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Heart rate and skin conductance analysis of antecendents and consequences of decision making

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Abstract

The current study examined the pattern of heart rate and skin conductance changes preceding risky choices and following outcome for bad, moderate, and good performers on an analogue of the Iowa gambling task (Bechara, Damasio, Damasio, & Anderson, 1994). The task required a choice between four options; two options were followed by a high reward and, unpredictably, an even higher loss (disadvantageous options) and two other options were followed by a small reward but the unpredictable loss was also small (advantageous options). Anticipatory heart rate slowing and skin conductance level were higher preceding disadvantageous relative to advantageous options, but only for good performers. In contrast, heart rate slowed and skin conductance level increased following loss relative to reward outcomes, and these changes were similar for all performance groups. These findings were interpreted to suggest that decision-making impairments in bad performers arise from a weak somatic response generated by secondary inducers (i.e., somatic markers), rather than a weak somatic response generated by primary inducers of reward and punishment.

Descriptors: Heart rate, Skin conductance, Gambling, Decision making, Feedback, Anticipation

Decision making is required for a variety of behaviors and often involves consideration of multiple alternatives and reasoning about distant future consequences. In these situations one often compares the obtained outcomes of a decision against beliefs about the likelihood of the obtained outcomes; therefore both experienced and anticipated emotions influence the decision-making process. In case the situation is complex and there is remarkable uncertainty about the future, it is no longer manageable to base decisions solely on a logic-based, cost–benefit analyses of performance outcomes. Damasio (1994) proposed that in these situations individuals use somatic markers, which constrain the complexity of the situation and permit the individual to decide efficiently within short time intervals.

The system network necessary for somatic markers to operate assigns an important role to the ventromedial prefrontal cortex. The ventromedial cortices are presumed to contain convergence zones, hailing from both external and internal stimuli, which hold a record of temporal conjunctions of activity in varied neural units (e.g., the sensory areas). Autonomic effectors are presumed to be the critical output of the ventromedial convergence zones. When parts of certain exteroceptive–interoceptive conjunctions are reprocessed, their activation is signaled to the ventromedial cortices, which in turn activate somatic effectors in amygdala, hypothalamus, and brain-stem nuclei, which can be interpreted as an attempt to reconstitute the kind of somatic state that belonged to the conjunction (cf. Damasio, 1995). The reenacted somatic states can then be signaled to cortical and subcortical somatosensory areas, and can trigger a covert process modifying appetitive or aversive behavior.

Empirical support for the operation of autonomic signals comes from studies using a gambling task, which mimics real-life decisions in the way it factors reward, punishment, and uncertainty of outcomes (Bechara, Damasio, Damasio, & Anderson, 1994). This task requires individuals to sample from four decks, from which two can result in immediate high gain whereas two others result in immediate low gain. The uncertainty in outcomes lies in the way delayed punishment is presented. The two decks that result in high gain are accompanied by large delayed punishment, from which one of the decks is accompanied by frequent, relatively small punishment (50%), whereas the other is accompanied by infrequent, relatively large punishment (10%). Similarly, from the decks that result in low immediate gain, one is accompanied by frequent, but small delayed punishment (50%) and the other by infrequent, but large delayed punishment (10%). The first two decks (A and B) are disadvantageous in the long run, because they result in a net loss, whereas the other two decks (C and D) are advantageous in the long run, because they result in net gain.

Intact individuals typically learn to adopt an advantageous response strategy during the course of the task, and develop anticipatory skin conductance responses preceding disadvantageous choices (Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, Damasio, & Damasio, 1996; Tomb, Hauser, Deldin, &

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Caramazza, 2002). Patients with damage to the ventromedial regions, on the other hand, keep betting on the disadvantageous decks, and somatic markers in the form of skin conductance responses preceding disadvantageous options are absent. Thus, the anticipatory skin conductance arousal preceding disadvantageous choices on the Iowa gambling task is suggested to represent risk-related behavior that is associated with the magnitude of future loss, and is presumed to rely on ventromedial prefrontal convergence zones (Bechara et al., 1996; Damasio, 1995).

However, this interpretation is challenged by reports from Bechara, Damasio, and Damasio (2000) showing that a proportion of the normal population performs as impairedly as ventromedial patients (mostly disadvantageous choices) but does develop anticipatory skin conductance responses. This finding suggests that in the normal population anticipatory somatic markers develop independently from performance strategy. For example, in a recent study that focused on performance of ventromedial prefrontal patients, substance abusers, and normal control participants, Bechara and Damasio (2002) reported that 7 of 22 control participants performed as impairedly as patients with ventromedial prefrontal damage. These individuals developed anticipatory skin conductance responses that were somewhat smaller than anticipatory skin conductance responses seen in nonimpaired control participants, but remarkably larger than those seen in ventromedial prefrontal patients. Bechara and Damasio characterized these subjects as "high risk-takers," because they may have overridden their somatic signals by conscious deliberation. However, it was also observed that skin conductance rise following loss was less pronounced for poor performing control subjects, suggesting that poor performance could also have resulted from decreased somatic activity to performance outcomes, resulting in less efficient updating of context information to adjust performance strategy.

Notably, the gambling task requires that, in order to improve performance, individuals should first process the outcome of their decisions, then update their decision strategy according to the outcome, and finally implement these acquired stimulus–outcome associations in their future strategy. The interplay between these processes emphasizes that decision making is not mediated by ventromedial prefrontal cortex alone, but arises from large-scale systems that also include other cortical and subcortical components, such as the amygdala, the insular/somatosensory cortices, and the peripheral nervous system. Within this system, the amygdala is presumed to play a central role in processing the emotional content of negative performance outcomes (Bechara, Dolan, & Hindes, 2002), whereas the ventromedial region is critical for reenacting the somatic state that previously belonged to the situation (Bechara et al., 2002; Damasio, 1995). The contributions of these separate processes account for the finding that ventromedial prefrontal patients have normal somatic responses following punishment, while showing a pronounced deficit on the gambling task (Bechara et al., 1996). In contrast, the deficit to perform advantageously on the gambling task by patients with amygdala damage is accounted for by the absence of somatic activity following any type of punishment outcome (Bechara, Damasio, Damasio, & Lee, 1999).

