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Increased activation in the right insula during risk-taking decision making is related to harm avoidance and neuroticism

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Abstract

Decision making and risk taking are interrelated processes that are important for daily functioning. The somatic marker hypothesis has provided a conceptual basis for processes involved in risk-taking decision making and has been used to link discrete neural substrates to risk-related behaviors. This investigation examined the hypothesis that the degree of risk-taking is related to the degree of activation in the insular cortex. Seventeen healthy, right-handed subjects performed a risk-taking decision-making task during functional magnetic resonance imaging (fMRI) using a fast event-related design. This investigation yielded three main findings. First, right insula (BA 13) activation was significantly stronger when subjects selected a "risky" response versus selecting a "safe" response. Second, the degree of insula activation was related to the probability of selecting a "safe" response following a punished response. Third, the degree of insula activation was related to the subjects' degree of harm avoidance and neuroticism as measured by the TCI and NEO personality questionnaires, respectively. These results are consistent with the hypothesis that insula activation serves as a critical neural substrate to instantiate aversive somatic markers that guide risk-taking decision-making behavior.

Introduction

Decision making, i.e., selecting an action from a set of alternatives with an uncertain outcome, consists of several component processes. A particularly important component of decision making is risk taking, which can be defined as the propensity to select an action with the potential for a relatively large beneficial or adverse outcome over an alternative action that results in a relatively small beneficial outcome (Slovic, 1987; Mellers et al., 1997). Risk taking itself, however, can be broken down into several components, including anticipation, reward, and punishment-related processing.

Abnormalities of risk-taking aspects of decision-making behavior have been observed in several psychiatric disorders (Mogg et al., 1991; Rahman et al., 2001; American Psychiatric Association, 1994; Rahman et al., 1999), including substance-related syndromes (Rogers et al., 1999a). For example, substance dependent subjects are more likely to select a high gain/high-risk alternative (Bechara, 2001) over a low-gain/low-risk alternative even when the former alternative is associated with a disadvantageous long-term outcome.

Experimentally, risk-taking decision-making behavior appears to be highly sensitive to context. For example, the selection of the risky alternative is dependent on the number of other available outcomes (Weber et al., 1992), on the stimulus context (Mellers and Chang, 1994), and on cultural background of the subject (Hsee and Weber, 1999). One common approach to examine risk-taking behavior is to present subjects with a choice between a sure thing and a gamble (Yates and Stone, 1992). By varying the expected value of each action alternative (i.e., the magnitude of the beneficial effect multiplied by the likelihood of the out-

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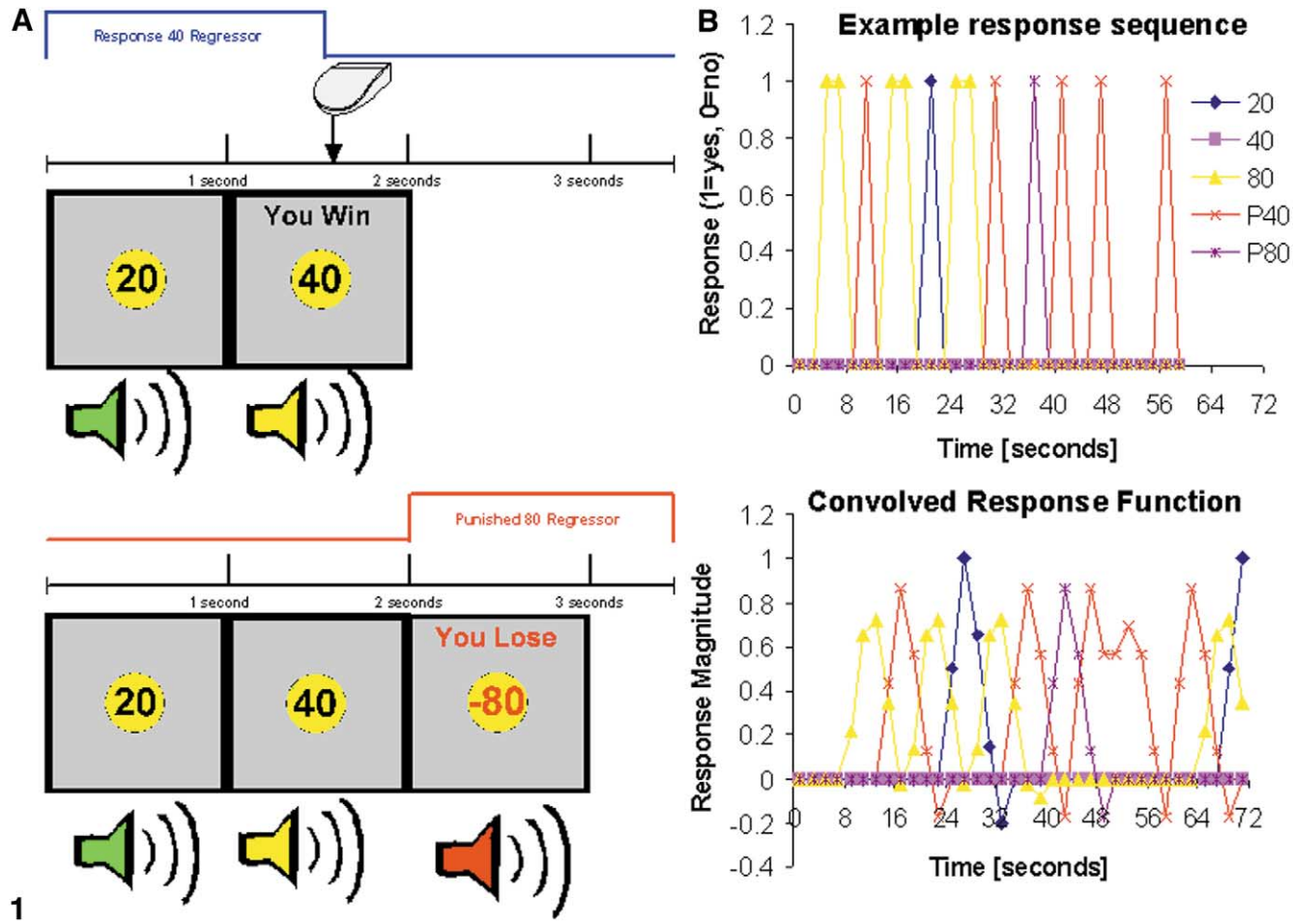


