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Research Article

Neural Correlates of Adaptive Decision Making for Risky Gains and Losses

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ABSTRACT—*Do decisions about potential gains and potential losses require different neural structures for advantageous choices? In a lesion study, we used a new measure of adaptive decision making under risk to examine whether damage to neural structures subserving emotion affects an individual's ability to make adaptive decisions differentially for gains and losses. We found that individuals with lesions to the amygdala, an area responsible for processing emotional responses, displayed impaired decision making when considering potential gains, but not when considering potential losses. In contrast, patients with damage to the ventromedial prefrontal cortex, an area responsible for integrating cognitive and emotional information, showed deficits in both domains. We argue that this dissociation provides evidence that adaptive decision making for risks involving potential losses may be more difficult to disrupt than adaptive decision making for risks involving potential gains. This research further demonstrates the role of emotion in decision competence.*

Throughout life, every individual is confronted with decisions that bear a certain degree of risk. Despite its ubiquity, many individuals approach risk with trepidation, as the term *risk* itself often brings to mind thoughts of potential peril and danger. In fact, the dictionary definition of the term is "the possibility of suffering harm or loss" (American Heritage Dictionary of the English Language, Fourth Edition, cited by Dictionary.com, http://dictionary.reference.com/browse/risk). It follows that the ability to make advantageous choices in the face of risk, especially when undesired consequences may result from an imprudent choice, is an essential aspect of human survival.

Given the potentially detrimental consequences that an ill-considered choice may bring, it is important to understand the underlying psychological mechanisms that help people arrive at advantageous decisions. Emotions may be one such substrate. Indeed, research from a wide range of disciplines suggests that emotions play a critical role in decision making under uncertainty (e.g., A.R. Damasio, 1994; Hsu, Bhatt, Adolphs, Tranel, & Camerer, 2005; Huettel, Stowe, Gordon, Warner, & Platt, 2006; Kuhnen & Knutson, 2005; Loewenstein, Weber, Hsee, & Welch, 2001; Mellers, Schwartz, & Ritov, 1999; Peters & Slovic, 2000; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003; Schwarz & Clore, 1983; Shiv, Loewenstein, Bechara, Damasio, & Damasio, 2005). Further, damage to neural structures that subserve emotion can impair an individual's decision-making capabilities (e.g., Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Damasio, Damasio, & Lee, 1999). Although this research has provided valuable insights into the role emotion plays in the decision process, one question that has not been clearly answered is whether damage to the brain's emotional circuitry differentially impairs decision making as a function of whether the risk is presented in terms of potential gains or potential losses. In the lesion study reported here, we used a novel risky-decision task that separates these two decision domains in order to test whether damage to neural structures subserving emotion would differentially affect the ability to make adaptive decisions in these two domains. The specific brain regions we examined were the amygdala, an area responsible for processing emotional responses to environmental stimuli, and the ventromedial prefrontal cortex (VMPC), an area responsible for integrating cognitive and emotional information.

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AFFECT AND DECISION MAKING: A NEURAL PERSPECTIVE

According to *affect-as-information* theories of decision making (e.g., Loewenstein et al., 2001; Schwarz & Clore, 1983), affect that is experienced as reactions to an impending judgment may guide decisions via a "how do I feel about it?" strategy. Research suggests that in risky decision making, an "affect heuristic" (Finucane, Alhakami, Slovic, & Johnson, 2000; Slovic, Finucane, Peters, & MacGregor, 2002) directs judgments of risks and benefits. For example, Alhakami and Slovic (1994) found that when making judgments involving perceived benefits and risks of an activity or a technology, people are influenced not only by cognitive information, but also by the strength of positive or negative affect associated with that activity or technology.

Taking a neuroscience perspective, we propose a model that is consistent with affect-as-information models of decision making under uncertainty. According to this model, risky decision making is guided by two separate neurological processes, both generating emotion. The first process generates an automatic judgment and is focused on immediate outcomes. This judgment arises from the processing of a *primary inducer*, a stimulus in the environment (i.e., risk) that evokes an emotional response. Primary induction, the process of coupling stimulus features with affective value, is believed to be mediated by the amygdala (e.g., LeDoux, 2000; Malkova, Gaffan, & Murray, 1997).