The primary goal of this study was to examine the physiological responsiveness to gain and loss outcome in badly and well performing participants, and unravel the possible dissociation with developing somatic markers. Although we accept the distinction between outcome-related somatic activity and anticipatory somatic activity, on the basis of previously reported data (Bechara & Damasio, 2002), it is not directly clear if these processes function independently in Bechara's gambling task. The evidence so far is rather indirect, because it is derived from studies examining performance of subjects with brain damage (Bechara et al., 1996, 1999), and these studies, at least to our knowledge, did not directly examine the relation between anticipatory and outcome-related somatic activity within individuals. The present study sought to remedy this shortcoming by examining anticipatory and outcome-related somatic activity in normal healthy individuals who were categorized on the basis of their performance in bad (1/3) moderate (1/3), and good performers (1/3).

This categorization was based on Bechara and Damasio's (2002) observation that approximately 33% of their normal control group performed as impairedly as ventromedial prefrontal patients. Three possibilities were considered for why a subgroup of intact individuals performs badly on the gambling task. The first is that outcomes (reward and punishment) trigger less autonomic activity, resulting in inefficient context updating (e.g., Bechara et al., 1999). Second, poorly performing subjects may show normal autonomic activity following the outcome of choices, but may fail to integrate the acquired stimulus–response associations in the formation of somatic markers. This would be the most prominent interpretation in terms of the somatic marker hypothesis (Damasio, 1994). Bechara and Damasio (2002) considered a third possibility, which claims that intact poor performers show normal activity following outcomes and develop anticipatory somatic responses, but nonetheless consciously override the information derived from somatic markers because they are high-risk takers.

In previous studies, skin conductance reactivity has been used as the primary index of sympathetic bodily arousal, but the somatic marker hypothesis suggests a general sensitivity of the autonomic system to activity associated with cognitively driven changes in body states of arousal (cf. Damasio, 1996). We examined whether somatic marker activity could be extended to the cardiovascular domain of arousal. Prior work has robustly shown that heart rate slows when an individual prepares for a voluntary response (Lacey & Lacey, 1974; Van der Molen, Somsen, Jennings, & Orlebeke, 1985). Somsen, Van der Molen, and Orlebeke (1983) demonstrated that anticipatory heart rate slowing was much more pronounced when individuals were preparing for an aversive event (a shock threat), and this slowing was largest when the shock was unavoidable. These findings led to the hypothesis that heart rate slowing is associated with the extent to which individuals restrict attention to potentially aversive stimuli. Following this assumption, there may be an important relation between risk anticipation and anticipatory heart rate slowing, that is, we expected larger heart rate slowing preceding disadvantageous decisions. Following Bechara and Damasio (2002), we expected that all participants would develop somatic markers during the course of the task, and somatic activity should be larger for good performers (see also Damasio, 1994). Thus, good performers should have higher anticipatory skin conductance rise when preparing a disadvantageous decision, and their heart rate slowing should differentiate more preceding advantageous and disadvantageous decisions.

By comparing somatic responses following outcomes of choices for bad, moderate, and good performers, we expect to unravel the inconsistency in the literature concerning the relation between outcome responsiveness and future performance adjustment. Immediate reward and punishment should result in larger skin conductance (Bechara et al., 1996) and more

pronounced heart rate slowing following punishment trials (Crone et al., 2003), and these responses should be positively related to the magnitude of punishment (Bechara et al., 2000; Damasio, 1994). On the basis of the literature, we tested two hypotheses to account for a disadvantageous strategy.

Bechara et al.'s (1996) observation that patients with damage to ventromedial regions show intact skin conductance reactivity to punishment outcomes but perform disadvantageously in the long term led us to predict that direct reactivity to feedback outcome is independent from long-term response strategy. In a similar vein, previous reports showed that ventromedial prefrontal patients switched decks following punishment similarly to control subjects (Bechara et al., 2000), which was interpreted to suggest that the patients respond to immediate effects of punishment, and therefore do not show a deficit in reversal-learning or inhibition. The interpretation of this deficit is that bad performers fail to integrate acquired stimulus–response associations in a long-term strategy.

Alternatively, the performance patterns and autonomic outcome responsiveness of poorly performing participants in the Bechara and Damasio (2002) study suggests that a disadvantageous strategy could also result from less somatic activity following the outcome of choices. Further support for this hypothesis comes from studies showing that patients with orbitofrontal damage in similar types of tasks have difficulty with response reversal (i.e., leaving the previously rewarded trial despite current loss at that trial; Rolls, 2000). Thus, following this prediction, less pronounced skin conductance rise and heart rate slowing following outcomes might account for a disadvantageous performance strategy.

A further goal of this study was to examine somatic activity in relation to the predictability of outcomes. Previous studies collapsed over options that result in high versus low frequent punishment, based on the assumption that somatic markers are related to the total magnitude of anticipated future loss (Bechara et al., 1996; Tomb et al., 2002). However, in behavioral studies, we found that individuals typically opt for choices in which punishment frequency is low (10%), suggesting that local choices are affected by outcome certainty and are mainly reward driven (Crone & Van der Molen, 2004). Given that individuals mostly avoid trials on which punishment occurs frequently, this may indicate that anticipatory arousal is also related to the predictability of feedback outcome (see also Damasio, 1994). This assumption is consistent with findings by Critchley, Mathias, and Dolan (2001), who developed an alternative gambling task and found that anticipatory arousal was larger when chances for punishment increased (e.g., 50% chance vs. 10% chance). Likewise, heart rate has previously been proposed to be especially sensitive to predictability of threat (Somsen et al., 1983). We examined skin conductance and heart rate preceding options that could result in (smaller) frequent punishment and (larger) infrequent punishment. Following Critchley et al. (2001), it was anticipated that skin conductance responses would be higher and heart rate slowing larger more preceding decks with higher probability of punishment (50%).

Method

Participants

Ninety-six students participated in the study, ranging in age between 18 and 31 ($M = 20.2$, $SD = 2.4$; 30 men, 66 women). Another 4 subjects were excluded because of equipment malfunction. The three performance groups did not differ in age, $F(2,93) = 0.85$, $p = .43$, or gender distribution, $\chi^2(2) = 2.70$, $p = .84$ (bad performers: 8 men, 21 women; moderate performers: 11 men, 23 women; good performers: 11 men, 22 women). All subjects reported they were healthy, with no history of brain injury, and had normal or corrected-to-normal vision.

