

Introduction

Theories of Peripheral Feedback and Emotion

Charles Darwin, a man whom many diverse fields rightfully claim as one of their founders, made lasting contributions to the field of psychology. Many of the main contributions are detailed in *The Expression of the Emotions in Man and Animals* (Darwin, 1965), a work that emphasized such seminal ideas as cross-species comparison for better understanding of human psychology. A particularly tantalizing idea introduced in this work, one that has proven to be irresistible to generations of scientists concerned with emotion, is that certain bodily responses (in this case, responses of the “sensorium”) play a key role in causing emotional states.

Much of Darwin’s work in *The Expression* focused on the habitual connection of a certain body state with a particular emotional state. After years of connecting the excretion of tears with grief, for example, these responses become strongly linked. Further, Darwin even suggested that in some cases, bodily responses to an emotional situation might cause cognitive and behavioral changes. When describing a child who jumps and squeals when reacting joyfully, Darwin draws causal arrows in the following fashion: “Joy quickens the circulation, and this stimulates the brain, which again reacts on the whole body” (Darwin, 1965). An excited sensorium thus causes an overflow of “nerve-force,” resulting in emotional behaviors. The idea that bodily responses could precede and impact the cognitive experience of emotion was one that influenced a number of researchers.

Inspired by Darwin's exploration of the body's influence on emotion, William James and Carl Georg Lange (James, 1893; Lange & James, 1922) developed what is known as the James-Lange Theory of Emotion, which sparked the debate about whether emotions drive their associated body states, or vice-versa. For those taking a "common sense" view of emotion, it seems reasonable that bodily responses such as a pounding heart or sweaty palms associated with fear, anger, or other intense feelings are merely byproducts of the emotion itself. However, in the late nineteenth century, James (1893) proposed the following counterintuitive hypothesis concerning the more intense, or "coarse" emotions:

Our natural way of thinking about these coarser emotions is that the mental perception of some fact excites the mental affection called the emotion, and that this latter state of mind gives rise to the bodily expression. My theory, on the contrary, is that *the bodily changes follow directly the perception of the exciting fact, and that our feeling of the same changes as they occur IS the emotion.*

In other words, emotion is caused by changes in body state. Lange simultaneously developed similar ideas (Lange & James, 1922). According to this view, the body was seen as a "sounding board," one sufficiently complex to produce every conceivable variation in intense emotions (Lange & James, 1922).

However, the James-Lange theory was generated solely from introspection. The main thought-experiment James referred to as evidence for his theory was that if one thought of an emotional experience, such as finding an absurd object humorous, but removed all bodily symptoms, such as laughter, no conscious phenomena were left to associate with that emotion. Although the James-Lange theory apparently provides a good deal of introspective appeal (indeed, James noted that those individuals who disagreed with the conclusions of his thought-experiment hadn't understood it correctly), it is not supported by any experimental proof, a fault acknowledged by its creators (Lange & James, 1922).

More recent theories have attempted to causally relate bodily state to emotion by varying the logic of the James-Lange theory. Schachter (1971) argued that the type of emotion (fear as opposed to anger, for example) was determined by cognitive thought, but that the intensity of this emotion was determined by bodily responses. This conclusion was based on experimental research suggesting that injection of epinephrine did not cause any emotion in particular, but increased subjective reports of the intensity of different kinds of emotions influenced by laboratory stimuli, including fear, anger and amusement (Schachter, 1971).

Others have argued for theories of “facial-feedback,” hypothesizing that facial expressions themselves can contribute to the body state and cognitive experience of the associated emotion. When people make facial movements associated with different emotions, such as smiling or frowning, their cognitive experience can change accordingly. For example, when individuals are instructed to contract certain facial muscles, forming a smile, they find certain stimuli, such as cartoons, more pleasurable, and report happier general moods than when they contract other facial muscles, forming a frown, even though they remain unaware of the nature of their expressions (Laird, 1974; Zajonc, Murphy, & Inglehart, 1989). Other evidence suggests that performing these directed facial actions in order to mimic different emotional facial expressions can produce physiological changes that are similar to (though much stronger than) those seen when the associated emotion is produced by cognitive instruction, such as imagining an appropriate emotional situation (Ekman, Levenson, & Friesen, 1983).