Such amygdala-generated responses are rapid, automatic emotional responses to stimuli; they are short-lived and habituate quickly (Bechara, 2005). Moreover, in a lesion study, Bechara et al. (1999) found that individuals with bilateral damage to the amygdala performed poorly on the Iowa gambling task (IGT). The authors proposed that amygdala patients' poor performance resulted from the inability to generate physiological responses to experienced rewarding and punishing consequences of their behavior (Bechara et al., 1999). In other words, the inability to generate a primary response to experienced punishments precluded any opportunity to effectively utilize such responses in a prospective manner.

A second process that guides risky decision making is more deliberative and focuses more on emotional responses associated with anticipated outcomes. In this conceptualization, amygdala-generated responses trigger the VMPC, a structure believed to mediate decision making by linking together working memory and emotional systems (A.R. Damasio, 1994). The VMPC subsequently prompts a more careful, deliberative analysis of the decision, an analysis that is believed to trigger *secondary* emotional responses generated from thoughts about the risky decision. These emotional responses are believed to be essential for advantageous prospective decision making. Individuals with VMPC damage (but with an intact amygdala) are unable to generate *anticipatory* emotional responses—which may provide an early-detection system that warns of future punishments—even though these patients generate emotional responses to experienced punishments and rewards (Bechara et al., 1997).

This framework suggests that both the amygdala and the VMPC are vital to risky decision making. We propose that for adaptive decision making to occur when an individual approaches a risky decision, the amygdala must first process an instinctive, automatic response to the risk or uncertainty. This response then engages the VMPC, which conducts a more deliberative evaluation of the risk and evokes a secondary emotional response that helps guide decision making. If the amygdala does not evoke a primary response, the VMPC will not engage as it does in normal individuals. In other words, an amygdala response is a necessary first step for engaging the VMPC. However, even if the amygdala responds to risk, decision making is ultimately dependent on the integrity of the VMPC.

Accordingly, this model suggests that if the integrity of these structures is compromised, decision-making deficits will occur. Indeed, previous research using lesion patients supports this theory (Bechara et al., 1997, 1999). However, because previous investigations have employed tasks like the IGT, which involve "mixed" gambles (i.e., risky decisions that involve elements of both potential gains and potential losses), it is difficult to tease out whether these structures are vital to adaptive decision making both when people make choices to achieve gains and when people are faced with choices involving potential losses. Although a mixed-gambles task can provide valuable insights about decision-making behavior, there is strong reason to believe that individuals approach gain- and loss-related decisions differently. For instance, behavioral research suggests that the psychological impact of potential losses is stronger than the psychological impact of potential gains (e.g., Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001), perhaps because realized losses may carry harsher implications for survival. Moreover, prospect theory's value function, which is concave for potential gains and convex (and steeper) for potential losses, predicts that when a decision is framed in terms of losses, individuals will seek risk, but when a decision is framed in terms of gains, individuals will be risk averse (Kahneman & Tversky, 1979). Thus, preference for risk differs between these two decision domains (Levin, Schneider, & Gaeth, 1998).

In the current study, we used a new methodology that allowed us to analyze adaptive decision making separately for decisions involving potential gains and decisions involving potential losses. Specifically, we assessed subjects' capacity to adjust choices on the basis of differences in the choice options' expected value (EV), comparing performance of two groups of lesion patients with performance of a healthy comparison group. Thus, we tested directly whether damage to the amygdala or the VMPC is sufficient to disrupt decision making, regardless of whether the decision is presented in terms of potential gains or potential losses.

METHOD

Subjects

Patients

Subjects with brain lesions were selected from the patient registry of the University of Iowa's Division of Cognitive Neuroscience. Basic neuropsychological and neuroanatomical characteristics of these patients were evaluated according to the standard protocols of the Benton Neuropsychology Laboratory (Tranel, 1996) and the Laboratory of Neuroimaging and Human Neuroanatomy (H. Damasio, 1995; H. Damasio & Frank, 1992). Clinical interviews indicated that none of the subjects had a history of mental retardation, psychiatric disorder, substance abuse, learning disability, or systemic disease that would affect the central nervous system. All data were collected when the subjects were in the chronic phase of recovery (i.e., at least 3 months after lesion onset).

The amygdala patients ($n = 16$; 6 males, 10 females) had either unilateral (6 left, 7 right) or bilateral ($n = 3$) damage in an anterior temporal region including the amygdala. The degree of damage in adjacent and surrounding hippocampal and temporal cortices varied. For the VMPC group ($n = 7$; 5 males, 2 females), the damage included the ventral and low mesial sectors of the frontal lobe in both the right and left hemispheres (i.e., bilateral damage). In all subjects, the dorsolateral sectors of the prefrontal cortex were intact.