Task Format

Participants sat in front of a computer monitor. The trial sequence started with the presentation of a stimulus display, followed by a 6,000 ms delay in which the subject could ponder which decision to make, followed by a cue indicating that the subject could respond. Following the response, a 1,000-ms blank screen, followed by a 2,500-ms outcome display, replaced the stimulus display. Responses were made approximately 500–1,000 ms following the cue, resulting in an intertrial interval of approximately 10 s. This interval was similar to studies of Bechara et al. (1996, 2002), and was chosen to let skin conductance responses return to baseline following punishment feedback and at the same time to create a reasonable length to keep subjects involved in the task.

The stimulus display consisted of four doors presented on a horizontal row, A, B, C, and D, followed by a donkey (the cue) in front of the doors (for a detailed description, see Crone & Van der Molen, 2004). Subjects were told to assist the hungry donkey to collect as many apples as possible by pressing one of four keys corresponding to the doors. The fingers from the dominant hand were assigned to the "C," "V," "B," and "N" keys of the computer keyboard. The four keys were mapped onto the doors from left to right. Upon pressing one of the keys, the stimulus display was replaced by a blank screen, followed by the outcome display showing the number of (intact) apples gained or the number of (crossed) apples lost. As a feedback tracking, a large horizontal bar was presented just below the donkey. At the beginning of the task, the left half was colored green and the right half was colored red. During the course of the task, the color change of the bar corresponded to the amounts of apples won or lost, averaged across options.

Task Description

All subjects performed a standard task that contained 100 trials. In the standard task, the win and loss schedule was similar to the one used by Bechara et al. (1994). That is, the relative proportions of wins and losses were identical to those used by Bechara et al. but the absolute amounts were reduced by a factor of 25. The ultimate future yield of each door varied, because the wins were higher at the high-paying doors (A and B) and lower at the low-paying doors (C and D). The punishment frequency of each door varied, because punishment frequency was higher for Doors A and C (50% of the trials) and lower for Doors B and D (10% of the trials). Selecting Door A or B resulted in a gain of four apples, whereas Door C or D resulted in a gain of two apples. After selecting 10 A doors, the subject received 40 apples, but had also encountered five unpredicted losses of either 8, 10, 10, 10, or 12 apples, bringing the total cost to 50 apples, thus incurring a net loss of 10 apples. After selecting 10 B doors, the subject received 40 apples but had encountered one unpredicted loss of 50 apples, also incurring a net loss of 10 apples. After selecting 10 C doors, the subject received 20 apples, but had encountered five unpredicted losses of 1, 2, 2, 2, or 3 apples, bringing the cost to 10 apples, incurring a net gain of 10 apples. The same happened at

Door D, except that instead of encountering five losses, there was one larger unpredicted loss of 10 apples. Thus, Door D also resulted in a net gain of 10 apples. In sum, Doors A and B were equivalent in terms of overall net loss over the trials. The difference was that at Door A, the probability of loss was frequent, but of smaller magnitude, whereas at Door B, the probability of loss was less frequent but larger. Doors C and D were also equivalent in terms of overall net loss. At Door C, the probability of loss was frequent and of smaller magnitude whereas at Door D the probability loss was less frequent and of higher magnitude.

Data Recording and Reduction

The experimental task was presented using a PC with a 17-in. monitor. The computer registered response speed to the nearest millisecond. Participants sat in a comfortable chair in a dimly lit laboratory at a distance of approximately 75 cm from the monitor.

During the task, the electrocardiogram (ECG), skin conductance level (SCL), and respiration were continuously recorded. The ECG was recorded from three AgAg/CL electrodes, attached via the modified lead-2 placement. Skin conductance activity was recorded using a constant voltage (0.5 V) with 1-cm^3 AgAg/Cl electrodes attached to the medial phalanx surfaces of the middle and index fingers of the nondominant hand. Respiration was recorded through a temperature sensor placed under the nose. The signals were amplified by a Nihon Kohden polygraph and sampled by a Keithley AD converter at a rate of 400 Hz. The recorded interbeat intervals (IBIs) were screened for physiologically impossible readings and artefacts. These were corrected by adjusting specific parameters in the program that extracted the IBIs from the digitized ECGs. Skin conductance values were transformed to microsiemen values using Psylab software. The respiration signal was used to eliminate heart rate and skin conductance changes associated with gross respiratory maneuvers. Each time the subject pressed a response key, this action was synchronized with the sampling computer to the nearest millisecond.

Results

Performance

On the basis of total number of advantageous choices (out of 100), subjects were categorized into three levels of performance; bad ($n = 29$; M advantageous = 35, $SE = 3.4$), moderate ($n = 34$; M advantageous = 48, $SE = 3.1$), and good ($n = 33$; M advantageous = 58, $SE = 3.2$). To examine whether participants differentially learned to make advantageous choices during the course of the task, we submitted the number of each of four choices to a 3 (Performance Group) $\times$ 2 (Gain: A/B vs. C/D) $\times$ 2 (Punishment Frequency: A/C vs. B/D) $\times$ 5 (Task Block) ANOVA, where "block" represents the division of the task in segments of 20 trials. This analysis resulted in the expected interaction between Gain and Task Block, $F(4,372) = 22.88$, $p < .001$, $\varepsilon = .832$, which was altered by interactions between Performance Group and Gain, $F(2,93) = 11.85$, $p < .001$, and Performance Group, Gain, and Task Block, $F(8,372) = 6.17$, $p < .001$. As can be seen in Figure 1, bad performers showed no increase in number of advantageous choices during the course of the task, verified statistically by the absence of an interaction between Gain and Block ($p > .10$), and made more disadvantageous choices than advantageous choices, $F(1,28) = 23.37$, $p < .001$, $\varepsilon = 1.000$. Moderate performers showed improvement,