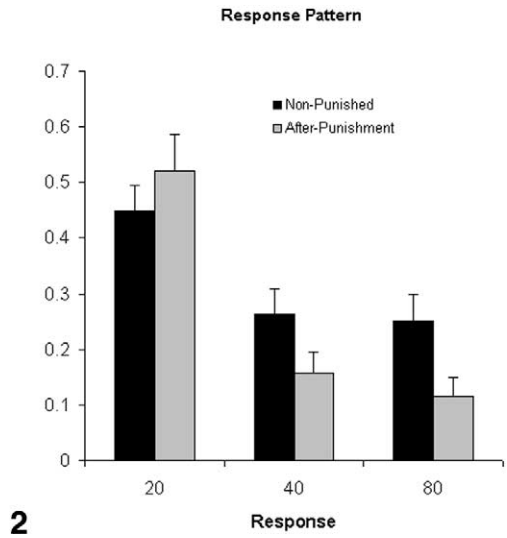
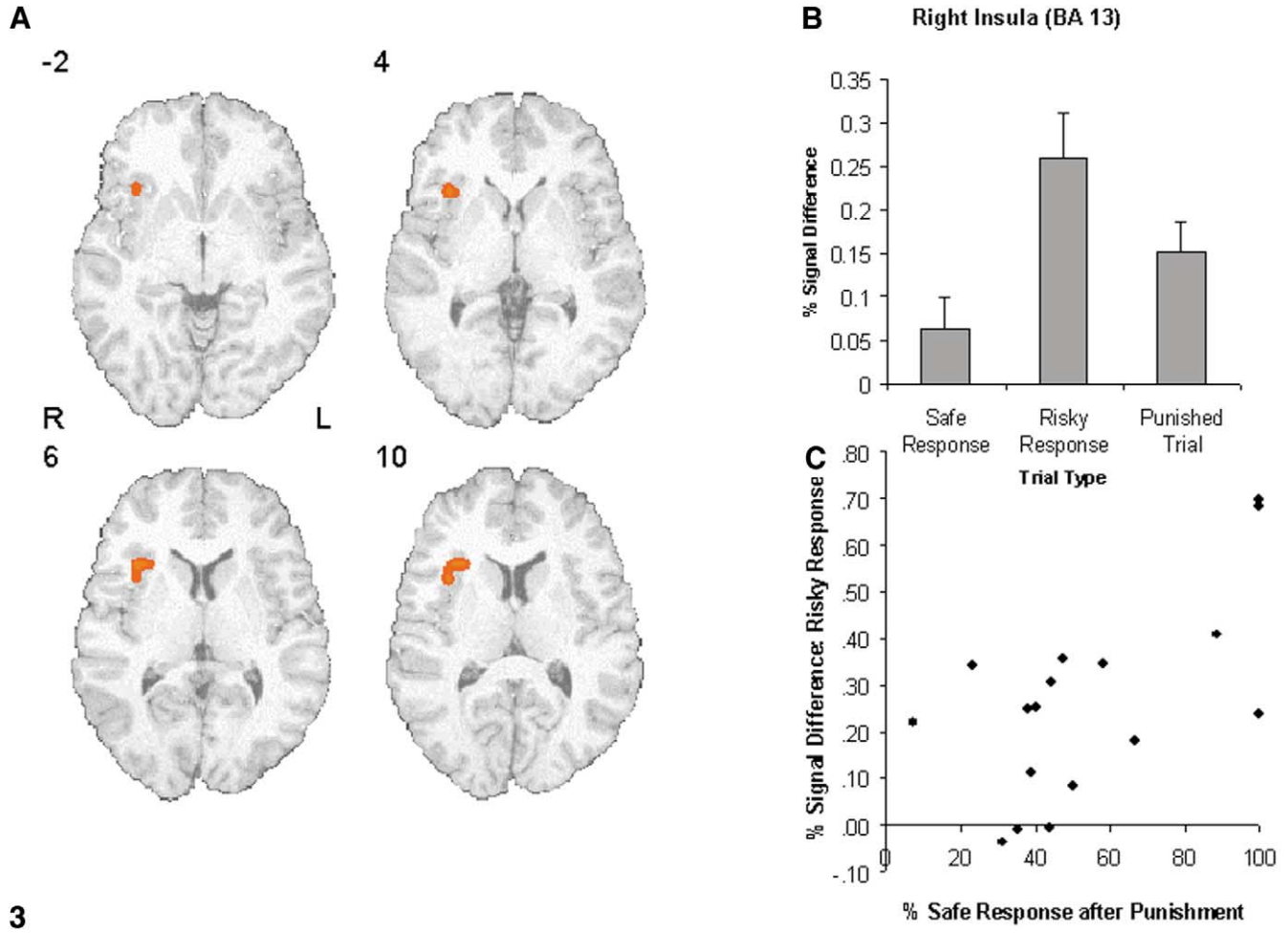