Theories of facial-feedback thus form a subset to the broad claims of earlier researchers. However, no matter what their scope, theories of causal bodily influence on emotion, or theories of “peripheral feedback,” have been challenged on a number of different grounds. First, a number of studies have suggested that destroying bodily responses does little to affect emotion.

In early experiments, destruction of the vagus nerve in dogs, which disconnects the main connections between the brain and most major organs, produced no discernible effect on emotional behavioral responses such as barking, whining, or expressions of fear (Cannon, 1927; Cannon, 1931). Similarly, destruction of the entire sympathetic division of the autonomic system in cats produced no changes in emotional displays (Cannon, 1927). Although it remains unclear whether the experience of emotion in these animals remained unchanged, or even whether normal dogs and cats can experience emotion in a way at all similar to human cognition, these results are striking. Further, more recent studies with humans support these suggestive results. Individuals with spinal cord damage (resulting in the loss of many connections between body and brain) were found to have few bodily correlates of emotion, but reported that their emotions, and specifically the intensity of these emotions, were unchanged by their injury (Bermond, Nieuwenhuys, Fasotti, & Shuerman, 1991). Additionally, numerous experiments with beta-blockers, drugs that reduce the activity of the sympathetic nervous system, have shown that these drugs inhibit peripheral responses, but do not inhibit the cognitive experience of emotion (Reisenzein, 1983).

Theories of peripheral feedback all depend on a close relationship between a particular body state and a corresponding emotional state. In the James-Lange theory, the body was viewed as a complex instrument capable of representing any intense emotion (Lange & James, 1922). Later theories of peripheral feedback necessarily retain similar, though perhaps more limited, claims. However, numerous theorists have challenged the view that emotions are so tightly bound to the body's particular state. Indeed, given the seemingly infinite shades of human emotion, it is unclear that one or more bodily states could possibly correspond to each (Rolls, 2000; Wagner, 1989). It is also telling that a wide range of emotions is still possible

during abnormal physiological states such as chills, fever and hypoglycemia. If body states were tied closely with emotional states, it would be expected that these states would be associated consistently with large emotional responses, perhaps manifesting as rage or fear (Cannon, 1927), and that these states would continue for the duration of the condition. However, this is not the case.

Finally, theories of peripheral feedback predict that emotions caused by external sources, such as direct changes in body state, will be associated with the same peripheral effects as if these same emotions were caused by internal sources, such as cognitive imagery. Because the same emotion is evoked, a similar body state must be the cause. This has not been supported. Studies comparing the physiology of emotions created by external versus internal stimulation of subjects have uncovered a greater bodily response in the externally generated emotions. For example, when subjects are told to contract certain facial muscles into emotional expressions, their autonomic physiological response, as measured by skin conductance, heart rate, finger temperature and muscle activity, was significantly more pronounced than during analogous imagery tasks, even though the strength of the experienced emotion was judged to be similar (Levenson, Ekman & Friesen, 1990). Similarly, a number of different physiological variables were significantly higher when subjects were presented with emotional stimuli such as anger-producing tasks or fearful stimuli, compared to cognitive imagery in which subjects imagined emotional autobiographical situations, though the emotions produced were rated nearly equally (Stemmler, 1989).

In recent years, theories of peripheral feedback have lost sway because of these criticisms. Despite this wide-ranging evidence, however, a new theory of emotion-regulated decision-making has invoked the same basic tenets of peripheral feedback upon which the

James-Lange theory of emotion was based. This theory, called the somatic marker hypothesis, posits that bodily responses are necessary for decision-making, and that they guide an individual, consciously or unconsciously, to choose correct alternatives (Damasio, 1994). This thesis will attempt to challenge the validity of this theory by theoretically and experimentally critiquing its supporting arguments.

The Somatic Marker Hypothesis

The somatic marker hypothesis, proposed by a research group led by Antonio Damasio, can be briefly described as follows (e.g., Tranel, Bechara, & Damasio, 2000; Damasio, 1994). Structures in the prefrontal cortex, especially the orbitofrontal cortex, associate differing body states to different higher-order stimuli. Body state information includes all bodily components, including the viscera (the basis of the James-Lange theory of emotion) and two additional parts: musculoskeletal areas and the internal milieu. Associations between the body and various stimuli are represented by the prefrontal cortex and a wide range of connecting neural circuitry.

