLPFC, but this difference did not reach statistical significance. Maybe impairments to both the VMPFC and the DLPFC account for the increases in risk-taking behaviour, but at the moment this is a rather speculative idea. Further examination of the data revealed that the *risky* groups paid more attention to what they could get (cf. increased sensitivity to *reward*) whereas the *safe* groups focused on the amounts of money that they could lose (cf. increased sensitivity to *punishment*). The overall results prompt the hypothesis that the poor performance of VMPFC patients in the original IGT may be traced to an increased preference for risky decisions as measured by variance.

10. A computational cognitive model of IGT performance

The first two sections outline the characteristics of our derived[23] computational cognitive model of IGT performance and explain the different modules modelling two distinct players: (1) A rational player that bases its decisions on expected values and (2) an emotional player that is able to use affective information (see Figure 8). The only difference between the two players is the appraisal system that calculates the "desirability" of the four different decks (the appraisal system is missing in the rational player). The characteristics and modules of both players are described in

[23] The neurocognitive framework of decision making under uncertainty and risk developed by Bechara (2005) and described in detail in Chapter 4 (cf. Figure 1) provides the basis for our computational cognitive model.

detail in the two subsequent sections. The fourth section compares the card drawing behaviour of both players to that of the Expectancy–Valence Model (described in detail in section 3) developed by Busemeyer and Stout (2002) to evaluate their explanatory power with respect to IGT results. The last section introduces the proposed refinement of the Expectancy-Valence Model and discusses its assumed benefits with respect to the interpretation of IGT results.

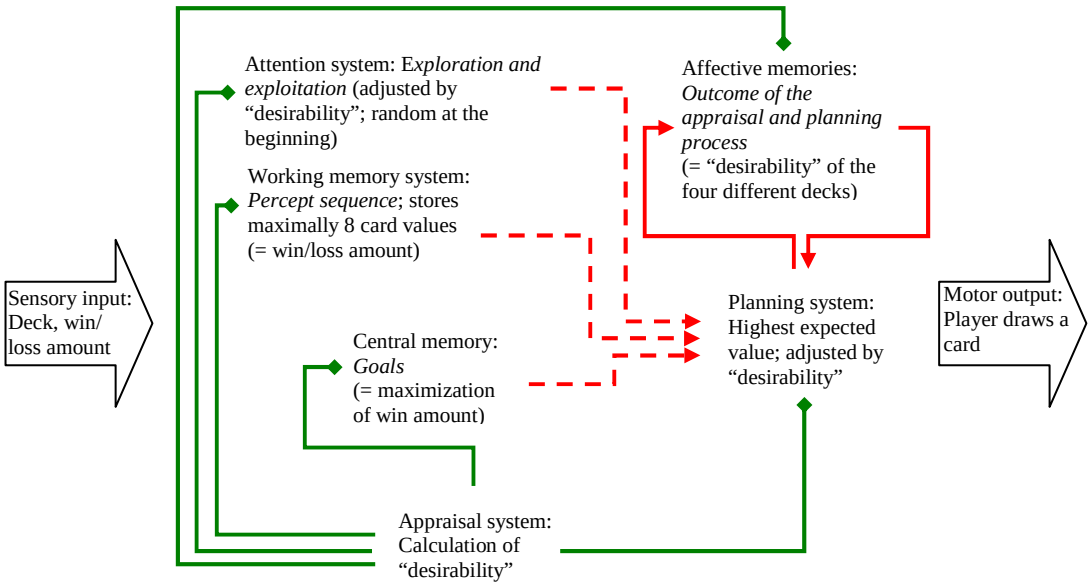

Figure 8: Computational cognitive model of the Iowa Gambling Task as abstraction of the neurocognitive framework: Red lines (no line marker) indicate excitatory connections; green lines (diamond ◆ line marker) indicate modulatory connections; and dashed lines indicate modulated excitatory influences.

