

General Discussion

Counterarguments

The results of the anticipatory SCRs recorded in experiments 1 and 2 suggest, contrary to previous gambling task studies (Bechara et al., 1996; Bechara et al., 1997), that these SCRs cannot be correlates of somatic markers. Indeed, given the switch in anticipatory SCR magnitude between experiments 1 and 2, if these SCRs were correlates of somatic markers, these markers would be sending contradictory signals. Instead, it is suggested that anticipatory SCRs are associated with conscious processes that may be related to the magnitude of punishment and reward in each deck. If it is true that anticipatory SCRs are not correlates of somatic markers, then the Damasio group has yet to produce any direct evidence for the existence of these markers.

Arguments could be made, however, challenging the validity of the results obtained in experiment 2. Table 3 lists a number of differences between the methodology used in, and results obtained from, experiment 2 versus previous experiments (Bechara et al., 1996; Bechara et al., 1997). Each of these differences will be examined in turn. It is argued that while these differences may be important, none alter the conclusion that the somatic marker hypothesis is unable to account for the results of experiment 2, and thus that previous gambling task experiments may have failed to produce direct evidence of somatic markers.

Subjects in previous gambling task studies were significantly older than subjects in the present experiments. Subjects in Bechara et al. (1997) ranged from 51 to 65

years old while subjects in experiments 1 and 2 ranged from 18 to 22 years old.

Although this clearly is a significant age difference between subject groups, the somatic marker hypothesis does not predict different performance on the gambling task because of this age difference. Indeed, all age-related impairments on card choice performance in the gambling task have been found in individuals who were over the age of 64 (Bechara et al., 2000a). Another difference between subjects in previous studies compared to those in experiment 2 is that those in the present study are drawn from a Harvard University population, whereas individuals from previous studies were not. Although this may result in certain cognitive differences between subject populations, the gambling task has been shown to be relatively invariant to similar factors, including years of education (Bechara et al., 2000a). Further, a difference in performance between subjects in previous experiments and subjects in experiment 1 was *not* found: subjects picked cards from the good and bad decks in proportions that were very similar to previous data (Bechara et al., 1994). Additionally, SCR data from experiment 1 were similar to data found in previous studies (Bechara et al., 1996; Bechara et al., 1997), which suggests that subject differences did not have a major effect on SCR recording.

A few differences between the procedures and results of SCR analysis can be found between experiment 2 and previous gambling task studies. Different recording equipment was used, and the magnitudes of anticipatory SCRs recorded in previous studies were higher than the magnitudes recorded in experiments 1 or 2. It is well known that differences in recording equipment can cause changes in SCRs (Fowles et al., 1981), and it may be the case that equipment was important in causing the corresponding anticipatory SCR amplitude difference. However, neither of these factors

can account for the switch in anticipatory SCRs for the good and the bad decks between experiments 1 and 2. Although differing equipment may have absolutely decreased, or perhaps increased, the magnitude of SCRs recorded in the present studies, there is no reason to suggest that it could have selectively changed the amplitudes of certain decks but not others. Further, the replication of the crucial anticipatory SCR difference in experiment 1 demonstrates that recording equipment differences could not have caused the results of experiment 2.

In a previous gambling task experiment, anticipatory SCR were established to be higher for selections of cards from the bad decks versus the good decks by a one-way ANOVA. Further, post-hoc tests compared anticipatory SCRs for each good deck versus each bad deck individually (Bechara et al., 1996). In the present experiments, however, anticipatory SCRs from both good decks and both bad decks were grouped together. Then, t-tests were used to demonstrate statistically significant differences between decks. In particular, in experiment 2, post-hoc Tukey HSD tests revealed that anticipatory SCRs from deck B were higher than those anticipatory SCRs associated with either decks C or D. No other post-hoc tests were found to be statistically significant. However, a t-test comparing anticipatory SCRs from the good decks relative to the bad decks showed a highly statistically significant effect.

Given these differences in statistical analysis between the present experiments and previous gambling task studies, the argument could be made that the anticipatory SCR differences found in experiments 1 and 2 are not valid because they do not strictly replicate the individual deck comparisons of earlier studies. However, this view presupposes that individual decks of cards are the only appropriate groups for statistical

comparison. Grouping the four decks into two separate subgroups of “bad” and “good” decks is also a valid way of analyzing anticipatory SCR data, one that has been used in other studies of the gambling task (Bechara et al., 1997).