When similar stimuli are subsequently experienced, the orbitofrontal cortex then reactivates these stored associations, either triggering the associated bodily response which, in turn, is processed again by neural circuitry (the “body loop”), or triggering only the neural representation without any actual bodily change (the “as-if body loop”). These reactivated representations serve as positive or negative “somatic markers,” which bias the individual’s decision-making. Somatic markers may be expressed in physical terms as sweaty palms or a churning stomach, and may operate consciously or unconsciously. An example of a somatic marker “in action” is that an individual might experience sweating on the palms while contemplating a particular decision, thus biasing the individual against that option.

A few observations are notable at this point. First, the somatic marker hypothesis differs from some earlier theories of peripheral feedback (such as the James-Lange theory of emotion) in that the brain is seen as partly responsible for creating emotions in the case of the “as-if body loop” (Damasio, 1999). That is, the body is not seen as the only causal agent in creating somatic markers. Second, it is clear that the somatic marker hypothesis is strongly dependent on peripheral feedback, and as such, is subject to many of the critiques mentioned earlier.

Evidence for the Somatic Marker Hypothesis: Individuals with Prefrontal Damage

The majority of evidence taken to support the somatic marker hypothesis comes from studies comparing the behavior of patients with damage to the ventromedial prefrontal and orbitofrontal sectors of the cortex to that of healthy individuals. Case studies and laboratory experiments involving moral rule judgment tasks have suggested that the behavior of individuals with such damage is characterized by a lack of decision-making ability concerning social situations and decisions about the future (Damasio, Tranel, & Damasio, 1991; Anderson, Bechara, Damasio, Tranel & Damasio, 1999; Hornak, Rolls, & Wade, 1996; Rolls, 1996; Rolls, Hornak, Wade, & McGrath, 1994; Shallice & Burgess, 1991). Further, an abnormal lack of somatic responsiveness (as measured by Skin Conductance Responses, or SCRs) was seen in individuals with frontal damage when presented with pictures of emotionally arousing scenes, such as nudity or social disaster (Damasio, Tranel, & Damasio, 1990; Damasio, Tranel, & Damasio, 1991). Individuals with frontal damage, then, do not generate normal somatic responses to emotional stimuli, and have deficits in their social decision-making. It is thus hypothesized that normal somatic markers are necessary for proper decision-making, and that the

deficits in decision-making seen in individuals with frontal damage are directly due to the loss of these somatic markers.

Although this interpretation of results is certainly not the only one (as will soon be discussed), there is at least one tenet of the somatic marker hypothesis that is supported by a number of papers in the literature.

The Orbitofrontal Cortex and Decision-Making

A number of studies generally agree that certain areas in the prefrontal cortices (especially the orbitofrontal cortex), usually in concert with the amygdala, are important in the evaluation of reward and punishment in the process of decision-making. This general agreement is based on a wealth of experiments using many different perspectives and techniques.

First, studies suggest that areas in the prefrontal cortex are involved in processing the punishing or rewarding value of stimuli. In a functional magnetic resonance imaging (fMRI) study with regular cigarette smokers, administration of nicotine was associated with increases in blood flow to areas of the frontal lobes and the limbic system, accompanying a feeling of “drug liking” in the subjects (Stein, Pankiewicz, Harsch, Cho, Fuller, Hoffmann, Hawkins, Rao, Bandettini, & Bloom, 1998). Similarly, increased prefrontal cortical activation was seen in an fMRI study after cocaine administration (Breiter, Gollub, Weisskoff, Kennedy, Makris, Berke, Goodman, Kantor, Gastfriend, Riorden, Mathew, Rosen, & Hyman, 1997). In addition to pleasant stimuli, the prefrontal cortex has also been shown to be selective for unpleasant stimuli. In a rare human single-unit recording study involving an epileptic man who tested normally on tests of frontal function, a number of prefrontal neurons were discovered to be selective for

emotionally aversive pictures (Kawasaki, Adolphs, Kaufman, Damasio, Damasio, Granner, Bakken, Hori, & Howard, 2001).