Nonpatient Comparison Subjects

Thirty individuals were recruited as nonpatient comparison subjects (11 males, 19 females). These subjects were recruited through advertisements in local newspapers and were compensated for their time. They were screened for history of neurological or psychiatric disease.

Procedure

To assess individuals' decision propensities under risk, we used a modified, computerized version of Levin and Hart's (2003; Levin, Weller, Pederson, & Harshman, 2007) cups task. This task was originally designed to provide a simple way of showing subjects the probability of an outcome (by depicting a particular number of cups from which to choose) in a risky choice. In our modification, we added a within-subjects manipulation of the relative EV of the risky and riskless options; with this manipulation, we were able to assess decision makers' sensitivity and adaptability to contingencies that make a risky choice advantageous or disadvantageous.

In the current version of the cups task, subjects made choices with real monetary outcomes. The independent variables were domain (gain or loss), probability of a nonzero outcome if the risky option was selected (.20, .33, or .50), and outcome magnitude for the risky option (two, three, or five quarters vs. one quarter for the riskless option). Subjects received three trials of each combination of probability level and outcome level, for a total of 54 trials. Some combinations of probability and magnitude created equal EV for the risky and riskless options: This was the case for .20 chance of five quarters, .33 chance of three quarters, and .50 chance of two quarters, on both gain and loss trials. Other combinations were risk advantageous (i.e., the EV was more favorable for the risky than for the riskless option): This was the case for .33 chance of five quarters, .50 chance of three quarters, and .50 chance of five quarters on gain trials, and for .20 chance of two quarters, .20 chance of three quarters, and .33 chance of two quarters on loss trials. Finally, still other combinations were risk disadvantageous (i.e., the EV was more favorable for the riskless than for the risky option): This was the case for .20 chance of two quarters, .20 chance of three quarters, and .33 chance of two quarters on gain trials, and for .33 chance of five quarters, .50 chance of three quarters, and .50 chance of five quarters on loss trials. Gain and loss trials were presented as separate blocks, with order counterbalanced across subjects in each group. Within a block, combinations of probability and outcome were presented in random order.

Gain trials involved the choice between an option that offered a sure gain of one quarter and an option that offered a designated probability of winning multiple quarters. Loss trials involved the choice between a sure loss of one quarter and a designated probability of losing multiple quarters. On each trial, an array of two, three, or five cups was shown on each side of the screen. One array was identified as the certain side: If any cup on this side was selected, one quarter was gained or lost, depending on the domain for that trial. The other array was identified as the risky side: Selection of one cup on this side led to a designated number of quarters being gained or lost, and selection of the other cups led to no gain or loss. At the bottom of the screen was a depiction of a bank; coins were added to or subtracted from this bank according to a trial's outcome. At the start of each loss trial, the bank contained the maximum number of quarters that could be lost on that trial. The outcome on each trial depended on which side was selected and, if it was the risky side, the choice of one cup. For a risky choice, whether the cup selected led to a nonzero outcome was determined by a random process with p equal to 1 divided by the number of cups. When a subject completed all 54 trials, the total amount he or she had won appeared on the screen. Subjects were compensated with the amount that they had won on the task.

Dependent Variables

To assess decision making under risk, we calculated overall risk taking in both the gain and the loss domains. We also measured how subjects adapted to differences in EV between the riskless and risky options, by calculating the proportion of risky choices for trials at each of three EV levels: risk-advantageous trials (the EV of the risky choice was more favorable than that of the riskless choice), equal-EV trials (the EVs for the riskless and risky options were equal), and risk-disadvantageous trials (the EV was more favorable for the riskless option than for the risky

option). These proportions were computed separately for the gain and loss domains.