Fig. 1. Risky-Gains decision-making task. (A) Individual regressor functions based on subject's responses; 20, 40, 80 = selected 20, 40, or 80 respectively; P40, P80 = punished with 40 or 80 (B).
Fig. 2. Response probability during the Risky-Gains task following nonpunished (black) and punished (gray) trials.

come), one can determine whether subjects are risk seeking (selecting the gamble even when the expected value is lower than the sure thing) or risk averse (selecting the sure thing even when the expected value of the gamble is higher).

Functional neuroimaging studies have shown that risk-taking decision making is critically dependent on the activation of inferior prefrontal cortex (Paulus et al., 2001; Ernst et al., 2002), ventromedial and ventrolateral frontal



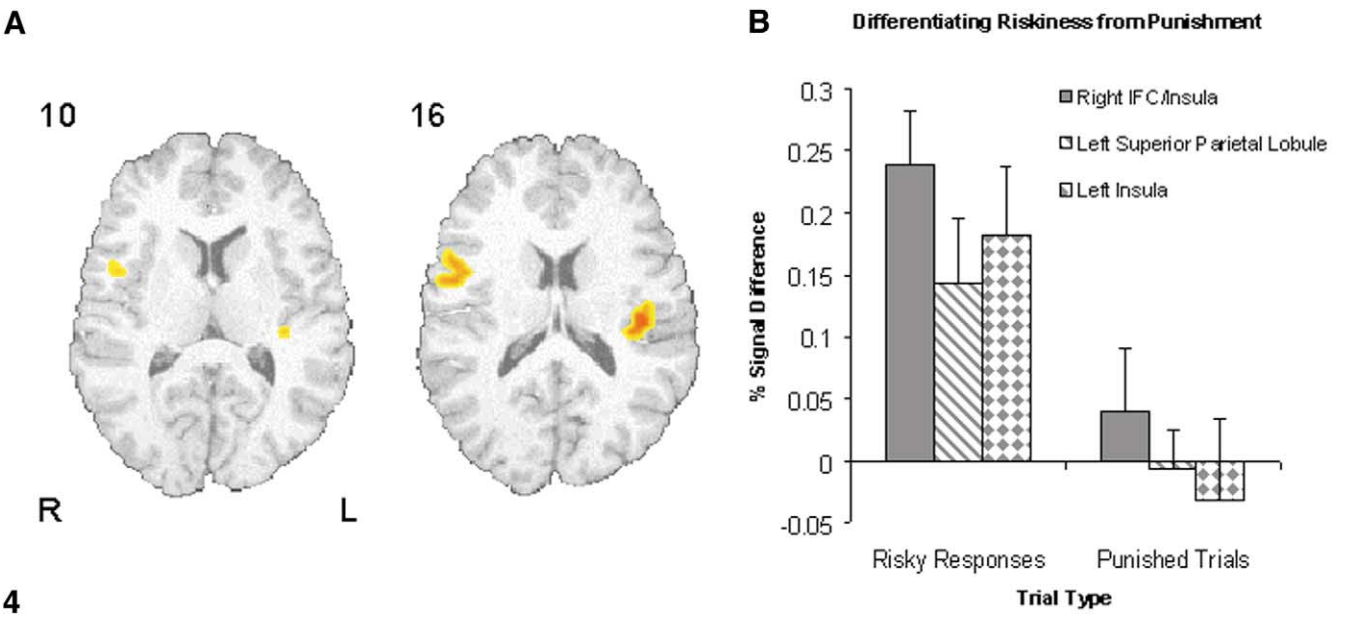


Fig. 3. (A) Contrast analysis "risky"–"safe" responses: Volume thresholded cluster of activation in the right insula, numbers indicate *z* coordinate. (B) Percentage of activation over rest during the "safe," "risky," and "punished" trials. (C) Scatter plot between the percentage of "safe" responses following a punished trial and the degree of activation in the anterior insula during a "risky" response.

Fig. 4. (A) Contrast analysis "risky" responses—punished trials: volume threshholded cluster of activation in the right inferior frontal gyrus/insula and the left insula. (B) Percentage of activation above rest during "risky" responses and "punished" trials.

cortex (Elliott et al., 1999, 2000a; Rogers et al., 1999b), anterior cingulate (Elliott et al., 2000a), insula (Critchley et al., 2001), and parietal cortex (Paulus et al., 2001). The anterior cingulate has been implicated in the response selection process when the reward magnitude is altered (Bush et al., 2002), whereas the nucleus accumbens has been shown to activate during anticipation of reward (Knutson et al., 2001). Others have argued that anterior cingulate activation during decision making is related to the degree to which the outcome is uncertain (Elliott and Dolan, 1998), whereas the activation in the nucleus accumbens is due to the calculation of an error signal between an expected and received reward (Pagnoni et al., 2002).

The precise role of the neural substrate underlying risk-taking decision making is not fully understood. The somatic marker hypothesis (Damasio, 1996) has provided a conceptual basis for processes involved in risk-taking decision making and has been used to link discrete neural substrates to risk-related behaviors. This hypothesis poses that external or internal stimuli initiate a state that is associated with pleasurable or aversive somatic markers. These markers function to guide the person's behavior by biasing the selection toward actions that result in an increase in pleasurable somatic markers (while avoiding actions resulting in aversive somatic markers).