Evidence also suggests that the orbitofrontal cortex responds differently depending on the affective nature of stimuli. For example, using fMRI, it was discovered that the orbitofrontal cortex is more strongly activated when one hand was touched pleasantly with velvet compared to neutrally with wood (Francis, Rolls, Bowtell, McGlone, O'Doherty, Browning, Clare, & Smith, 1999). This graded activity of the orbitofrontal cortex with respect to the affective nature of the stimulus has been thoroughly investigated by studies comparing orbitofrontal responses to pleasant and aversive tastes. In an fMRI study, different regions of the orbitofrontal cortex were activated during oral administration of a pleasant solution (glucose) compared to an aversive solution (salt; O'Doherty, Rolls, Francis, Bowtell, & McGlone, 2001). Single-unit recording techniques in animals have also revealed that taste-sensitive neurons in the orbitofrontal cortex respond more strongly when the animal is hungry than when satiated (Mora, Avrith, Phillips, & Rolls, 1979; Rolls, Yaxley, & Sienkiewicz, 1990). Further, this effect has been replicated in humans (and while examining a much greater number of neurons at a time) using fMRI: a region of the orbitofrontal cortex was shown to become less active as hungry subjects ate a food to satiety (O'Doherty, Rolls, Francis, Bowtell, McGlone, Kobal, Renner, & Ahne, 2000). The prefrontal cortex has also been broadly implicated in organizing associative learning; for example, single-unit recording in animals has shown that different groups of neurons in the prefrontal cortex fire differently during presentation of different stimuli associated with rewarding or aversive responses (Watanabe, 1990; Asaad, Rainer, & Miller, 1998).

Studies involving naturalistic stimuli such as different tastes so far have suggested that the orbitofrontal cortex is involved in the processing of punishing and rewarding stimuli.

Recently, a number of studies have examined the orbitofrontal cortex's activation patterns for more abstract, perhaps "higher-order" cognitive stimuli not involving basic stimuli such as taste and touch, and have found similar results. In these studies, cognitive experiences such as winning and losing during a game are assumed to be in some sense rewarding and punishing, respectively. In one experiment using positron emission tomography (PET), it was discovered that increased activity in the orbitofrontal cortex was induced by positive and negative feedback when subjects were given a guessing task (Elliott, Frith, & Dolan, 1997). In an fMRI study, subjects in a pseudo-competitive response time task displayed increased orbitofrontal cortex activation for winning, but not losing (Zalla, Koehler, Pietrini, Basso, Aquino, Sirigu, & Grafman, 2000).

Subjects' neural responses have also been investigated while subjects perform tasks that use money, or play money, as the rewarding or punishing stimulus. In one PET experiment, subjects participated in a go-no go decision task in which money was given as a reward for correct responses. In this study, the orbitofrontal cortex was significantly activated for reward (Thut, Schultz, Roelcke, Nieunhusmeier, Missimer, Maguire, & Leenders, 1997). In another study in which the task involved both acquiring money for correct decisions and losing money for incorrect decisions, the orbitofrontal cortex was significantly activated for both financial punishment and reward (Elliott, Friston, & Dolan, 2000), a result that was later replicated with play money in a similar task (O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001).

In addition to processing the rewarding and punishing value of stimuli, the orbitofrontal cortex has also been implicated in decision-making based on these reward values. The prefrontal cortex has also been shown to be important in goal-directed behavior, and surgical removal of areas in the prefrontal region leads to deficits in such behavior (Petrides, 1996; Goldman-Rakic,

1996). Additionally, decision-making in a risk-taking decision task has been associated with increased orbitofrontal activity in a recent PET study (Rogers, Owen, Middleton, Williams, Pickard, Sahakian, & Robbins, 1999).