10.1. The rational player

One possibility to predict the performance of participants in the IGT is to consider their behaviour as rational[24] simultaneously taking into account the limitations of

[24] Certain affect and personality characteristics can also influence the behaviour in the IGT. For example, negative affect and high scores in fun seeking are correlated with less advantageous decision strategies in the IGT (Suhr and Tsanadis, 2007). To keep the computational cognitive model simple, such exogenous influences on IGT performance were factored out.

human rationality (cf. bounded rationality). According to Russel and Norvig (2003, p.36), “a rational agent should select an action that is expected to maximize its performance measure, given the evidence provided by the percept sequence and what ever built-in knowledge the agent has”. Thus, a rational player of the IGT should base its decision on the *expected value* of the four different decks (A-D). We assume that three different systems need to be considered with respect to this calculation:

(1) A working memory system (cf. DLPFC and hippocampus), i.e. the player has to keep in mind the values of the drawn cards (cf. percept sequence). It stores the values of each card that is drawn in the IGT. A decay mechanism is implemented to simulate the limited capacity of human working memory (cf. Miller, 1956). Thus, this module can store maximally eight card values at a time. If the card memory runs out of card values for a certain deck, the expected value for this deck is set to 0.

(2) A calculating and planning system (cf. VMPFC) that enables the player to draw inferences about the expected value (cf. maximize its performance measure) of the next card with respect to the four decks (cf. action selection). It computes the expected values (i.e., the objective statistics) for the four different decks. An error mechanism is needed to adequately simulate human inference capabilities. This is realized through the constrained working memory system (see above). Another possibility would be to use a version of running average to smooth the data.

(3) An attention system (cf. DLPFC) that permits *exploration* (especially at the beginning of the task) and *exploitation* of all of the four decks. The attention for all four decks is equal at the beginning of the task (cf. exploration phase). After a card is drawn from each of the four different decks, the planning system calculates the expected values for the respective decks based on the card values that are stored in the working memory system thus shifting the focus towards the deck with the highest

expected value for the next card. Thus, the outcome of the last action adapts the decision strategy for the next card that has to be drawn[25].

10.2. The emotional player

Emotion-related signals seem to play an important role in the decision process of human subjects through providing a qualitative bias in terms of the "desirability" of the outcomes (Damásio, 1994). Together with the likelihood of certain events, emotions may facilitate and accelerate the decision process. Thus, emotions may enable a more efficient decision strategy compared to a purely rational player (Bechara et al., 1994). Hence, the objective statistics calculated by the rational player are modulated by the "desirability" of the last outcome of the respective deck leading to some sort of subjective statistics about the inherent structure of the IGT. This idea is realized through the appraisal system. This system associates each stimulus with its affective information (= "desirability" of the deck) dependent on its monetary value (cf. primary emotions[26]). The "desirability" of the four different decks is calculated separately for rewards and punishments: (1) It remains the same if the current reward (*punishment*) corresponds to the anticipated reward (*punishment*) of a deck. (2) If the current reward is higher[27] than the anticipated reward, the "desirability" for this deck increases. (3) The "desirability" decreases if the current reward is lower than the

[25] In the computational model, the planning system includes the attention system with an exploration and exploitation phase and the working memory system that holds a maximum of 8 card values. The planning system computes the expected value of the next card for each of the four different decks. Besides, an expectation function returns a list with actual and anticipated values that could be used for further refining the drawing function of the rational player in future studies.

[26] Primary emotions are immediate emotional reactions to situations, objects, or persons (Damásio, 1994, p. 131-134).

[27] The "desirability" of punishments is calculated complementarily: If the current punishment is lower (*higher*) than the anticipated punishment then the "desirability" increases (*decreases*).

anticipated reward. The expected value for each deck is multiplied by the combined "desirability" for rewards and punishments (i.e., a standardized value between 0[28] and 1) and the drawing behaviour of the emotional player is adapted accordingly. The working memory, attention, and planning system are equal to the rational player.

To summarize, the decision strategy of the emotional player consists of three major parts: (1) The expected value before drawing a card, (2) the "desirability" of reward and punishment (after drawing), and (3) the anticipated "desirability" of reward and punishment. For example, if the player draws a card with the expected value +$50 without punishment, and actually gets +$50 but with a punishment of -$250, the player will update its decision strategy to represent that drawing a card from this deck indeed led to a gain of +$50 but also resulted in an unexpected loss of -$250.

10.3. The Expectancy–Valence Model

The Expectancy–Valence Model is based on a model developed originally by Busemeyer and Myung (1992) to integrate learning and decision-making processes and described in detail in Busemeyer and Stout (2002). It assumes that the player integrates information about experienced rewards and punishments on each trial of the IGT into an affective reaction called a *valence*. The expectancies about these valences for each of the four different decks (A-D) are learned through an adaptive learning mechanism and serve as the input into a probabilistic choice mechanism that selects one of the decks in each trial.