The neural systems underlying the somatic marker hypothesis comprise the ventromedial and orbitofrontal cortex, amygdala, insula, and ventral striatum. In particular, the insula acts as a critical interface between affective inputs from limbic structures such as the orbitofrontal cortex, amygdala (McDonald et al., 1999), and anterior cingulate and the attentional prefrontal-parietal network in the processing of somatic states associated with risk-taking decision making (Bechara, 2001). Within the context of the somatic marker hypothesis, the insula has been conceptualized as part of both the "body" and the "as-if" loop system that is critical for the initial representation and the reenactment of somatic markers (Bechara, 2001). In this scenario, increased activation in the insula may signal the strength of the somatic state. If the insula signal is associated with aversive somatic markers, a relatively large activation during a decision-making situation would signal a potentially aversive outcome and may guide the subject to avoid the selection of a risky action alternative.

This investigation examined the hypothesis that the degree of risk-taking is related to the degree of activation in the insula. Specifically, it was hypothesized that a large activation in the insula during a risky response, which would correspond to a potent aversive representation of a somatic state, is associated with a lower propensity to select a risky response. Moreover, if insula activity were related to risk-taking behavior and not to response to punishment, one would expect to observe differential activation during risk-taking trials that were not punished versus those that were punished. Finally, to examine the external validity of this approach, two temperament or personality measures were obtained and correlated with activation in the insula. Cloninger (1987) developed the Temperament and Character Inventory (TCI) to quantify several dimensions of personality. These temperamental dispositions are defined in terms of the basic stimulus–response characteristics and comprise novelty seeking, harm avoidance, and reward dependence. In particular, harm avoidance has been used as a measure of anxiety proneness and reduced risk-taking propensity (Cloninger et al., 1998). In addition, the NEO personality inventory (Costa and McCrae, 1992), a measure of five personality factors, was used to obtain convergent validity that temperamental sensitivity to negative stimuli is closely associated with harm avoidance. In combination, it was hypothesized that a high degree of harm avoidance (i.e., the opposite of risk taking) or neuroticism is associated with a large activation in the insula during a risky response.

Methods

Subjects

Seventeen healthy, right-handed subjects (6 females and 11 males) age 38.3 years ± 1.4 (range 27–53) with an average education level of 14.7 ± .5 years (range 11–18) without a life-time history of Axis I DSM-IV disorders based on a structured clinical interview for DSM-IV diagnosis (Spitzer et al., 1992) participated in this study, which was approved by the UCSD Human Research Protection Program. These subjects gave their informed, written consent and performed a Risky-Gains decision-making task during functional magnetic resonance imaging. All subjects were given the Temperament and Character Inventory (Cloninger, 1987) as well as the Neuroticism Extraversion Openness Five Factor Inventory (NEO-FFI) (Costa and McCrae, 1992) and asked to return the completed questionnaire in a stamped envelope, we received completed questionnaires from 15 of 17 subjects.