Animal research has been particularly instructive concerning the importance of the orbitofrontal cortex in decision-making. In particular, lesion techniques in monkeys have allowed researchers to examine the connections between the prefrontal cortex and the amygdala, a connection that has proven to be particularly important for different kinds of decision-making. Monkeys with lesions of the prefrontal cortex but not the amygdala on one hemisphere, and of the amygdala but not the prefrontal cortex on the opposite hemisphere, followed by destruction of the corpus callosum and anterior commissure are left with unilateral amygdala and prefrontal cortical structures that are functional, but unable to communicate with each other (Gaffan, Murray, & Fabre-Thorpe, 1993). Behavioral deficits in these animals are then attributed to a lack of connection between the amygdala and prefrontal cortex. Using this surgical technique, researchers have shown that the ventromedial prefrontal cortex, along with the amygdala and interaction between these two structures, is necessary for association learning (Gaffan et al., 1993) and reinforcer devaluation learning, in which an adjustment in decision-making behavior is necessary after a change in reinforcer value (Malkova, Gaffan, & Murray, 1997; Baxter, Parker, Lindner, Izquierdo, & Murray, 2000). Single-unit recording studies in rats support these results convincingly. When rats gradually learn to associate certain odors with either pleasant or aversive stimuli, certain neurons in both the orbitofrontal cortex and amygdala gradually become selective to either pleasant or aversive conditions (Schoenbaum, Chiba, & Gallagher, 1998; Schoenbaum, Chiba, & Gallagher, 1999). That is, certain neurons in both areas fire more during the presentation of odors that are associated with pleasant or aversive stimuli, and this increase in

firing rate becomes more pronounced as training proceeds, and rats improve behaviorally on the task.

These studies have suggested that the prefrontal and orbitofrontal cortices play an important role in both evaluating the rewarding or punishing nature of stimuli and making decisions based on this information. Indeed, one theory of decision-making proposed by Edmund T. Rolls, which, unlike the somatic marker hypothesis, does not invoke any processing of peripheral responses, is based on the function of the orbitofrontal cortex (Rolls, 1990; Rolls, 1996; Rolls, 1999). This theory is grounded in findings from single-unit recording studies in primates in which neurons in the orbitofrontal cortex selective for either rewarding stimulus associations (“reward neurons”) or non-rewarding stimulus associations (“non-reward neurons”) can be identified (Rolls, 2000; Thorpe, Rolls, & Maddison, 1983). Briefly, this theory states that the orbitofrontal cortex identifies the rewarding or punishing values of stimuli and identifies associations between this set of stimuli and others. Decision-making is thus theorized to be based on firing of either reward neurons or non-reward neurons, with the individual choosing the alternative to which reward neurons are attuned (Rolls, 1999; Rolls, 2000). Although this is only one theory involving orbitofrontal cortical functioning, it is instructive in noting that the orbitofrontal cortex may be involved in decision-making without necessarily processing peripheral inputs.

The Somatic Marker Hypothesis: Points of Support/Non-Support

In this review of the literature, two main arguments behind the somatic marker hypothesis have been widely supported. First, damage to the orbitofrontal cortex has indeed been found to create behavioral deficits in emotion and decision-making. Second, a wide variety of studies

have emphasized the importance of the orbitofrontal cortex in processing rewarding and punishing stimuli and making decisions based on this information. However, as reviewed, there is no evidence supporting the existence of somatic markers and the theory that they precede decision-making. In order to argue effectively for the somatic marker hypothesis, it is necessary to demonstrate the existence of these markers and to show that they play a primary role in mediating outcomes on a decision-making task. If, in contrast, such evidence is lacking, other theories that do not posit a role for peripheral feedback would, by Occam's Razor, be preferable. For example, theories of prefrontal function and decision making such as Rolls' do not create the complicated peripheral response pathway that is the main feature of the somatic marker hypothesis. Without direct evidence of somatic markers, the somatic marker hypothesis remains partly speculative.

The Damasio group has addressed the lack of evidence for the existence of somatic markers by using a novel paradigm called "the gambling task." This task allows for exploration of the somatic marker hypothesis not only with frontal patients, but with normal subjects as well. The gambling task is the sole paradigm demonstrating what the Damasio group argues are the correlates of somatic markers and the mechanism of body state preceding decision-making. By exploring real-world decision-making with this paradigm while recording SCRs, they believe that they are able to adequately address the counter-argument against data from individuals with prefrontal cortex damage. In this thesis, however, it is argued that the methodology of this paradigm is flawed, and that results involving normal subjects that are taken by the Damasio group to support the somatic marker hypothesis may actually be accounted for by a logical, purely cognitive explanation. Further, if this potential flaw proves to be real, this may pose a

substantial problem for the somatic marker hypothesis, because little evidence for that theory comes from studies of non-brain-damaged individuals.