[28] 0 corresponds to the highest possible punishment in the IGT (-$1250) and 1 corresponds to the highest possible reward (+$100). We are aware that these boundaries are rather arbitrary and we suggest to estimate more reasonable values from empirical data sets in future studies.

10.3.1. Valences

The experienced rewards and punishments produce an affective reaction, which is represented as a weighted average of gains and losses (see Equation 1).

$$v(t) = \{(1 - w) \cdot R[D(t)] + w \cdot L[D(t)]\}$$

Equation 1: The valence v(t) experienced after drawing a card from deck D on trial t is calculated as the weighted average of rewards (R) and losses (L). The attention weight parameter w (which has to be estimated from empirical data sets) can vary between 1 and 0 and reflects the amount of attention given to rewards and punishments, respectively (Busemeyer and Stout, 2002, Equation 3a, p. 257).

10.3.2. Expectancy learning

The expectancies about the valences for the four different decks are learned through experience: The expected valence for deck D_i on trial t is updated through an adaptive learning mechanism. If valence v(t) is experienced because deck D is chosen on trial t, the expected valence is updated according to the following equation 2:

$$Ev[\mathrm{D}_i|t] = (1 - a) \cdot Ev[\mathrm{D}_i|t - 1] + a \cdot v(t)$$

Equation 2: The update of expected valences resembles a weighted average of past valences. If the updating rate a ($0 < a < 1$) is high, then changes happen fast, which means rapid forgetting, strong recency effects, and short associative memories. If a has a numerical value near 0, then the update is very slow, which means slow forgetting, weak recency effects, and long associative memories (Busemeyer and Stout, 2002, Equation 3b, p. 257).

The expected valences for the three decks that are not chosen on trial t remain unchanged. It is important to note that recently experienced valences receive more weight than remote affective reactions.

10.3.3. Probabilistic choice

The probability that a deck is chosen on the next trial is an increasing function of the expected valence for this deck and a decreasing function of the expected valences for

the other three decks. The following equation 3 represents the rule for choice probabilities according to the Expectancy–Valence Model as described in Busemeyer and Stout (2002):

$$Pr[\mathrm{D}_i|t+1] = \frac{e^{Ev[\mathrm{D}_i|t]\theta(t)}}{\sum_{i=1}^{4} e^{Ev[\mathrm{D}_j|t]\theta(t)}}$$

Equation 3: Probabilistic function of expected valences associated with each of the four different decks. The parameter θ(t) — the sensitivity parameter — controls the sensitivity of the choice probabilities to the expected valences. If θ(t) is set to 0, then the choice is completely random and therefore independent from expected valences. If θ(t) is high in magnitude, the choices strongly depend on the expected valences (and can even become deterministic) (Busemeyer and Stout, 2002, Equation 3c, p. 257).

The sensitivity parameter θ(t) that controls the sensitivity of the probabilistic choice function is assumed to vary with experience, being random at the beginning of the IGT (cf. *exploration* phase of the game) and increasing during the task (cf. *exploitation* phase of the game). Busemeyer and Stout (2002) argue that brain damaged individuals may experience a loss of concentration and get tired sooner than normal controls which can lead to differences in the sensitivity parameter across these groups. This assumption is described in the following equation 4:

$$\theta(t) = (t/10)^c$$

Equation 4: The sensitivity parameter θ(t) is controlled by parameter c, which is the third parameter that has to be estimated from empirical data sets. Positive c values indicate increasing sensitivities to expected valences, whereas negative c values indicate decreasing sensitivity to the expectancies that are used for the probabilistic choice function (Busemeyer and Stout, 2002, Equation 3d, p. 257).

10.4. Critical evaluation of the Expectancy–Valence Model

The predictions of the Expectancy–Valence Model were tested by implementing[29] the described equations into a computer algorithm and comparing the results (i.e., number of cards drawn from the four different decks) to the performance of the algorithms for the rational and the emotional player, respectively. Both are mathematically simpler than the Expectancy–Valence Model.