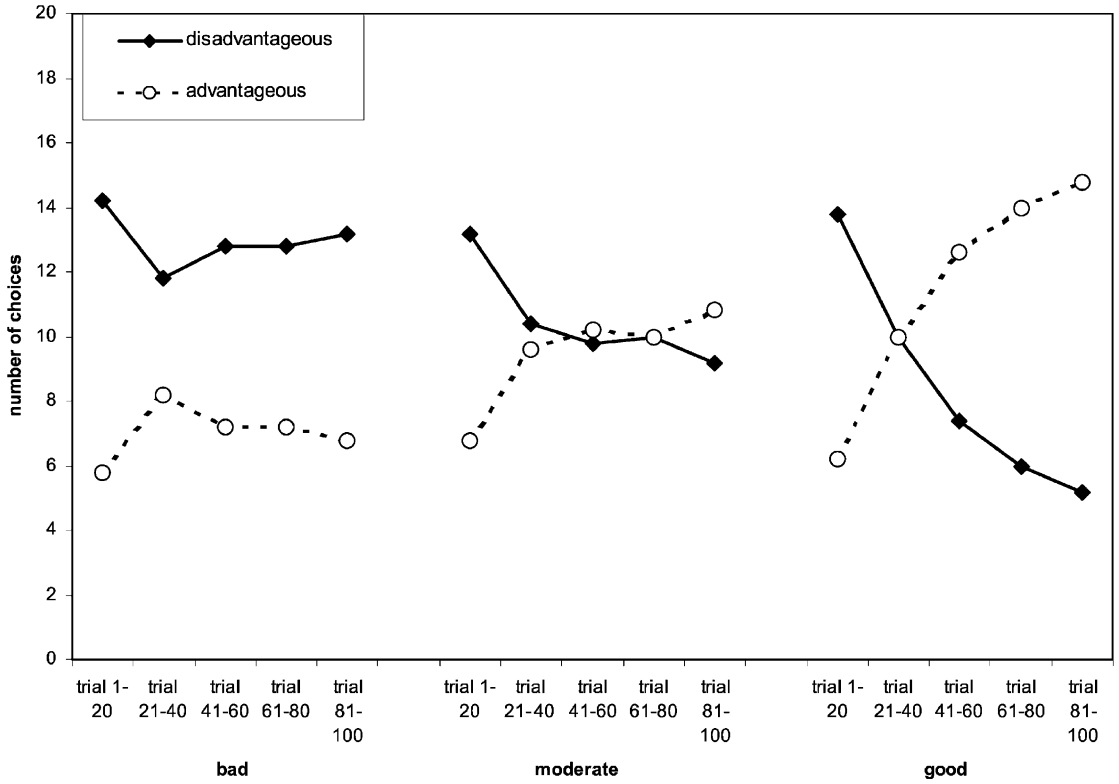

Figure 1. Number of advantageous choices for each performance group (both the courses of advantageous and disadvantageous choices are depicted, although these are dependent).

as can be seen by an interaction between Gain and Block, $F(4,132) = 5.72$, $p < .001$, $\varepsilon = .697$, but the main effect of Gain ($p > .01$) was absent, showing that learning strategy was not optimal. Good performers, finally, increased number of advantageous choices during the course of the task, as seen by a significant Gain × Block interaction, $F(4,128) = 21.40$, $p < .001$, $\varepsilon = .787$, and the main effect of Gain revealed that in general good performers made more advantageous than disadvantageous choices, $F(1,32) = 4.10$, $p < .05$, $\varepsilon = 1.000$.

As predicted, there was a main effect of Punishment Frequency, $F(1,93) = 20.83$, $p < .001$, $\varepsilon = 1.000$, and interactions between Punishment Frequency and Gain, $F(1,93) = 68.54$, $p < .001$, $\varepsilon = 1.000$, and between Performance Group, Punishment Frequency, and Gain, $F(2,93) = 6.48$, $p < .001$. This last interaction is plotted in Figure 2. Separate post hoc ANOVAs for disadvantageous choices indicated that subjects preferred options with low punishment frequency over options with high punishment frequency, $F(1,93) = 124.04$, $p < .001$, $\varepsilon = 1.000$, whereas this effect was absent for advantageous choices, $F(1,93) = 0.90$, $p = .35$, $\varepsilon = 1.000$. Separate comparisons for each choice showed that good performers made less (disadvantageous) A ($p < .01$) and B ($p < .01$) choices and more (advantageous) C ($p < .01$) choices, but did not differ in the number of D choices ($p = .36$) compared to both moderate and bad performers. Effects of Punishment Frequency did not interact with Task Block.

The next performance analysis examined if subjects switched response options when they received punishment. Trials were selected on the basis of previous feedback, which could be reward trials or punishment trials. Switch percentage was computed for disadvantageous and advantageous trials separately. Switch percentage following punishment was computed by calculating the total number of response switches following punishment as a function of the total number of punishments (*n* switch following punishment/ [*n* switch following punishment+*n* stay following punishment]). A similar procedure was used to achieve the switch percentage following reward (*n* switch following reward/[*n* switch following reward+*n* stay following reward]). The 3 (Performance Group) × 2 (Gain) × 2 (Previous Feedback) ANOVA resulted in a main effect of Previous Feedback, $F(1,92) = 90.35$, $p < .001$, $\varepsilon = 1.000$, showing that subjects switched responses more often following punishment (72%) than following reward (50%). The interaction between Gain and Previous Feedback, $F(1,90) = 14.78$, $p < .001$ $\varepsilon = 1.000$, showed that this effect was somewhat larger following disadvantageous choices (75% vs. 48%) than following advantageous choices (68% vs. 52%). Performance Group did not interact with Previous Feedback, suggesting that all groups were similarly sensitive to immediate loss.

Finally, to examine if subjects responded with similar speed to the four different types of choices, median RTs were computed for each choice separately and submitted to a 3 (Performance Group) × 4 (Choice) ANOVA. This analysis revealed that RTs generally varied between 768 and 947 ms with a 95% confidence interval, but there was no significant difference in response speed to the four response options, $F(3,270) = 3.02$, $p = .09$ ($M = 858$, $SD = 45.2$) or between performance groups ($p > .10$).

Heart Rate and SCL in Anticipation of Risky and Safe Choices

The next set of analyses focused on two questions. First, we examined whether both heart rate and skin conductance level are sensitive to outcome anticipation. Second, we examined whether this activity was larger for good performers. For this purpose, two analyses were performed, one for heart rate responses preceding advantageous and disadvantageous choices and the second for skin conductance activity preceding advantageous and disadvantageous choices. For both analyses, performance group was added as a between-subjects factor, representing subjects that were categorized as bad, moderate, and good performers on the basis of their total number of advantageous choices.