The Gambling Task: Evidence from Both Orbitofrontal Patients and Normal Subjects

The gambling task is meant to simulate real-world decision-making in the laboratory, in that the premises and outcomes are not made clear to the subject at the beginning of the task, but must be determined through experience (Bechara, Damasio, Damasio, & Anderson, 1994). In this task, subjects choose cards that give play money punishments and rewards, distributed as follows (See Figure 1; Bechara, Damasio, Damasio & Lee, 1999). Subjects are given an initial amount of \$2000 in play money and are presented with four decks of cards, which, unbeknownst to the subjects, are comprised of two “bad” decks (decks A and B, from which the subject would ultimately lose money) and two “good” decks (decks C and D, from which the subject would ultimately earn money), and are asked to choose cards one at a time from any of the decks in order to maximize their profits. After a total of 100 cards have been picked, the task ends.

Each time a card is chosen from one of the good decks, the subject receives \$50, while each time a card is chosen from one of the bad decks, the subject receives \$100. However, there are occasional punishments in addition to these automatic rewards. For every 10 cards chosen from the bad decks, in addition to giving \$1000 in automatic reward, the bad decks punish the subject with a total of \$1250 in loss. The difference between the decks is that in deck A, over the course of 10 cards, the subject

receives five unpredictable punishments ranging from \$150 to \$350, for a total of \$1250, whereas in deck B, the \$1250 loss comes in the form of one large punishment. For every 10 cards chosen from the good decks, in addition to giving \$500 in automatic reward, the good decks punish the subject with a total of \$250 (in the form of 5 punishments ranging from \$25 to \$75 in deck C and one large punishment of \$250 in deck D). Thus, decks A and B are equivalent in terms of net loss. Similarly, decks C and D are equivalent in terms of net gain. The difference is that in decks A and C, punishment is of smaller magnitude and is more frequent than punishment in decks B and D.

Initially, the gambling task was conducted without concurrently measuring SCRs. It was found that, indeed, patients with prefrontal damage picked significantly fewer cards from the good decks and significantly more cards from the bad decks, than did normal controls (Bechara, et al., 1994). Subsequently, experimenters measured SCRs from individuals with prefrontal damage and control subjects during the gambling task, and divided these physiological responses into three classes: 1) reward SCRs: recorded for a 4-second period starting immediately after the subject received a card for which there was a reward and no penalty, 2) punishment SCRs: recorded for a 4-second period starting immediately after the subject received a card for which there was reward immediately followed by punishment, and 3) anticipatory SCRs: recorded during the time between the end of the money exchange and the next card selection (Bechara, Tranel, Damasio, & Damasio, 1996). Thus, reward and punishment SCRs are expected to reflect a subject's reaction to receiving a card, while anticipatory SCRs are expected to reflect the subject's consideration of which card to select next. All three classes of

SCRs may be associated with orbitofrontal cortex activity, which, as discussed earlier, is important not only for the evaluation of reward and punishment, but also for decision-making based on this information.

In gambling task studies in which SCRs were recorded, experimenters found that after a number of cards, normals began to generate anticipatory SCRs before card selections. Additionally, normals generated higher-magnitude anticipatory SCRs before selections of cards from the bad decks compared to SCRs recorded before selections of cards from the good decks (Bechara et al., 1996). Frontal patients did not develop anticipatory SCRs at all, let alone differences between decks (Bechara et al., 1996). Finally, there was no significant effect of group (normal subjects versus patients) when magnitude of reward and punishment SCRs were compared (Bechara et al., 1996).

In an extension of this work, experimenters modified the Gambling task paradigm such that after 20 cards and every 10 cards thereafter, the subjects were asked about their knowledge of the game (Bechara, Damasio, Tranel, & Damasio, 1997). In this experiment, again normal subjects generated higher-magnitude anticipatory SCRs prior to selections of cards from the bad decks relative to the good decks (Bechara et al., 1997). Further, after this anticipatory SCR difference had been established (although not to a level of statistical significance), by approximately card 50, subjects began to say that they had a “hunch” about the nature of the task, which later solidified into conceptual knowledge concerning the good and bad decks (Bechara et al., 1997).