Generally, computational modelling can be used to disentangle the different cognitive (e.g., expected value) and motivational (e.g., sensitivity to reward and punishment) mechanisms that are operating in complex behavioural and cognitive tasks like the IGT. It can provide a theoretical basis for identifying and measuring hidden processes underlying the performance in these tasks. Within the IGT paradigm, computational modelling could be used to evaluate what happens to the player's decision strategy if one of the proposed mechanisms or systems fails (like in VMPFC patients) or to investigate what happens if one of the proposed systems (e.g., the impulsive system) gets overactive and inhibits the activity in one of the other systems (as it is assumed for the reflective system in SDIs). However, the present evaluation predominantly aims to answer the following question: Does the Expectancy–Valence Model have an obvious advantage in explanatory power when compared to a decision model that uses expected values and a limited working memory for the calculation which card to draw next (i.e., the rational player)? The rational player calculates expected values for the four different decks on the basis of the numerical net values of the last eight cards that are drawn. In each trial the deck with the highest expected value for the next card is

[29] It has to be noted that the values for the three free parameters (i.e., attention weight, update rate, and sensitivity parameter) were taken from the literature (Busemeyer and Stout, 2002), since no suitable behavioural data sets were available to our laboratory at the time of the current investigation to estimate the values by ourselves.

chosen. If there are no cards left for a deck, the expected value for this deck is set to 0. The algorithm selects a deck randomly when there is more than one deck with the highest expected value[30]. The algorithm that implements the equations described by Busemeyer and Stout (2002) computes the expected valences of the four decks for its decision strategy which card to draw next. The attention weight parameter that regulates the sensitivity to reward and punishment can vary between 0 and 1. A value near 0 is associated with hypersensitivity to reward and a value near 1 represents hypersensitivity to punishment. Thus, an attention weight of 0.5 indicates that gains and losses weigh equally in the decision process. According to Busemeyer and Stout (2002) the decision strategy of healthy human subjects can be modelled by setting the attention weight parameter to 0.35[31].

Table 6: The table shows the mean values and standard deviations (SD) of the drawn cards (100 runs) for deck A, B, C, and D with respect to the three different models implemented.

Decks	Mean	SD	Model
A	7.28	1.64	The rational player
B	15.64	2.48	
C	39.69	1.77	
D	37.39	2.03	
A	7.88	2.87	The emotional player
B	15.43	3.18	
C	39.10	2.66	
D	37.59	2.23	
A	11.00	0.00	Expectancy–Valence Model
B	9.00	0.00	Attention weight = 0.35
C	40.00	0.00	Update rate = 0.34
D	40.00	0.00	Sensitivity parameter = 0.32

The most interesting finding was that the decision strategy of the rational player seemed to be similar to the one observed in healthy human subjects, albeit with a

[30] It chooses one of the decks with equal highest expected value randomly.

[31] This value is taken from the literature (Busemeyer and Stout, 2002).

sharper separation between "good" and "bad" decks, which means that the implemented computational algorithm draws more cards from the advantageous decks than a participant without neurological impairments would do (see Table 6). There was *no* statistically significant difference between the card drawing behaviour of the rational and the emotional players. However, there were some differences, although not statistically significant, between the results obtained with the algorithm that uses expected values for its decision strategy (i.e., the rational player)[32] and the algorithm that implements the equations described by Busemeyer and Stout (2002): (1) There was no variation in the card drawing behaviour of the algorithm for the Expectancy–Valence Model. Each of the 100 complete trials led to exactly the same distribution of drawn cards with respect to the four different decks (see Table 6). This result could be due to the reported estimated value for the sensitivity parameter in healthy human subjects. The value of 0.32 ultimately led to a probability to draw a card from the deck with the highest expected valence near 1 and thus the player chose almost with certainty from the deck with the highest expected valence. However, a more conclusive evaluation of this finding would need actual behavioural data to re-estimate and evaluate the (validity of the) three free parameters reported in Busemeyer and Stout (2002). Without this further step no conclusive interpretations are warranted. (2) Another interesting result in the evaluation of the Expectancy–Valence Model was that "only" nine cards were drawn from deck B (see Table 6). This result could be due to the fact that card number 9 represents the first punishment for deck B and the highest possible punishment in the IGT, which means that the participants never encounter a loss of comparable magnitude while playing the IGT.

[32] Similar results were obtained when comparing the results of the Expectancy–Valence Model with the card drawing behaviour of the emotional player (see Table 6).

