Neurocognitive mechanisms underlying identification of environmental risks

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\textbf{Abstract}

Environmental risks threaten a large population and are more dreadful than personal risks that bring physical or health problems to individuals. To assess the neurocognitive processes involved in environmental risk identification, we recorded brain activities, using event-related potential (ERP) and functional magnetic resonance imaging (fMRI), from human adults while they identified risky and safe environmental and personal events depicted in words. We found that, relative to safe environmental events, the identification of risky environmental events induced larger amplitudes of an early positive ERP component at 180–260 ms over the frontal area (P200) and of a late positive wave at 420–660 ms over the central–parietal area (LPP). fMRI results showed that the identification of environmental risks was associated with increased activations in the ventral anterior cingulate cortex (vACC) and posterior cingulate cortex (PCC). The amplitudes of the LPP/P200 and the PCC activity positively correlated with subjective ratings of risk degree of and emotional responses to the risky environmental events. However, the identification of personal risks induced positive shift of ERPs at 280–320 ms over the frontal and parietal areas and increased activity in the left inferior and medial prefrontal cortex. Our findings suggest that identification of dreadful environmental risks is subserved by an early detection in vACC and a late retrieval of conscious experience in PCC.

\section{1. Introduction}

Natural disasters such as floods and earthquakes may induce serious damages to a large population and constitute severe risks to the public. After entering the 20th century, human beings are also confronted with potential artificial disasters such as nuclear explosion and chemical pollution that can damage the environment and lead to catastrophic consequences to human society. These environmental risks have increasingly dominated individual and collective consciousness (Denney, 2005; Laudan, 1994) since perception of these environmental risks is crucial for making decisions on both individual behaviors and public policies.

Psychometric studies showed that risk perceptions are highly domain specific (Blais & Weber, 2001; Weber, Blais, & Betz, 2002). For example, risks related to an individual can be decomposed into subcategories such as those related to personal health/safety and social decisions (Weber et al., 2002). Our recent functional magnetic resonance imaging (fMRI) study showed that distinct neural substrates engage in identifications of personal risks that arise from interpersonal interactions in social contexts (social risks) and that come from situations that may give rise to physical discomfort (physical risks) (Qin & Han, in press). Specifically, the identification of social risks induced increased activities in the medial prefrontal cortex (MPFC), the dorsal anterior cingulate cortex (dACC), and bilateral posterior insula, whereas the identification of physical risks resulted in activations in the MPFC, the ventral anterior cingulate cortex (vACC), the right cuneus/precuneus and bilateral amygdala. The fMRI findings suggest that identifications of risks in the social and physical domains are different in both cognitive processes and emotional responses.

Researchers also categorized risks into environmental and individual personal domains (Gattig & Hendrickx, 2007; Schütz, Wiedemann, & Gray, 2000). The environmental risks arise from the natural processes and the use of technology, lack direct control by individuals (Schütz et al., 2000), and may generate catastrophic consequences relevant to the survival of a large population (Böhm & Pfister, 2000). In contrast, personal risks result from individual activities (e.g., smoking, drinking, or car driving) that influence individual health and safety (Schütz et al., 2000). It has been shown that humans may discount the ponderance of the same personal risks that may happen in the far than near future (Chapman, 1996; Chapman & Elstein, 1995), whereas evaluation of the severity of...
Most of contemporary research on risk perception/evaluation emphasizes both probability and consequences of risks during decision making (Kahneman & Tversky, 1979; Sanfey, Loewenstein, McClure, & Cohen, 2006). Neuroimaging studies have shown evidence that the processing of probability and negative outcome are associated with the prefrontal cortex (ventral and medial prefrontal cortex:  
Longe, Elliott, & Deakin, 2001; ventral and dorsal prefrontal cortex:  
Casey et al., 2001; dorsal lateral prefrontal cortex:  
Huetter, Song, & McCarthy, 2005) and the ACC (Gehring & Willoughby, 2002; Yeung & Sanfey, 2004), respectively. However, memory of emotional experience and other factors may influence the way people evaluate risks in everyday life so that the probability of risky events may be ignored (Bottrell & Mazur, 2004; Loewenstein, Weber, Hsee, & Welch, 2001; Sunstein, 2003). In this case, the evaluation of potential consequences or consequences that have already taken place may become extremely important for risk perception. The psychometric approach on risk perception showed that subjective rating of risks correlated with the severity and dreadfulness of hazards that reflect the consequences associated with risks (Slovic, 1987). These findings suggest that feelings of dread play an important role in risk perception (Fischhoff, Slovic, Lichtenstein, Read, & Combs, 1978; Slovic, 1987) and risk perception may be associated with emotional reactions (Loewenstein et al., 2001; Slovic, Finucane, Peters, & MacGregor, 2004).