Heart rate. IBI responses were computed on the basis of the difference between the IBI concurrent to the response and the IBI preceding the response. This selection was based on previous

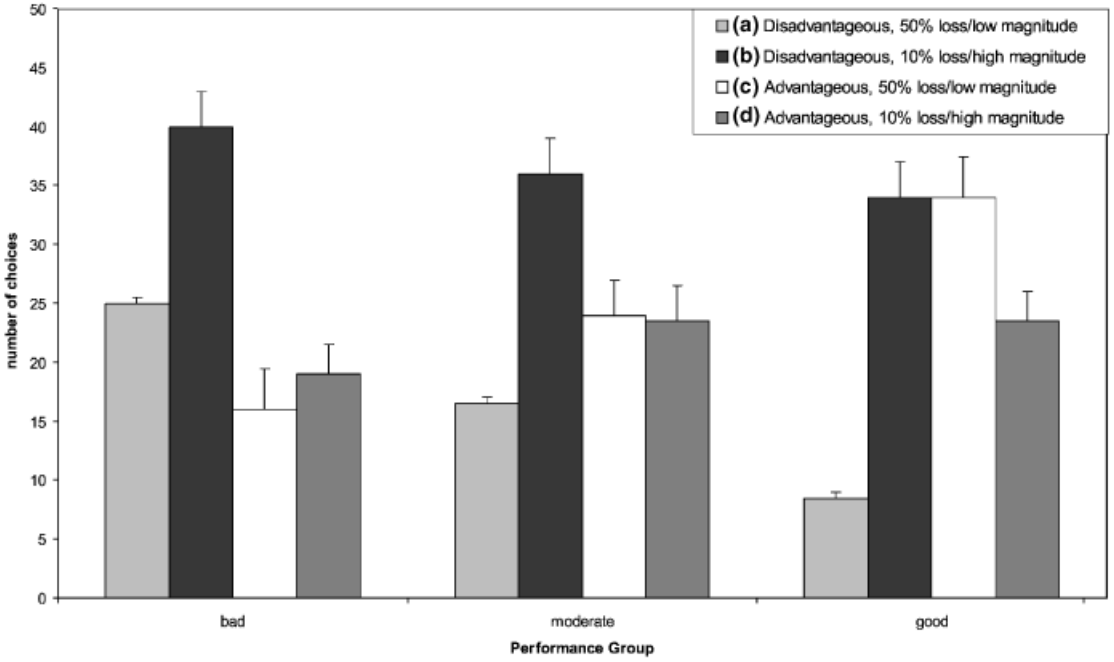

Figure 2. Number of choices for each option, for bad, moderate, and good performers.

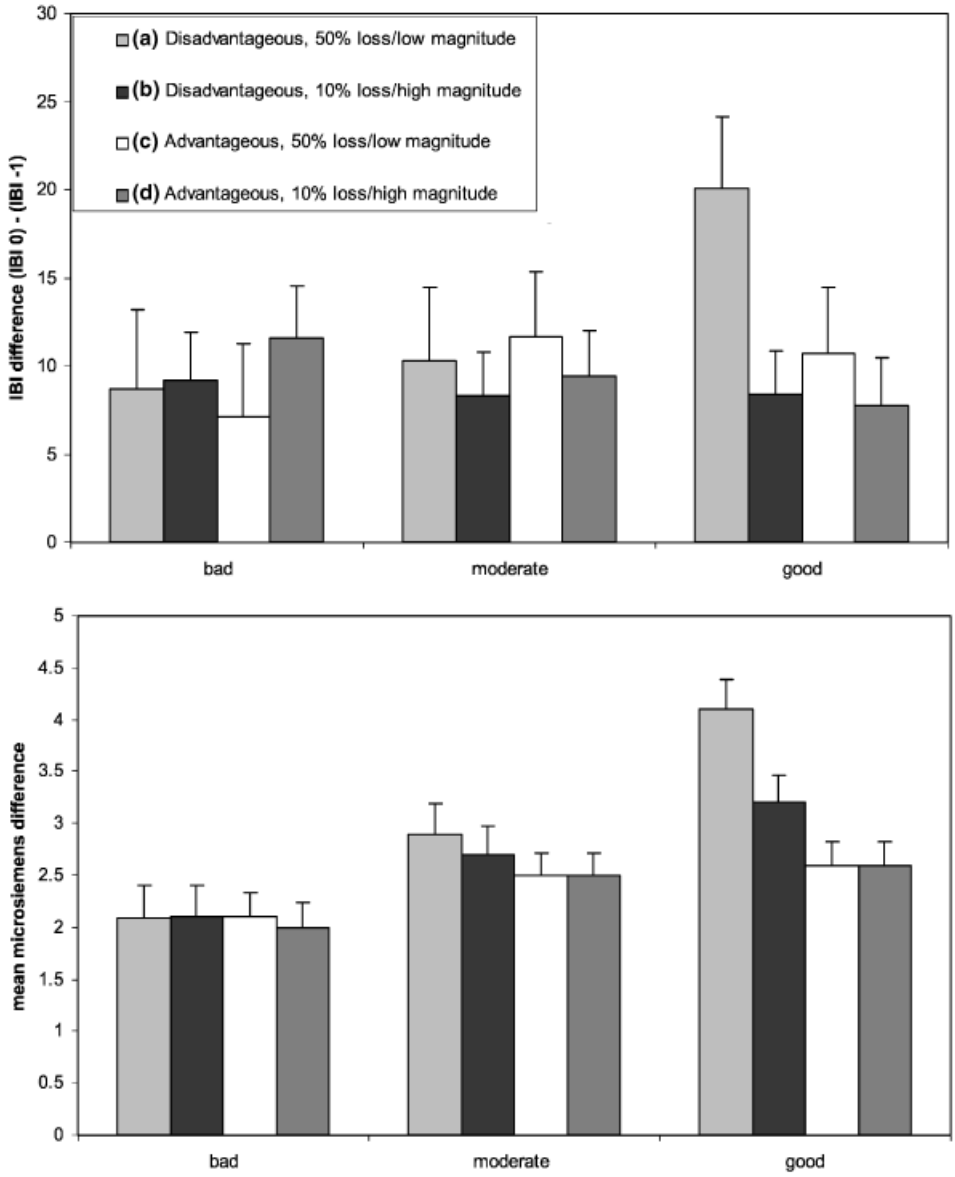

Figure 3. Heart rate and mean skin conductance level preceding each performance choices for bad, moderate, and good performers.

reports showing that response uncertainty effects become prominent at the IBI in which the response is committed (Somsen et al., 1983). As can be seen in the upper panel of Figure 3, heart rate slowed in anticipation of the response, but for good performers this slowing differentiated between anticipation on disadvantageous and advantageous choices. Choices could be risky in terms of future gain (A, B vs. C, D) and in terms of punishment frequency (A, C vs. B, D). IBI difference scores were submitted to a 3 (Performance Group) × 2 (Gain) × 2 (Punishment Frequency) ANOVA. This analysis resulted in a significant interaction between Performance Group and Frequency, $F(2,92) = 3.18$, $p < .05$, $\varepsilon = 1.000$, but a nonsignificant interaction between Performance Group, Gain, and IBI, $F(2,92) = 1.20$, $p = .30$. The interaction between Performance Group, Frequency, and IBI indicated that for good performers, heart rate slowed more preceding choices with a high frequency of punishment, $F(1,32) = 4.89$, $p < .05$, $\varepsilon = 1.000$, whereas this effect was not consistent for moderate performers ($p = .37$) and bad performers ($p = .33$).