Since the algorithm chooses almost with certainty from the deck with the highest expected valence for the next card and since the very high punishment leads to an immediate switch from deck B to one of the other decks, the expected valence for deck B remains low throughout the remaining trials. Thus, one of the other three decks always has a higher expected valence for the next card and is therefore chosen by the algorithm. There is no chance to get back to deck B after the first punishment is experienced. In contrast, both the rational and the emotional player use a limited working memory system, which ensures that the algorithm is able to get back to deck B, if with reduced probability.

10.5. Refinement of the Expectancy–Valence Model

The Expectancy–Valence Model in its current formulation seems to lack any obvious advantage in explanatory power when compared to other strategies that use either the expected values and a limited working memory or additional emotional information for the computation of card drawing behaviour in the IGT. Nevertheless, its results are promising enough to do further investigations. As already mentioned in previous chapters, normal controls tend to show a high variability in their behavioural results, dependent on the actual reward/punishment schedules that are used (Fum et al., 2008; Rakovský, 2009), their individual risk-taking behaviour (Lerner and Keltner, 2000; Loewenstein et al., 2001; Raghunathan and Pham, 1999), and their individual levels of anxiety and neuroticism (Carter and Smith-Pasqualini, 2004; Suhr and Tsanadis, 2007)[33]. Thus, it seems that the model could be advanced by splitting the unimodal

[33] Another relevant aspect might be apathy or lack of motivation. Impaired performance on the IGT can be associated with decreased sensitivity to punishment but another possible interpretation could be that the losses simply not bother the participants enough to actively avoid the riskier decks. Empirical evidence (Carter and Smith-Pasqualini, 2004) suggests improved performance of healthy subjects on

dimension[34] of the attention weight parameter into two separate dimensions of valence, i.e. independent sensitivities to reward and punishment[35]. Such a refinement would make the assumptions of the Expectancy–Valence Model more consistent with the current experimental findings in the IGT literature. Such a refined version might further be able to model the observed variability in healthy human subjects[36]. We suggest the following modification of the first equation (Equation 5):

$$v(t) = r * R\,[Dt] + l * L\,[Dt]$$

Equation 5: The valence v(t) experienced after drawing a card from deck D on trial t is calculated as the added separate sensitivities to reward and punishment. The attention weight parameters r and l both can vary between 1 and 0 and reflect the distinct amount of attention given to reward and punishment. Both parameters have to be estimated from experimental data.

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the IGT when play money is replaced by real money indicating that motivation (or rather a lack of motivation) might be another important factor that should be considered in future studies. In contrast, Bowman and Turnbull (2003) did not find any significant differences in performance when replacing play money with real money.

[34] If the sensitivity to reward is high (a = 0.7), then the sensitivity to punishment is low (1 - a = 0.3). Since several studies report that high sensitivity to punishment can co-occur with high sensitivity to reward such a refinement seems necessary to capture the full range of observed (healthy) human behaviour in the IGT.

[35] Interestingly, Hornak and colleagues (2004) were able to show that sensitivity to punishment and sensitivity to reward can be *anatomically distinguished*. Sensitivity to reward is impaired in patients with *medial* orbitofrontal cortex lesions, whereas sensitivity to punishment is impaired in *lateral* orbitofrontal cortex patients. Furthermore, the right hemisphere has been associated with punishment learning and the left hemisphere has been associated with reward learning (Davidson and Irwin, 1999).

[36] Since a model is by definition partial and abstract, it is very important to clearly define the purpose and scope of the model and to outline what it might be able to explain and what it might not be able to explain (for a thorough review of the advantages and disadvantages of computational modelling see Fum et al., 2007). The refined version of the Expectancy–Valence Model predominantly aims to shed light on the high variability in card drawing behaviour in healthy human subjects in the IGT. We argue that such a refined version would be more consistent with recent empirical findings within the IGT paradigm than its original formulation.

Nevertheless, without actual behavioural data sets to estimate the two new free parameters r and l, the proposed refinement remains speculative, although quite promising, given experimental results obtained within the IGT paradigm in the last decade (Bechara and Damásio, 2002; Bechara et al., 2002; Carter and Smith-Pasqualini, 2004). Future studies will have to evaluate whether such a refinement is able to predict the performance of normal controls and different patient groups in the IGT.