Task

For the Risky-Gains task, subjects are presented with three numbers in ascending order (20, 40, and 80; see Fig. 1). Each number is presented on the screen for one second and if the subject presses a button when the number is shown on the screen, he/she receives the number of points shown on the screen. The subjects are informed that for both 40 and 80 points there is a chance that a 40 or 80 in red color may appear on the computer screen which signals that the subject loses 40 or 80 points, respectively. Thus, although the subject may gain more points per trial by waiting until a 40 or 80 appears on the screen, there is also a risk of losing 40 to 80 points. The probabilities of presenting a negative 40 or 80 are such that a subject's final score would be identical were they to consistently select 20, 40, or 80. Thus, there was no inherent advantage to select the risky response

(40 or 80) over the safe response (20). Each trial lasts 3.5 s irrespective of the subject's choice and the subject receives rewarding feedback (stimulus on the screen and auditory sound) immediately after selecting a response.

Behavioral measures

The 96 trials of the Risky-Gains task consist of three trial types, which were presented in randomized order: (1) a nonpunished trial type ($n = 54$), (2) a 40 punished trial type ($n = 24$), and (3) an 80 punished trial type ($n = 18$). The primary dependent measure to assess the degree of risk taking and the response to punishment is the probability of selecting 20, 40, or 80 as a function of the previous trial outcome (punished versus nonpunished).

Personality questionnaires

Of the 17 subjects, 15 subjects completed a TCI 125, which consists of 125 true/false statements to assess harm avoidance, reward dependence, novelty seeking, persistence, self-directiveness, cooperativeness, and self-transcendence (Cloninger, 1987; Cloninger et al., 1991) and the NEO Five Factor Inventory (FFI) (Costa and McCrae, 1992; Costa Jr. and McCrae, 1997), which consists of 60 statements that are rated by the subjects on a 5-point scale from "strongly agree" to "strongly disagree." The NEO-FFI can be used to extract five personality factors: neuroticism, extraversion, openness, agreeableness, and conscientiousness. These NEO-FFI factors were transformed to normalized *z* scores.

Functional magnetic resonance imaging

During the decision-making task a functional imaging run sensitive to blood oxygenation level—dependent (BOLD) contrast was collected for each subject using a 1.5-T Siemens (Erlangen, Germany) scanner (T2*-weighted echo-planar imaging, TR = 2000 ms, TE = 40 ms, 64 × 64 matrix, 20 4-mm axial slices, 256 scans). fMRI volume acquisitions were time-locked to the onset of each trial. During the same experimental session, a T1-weighted image (MPRAGE, TR = 11.4 ms, TE = 4.4 ms, flip angle = 10°, FOV = 256 × 256, 1 mm^3 voxels) was obtained for anatomical reference. For preprocessing, voxel time series were interpolated to correct for nonsimultaneous slice acquisition within each volume and corrected for three-dimensional motion. One subject was excluded due to large movement artifacts apparent during systematic visual inspection of the voxel time series.

FMRI analysis pathway

The data were preprocessed and analyzed with the software AFNI (Cox, 1996). The echo-planar images were realigned to the 128th acquired scan and time corrected for slice acquisition order. To exclude the voxels showing an artifact related signal drop, a combined threshold/cluster-growing algorithm was applied to the mean of the functional images to compute a whole brain mask. This screened out nonbrain voxels and voxels falling within the artifact region. A randomized fast-event related design was used with 6 resting trials interspersed between the 96 Risky-Gains trials. The preprocessed time series data for each individual were analyzed using a multiple-regression model consisting of 10 regressors. Five regressors of interest were constructed from the behavioral data obtained during the task. As shown in Fig. 1, the response regressors are set to 1 from the beginning of the trial to the time when the subject is making a response. The punishment regressors are set to 1 from the reception of punishment until the beginning of the next trial. These five regressors are referred to as (1) selecting 20 (safe response), (2) selecting 40 (risky response), (3) selecting 80 (risky response), (4) punished with −40, and (5) punished with −80. These regressors were convolved with a modified gamma variate function modeling a prototypical hemodynamic response (Boynton et al., 1996) prior to inclusion in the regression model. In addition three regressors were used to account for residual motion (in the roll, pitch, and yaw direction). Regressors for baseline and linear trends were used to eliminate slow signal drifts. The AFNI program 3dDeconvolve was used to calculate the estimated voxelwise response amplitude. A Gaussian filter with FWHM 4 mm was applied to the voxelwise percent signal change data to account for individual variations of the anatomical landmarks. Data of each subject were normalized to Talairach coordinates.