The Damasio group has made several conclusions about these data. First, the SCRs recorded during these experiments are taken to be correlates of the proposed somatic markers. Second, the Damasio group has interpreted the data as evidence that

not only do somatic markers guide decision-making, but also that in some cases these somatic markers can guide decision-making before conscious knowledge does (Bechara et al., 1997). Finally, they concluded that the failure to generate somatic markers seen in individuals with ventromedial prefrontal damage is the direct cause of the behavioral deficit of these individuals on the card task (Bechara et al., 2000a).

Problems with the Gambling Task

The hypothesis of this thesis is that normal subjects' increased anticipatory SCRs to selections of cards from the bad decks can be explained by consciously-driven processes rather than the somatic marker hypothesis advocated by the Damasio group. Further, it is argued that it is unnecessary to appeal to the somatic marker hypothesis to explain the behavioral deficits of patients with ventromedial prefrontal damage, both in general and as displayed in the gambling task. Three main problems with the logic of the Damasio group concerning the gambling task are identified.

Magnitude Differences Between the Good and Bad Decks

In each of the gambling task experiments described so far (Bechara et al., 1994; Bechara et al., 1996; Bechara et al., 1997), an important difference is found between the good and bad decks. In these experiments, the "per-card" magnitude of reward and punishment is higher for the bad decks than for the good decks (see Table 1). Every time a subject turns over a card from the bad decks, an automatic reward of \$100 occurs, whereas the automatic reward for cards from the good decks is only \$50. Likewise, the magnitude of "per-card" punishment is higher for cards from the bad decks

compared to cards from the good decks. This fact, in combination with studies showing that higher-magnitude SCRs can be generated in response to arousal (e.g., Matzman, Kantor, & Langdon, 1966), makes it easy to see that subjects' increased SCRs to selections from bad decks may simply result from higher arousal coinciding with a higher-magnitude choice. Although further studies using the gambling task have been conducted more recently (Bechara, Damasio, Tranel, & Anderson, 1998; Bechara, Damasio, Damasio & Lee, 1999; Bechara, Tranel, & Damasio, 2000), the methodology used in these studies has, in some cases, not been changed, or if it has been modified, anticipatory SCRs have not been recorded. Thus, the interpretation that the anticipatory SCR difference may be the result of a reaction to magnitude difference rather than a correlate of somatic markers remains unaddressed.

The argument could be made, however, that by having two good and bad decks that differed in magnitude of punishment, the Damasio group has already tested for magnitude differences. For example, because one bad deck costs the subject money through five small punishments, each ranging from \$100 to \$350, while the other bad deck costs the subject money through one much larger loss of \$1250, it may seem as though this controls for magnitude differences. By this logic, if per card magnitude is the driving factor behind anticipatory SCR differences, then differences between the SCR data of the differing bad and good decks (between A and B or C and D) should have been found (none were). However, it is argued that this manipulation is insufficient to test for the effects of magnitude differences.

The Damasio group's design does not vary the magnitude of the reward, but only the magnitude of punishment. This seems particularly problematic if one views the

design with an eye towards the subject's conscious knowledge of the game. That is, the difference in the magnitude of reward of the good and bad decks is especially salient to the subjects because each time the subject turns over a good card, he or she automatically receives a consistently lesser amount than when a bad card is turned. Punishments are much less consistent in terms of both occurrence (punishments do not occur upon the turning of most cards, whereas rewards occur with each card turn) and amount (amount of punishment varies whereas amount of reward remains constant for each deck), and thus harder for the subject to keep track of. During the task, subjects should be juggling information about their current game status, potential strategies, and other factors influencing the game, and it is likely that the consistent difference in reward between the bad and good decks will be much more salient to the subjects than the difference in punishment magnitude.