11. Discussion and conclusions

Since anticipatory SCRs and the associated card drawing behaviour in the IGT may correspond to a variety of psychological mechanisms involved in the decision process, like affective responses to feedback, risk-related signals, indicators of post-decision emotional state, or markers of the ”goodness” and “badness” of different alternatives, it seems necessary to develop computational cognitive models that help explain human decision-making under uncertainty and risk in the IGT and disentangle the contributions of the assumed cognitive and motivational factors. Such models should be consistent with the IGT literature and should allow formulating new hypotheses that can be tested in future investigations.

We argue that anticipatory SCRs (i.e., somatic markers) embody the perceived risk of the four different decks in the IGT and that further autonomic parameters are needed in addition to the measurement of anticipatory SCRs (e.g., heart rate deceleration) to differentiate between the putative role of somatic markers as a stopping signal (i.e., negative somatic marker) or an approach signal (i.e., positive somatic marker) in the decision process under uncertainty and risk. Furthermore, we assume that differences in risk perception cause the inconsistencies observed in the card drawing behaviour of

healthy human subjects. We propose that a refined version of the Expectancy–Valence Model could be able to account for these inconsistencies. Since the model still needs to be fully evaluated by means of empirical data sets in future studies, these arguments are rather speculative at the moment. However, the proposed refinement seems promising, because it captures important characteristics of IGT performance in normal controls, SDIs, and VMPFC patients. For example, healthy individuals with high scores in neuroticism, who show both high sensitivity to punishment and high sensitivity to reward (Torrubia et al., 2001), tend to do well on the IGT (Carter and Smith-Pasqualini, 2004). Since it can be argued that participants only have to pay attention to the punishment component of the payoff schedule[37] to succeed in this task, it may seem unsurprising that neuroticism correlates positively with IGT performance, since neuroticism is characterized by high sensitivity to punishment. Thus, it may be assumed that the IGT predominantly measures behavioural[38] and psychophysiological reactions to punishment and that more attention to this component in the decision process within the IGT paradigm is required. The predictions of such hypotheses could be tested using the proposed refined version of the Expectancy–Valence Model. The following two sections qualify the two main hypotheses within this thesis: (1) Differences in risk aversion can account for the high variability in performance of normal controls. (2) Decreased emotional awareness of risky situations in VMPFC patients can explain their poor performance in the IGT and their superior performance when risk taking is rewarded.

[37] Only the punishment schedule varies in the IGT, whereas the magnitude of rewards is fixed across trials: \$50 for the "good" decks and \$100 for the "bad" decks.

[38] Deck position is not varied across participants in the IGT and thus can lead to the emergence of deck preferences that reflect a location bias rather than advantageous and disadvantageous decision-making, respectively. Thus, future studies should vary deck position systematically to rule out this possibility.

11.1. Healthy subjects: Are somatic markers indicators of risk?

Since it can be argued that distinct sensitivities to punishment and reward are likely to play a role in the decision process of healthy human subjects in the IGT (Carter and Smith-Pasqualini, 2004), future studies should systematically investigate this possibility in healthy subjects. For example, the behavioural performance and psychophysiological responses of healthy participants in the Rogers Decision-Making Task (RDMT) in which subjects choose between higher and lower probability gambles and the IGT could be compared. According to Monterosso and colleagues (2001), IGT performance correlates significantly with performance in temporal discounting tasks but is less clearly associated with RDMT performance. They argue that their participants (i.e., cocaine-dependent individuals) seem to have problems in considering future outcomes accurately rather than having problems with risk perception. However, it remains open to what extent these results hold for healthy individuals. In our view, it seems that SDIs show an *increased* sensitivity to reward paired with a *decreased* sensitivity to punishment. Thus, a comparison of both tasks in normal controls is highly encouraged. The proposed refined version of the Expectancy–Valence Model could be used to evaluate the contribution of both factors independently, since it regards the two dimensions — sensitivity to reward and sensitivity to punishment — separately. However, the integration of neuroscientific and psychological experimental results together with insights gained from computational modelling can help to qualify existing decision-making theories and can be used to generate testable hypotheses. Such an interdisciplinary account has the potential to answer questions that can not be solved within the boundaries of each academic discipline alone.