Skin conductance. For the following anticipation analysis, skin conductance responses were measured in a time interval 5,000 ms before the response (Bechara et al., 1996, 2002). Mean skin conductance level was referred to the lowest microsiemens value (baseline) for each participant separately. The difference scores were submitted to a 3 (Performance Group) × 2 (Gain) × 2 (Punishment Frequency) ANOVA. The analysis resulted in main effects of Gain, $F(1,92) = 21.05$, $p < .001$, $\varepsilon = 1.000$, Frequency, $F(1,92) = 4.35$, $p < .05$, $\varepsilon = 1.000$, and Performance Group, $F(2,92) = 6.09$, $p < .05$. These effects were qualified by interactions between Performance Group and Gain, $F(2,92) = 8.58$, $p < .05$, Gain and Frequency, $F(1,92) = 4.56$, $p < .05$, $\varepsilon = 1.000$, and Performance Group, Gain, and Frequency, $F(2,92) = 3.69$, $p < .05$

The last interaction is plotted in the lower panel of Figure 3 and shows that bad performers show no differential skin conductance activity preceding advantageous and disadvantageous choices, whereas moderate performers show somewhat larger skin conductance activity preceding disadvantageous choices,

and largest skin conductance activity preceding disadvantageous choices is seen for good performers. These visual impressions were statistically verified by a significant Gain effect, $F(1,32) = 16.67$, $p < .05$, and Gain × Frequency interaction, $F(2,32) = 5.98$, $p < .05$, for good performers and a significant Gain effect for moderate performers, $F(1,33) = 6.57$, $p < .05$. For bad performers these effects were not significant. A separate comparison for moderate and good performers showed that the anticipatory skin conductance increase preceding bad choices was larger for good performers than for moderate performers, as seen by a Gain × Performance Group interaction, $F(1,65) = 7.52$, $p < .05$. The Gain × Frequency interaction for good performers showed that anticipatory skin conductance increase was larger preceding choices with high frequent punishment (50%) when choices were disadvantageous, $F(1,32) = 8.92$, $p < .05$, but not when choices were advantageous ($p = .98$). The relation between performance and anticipatory skin conductance arousal was further confirmed by a significant positive correlation between the number of advantageous choices and the difference between skin conductance activity preceding disadvantageous and advantageous choices ($r = .49$, $p < .001$).

Given that bad performers made more disadvantageous choices and therefore also received more negative feedback, anticipatory responses could have been influenced by responses to previous feedback. To exclude the possibility that anticipation differences were influenced by previous feedback responses, the preceding analysis was performed again including a factor Previous Feedback (reward vs. punishment) to examine if the interactions were modulated by trials that were preceded by punishment feedback versus trials that were preceded by reward feedback. The 3 (Performance Group) × 2 (Gain) × 2 (Frequency) × 2 (Previous Feedback) ANOVA resulted, besides an interaction between Gain and Previous Feedback, in no further effects of previous feedback, whereas the interactions including Performance Group described in the previous analysis remained significant. The Gain × Previous Feedback interaction showed that, in general, the anticipatory skin conductance activity preceding disadvantageous choices was somewhat less pronounced following punishment feedback (2.6 vs. 2.4) compared to choices following reward feedback (2.9 vs. 2.3), but the Gain × Performance group interaction remained significant for both types of trials in the expected direction, $F(1,91) = 7.71$, $p < .05$ for reward trials and $F(1,78) = 4.62$, $p < .05$ for punishment trials. Thus, performance group differences in anticipatory skin conductance activity were not confounded by activity from the preceding feedback.

Heart Rate and SCL Following Performance Feedback

The next set of analyses focused on immediate effects of reward and punishment on autonomic arousal and its relation to performance. First, analysis of heart rate will be presented, followed by a presentation of the effects of performance feedback on skin conductance responses.

Heart rate. Figure 4 shows the pattern of heart rate responses following reward and punishment feedback for each performance choice. To examine whether heart rate was differentially sensitive to the magnitude and frequency of punishment, the first IBI following the presentation of feedback was selected and was referred to IBI −2. Analysis of IBI −2 revealed no differences in baseline heart rate between reward and punishment outcomes; neither was there a difference in baseline between performance groups. The IBI difference scores were submitted to a 3 (Performance Group) × 2 (Feedback) × 2 (Gain) × 2 (Punishment Frequency) ANOVA. This analysis resulted in main effects of Feedback, $F(1,85) = 34.93$, $p < .001$, $\varepsilon = 1.000$, and Punishment Frequency, $F(1,85) = 10.46$, $p < .005$, $\varepsilon = 1.000$. The main effect of Feedback indicated that heart rate slowed following punishment (6.9), but recovered to baseline following reward (−7.5), and the main effect of Punishment Frequency showed that heart slowing was larger following trials with infrequent punishment (3.1) compared to trials with frequent punishment (−3.8). To examine whether heart rate was more responsive to frequency effects than to magnitude of punishment, IBIs that followed punishment from disadvantageous choices with high frequency (50% punishment) were compared with IBIs that followed punishment from advantageous choices with low frequency (10% punishment). The 3 (Performance Group) × 2 (Frequency) ANOVA resulted in a nonsignificant effect of Punishment Frequency ($p = .35$), suggesting that heart rate is sensitive to the magnitude of punishment, rather than its frequency. Performance Group did not result in a main effect; neither did it alter any of the above described interactions, suggesting that performance groups were similarly sensitive to feedback. The correlation analysis between number of advantageous choices and IBI difference following punishment and reward was not significant.