Previous studies suggested that strong feelings of dread are induced by the risks that lack control by individuals and may induce severe consequences (Slovic, 1987). Environmental risks are out of control in most cases (Schütz et al., 2000) and may produce catastrophic consequences to the survival of a large population (Böhm & Pfister, 2000). In these senses, environmental risks are more dreadful than personal risks (Slovic, 1987). This is consistent with the stress-related theory of risk perception, which claims that perception of high risk or anticipation of serious negative consequences may elicit intense emotions such as dread or fear (Stallen & Tomas, 1985). Moreover, Böhm (2003) suggested that prospective consequence-based feelings such as dread and fear are the most intense emotion associated with the consequence-based evaluation of environmental risks. Based on these studies, we hypothesized that, relative to the process of personal risks, the identification of environmental risks may enhance neural activities in brain regions such as vACC and PCC since environmental/personal event was initially rated by an independent group of 11 subjects, and the same stimuli and stimulus duration were used in both the ERP and fMRI experiments.


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with the addition of two mastoid electrodes. The electrode at the right mastoid was mounted on an elastic cap according to the extended 10-20 system, the risk degree ("How risky is this event?" 0 = safe, 6 = extremely risky).

2.3. ERP experiment

2.3.1. Procedure

Each participant participated in eight blocks of trials, in which the stimuli and tasks varied. In each two blocks of trials, subjects either (1) were presented with words/phrases depicting environmental events (half safe and half risky) and were asked to judge risky vs. safe environmental events (environmental risk identification task); (2) were presented with half words/phrases depicting environmental events and half pseudo words/phrases, and were asked to judge real vs. pseudo words/phrases (semantic control task); (3) were presented with words/phrases depicting personal events (half safe and half risky) and were asked to judge risky vs. safe personal events (personal risk identification task); or (4) were presented with half words/phrases depicting personal events and half pseudo words/phrases, and were asked to judge real vs. pseudo words/phrases (semantic control task). Subject pressed one of the two buttons to indicate risky/safe in the risk identification task or real/pseudo words/phrases in the control task using the index or middle finger. The responding hand corresponding to ‘yes’ and ‘no’ responses was counterbalanced across subjects. Each block of trials began with the presentation of instructions for 2000 ms, which defined the task (i.e., risk identification or semantic control tasks) for each of which consisted of eight sessions. Each session began with a block of 80 trials in each block. Each trial a word/phrase was presented for 1500 ms at the center of the screen, which was followed by a fixation cross with a duration varying randomly between 800 and 1200 ms. The stimuli in each block of trials were presented in a random order and the order of risk identification or semantic control tasks was counterbalanced using the Latin-square design for each subject. After the EEG recording session, subjects were asked to evaluate each stimulus item using a seven-point Likert scale on the emotional impact ("How strong is your emotional response to this event?" 0 = no, 6 = extremely high) and on the risk degree ("How risky is this event?" 0 = safe, 6 = extremely risky).

2.3.2. Data recording

The electroencephalogram (EEG) was continuously recorded from 60 scalp electrodes that were mounted on an elastic cap according to the extended 10-20 system, with the addition of two mastoid electrodes. The electrode at the right mastoid was used as reference. Eye blinks and vertical eye movement were monitored with electrodes located above and below the left eye. The horizontal electro-oculogram was recorded from electrodes placed 1.5 cm lateral to the left and right external canthi. The electrode impedance was kept less than 5 kΩ. The EEG was amplified (band pass 0.01–100 Hz) and digitized at a sampling rate of 250 Hz.