To conclude, it seems that somatic markers, as operationalized by means of anticipatory SCRs, may represent emotion-related signals that indicate the riskiness of the different decks in terms of different sensitivities to reward and punishment. Thus, variations in these sensitivities may account for the high variability in behavioural and psychophysiological results obtained in healthy human subjects in the IGT and related decision-making tasks. The proposed refined version of the Expectancy–Valence Model may help explain the experimental findings within the IGT paradigm.

11.2. VMPFC patients: Do they show decreased emotional awareness of risky situations?

Decreased emotional awareness of risky situations in VMPFC patients may explain their bad performance in the IGT and superior performance when risk-taking is rewarded (Shiv, Loewenstein and Bechara, 2005). This hypothesis is encouraged by current empirical findings and consistent with the proposed refined version of the Expectancy–Valence Model, although the predictions for VMPFC patients are less clear than for healthy subjects. In VMPFC patients, an important structure for decision-making is destroyed. Thus, a variety of explanations for the observed deficits are possible. The most robust finding in VMPFC patients is the fact that anticipatory SCRs are entirely missing and that these psychophysiological null-findings correlate significantly with impaired decision-making in the IGT. However, it could be argued that the observed relationship is “only” correlational, since there is no need to infer a causal relationship between measured autonomic responses and observed behavioural results (Amiez et al., 2003; Crone et al., 2004). Furthermore, other psychological mechanisms (e.g. working memory, affective shifting, contingency reversal learning, and motivation) necessary for accurate decision-making could be affected instead.

Thus, several other possible explanations exist that may account equally well for the observed deficits. For example, disruption of working memory functionality might be able to explain why VMPFC patients do worse on the IGT (Bechara et al., 1998). Moreover, information about individual differences in decision-making before lesion onset is generally not available. Therefore, individual behavioural responses cannot be directly compared to the behaviour before the accident, surgery, or stroke. However, it could be shown that VMPFC patients display decreased emotional awareness of risky situations (Shiv, Loewenstein and Bechara, 2005) indicating that at least one of the proposed affective dimensions (i.e., sensitivity to punishment) is affected. We argue that the proposed refined version of the Expectancy–Valence Model could be used to study the behavioural deficits in these patients in more detail. Furthermore, we assume that this model would be able to help explain why VMPFC patients have difficulties to overcome their tendency to choose the immediate reward options in spite of the experienced high losses.

11.3. Final remarks

To conclude, anticipatory SCRs that are triggered by certain situations, like the IGT, are involved in aiding the decision process under uncertainty and risk (Bechara et al., 1997; Bechara et al., 1996). Furthermore, they might assist this process by narrowing down the possible alternatives how to (re)act. This might be accomplished by refuting disadvantageous and endorsing advantageous options. Thus, emotion-related markers might act as highly relevant signals in decision-making under uncertainty and risk that speed up the decision process. However, these affective reactions can be both beneficial and disruptive depending on the circumstances (Shiv, Loewenstein and Bechara, 2005). Distinct sensitivities to reward and punishment might be able to

explain their adaptive role in decision-making more thoroughly. We argue that a two-dimensional conceptualization of emotion-related signals might be able to explain the huge range of experimental findings obtained within the IGT paradigm in the last decade. Furthermore, we assume that the proposed refined version of the Expectancy–Valence Model is able to contribute significantly to the explanation of these empirical results. Finally, we propose that the refined version is able to contribute to the on-going discussion on the inconsistent empirical results in healthy subjects and to a better understanding of the actual role of emotion-related signals in decision-making, in particular under uncertainty and risk.

The SMH is not refuted but seriously challenged by recent research. New sophisticated experiments are required to study the function of anticipatory SCRs more thoroughly. Other psychophysiological measurements should be included, especially heart and respiratory rate, since these autonomic parameters are known to be able to distinguish between positive and negative emotions (Rainville et al., 2006) and thus between positive and negative somatic markers. The current investigation suggests that different sensitivities to punishment and reward play a major role with respect to the card drawing behaviour in the IGT. The proposed refined version of the Expectancy-Valence Model can be used to model the behaviour of healthy individuals and VMPFC patients in the IGT and to develop new experimental designs for future studies.