Skin conductance. A similar analysis was performed for skin conductance activity. Figure 4 presents mean skin conductance in a 5,000-ms level following the feedback for each performance choice (Bechara et al., 1996; Tranel, 2000). To examine whether skin conductance level was differentially sensitive to magnitude and frequency of punishment, for each participant the peak latency of skin conductance value in the 0–5,000-ms time window following the feedback was selected (Bechara et al., 1996), and these values were referred to the mean SCL 1,000 ms preceding the presentation of feedback. There was no difference in skin conductance baseline level for reward versus punishment outcomes. The difference scores were submitted to a 3 (Performance Group) × (Feedback) × 2 (Gain) × 2 (Punishment Frequency) ANOVA. This analysis resulted in main effects of Feedback, $F(1,86)=21.60$, $p < .001$, $\varepsilon=1.000$, Gain, $F(1,86)=31.49$, $p < .001$, $\varepsilon = 1.000$, and Punishment Frequency, $F(1,86) = 14.00$, $p < .001$, $\varepsilon = 1.000$. These main effects were qualified by interactions between Gain and Feedback, $F(1,86) = 28.20$, $p < .001$, $\varepsilon = 1.000$, and between Punishment Frequency and Feedback, $F(1,86) = 15.90$, $p < .001$, $\varepsilon = 1.000$.

Post hoc ANOVAs showed that the interaction between Gain and Feedback indicated that when participants received reward feedback, skin conductance level did not differ between advantageous and disadvantageous choices, $F(1,90) = 0.90$, $p = .34$. However, when participants received punishment, skin conductance rise was much larger following feedback from disadvantageous choices compared to advantageous choices $F(1,88) = 36.22$, $p < .001$. Similarly, the interaction between Punishment Frequency and Feedback indicated that when participants received reward, skin conductance level did not differ between choices with high versus low punishment frequency, $F(1,92) = 0.26$, $p = .61$; in contrast, when participants received punishment, skin conductance level was higher when punishment was infrequent but high in magnitude, $F(1,88) = 16.01$, $p < .001$. A separate analysis of disadvantageous choices followed by high frequent punishment (50%) and advantageous choices with low frequent punishment (10%) did not result in a significant effect,

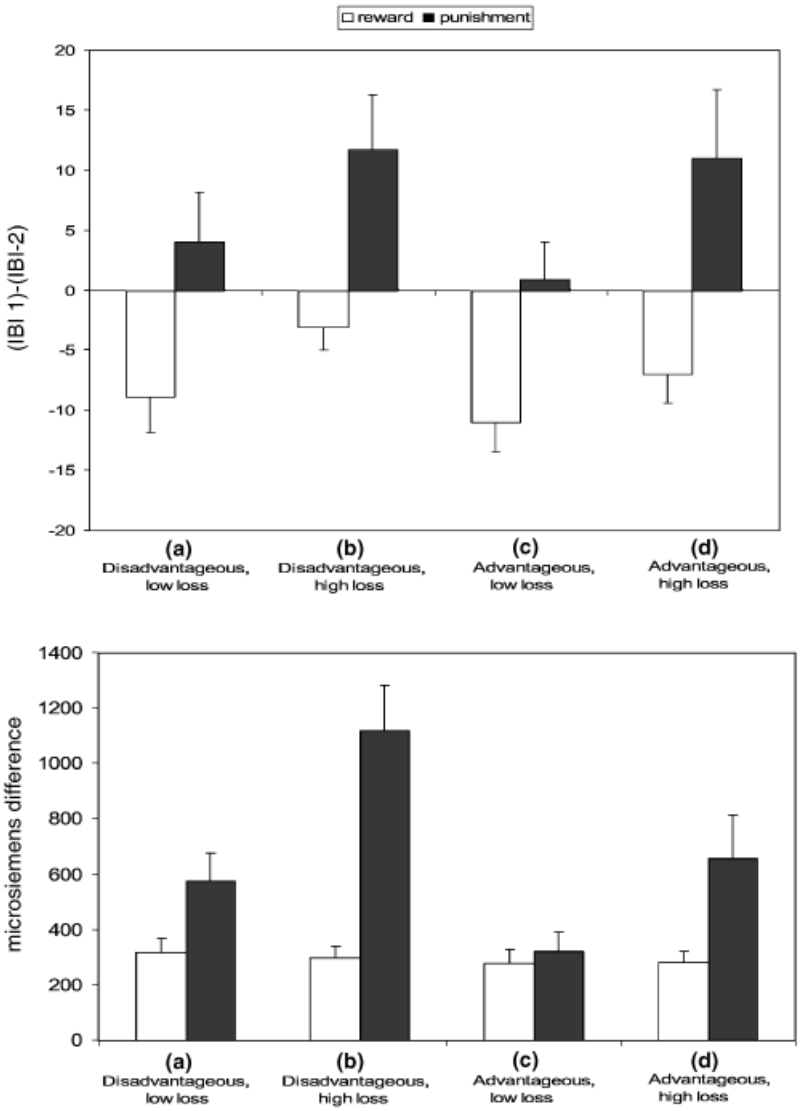

Figure 4. Heart rate and skin conductance level following reward and punishment.

suggesting that skin conductance was primarily sensitive to magnitude of punishment rather than to its frequency.

There was no main effect of Performance Group, and this factor did not alter any of the interactions. The correlation analysis between number of advantageous choices and difference between skin conductance rise following punishment and reward also did not result in a significant association. The absence of a modulating effect of Performance Group suggests that immediate effects of punishment were similar for all subjects and did not affect long-term performance strategy.

Discussion

The distinction in performance groups was successful, resulting in one third of the participants that were less likely to avoid the disadvantageous decks, one third that improved moderately, and one third that avoided disadvantageous decks relatively fast. In line with this distinction, the group of bad performers showed no differentiation in autonomic activity preceding disadvantageous and advantageous choices, suggesting that they received no somatic warning signals preceding risky choices. Moderate and good performers, in contrast, showed larger skin conductance activity preceding disadvantageous choices, and for good performers, both heart rate and skin conductance responses were larger when the disadvantageous choices could result in frequent punishment. These results are consistent with reports by Bechara et al. (1996, 2002) and Tomb et al. (2002), and show that for good performers risky choices are preceded by somatic warning signals.