RESULTS

Demographic Statistics

The ages of the amygdala patients ($M = 40.56$, $SD = 14.89$), VMPC patients ($M = 45.86$, $SD = 24.14$), and comparison group ($M = 39.37$, $SD = 12.69$) did not differ significantly. The comparison group had significantly more years of education ($M = 16.10$, $SD = 2.42$) than the amygdala patients ($M = 14.13$, $SD = 2.42$) and the VMPC patients ($M = 11.43$, $SD = 2.94$), $F(2, 52) = 11.20$, $p < .01$, but this difference did not have a significant influence on our results.[1]

Preference Shifts in Risky Decision Making Involving Gains Versus Losses

To test for evidence of preference shifts in risky decision making (i.e., greater risk taking in the loss domain than in the gain domain), we conducted three repeated measures analyses of variance (ANOVAs), with overall risk taking in each domain (gain, loss) as the dependent variable. Although the comparison group demonstrated the predicted preference shift, $F(1, 30) = 9.07$, $p < .01$, $\eta^2 = .24$, neither amygdala nor VMPC patients were significantly more risk seeking in the loss domain than in the gain domain (Fs < 1.0). These findings suggest an altered response pattern for lesion patients compared with healthy adults.

Adaptive Decision Making

Figure 1 plots the proportion of risky choices for each group as a function of EV level, separately for gain and loss trials. Inspection of the figure reveals several group differences. Regardless of decision domain, VMPC patients not only made more risky choices than the other groups, but also displayed insensitivity to differences in EV between choice options. In contrast, the amygdala patients' performance differed across domains. Their responses to loss trials were indistinguishable from those of the healthy comparison group in terms of both overall level of risk taking and sensitivity to EV differences between choice options. However, like VMPC patients, amygdala patients displayed elevated levels of risk-seeking behavior and impaired adaptive decision making in the gain domain.

We conducted a 3 (EV level) × 2 (domain) × 3 (group: amygdala,[2] VMPC, healthy) ANOVA to compare the groups' risk taking as a function of EV differences between choice options in each domain. As expected, we found a main effect for group, $F(2, 52) = 4.96$, $p = .01$, $\eta^2 = .17$, in that lesion patients, regardless of lesion type, showed more overall risk taking than healthy adults. As Figure 1 suggests, group differences in risk taking were especially apparent on the risk-disadvantageous trials. To test the simple effects, we conducted parallel t tests comparing each lesion group with the comparison group on these trials, in both domains. We found that compared with healthy adults, both VMPC and amygdala patients displayed elevated risk taking on risk-disadvantageous gain trials, $t(35) = 4.53$, $p < .01$, $d = 1.73$, and $t(44) = 4.39$, $p < .01$, $d = 1.32$, respectively. In the loss domain, VMPC patients were more risk taking on

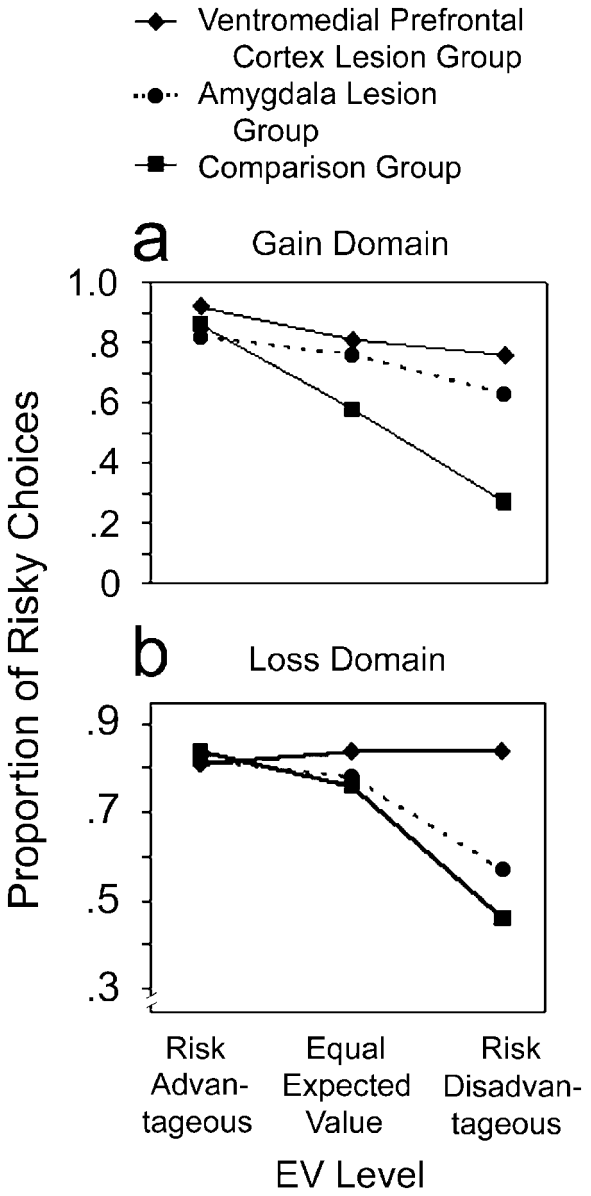

Fig. 1. Proportion of risky choices involving (a) gains and (b) losses, as a function of subject group and expected-value (EV) level. Subjects received nine gain trials and nine loss trials for each of the three EV levels.