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14. Appendix

14.1. Abstract (English)

Damásio's Somatic Marker Hypothesis (SMH) provides a plausible neurobiological explanation for the deficits observed in real-life decision-making, and for impairments found in patients with ventromedial prefrontal cortical (VMPFC) lesions in Bechara's Iowa Gambling Task (IGT). Roughly, the SMH assumes that overt reasoning processes are preceded by covert emotional biases (i.e., somatic markers) that help to decide advantageously under uncertainty and risk. Recent studies suggest that similar mechanisms are responsible for the inferior performance of substance dependent individuals (SDIs) and the superior performance of players with high scores in neuroticism. Bechara proposes an imbalance between reflective and impulsive processes in decision-making as the cause of observed deficits: In VMPFC patients, the reflective processes would be directly affected, whereas hyperactivity in the amygdala in SDIs would lead to an attenuation of reflective processes and thereby to sensitivity to immediate reward, and indifference to possible negative future consequences of decisions. Basing our conclusions on a refined version of the Expectancy-Valence Model (originally developed by Busemeyer and Stout), we argue that distinct sensitivities to punishment and reward might explain the overall performance of VMPFC patients and normal controls in the IGT. The following two hypotheses are endorsed by the model thus derived: (1) Differences in risk aversion can account for the high variability in performance of normal controls. (2) Decreased emotional awareness of risky situations in VMPFC patients can explain their poor performance in the IGT and their superior performance when risk taking is rewarded.

14.2. Abstrakt (Deutsch)

Damásio's Hypothese der Somatischen Marker (SMH) stellt eine plausible neurobiologische Erklärungsmöglichkeit für die in Patienten mit Läsionen im ventromedialen präfrontalen Kortex gefundenen Defizite im Entscheidungsverhalten dar. Diese Beeinträchtigungen betreffen vor allem die Performanz dieser Patienten in der von Bechara entwickelten experimentellen Spielsituation, genannt „Iowa Gambling Task" (IGT). Die SMH besagt, dass Entscheidungsprozeße durch unbewußte emotionale Signale (d.h., Somatische Marker) beeinflußt werden können. Aktuelle Studien belegen, dass ähnliche Mechanismen auch für die Beeinträchtigungen von substanzabhängigen Personen in der IGT verantwortlich sind. Interessanterweise, zeigen Personen mit hohen Neurotizismus Werten vorteilhaftes Entscheidungsverhalten in diesem experimentellen Paradigma. Bechara erklärt sich diese Befunde dadurch, dass es bei Entscheidungsprozessen zu einem Zusammenspiel zwischen reflektiven und impulsiven Prozessen kommt. In Patienten mit Läsionen im ventromedialen präfrontalen Kortex sind die reflektiven Prozesse direkt beeinträchtigt, während bei substanzabhängigen Personen eine Überaktivierung, der für die impulsiven Prozesse relevanten Gehirnstrukturen (insbesondere der Amygdala), die reflektiven Prozesse im ventromedialen präfrontalen Kortex schwächt: Es kommt zu einer Überempfänglichkeit für die belohnenden Effekte von Gewinnen und zu einer indifferenten Haltung gegenüber möglichen künftigen Verlusten in der IGT. Unsere Schlußfolgerungen basieren auf einer überarbeitenden Version des von Busemeyer und Stout vorgeschlagenen „Expectancy-Valence Models" zur Erklärung des Spielverhaltens in der IGT. Wir behaupten, dass unterschiedliche Sensitivitäten für Gewinne und Verluste die beeinträchtigte Performanz von Patienten mit Läsionen im ventromedialen präfrontalen Kortex und

die Variabilität in den Befunden gesunder Probanden in der IGT erklären können. Die folgenden zwei Hypothesen werden durch dieses Model nahe gelegt und in der vorliegenden Arbeit diskutiert: (1) Die gefundenen Performanzunterschiede im Spielverhalten gesunder Versuchspersonen in der IGT lassen sich durch Unterschiede in ihrer Risikowahrnehmung (d.h., durch unterschiedliche Sensitivitäten für Gewinne und Verluste) erklären. (2) Eine verminderte emotionale Wahrnehmung des Risikoaspektes von Spielsituationen in Patienten mit Läsionen im ventromedialen präfrontalen Kortex kann die gefundenen Beeinträchtigungen in der IGT und die gefundene überlegene Spielstrategie in Spielsituationen, die risikoreiches Verhalten belohnen, erklären.

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