Statistical analyses

The voxelwise percentage signal change data were entered into a mixed-model ANOVA with response type as a fixed factor and subjects as a random factor. First, to determine areas that significantly activated with risky versus safe responses, a within-subjects contrast was computed between the "40 and 80" −20 regressors. Second, to differentiate the risk-taking behavior from punishment related activation, a contrast was computed between the "40 and 80" and the "punished with −40 and punished with −80" regressors. A threshold adjustment method based on Monte-Carlo simulations was used to guard against identifying false-positive areas of activation (Forman et al., 1995). Based on these simulations, it was determined that a voxel wise a priori probability of 0.05 would result in a corrected clusterwise activation probability of 0.05 if a minimum volume of 500 μl and a connectivity radius of 4.0 mm was considered. Finally, the average percentage signal difference was extracted from regions of activation that were found to survive this threshold/cluster method.

All analyses for the behavioral data were carried out with SPSS 10.0 (Norusis, 1990). A mixed-model ANOVA (fixed factor: task conditions; random factor: subjects) was used to

Table 1
Contrast analysis "risky" − "safe" responses: volume-thresholded cluster center of mass coordinates

Volume (μl)	x	y	z	L/R	Area	BA
768	32	18	7	R	Insula	13
1088	−7	−74	32	L	Cuneus	7
576	−17	−70	40	L	Precuneus	7
512	10	−65	27	R	Precuneus	7
512	37	7	33	R	Middle frontal gyrus	9

analyze the behavioral measures. The planned comparisons were evaluated using the least significant difference (LSD) post hoc analysis.

Results

Behavioral results

Subjects selected the "safe" 20 response 46% ± 4 of the time, the "risky" 40 response 26% ± 5 of the time, the "risky" 80 response 23% ± 5 of the time [$F(2, 32) = 4.75$, $P < 0.05$] and failed to respond 4% of the time. Therefore, subjects selected "safe" and "risky" responses with similar frequencies. As shown in Fig. 2, there was a significant interaction between prior punishment and response type [$F(2, 32) = 9.0$, $P < 0.01$] wherein subjects made fewer 40 and 80 (risky) responses after punishment.

Neuroimaging results

As shown in Table 1, based on the first contrast analysis five areas showed a significant differential activation between "safe" and "risky" responses. These areas included posterior parietal cortex (BA 7), dorsolateral prefrontal cortex (BA 9), and the insula (BA 13). As shown in Fig. 3, activation in the right anterior insula was significantly higher during the "risky" than during a "safe" responses [$F(2, 32) = 7.7$, $P < 0.01$] but also showed significant activation during punished trials [punished −40 regressor $t(16) = 2.61$, $P < 0.05$; punished −80 regressor $t(16) = 3.88$, $P < 0.01$]. Moreover, the larger the activation to a "risky" response in the right anterior insula, the more likely the subject selected the "safe" response after punishment ($r = 0.62$, $P < 0.05$).

Right inferior frontal gyrus (BA 44) and insula (BA 13), left insula (BA 13), and left superior parietal lobule (BA 7) were significantly more active when comparing risky trials (40 and 80 regressor) versus those trials that were punished (punished with −40 and punished with −80 regressor; Fig. 4, Table 2). Moreover, the larger the response in the right ($r = 0.54$, $P < 0.05$) and left ($r = 0.50$, $P < 0.05$) insula during the "risky" trials, the more frequently subjects responded with a safe response following a punished trial.

Relationship between insula and temperament

Harm Avoidance, Reward Dependence, Persistence, Self-Directiveness, Cooperativeness, and Self-Transcendence were entered separately into two stepwise regression analyses to predict the degree of insula activation during "risky" but nonpunished responses and during punished responses. Although these variables did not predict the degree of insula activation during a "risky" response ($r = 0.36$, $P = 0.1$), harm avoidance ($r = .54$) significantly predicted the degree of insula response during a punished response [$F(1, 14) = 5.30$, $p < 0.05$, $r^2 = 0.29$]. Similarly, neuroticism, extraversion, openness, agreeableness, and conscientiousness were entered into two separate stepwise regression analyses to predict the degree of insula activation during "risky" responses and during punished responses. The NEO neuroticism variable was not able to predict the insula activation during the selection of a "risky" response ($r = 0.18$, *ns*) but neuroticism (partial $r = 0.72$) and conscientiousness (partial $r = -0.60$) were able to predict the degree of insula activation during a punished response [$F(1, 14) = 7.71$, $p < 0.05$, $r^2 = 0.58$]. Moreover, the degree of neuroticism correlated significantly with the degree of right anterior insula response to punishment [$r = 0.59$, $P < 0.05$]. Finally, the degree of neuroticism and harm avoidance was highly correlated among subjects [$r = 0.59$, $P < 0.05$], which shows that these factors probe for similar personality domains. As shown in Fig. 5, a larger degree of insula activation during punishment was associated with higher levels of harm avoidance and neuroticism.