It could also be argued that because anticipatory SCRs from deck B were found to be significantly higher than decks C and D in experiment 2, deck B alone may be driving the effect that anticipatory SCRs from the bad decks are higher than those from the good decks. However, in experiment 2 anticipatory SCRs from deck A were absolutely higher than those from decks C and D and further, no significant difference was found in post-hoc tests between decks A and B. It is thus suggested that anticipatory SCRs from deck A also played a role in the good versus bad deck difference. Even if anticipatory SCRs from deck B were the main factors driving the difference between the good and bad decks in experiment 2, this would still render the results incompatible with the somatic marker hypothesis. That is, a pattern of results in which the anticipatory SCRs from one good deck were significantly higher than those from both of the bad decks, and the anticipatory SCRs from the other good deck were absolutely higher than those from both of the bad decks would never occur if these SCRs were driven by the inherent “goodness” or “badness” of each deck.

A small number of differences in the card schemes between previous gambling task experiments and experiment 2 can be identified. First, a higher absolute dollar amount of play money is used in experiment 2, and the loan of money at the beginning of the game is absolutely higher than in previous experiments. As described earlier, the ratios of reward and punishment in the good versus the bad decks are not identical to those in the previous studies. None of these changes in card scheme, however, have

any input into the somatic marker hypothesis. Because the somatic marker hypothesis predicts anticipatory SCR differences based solely on whether each deck will eventually win or lose the subject money, these differences in card scheme do not affect the interpretation of the results of experiment 2.

In both experiments 1 and 2, subjects' verbal reports concerning their knowledge of the game differed significantly with those reported in an earlier study (Bechara et al., 1997). As discussed previously, it is unknown whether this is due to an actual difference in knowledge between subjects in experiments 1 and 2 versus subjects in the previous study, or whether it is due to a different questioning procedure or perhaps increased tentativeness of the subjects in the present studies. If, as the Damasio group claims, conscious knowledge is an aid to decision-making in addition to the action of somatic markers (Bechara et al., 1997), subjects in the present study should have performed worse on the gambling task if they possessed less knowledge about the game. Instead, card choice in the present experiment is very similar to that of previous gambling task experiments, thus suggesting that conscious knowledge across experiments may be similar.

Despite this evidence, if it is assumed that subjects in the present studies actually possessed less knowledge about the game than those subjects in the previous gambling task study (Bechara et al., 1997), it could be argued that the anticipatory SCR differences seen in experiments 1 and 2 between the good and bad decks are systematically different from those reported earlier, in that they involve less interaction with conscious knowledge. However, this argument would falter because of certain tenets of the somatic marker hypothesis that posit that not only can somatic markers

exist without conscious knowledge about a particular decision, but that these markers can actually precede conscious knowledge of a decision (i.e., Bechara et al., 1997). Thus, if subjects in experiments 1 and 2 were only in the process of “figuring out” the game when the gambling task was terminated, they still should have been able to generate somatic markers. Thus, the comparison between experiments 1 and 2 would remain valid.

Finally, the card scheme of experiment 1 differs from experiment 2 in that presumably the bad decks are initially more attractive to the subject because of their higher rewards, and only later in the game does the subject discover that these decks are to be avoided. In experiment 2, the initial reward of the good decks is higher than that of the bad decks, and this remains true throughout the task. It could be argued, then, that in experiment 1 and previous gambling tasks, subjects are initially attracted to the bad decks and must learn to inhibit this response and eventually choose from the good decks. That is, in experiment 1, subjects must learn to switch their pattern of responding from pulling cards from the bad decks to pulling cards from the good decks, and since this switching process is not required in experiment 2, the decision-making tasks are not comparable.