[1]For all subsequent analyses, we conducted multiple regression analyses that tested whether education was a significant predictor of the dependent measures. The results of these analyses did not differ from those presented, and therefore are not discussed further.

[2]Because there were differences in the location of the amygdala lesion (i.e., bilateral, left, and right), it was possible that this variation affected the results. To test this possibility, we conducted one-way ANOVAs comparing the amygdala lesion subgroups (i.e., bilateral, left, and right) for all dependent variables reported in this study. There were no significant differences among these subgroups for any variable of interest. Thus, we collapsed the data for all amygdala subgroups into a unitary amygdala group.

risk-disadvantageous trials than were both the healthy subjects, $t(35) = 2.97, p < .01, d = 1.42$, and amygdala patients, $t(21) = 2.19, p < .05, d = 1.06$. However, amygdala patients did not differ significantly from the comparison group on these trials, $t(44) = 1.12$, n.s., $d = 0.35$.

Because our focus was on comparing risk-advantageous with risk-disadvantageous trials, we used a linear contrast between these two levels as a more powerful test of the effects of EV level. As expected, we found a main effect: The proportion of risky choices was greater on the former trials (i.e., when the EV was more favorable for the risky option than for the riskless option), $F(1, 52) = 45.10, p < .01, \eta^2 = .37$. We also found an EV Level × Group interaction, $F(2, 52) = 12.70, p < .01, \eta^2 = .21$; the level of risk taking among lesion patients, compared with healthy subjects, was particularly elevated when the EV for the risky choice was less favorable than the EV for the riskless option. Furthermore, we found a three-way Domain × EV Level × Group interaction, $F(2, 52) = 3.25, p < .01, \eta^2 = .11$; the VMPC group was insensitive to EV level in both domains, but the amygdala group demonstrated a comparable insensitivity only in the gain domain.

DISCUSSION

Our results strongly reinforce previous research and theory indicating that decision making is not mediated solely by cold cognitive processes (e.g., A.R. Damasio, 1994), and that arriving at an advantageous decision also requires affective information. VMPC-generated emotions appear to play a vital role in individuals' ability to make adaptive decisions in the face of risk, regardless of whether the goal of the decision is to achieve a gain or avoid a loss. VMPC patients displayed elevated risk-seeking behavior regardless of both EV considerations and domain. This deficit was especially visible in VMPC patients' propensity to take risks even when the odds of success were small. These findings add to a growing literature that implicates the VMPC in decision making under uncertainty (Bechara et al., 1997; Bechara et al., 1999). Notably, however, Leland and Grafman (2005) found that patients with VMPC lesions did not differ from healthy subjects on a variety of hypothetical scenario-based decision tasks in which the consequences of choices were not realized. In contrast, our task involved immediately realized consequences for choices. We believe that our results differ from those of Leland and Grafman (2005) because of increased emotional responses resulting from the temporal proximity between choice and outcome (e.g., Loewenstein, 1987).

The most intriguing finding of our study, though, is the dissociation in risky decision making between VMPC and amygdala patients across decision domains. Whereas VMPC patients exhibited a cross-domain deficit, amygdala patients did not. Instead, they showed elevated risk taking and insensitivity to differences in EV between choice options in the gain domain, but a pattern of decision making strikingly similar to that of healthy individuals in the loss domain (i.e., taking more risks when it was advantageous to do so and fewer risks when it was disadvantageous to do so).

These results accentuate the importance of distinguishing between gain- and loss-related decision making under uncertainty. We found that amygdala damage was associated with suboptimal decision making in the gain domain for the cups task, a result consistent with the findings of Bechara et al. (1999), who reported that amygdala damage is associated with impairment on the IGT. However, the absence of decision deficits in the loss domain represents a unique dissociation. One plausible reason for the apparent inconsistency between this previous study and our results for the loss domain lies in the decision tasks utilized in these two studies. Although the IGT can detect decision-making deficits within clinical populations, it is a complex task that blends many distinct components, including a mixture of rewards and punishments, and risk and ambiguity. Moreover, Bechara et al. found that amygdala patients demonstrated low reactivity to punishments and rewards. Yet the physiological responses they reported for punishments were not pure punishment responses. That is, they were responses to a reward followed by a punishment (e.g., "you won x amount, but you lost y amount"). For these reasons, it is difficult to interpret studies using the IGT in terms of decision deficits within specific domains. In contrast, the cups task was designed to separate decision domains, and this allowed us to delineate clearly whether damage to particular brain areas differentially influenced risk taking.