2.3.3. Data analysis

Both behavioral performance and ERPs data analysis focused on the responses to risky and safe stimuli presented in the environmental and personal risk identification tasks. Reaction times (RTs) were calculated as the time interval from stimulus onset to the subject’s response. Percentages of correct responses were 301.70 vs. 300.98, 0.44 vs. 0.38, 0.26 vs. 0.24, and 0.01–100 Hz) and digitized at a sampling rate of 250 Hz. The baseline for ERP measurements was the mean voltage of a 200 ms pre-stimulus interval and the latency was measured relative to the stimulus onset. Mean voltage of ERPs were obtained (a) at 20 ms intervals starting at 100 ms after stimulus onset and continuing until 340 ms post-stimulus and (b) at 40 ms intervals from 340 to 1060 ms post-stimulus. Statistical analysis were conducted on the mean voltage of ERPs at electrodes or each pairs of electrodes selected from the anterior frontal (AF1–AF4, AF7–AF8), frontal (Fz, F3–F4), frontal–central (FC2, FC3–FC4), central (Cz, C3–C4), central–parietal (CP2, CP3–CP4), parietal (Pz, P3–P4), temporal (T7–T8, TP7–TP8, P7–P8), parieto-occipital (P02, OZ, PO3–PO4) regions. The mean ERP amplitudes for environmental and personal events were subjected to ANOVAs with the factors being Valence and Hemisphere (electrodes over the left vs. right hemisphere) as within-subject independent variables. To confirm the possible different neural activities associated with identification of environmental and personal risks, we conducted ANOVAs with Risk (environmental vs. personal risks) and Valence (risky vs. safe) as independent variables for the mean ERP amplitudes.

Intracranial sources of ERP components related to environmental events were estimated by calculating current source density at each sampling point between 180 and 260 ms and between 460 and 600 ms. Analyses were conducted for grand average ERPs to risky environmental items, using low resolution electromagnetic tomography (LORETA) method embedded in Curry V5.0. A realistic volume conductor model, which was derived using a boundary element method with three layers [skin (10 mm), skull (9 mm), and brain (7 mm)], with conductivities of 0.3300, 0.0042, and 0.3300, respectively, was used in the LORETA analysis. To test for functional roles of the ERPs associated with identification of risky environmental events, correlation analysis was conducted between the rating scores of risky environmental events and the mean amplitudes of ERPs.

2.4. fMRI experiment

2.4.1. Procedure

A mixed design was used in the fMRI study. Four functional scans were obtained, each of which consisted of eight sessions. Each session began with the presentation of an instruction for 2000 ms, which defined the task (i.e., risk identification or semantic control tasks, which is similar to the ERP study). Participants pressed one of the two buttons to indicate risky/safe events in the risk identification task or real/pseudo words/phrases in the semantic control task using the index or middle finger. The responding hand was also counterbalanced between subjects. There were 10 trials in each session consisting of 5 risky and 5 safe events or 5 real and 5 pseudo words/phrases. Each item was presented for 1500 ms followed by an inter-stimulus interval that varied randomly among 500, 1000, 1500, 2000 and 2500 ms. Two adjacent sessions were intervened with a fixation of 8000 ms. The order of risk identification and semantic control tasks was counterbalanced using the Latin-square design. After the scanning procedure, subjects were asked to evaluate each stimulus item using a seven-point Likert scale on the emotional impact ("How strong is your emotional response to this event?" 0 = no, 6 = extremely high) and on the risk degree ("How risky is this event?" 0 = safe, 6 = extremely risky). The electroencephalogram (EEG) was continuously recorded from 60 scalp electrodes or each pairs of electrodes selected from the anterior frontal (AF3–AF4, AF7–AF8), frontal (Fz, F3–F4), frontal–central (FCz, FC3–FC4), central (Cz, C3–C4), central–parietal (CPz, CP3–CP4), parietal (Pz, P3–P4), temporal (T7–T8, TP7–TP8, P7–P8), parieto-occipital (PO2, O2, PO3–PO4) regions. The mean ERP amplitudes for environmental and personal events were subjected to ANOVAs with the factors being Risk (environmental vs. personal) and Valence (risky vs. safe) as within-subject independent variables. Two-tailed paired t-tests were conducted to compare the emotion and risk rating scores of the environmental and personal events.

SPM2 (Wellcome Department of Cognitive Neurology, London, UK) was used for imaging data processing and analysis. The time series for the voxels within each slice were realigned temporally to the acquisition of the middle slice. The functional images were realigned to the first scan to correct for the head movement between scans and the anatomical image was co-registered with the mean functional image produced during the process of realignment. All images were normalized to a 2 mm × 2 mm × 2 mm Montreal Neurological Institute (MNI) template in Talairach space (Talairach & Tournoux, 1998) using bilinear interpolation. Functional images were spatially smoothed using a Gaussian filter with a full-width-at-half maximum (FWHM) parameter set to 8 mm. A general linear model (GLM, y = f(x,e), where the response y is equal to a linear sum of weighted variables (fx) plus an error or residual value (e)) was used to construct the multiple time series regression design matrix. The image data were modeled using a canonical hemodynamic response function (HRF). The time derivatives and head motion parameters were included for accounting for extra variance in case the onsets are off by a little and capturing residual movement related artifacts (the three rigid-body translations and rotations determined from the realignment stage), respectively. All data were globally normalized with proportional scaling of the image means. High-pass filtering was used with a cutoff of 128 s. Effects at each voxel were estimated and regionally specific effects were compared using linear contrasts in individual participants using a fixed effect analysis. Items rated as risky were contrasted with those rated as safe to identify the regions activated by risky items during the environmental and personal risk identification tasks. The number of risky and safe trials was matched for each two blocks of trials, subjects either in each event-related analysis. The resulting set of voxel values for each contrast constituted a statistical parametric map of the r-stastic (SPM(t)) which was subsequently transformed to the unit normal distribution (SPM(Z)). Statistical inferences were based on the theory of random Gaussian fields. Random effect analyses were then conducted based on statistical parameter maps