While the assumption that subjects initially pick more cards from the bad decks from the good decks seems like a logical one, no statistical tests have shown that subjects pick more cards from the bad decks relative to the good decks during the early phases of the gambling task (Bechara et al., 1994; Bechara et al., 1996; Bechara et al., 1997). Indeed, in Bechara et al. (1997), subjects were found to pick absolutely more cards from the good decks than the bad decks even in the pre-hunch period, which is

an early phase of the gambling task. Thus, at least behaviorally, there is no evidence that such a “switch” occurs in normal subjects. Even if subjects did have to perform a switch from the bad decks to the good decks, such a switch is not necessary for the somatic marker hypothesis to be involved. The somatic marker hypothesis is posited to apply to a broad range of decision-making, including decisions that do not necessarily include a reversal of opinion about certain alternatives. Further, in experiment 2, the proportion of cards chosen from the good decks relative to the bad decks is very similar to that found in experiment 1, indicating that the difficulty of the subjects’ decision is relatively similar across experiments.

A number of differences exist between experiments 1 and 2 and previous studies of the gambling task. However, these differences do not invalidate the suggestion that the anticipatory SCRs observed in the present experiments, and indeed, in previous experiments, are not the correlates of somatic markers. It is unclear, in any case, why somatic markers would act in the manner suggested by the Damasio group in the gambling task. Supposedly, both positive and negative somatic markers exist that bias an individual towards, or away from, a particular choice or set of choices (Bechara et al., 2000a). It remains unexplained why positive somatic markers were not observed before card selections from the good decks. It is unknown whether the type of decision-making subjects use in the gambling task only incorporates negative somatic markers, or why this would be the case. Perhaps positive somatic markers are posited to exist in the gambling task, but are not measurable by anticipatory SCR recording, but no reason is given for why this should be true.

Theoretical Obstacles to the Somatic Marker Hypothesis

A number of different kinds of evidence argue against theories of peripheral feedback. As described earlier, studies examining spinal cord injury (Cannon, 1927; Cannon, 1931; Bermond et al., 1991), blockage of bodily responses by pharmacological agents (Reisenzein, 1983), and comparisons of externally-generated versus internally-generated emotions (Levenson et al., 1990; Stemmler, 1989) have suggested that peripheral response may play only a limited role in creating emotional experiences. This creates a problem for the somatic marker hypothesis, which depends on peripheral responses to mark a particular decision or group of decisions with a positive or negative value.

Other researchers have argued that emotions are too varied to each be associated with a particular body state (Rolls, 2000; Wagner, 1989; Cannon, 1927). This argument is also a large hurdle for the somatic marker hypothesis, because the decision-making process is assumed to depend on the analysis of bodily responses, the minutiae of which may change their effect on the decision to be made. Thus, it is unclear that each particular decision an individual makes corresponds to an associated bodily state, because of the complexity and variance possible in human decision-making.

Finally, the process of analyzing body state information and then grouping this information into different classes of biasing responses is one whose broadest features, let alone precise details, are unknown. Thus, it may be the case that no clear evidence for the existence of somatic markers has yet been found, and it certainly is the case that

no evidence has identified a mechanism for interpreting these markers in the manner that the Damasio group describes.

Future Research

In order to directly assess some of the complications that affected the results of experiments 1 and 2, further clarification studies may be carried out. For example, in order to directly address the question of whether the difference in the age of subjects between experiment 2 and previous gambling task studies impacted anticipatory SCR data, one could perform a study similar to experiment 2 in which subjects were approximately fifty years old. Likewise, one could conduct a study using a non-Harvard population of subjects to determine whether using only Harvard undergraduate students influenced the results of experiment 2.

In order to control for many of the card scheme differences between experiments 1 and 2, one could perform a different experiment using the card scheme of experiment 1. In this experiment, the card sheet of experiment 1 (Figure 1) would be used, except that the goal of the subject would be to *lose* as much money as possible. By this design, the good and bad decks would switch roles. Decks A and B would become the good decks and would be associated with higher magnitude of punishment and reward, and decks C and D would become the bad decks and would be associated with a lower magnitude of punishment and reward. Although in this design, the subject would therefore be encountering automatic punishments for every card pull and occasional rewards, many of the ratios of punishment and reward established in the card scheme of experiment 1 would be maintained. Thus, in this new study, the absolute dollar

amount of play money, as well as the initial loan amount would be identical to that of experiment 1.