An important question in this study was to what extent performance differences were related to processing of punishment outcomes. Both behavioral and autonomic measures revealed no such relation. First, performance groups did not show significant differences in immediate performance switches following punishment. These results are inconsistent with previous data investigating orbitofrontal cortex functioning in adult psychopathic individuals and patients with orbitofrontal damage who have indicated difficulties in response reversal (e.g., LaPierre, Braun, & Hodgins, 1995; Rolls, 2000), but are consistent with previous studies using the gambling task that reported no relation between gambling performance and response reversal (Blair, Colledge, & Mitchell, 2001; Crone, Vendel, & Van der Molen, 2003). Second,

consistent with Bechara et al. (1996) and Crone et al. (2003), skin conductance level increased and heart rate slowed following punishment, and these responses were positively related to the magnitude of punishment. Most important, autonomic activity following reward and punishment, as shown by both heart rate and skin conductance measures, was independent from performance strategy.

Damasio (1995) and Bechara and Damasio (2002) proposed a theoretical framework to account for the relation between immediate arousal following reward and punishment and long-term performance strategy. They suggest that unbalanced activities within the amygdala and orbitofrontal-insular cortex are interrelated and both have a critical role in encoding and using associative information about the motivational significance of stimuli (Gallagher, McMahan, & Schoenbaum, 1999; Schoenbaum, Chiba, & Gallagher, 1998, 2000). Within this circuit, the amygdala has a critical role in forming associations between unconditioned and aversive stimuli (Killcross, Robbins, & Everitt, 1997; LeDoux, 1998), and activity within this system may therefore be similar for the three performance groups. Orbitofrontal cortex encodes the motivational significance of the cues and the incentive value of expected outcomes (Gallagher et al., 1999; Schoenbaum et al., 2000; Thorpe, Rolls, & Maddison, 1983), and this system is expected to be differently involved within the three performance groups given the differences in response to changes in the value of reinforcement (Gallagher et al., 1999; Hatfield, Han, Conley, Gallagher, & Holland, 1996; Rolls, 2000).

The somatic states that are generated by recall and thought of future loss, for example, when pondering a decision with possible punishment consequences, have been referred to as secondary inducers because they are acquired only after associations with aversive stimuli (i.e., punishment) are formed (Bechara et al., 2002). In contrast, reward and punishment consequences were spelled about at the beginning of the task, and therefore induced a direct somatic response, which participants did not have to infer from previously acquired associations. This last type of somatic response has previously been referred to as primary inducers (Bechara et al., 2002). Thus, primary inducers are stimuli that are directly set as pleasurable or aversive, and when present in the immediate environment they automatically elicit a somatic response. The current results suggest that decision-making impairments in poor performers arise from a weak somatic response generated by secondary (i.e., acquired) inducers, rather than a weak somatic response generated by primary inducers of reward and punishment. In a previous study, Bechara et al. (1998) indicated that associations with secondary reinforcers are learned even before the participant is consciously aware of the differences in future outcomes, suggesting that these associations are learned implicitly. Within our design we did not examine participants' response strategy during the task, but an interesting avenue for future research would be to examine if poor performers rely more on an explicit learning mechanism than good performers, thereby possibly accounting for the slow acquisition or absence of acquiring somatic markers.

The pattern of bad performers is partly consistent with previous reports by Bechara et al. (2000, 2002), who showed that a subgroup of normal control participants performed disadvantageously on the gambling task but still acquired anticipatory skin conductance activity preceding bad choices. This group could be separated in a subgroup of normal control participants who showed no anticipatory skin conductance activity and a group having anticipatory skin conductance responses similar to good performing control participants. This last group was described by self-reports as high-risk takers and thrill seekers in real life. In these subjects the somatic state signals indexed by anticipatory skin conductance activity may have been overridden by conscious deliberation (Bechara & Damasio, 2002). The subgroup that performed disadvantageously and did not acquire anticipatory skin conductance activity preceding risky choices resembles the group of bad performers in the current experiment. The absence of somatic markers suggests defects in temporal integration between unconditioned stimuli and aversive outcomes, precluding effective display of knowledge representation required for the operations of reasoning. A second possibility is that these knowledge representations can be evoked, but they may be unstable (e.g., not held in working memory long enough to be implemented in reason strategies). The somatic markers are proposed to assist decision making in that they help inhibit the normal tendency to approach immediate reward and enhance and hold representation of future negative scenarios in working memory, and inefficiency of this process may account for disadvantageous performance.

Similarly as in our previous study (Crone & Van der Molen, 2004), we found that subjects developed a preference for choices associated with a low punishment frequency, suggesting that, although subjects' decision making is guided by a long-term goal (i.e., maximize yield) rather than immediate prospects, their local choices are still reward driven. Interestingly, good performers' choices associated with frequent but small punishment were preceded by larger skin conductance responses and larger heart rate slowing, suggesting that these choices are experienced as more aversive than choices associated with infrequent but large punishment. This assumption is consistent with a study performed by Critchley et al. (2001), who used fMRI to investigate the neural correlates of anticipation and autonomic arousal. They found that the orbitofrontal cortex and anterior cingulate cortex were active when the chances for punishment were higher (e.g., 50% chance vs. 33% chance). Interestingly, the anterior cingulate cortex was most associated with high arousal levels, as indexed by skin conductance activity, during the delay period between deciding and learning the outcome. Note that in this study participants were anticipating on the feedback and did not have an opportunity to adjust the response strategy. The anterior cingulate cortex has previously been associated with mediating conflict among possible options (Carter et al., 1998), as well as rapid outcome processing. These results suggest that, besides the orbitofrontal cortex, the anterior cingulated cortex may be a common region related to both bodily responses and cognitive appraisal of the chances and certainty of successful decision outcome.

The decision-making dysfunction seen in the bad performers may be a secondary consequence of some of the behavioral characteristics of impulsivity. For example, one aspect of impulsivity, sensation seeking, is often associated with higher rates of drug abuse, dependence, and multiple drug use (e.g., Hemphill, Hart, & Hare, 1994), and several studies have now reported that substance abusers show poor decision-making behavior (Grant, Contoreggi, & London, 2000; Schmitt, Brinkley & Newman, 1999) as well as decreased autonomic arousal preceding risky choices (Bechara & Damasio, 2002). Using a novel decision-making task, Rogers et al. (1999) assessed the quality of decision making and deliberation time of individuals with focal orbitofrontal cortex damage, and individuals who abused amphetamine or opiates. All three groups showed impaired performance on the task relative to comparison groups. Furthermore, chronic

amphetamine abusers showed a pattern of suboptimal decision making that correlated with their years of abuse. Future studies should examine which individual characteristics contribute to poor decision making in the normal population, because these individuals may be at risk for developing disorders related to impulsivity and dependence (Bechara & Damasio, 2002).

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