1 Both the ERP and fMRI data recorded in the semantic control task will be reported in another paper.
To exclude the effect of task and search for the specific activations linked to environmental and personal risks, we conducted the exclusive masking analysis that is used in the recent study to assess domain dependency of dorsomedial prefrontal cortex (Walter et al., in press). The main contrast of risky vs. safe environmental items was exclusively masked by the contrast of environmental vs. personal items and the main contrast of risky vs. safe personal items was exclusively masked by the contrast of personal vs. environmental items. All exclusive masking analyses used an uncorrected p-value of p < 0.05 for their masks.

To confirm the possible different neural activities associated with identification of environmental and personal risks, we calculated the percent signal change in the regions of interests (ROIs) defined as spheres with a 5 mm diameter centered at the peak voxel of specific activated brain areas identified in the contrast of risky vs. safe items in the random effect analysis, which was then subjected to ANOVAs with Risk (environmental vs. personal risks) and Valence (risky vs. safe) as independent variables. To test functional roles of the activations associated with identification of risky environmental events, correlation analysis was conducted between the rating scores of risky environmental events and the percent signal change of regions of interests (ROIs) which were spheres with a 5 mm diameter centered at the peak voxel of specific activated brain areas identified in the random effect analysis. The signal changes in the ROI were computed using MarsBaR 0.38 (http://marsbar.sourceforge.net).

3. Results

3.1. Behavioral performance

During the ERP recording procedure, subjects correctly identified 97.41 ± 1.93% (mean ± standard deviation) of the 40 risky environmental events, 93.66 ± 5.08% of the 40 safe environmental events, 88.21 ± 10.15% of the 40 risky personal events, and 97.86 ± 2.61% of the 40 safe personal events. ANOVAs of RTs showed a significant interaction of Risk × Valence (F(1, 13) = 18.24, p < 0.01, Fig. 1a), suggesting that the RTs were shorter to the risky than safe items in the environmental risk identification task (t(13) = 5.691, p < 0.001) but not in the personal risk identification task (t(13) = 1.432, p > 0.1). Paired t-test showed that the emotion rating scores of the stimuli obtained after the ERP recording procedure were significantly higher for risky environmental items than risky personal items (2.98 ± 0.94 vs. 2.42 ± 0.78, t(13) = 4.27, p < 0.001). However, there was no significant difference between the emotion rating scores of safe environmental and personal items (0.82 ± 0.74 vs. 0.77 ± 0.70, t(13) = 0.90, p > 0.05). Paired t-test also showed that the rating scores of risk degree were significantly higher for environmental than personal items (risks: 3.73 ± 0.39 vs. 2.87 ± 0.49, t(13) = 8.26, p < 0.001; safe events: 0.49 ± 0.32 vs. 0.30 ± 0.30, t(13) = 3.36, p < 0.01).

During the fMRI scanning procedure, subjects correctly identified 92.14 ± 7.26% of the 40 risky environmental events, 84.29 ± 10.58% of the 40 safe environmental events, 87.68 ± 12.80% of the 40 risky personal events, and 88.57 ± 6.77% of the 40 safe personal events. ANOVA analysis of RTs showed a significant main effect of Risk (F(1, 13) = 17.38, p < 0.001), indicating that RTs were shorter to the environmental than personal risk identification task. There was also a reliable interaction of Risk × Valence (F(1, 13) = 11.79, p < 0.01, Fig. 1b), suggesting that RTs were shorter to the risky than safe items in the environmental risk identification task (t(13) = 2.688, p < 0.05) but not in the personal risk identification task (t(13) = 1.817, p > 0.05). Consistent with the result of the ERP experiment, the emotion rating scores of stimuli obtained after the fMRI scanning procedure were significantly higher for risky environmental items compared with risky personal items (2.93 ± 0.94 vs. 2.53 ± 0.91, t(13) = 5.14, p < 0.001) whereas there was no significant difference in emotion rating scores between safe environmental and personal items (1.06 ± 0.64 vs. 0.91 ± 0.72, t(13) = 1.92, p > 0.05). Paired t-test also confirmed that the rating scores of risk degree were significantly higher for environmental than personal items (risks: 3.55 ± 0.53 vs. 2.78 ± 0.61, t(13) = 12.05, p < 0.001; safe events: 0.60 ± 0.45 vs. 0.44 ± 0.47, t(13) = 6.02, p < 0.001).