The absolute amount of play money and the initial loan amount in the gambling task could also be manipulated in card schemes in which subjects tried to win money. For example, results from a card scheme similar to experiment 2 in which each amount of money exchanged is ten times smaller than in experiment 2 could be compared to results from an experiment in which each amount of money exchanged is ten times larger than in experiment 2. Likewise, experiments differing in the amounts of loaned money could be compared to assess any differences.

Some of the differences in verbal responses between experiments 1 and 2 and previous gambling task experiments could be addressed by studies in which subjects were asked much more extensive questions about their knowledge of the game. Thus, instead of asking subjects open-ended questions every ten cards, subjects could be asked specific questions, such as “Do you have a hunch about which decks are good and which are bad?” and “Which decks do you think are the riskiest?” Subjects’ responses would be more specific, and could be more easily classifiable into categories such as “pre-hunch” and “hunch.” Further, this may improve the experimenters’ temporal acuity in determining when conscious knowledge about the game begins. That is, these questions may mitigate the effects that hypothesis-testing and a desire not to give the wrong answer have on subjects’ responses by encouraging subjects to immediately voice any hunches or ideas they have about the game. Even so, it would still be difficult to compare the temporal onset of conscious knowledge about the gambling task with the temporal onset of an anticipatory SCR difference between the

good and the bad decks. Further, as experiment 2 suggests that the anticipatory SCR difference between the good and bad decks cannot be due to somatic markers, a temporal comparison between anticipatory SCR onset and the onset of conscious knowledge about the game may be uninteresting to examine.

Finally, an interesting experiment involving individuals with characteristics of psychopathy could be performed using the original gambling task. In this study, individuals who score high, low, and normally on the Psychopathic Personality Inventory (PPI), a measure of psychopathy (Lilienfeld & Andrews, 1996) could be tested using the gambling task. It is argued by the Damasio group that psychopathic individuals' behavioral deficits, similar to those of ventromedial prefrontal patients, are due to a loss of somatic markers (Damasio, 1994; Damasio, Tranel, & Damasio, 1990). Thus, for individuals who score highly on the PPI, the somatic marker hypothesis predicts performance similar to that of ventromedial prefrontal patients: impaired behavioral performance and abnormal anticipatory SCR performance (Bechara et al., 1996; Bechara et al., 1997). This may be particularly interesting, because if high-scoring individuals perform abnormally on the gambling task, either behaviorally or psychophysiologicaly, this deficit could not be explained by brain damage to the prefrontal cortex (although an abnormally-functioning prefrontal cortex could not be discounted in these individuals).

Impairment of Ventromedial Prefrontal Patients on the Gambling Task

Although the present study only investigated the performance of neurologically normal subjects, other experiments done by the Damasio group suggest that individuals

with ventromedial prefrontal damage are impaired on the gambling task (Bechara et al., 1994; Bechara et al., 1996; Bechara et al., 1997). Specifically, these patients select more cards from the bad decks versus the good decks, and generate anticipatory SCRs that are significantly lower than those of controls and do not differentiate between the good and bad decks (though their reward and punishment SCRs are not significantly different from those generated by normals). Although the Damasio group explains these deficits in terms of the somatic marker hypothesis, alternate explanations are possible.

The card choice deficits seen in patients with ventromedial prefrontal damage are entirely consistent with literature reviewed earlier, which suggests that the prefrontal cortex is important in the evaluation of punishment and reward and decisions based on these outcomes. Additionally, activation in the ventromedial prefrontal cortex has been associated with the generation of SCRs (Critchley et al., 2000). It is possible, then, to explain the patterns observed simply on the basis that patients with damage to this cortical area have abnormal SCR responses. It is unknown why individuals with ventromedial prefrontal damage generate abnormal anticipatory SCRs but normal reward and punishment SCRs in the gambling task, but the somatic marker hypothesis remains undistinguished among several alternatives that may account for this phenomenon.

Conclusion

Literature concerning the function of the prefrontal cortex is unanimous in assigning that brain area an important role in decision-making, and in stating that

damage to this area can result in a variety of decision-making deficits. However, the lack of evidence showing “somatic markers in action” renders the somatic marker hypothesis as a theory that is intriguing but not yet supported by sufficient evidence, and that may indeed be at a disadvantage to less circuitous theories of decision-making until such evidence is discovered.