Additionally, our findings provide partial support for recent functional imaging studies that have found amygdala involvement in decision making (DeMartino, Kumaran, Seymour, & Dolan, 2006; Coricelli et al., 2005). It is of particular interest that DeMartino et al. found that bilateral amygdala activation was associated with risk-avoidant choice behavior in the gain domain, but with risk-seeking behavior in the loss domain. Extrapolating from DeMartino et al., one might predict that amygdala damage would result in risk-seeking behavior for gains and risk-averse choices for losses. Although our findings are consistent with this prediction for the gain domain, they are not consistent with this prediction for the loss domain.

One potential explanation for this discrepancy is that our study included patients with unilateral amygdala damage. However, this explanation seems unlikely to be correct because the patients with bilateral amygdala lesions did not differ significantly from the comparison group in their risk-seeking behavior in the loss domain, even on the most risk-disadvantageous trials. Nonetheless, future research should investigate and reconcile differences in results obtained by lesion and functional imaging studies whenever these results are conflicting, as in the case of our study and that of DeMartino et al. (2006).

To account for our data, we propose expanding the amygdala-VMPC neural model of decision making to accommodate the possibility of redundant neural systems that may process emotional information when a potential loss is at stake (see Fig. 2).

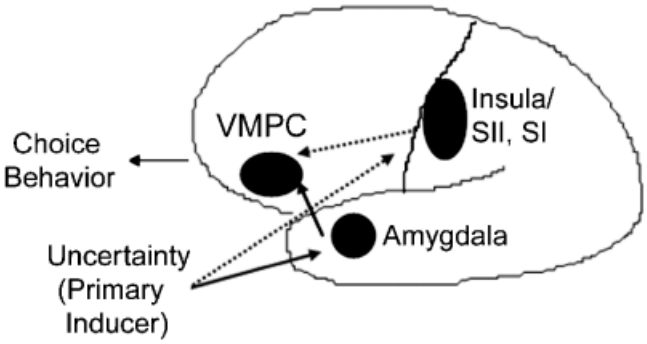

Fig. 2. Illustration of the expanded neural model of decision making under uncertainty. Processing of primary inducers, mediated by the amygdala, triggers the ventromedial prefrontal cortex (VMPC) system, which, in turn, conducts a more deliberative analysis of uncertainty. However, decisions involving potential losses may trigger redundant neural responding from structures such as the insula (anterior, posterior, or both) and the adjacent primary and secondary somatosensory cortices (SI and SII), which are independent of the amygdala; these backup processes are represented here by dotted lines. Thus, the VMPC system can still be engaged when the amygdala is damaged.

Although the amygdala may indeed be important for processing initial responses that are vital in decision making concerning potential losses (DeMartino et al., 2006; Coricelli et al., 2005), other structures, such as the anterior insula, may produce similar responses (Paulus & Stein, 2006) and thus render adaptive decision making involving potential losses more difficult to disrupt than adaptive decision making involving potential gains. Such redundancy may provide backup mechanisms in the event that one area, such as the amygdala, becomes damaged or dysfunctional. Future research is needed to test this proposition.

In sum, these results reveal that decision making under risk may depend on partially separate neural systems for dealing with potential losses and potential gains. These findings are important from the perspectives of both economics and neuroscience. In economics, researchers have long suggested that losses loom larger than gains (Kahneman & Tversky, 1979). Humans may have adapted to survival pressures by utilizing multiple neural systems to make decisions involving the loss of property, food, and other valuables. Therefore, exploring the neural correlates of risky decisions, as they relate to the domains of gains versus losses, will enrich scientific knowledge of the neural foundations of economic and social decisions. On the neuroscience front, elucidating the neural processes underlying different types of decisions will advance understanding of the neural "road map" for the physiological processes intervening between knowledge and behavior, and of the potential interruptions that lead to a disconnect between what one knows and what one does.

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