Discussion

This investigation yielded three main findings. First, right anterior insula (BA 13) activation was significantly larger when subjects selected a "risky" response versus selecting a "safe" response but also showed significant activation during punishment. Moreover, bilateral insula and left superior parietal lobule activation was larger during nonpunished "risky" responses than during punished trials. Second, in both cases the degree of insula activation was related to the probability of selecting a "safe" response following punishment. Third, the degree of right anterior insula activation was correlated positively with the subjects' degree of harm avoidance and neuroticism. In combination,

Table 2
Contrast analysis "risky" responses − "punished" trials: volume-thresholded cluster center of mass coordinates

Volume (μl)	x	y	z	L/R	Area	BA
1152	48	4	18	R	Inferior frontal gyrus/insula	44/13
640	−27	−47	55	L	Superior parietal lobule	7
512	−38	−21	15	L	Insula	13

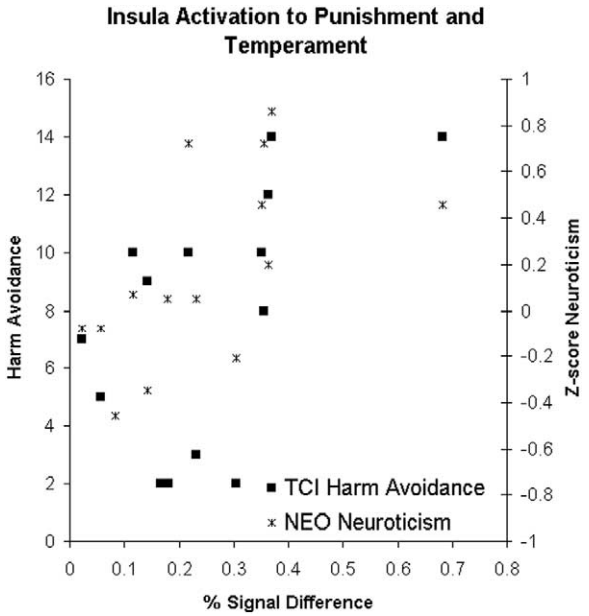


Fig. 5. Scatter plot of right insula response during punishment and neuroticism or harm avoidance.

these results support the hypothesis that the insula plays a critical role in the processing of risk during decision making and that the magnitude of the insula response is inversely related to personality measures of risk-taking behavior.

Several definitions of risk taking have been proposed in the context of experimental studies (Luce, 1967). Some investigators have suggested that risk-taking is involved whenever a decision is associated with uncertainty. Others have emphasized that risk implies a possible loss. In the risky gains decision-making task, risk-taking behavior is defined on a trial-by-trial basis as the selection of the high yield response (i.e., selecting 40 or 80) that is associated with a potential loss (−40 or −80) versus the selection of the low-yield response (20) that is never associated with a loss. In the current version of this task, subjects were informed that selecting a 40 or 80 response may be associated with a potential loss but were not informed that the expected value of each action alternative was set such that each pure strategy, i.e., selecting the safe response every time, or selecting one of the "risky" responses every time, would result in the same total payoff. Thus, unbeknownst to the subjects there was no inherent advantage of selecting a "risky" response over a "safe" response. Although subjects did not know the expected value of each response alternative, they selected the "safe" or "risky" responses with approximately equal frequency. The behavior of matching the frequency of selecting the "risky" or "safe" response to the expected values of these responses is consistent with general reward matching (Glimcher, 2002). Moreover, subjects selected fewer "risky" responses after punishment, which supports the aversive salience of the punishment. In combination, the behavioral results are consistent with the notion that subjects match the action selection to the expected value of the action alternatives and adjust briefly after punishment to select the "safe" response more frequently.

The right anterior insula showed risk-related activation. Risk-taking trials in this paradigm, however, were confounded with an expectation of higher reward (40 or 80 versus 20) and the expectation of punishment (−40 or −80). The contrast analysis between nonpunished "risky" responses and punished trials may help to clarify whether the insula response is related to expectation of reward or punishment. An area in the right insula, which partially overlapped with the area that showed significant differences in activation between safe and risky trials, and left insula as well as left superior parietal lobule showed activation that was significantly higher during nonpunished "risky" responses than during punished trials. Moreover, the degree of right insula activation identified in this contrast analysis was also correlated positively to the frequency of selecting a safe response following a punished trial. These findings support the hypothesis that, at least in part, areas in the right insula are related to expectation of punishment and not to punishment per se. Other regions in the anterior insula also showed activation during punished trials, which would support the hypothesis that the insula as a whole process both expectation and experience of punishment. In combination, insular cortex may be critical for the generation of anticipatory aversive somatic markers and for aversive outcome processing once a decision has been made.