Poor Temporal Resolution for Comparing Knowledge and SCR Differences

A main claim of the somatic marker hypothesis is that somatic markers are necessary for, and can precede, conscious decision-making (e.g., Damasio, 1994; Tranel et al., 2000). Evidence for this claim, provided from the gambling task experiments, is based on a tenuous temporal comparison of SCR activity and conscious knowledge (Bechara et al., 1997). After 50 card turns in the gambling task, subjects have usually reported a hunch about the good and bad decks, and by 80 card turns, most subjects are able to generate specific conceptual knowledge about the good and bad decks (Bechara et al., 1997). Experimenters also note that before either the "hunch" or "conceptual" periods, anticipatory SCRs for the bad decks are higher than

anticipatory SCRs for the good decks (Bechara et al., 1997). It is thus concluded that somatic markers, as displayed by SCR recordings, had already begun differentiating between the good and bad decks before the subject had consciously made the same realization.

Two main problems involving poor temporal resolution cloud this conclusion. First, the onset of an SCR difference between the good and bad decks is not a clearly identifiable point in time. Experimenters are able to say that, across the entire 100-card task, subjects generated greater-magnitude anticipatory SCRs relative to selections of cards from the bad versus good decks (Bechara et al., 1996). However, the difference in magnitudes of anticipatory SCRs for bad decks compared to good decks generated before the hunch period does not reach statistical significance (Bechara et al., 1997). Thus, for approximately 50 card turns, an SCR-based differentiation between good and bad decks is not possible to identify.

Second, the onset of conscious knowledge concerning the good and bad decks may be even harder to tie down temporally. After most subjects encounter their first loss in the gambling task, their spoken answers to the requests “Tell me all you know about what’s going in this game” and “Tell me how you feel about this game” were divided into three temporal categories: pre-hunch, hunch, and conceptual (Bechara et al., 1997). These categories are quite vague and defined loosely (Bechara et al., 1997). Additionally, it is not clear that all of the subjects’ conscious knowledge about the game would be revealed by these two questions. For example, during the pre-hunch period, the subject may tell the experimenter that he or she has no idea about the game, but may actually be hypothesis testing at the same time. Subjects’ verbal recounting of

their own knowledge about the game may also be generally conservative, in an effort to not give an incorrect response.

Thus, the temporal resolution concerning the onsets of both the SCR difference between the good and bad decks and conscious knowledge about the game is quite poor. An attempt to order these events temporally, then, may be seriously flawed.

Alternatives to the Somatic Marker Hypothesis

Given two pieces of evidence, that individuals with ventromedial prefrontal damage have a behavioral deficit on the gambling task, and that these individuals do not generate anticipatory SCRs, the Damasio group concludes that they are causally related by the somatic marker hypothesis. However, it is not necessary to invoke this explanation; somatic markers need not be involved to generate this pattern of results. Indeed, it is possible that damage to the ventromedial prefrontal cortex causes both the behavioral and SCR abnormality in the gambling task, but that these abnormalities are not related in the way the Damasio group suggests. For instance, the ventromedial prefrontal cortex has been shown to be active during the generation of SCRs (Critchley, Elliott, Mathias, & Dolan, 2000). Perhaps ventromedial prefrontal patients have abnormal SCRs because of an impairment in SCR generation, rather than a loss of somatic markers.

Although the complications just identified with the gambling task cloud the evidence surrounding the somatic marker hypothesis, they also create the opportunity to experimentally differentiate between that hypothesis and alternate explanations. The following experiments attempt to accomplish just that.

Experiments

Experiment 1

Experiment 1 is a strict replication of one of the gambling task experiments (Bechara et al., 1997). In this experiment, following the exact procedures described in the original paper, and in consultation with the first author, SCRs were recorded, and subjects were continuously asked about their knowledge of the game. This experiment confirmed a similarity in methods and equipment by replicating earlier results.

Experiment 2

In an extension of Experiment 1, this experiment uses identical methodology, with one exception: the money lost or gained in each deck is changed such that the good decks have a higher magnitude of reward and punishment than the bad decks (see Table 2). Given this situation, the somatic marker hypothesis predicts that SCRs will still be higher for the bad decks relative to the good decks, whereas the alternate hypothesis (i.e., that the SCR difference in the original gambling task experiments was driven by the magnitude of reward and punishment) predicts opposite results.