Insula activation occurs in a wide variety of task conditions. There is, however, an emerging consensus that insula activation is frequently associated with aversive states. Specifically, functional neuroimaging studies have shown insula related activation during the processing of fearful (Morris et al., 1998) or disgusted (Phillips et al., 1998) faces, during the anticipation of electric shocks (Chua et al., 1999), as well as during script-induced sad mood induction (Liotti et al., 2000). In fact, insula activation appears to be critical for the interface between cognitive and affective processing. For example, both aversive pavlovian conditioning (Buchel et al., 1998) and aversive trace conditioning (Buchel et al., 1999) were associated with increased insula activation. Moreover, insula activity was modulated by perceptual awareness of threat (Critchley et al., 2002), penalty (Elliott et al., 2000b), or error-related processes (Menon et al., 2001). Finally, support for the role of the insula in instantiating aversive somatic markers are based on findings linking insula activation to the degree of noxious heat (Becerra et al., 1999) or cold (Tracey et al., 2000; Davis, 2000) stimuli, which is consistent with pain intensity coding activation in the insula (Ploghaus et al., 2001).

Increased insula activation during risk-taking decision making is consistent with the function of the insula in linking cognitive and affective components (Sawamoto et al., 2000). Anatomical studies in rhesus monkeys have shown that the insula receives input from both dorsolateral prefrontal and posterior parietal cortex (Selemon and Gold-

man-Rakic, 1988). In particular, the rostral part of the posterior parietal lobe sends efferents to the insular cortex (Cavada and Goldman-Rakic, 1989). Moreover, the insula receives projections from the amygdala (McDonald et al., 1999). Therefore, it is not surprising that activations in the insula during "risky" versus "safe" responses were also associated with activations in the posterior parietal and the dorsolateral prefrontal cortex.

The computational processes underlying decision making appear to be distributed across various cortical and subcortical areas. Previous investigations have revealed a success-/failure-dependent activation pattern in the prefrontal (Elliott et al., 1999, 2000b), anterior cingulate (Elliott and Dolan, 1998), insula (Critchley et al., 2001), and posterior parietal cortex (Paulus et al., 2001). The current study shows that insula activation is related to the selection of a "risky" over a "safe" response and that the degree of insula activation is related to a trait variable of harm avoidance, a measure of "risk-aversion."

Harm avoidance has been described as a heritable tendency to learn to avoid punishment (Cloninger, 1987) and is thought to be related to central nervous system serotonergic turnover (Peirson et al., 1999; Gerra et al., 2000; Moresco et al., 2002). Several groups of patients with psychiatric disorders show altered levels of harm avoidance. For example, patients with anxiety and mood disorders scored higher than normal comparison subjects on harm avoidance (Kusunoki et al., 2000; Blairy et al., 2000; Starcevic et al., 1996). In comparison, subjects with substance use disorders scored lower on harm avoidance than normal comparison populations (Swendsen et al., 2002). Similarly, neuroticism (Costa and McCrae, 1992) refers to the general bias to respond more sensitively to external negative stimuli. Increased levels of neuroticism have been reported in depressed (Kendler et al., 1993), bipolar disorder (Solomon et al., 1996), and anxious (Kendler et al., 2002) subjects. The current investigation links harm avoidance and neuroticism to the degree of insula functioning in a risk-taking decision-making situation. Specifically, subjects with a relatively larger activation in the right anterior insula during punished trials were also more likely to score high on harm avoidance and neuroticism. In a previous study, Canli (Canli et al., 2001) found both positive and negative correlations between neuroticism and activation differences viewing negative relative to positive pictures in left frontal or temporal and right frontal gyrus, respectively. These findings provide emerging evidence that the brain response to affective processing is significantly modulated by trait personality variables (Canli and Amin, 2002). In combination, the degree to which the insula processes punishment appears to be directly related to the propensity to experience aversive situations and exhibit risk avoidant behavioral traits.

Several intriguing questions emerge from this investigation. First, is the insula reactivity during risk-taking decision-making paradigm a trait or endophenotypic marker, which could be used to identify "high" risk-takers or "high" avoiders? Second, do subjects who engage in risky behaviors such as continuous drug taking or gambling exhibit low insula reactivity? Third, can pharmacological manipulations affect the insula reactivity, and, if so, how would these influence risk-taking behavior?

In conclusion, this study showed that activation in the right anterior insula is associated with selecting a "risky" over a "safe" response and that the amplitude of the insula response during punishment is associated with the degree of harm avoidance and neuroticism as measured by the TCI and NEO, respectively. These results are consistent with the hypothesis that the insula activation serves as a critical neural substrate to instantiate aversive somatic markers that guide risk-taking decision-making behavior.

Acknowledgments

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