3.2. ERP results

To inspect the time course of the neural and cognitive processes involved in identification of environmental risks, we analyze the mean ERP amplitudes differentiating between risky and safe items using ANOVAs with Valence (risky vs. safe) and Hemisphere (electrodes over the left or right hemisphere) as within-subject independent variables. We found a significant main effect of Valence at 180–260 ms over the frontal and central electrodes (AF3–AF4: F(1, 13) = 5.46, p < 0.05; F3–F4: F(1, 13) = 13.39, p < 0.01; FC3–FC4: F(1, 13) = 15.49, p < 0.01, Fig. 2a). Relative to the safe environmental items, identification of risky environmental items elicited enlarged P200 amplitudes. In addition, the LPP with maximum amplitudes over the central and parietal area was of larger amplitudes to the risky than safe environmental items at 460–560 ms (CP3–CP4: F(1, 13) = 12.73, p < 0.01; P3–P4: F(1, 13) = 8.48, p < 0.05, Fig. 2b). We also found a reliable interaction of Valence × Hemisphere at 420–460 ms at anterior frontal electrodes (AF7–AF8: F(1, 13) = 6.30, p < 0.05) at 580–700 ms over the frontal–central area (FC3–FC4: F(1, 13) = 5.85, p < 0.05), due to the fact that the long-latency anterior positive activity associated with risky environmental items was of larger amplitudes over the right than left hemispheres. This hemispheric asymmetry suggests that the risky items induced stronger process in the right hemisphere, consistent with previous observation of the right lat-
Fig. 2. ERP results in the environmental and personal risk identification tasks. (a) P200 associated with risky environmental events relative to safe ones and its representative current sources identified in the vACC and medial occipital cortex at 228 ms; (b) LPP associated with risky environmental events relative to safe ones and its representative current sources identified in the PPC and PCC at 560 ms; (c) ERPs recorded at CPz differentiated between risky and safe personal events at 280–320 ms after stimulus delivery; (d) correlation between the difference of LPP amplitudes between risky and safe environmental events and the corresponding subjective rating scores of emotional impact; (e) correlation between the P200 amplitudes evoked by risky environmental events and the corresponding subjective rating scores of risk degree; (f) correlation between the LPP amplitudes evoked by risky environmental events and the corresponding subjective rating scores of risk degree. The mean rating score and ERP amplitude of each subject are indicated by a single disk. The lines represent the linear best fit; r refers to the correlation coefficient. LPP: late positive potential; PPC: posterior parietal cortex; PCC: posterior cingulate cortex; vACC: ventral anterior cingulate cortex.

eralized processing of negative information (Anderson et al., 2003; Cunningham, Espinet, DeYoung, & Zelazo, 2005).

The current sources of the P200 and LPP were estimated using LORETA. We found that two current sources, one located at the vACC and one at the medial occipital cortex (Fig. 2a), were able to account for over 90% of the variance of the topography at the time window corresponding to the P200. At a later time window corresponding to the LPP, the LORETA analysis showed an additional current source at the posterior parietal cortex and the PCC (Fig. 2b).

To assess whether the ERP effects were specific to the identification of environmental risks, the ERPs to personal items were analyzed similarly. Relative to safe personal items, risky personal items elicited a positive shift of ERPs at 280–320 ms, resulting in significant main effects of Valence over frontal–central (F3–F4: \(F(1, 13) = 6.28, p < 0.05\); FC3–FC4: \(F(1, 13) = 6.76, p < 0.05\); C3–C4: \(F(1, 13) = 6.98, p < 0.05\), Fig. 2) and central–parietal electrodes (CP3–CP4: \(F(1, 13) = 6.67, p < 0.05\); P3–P4: \(F(1, 13) = 8.45, p < 0.05\), Fig. 2c). However, neither the P200 nor the LPP was modulated by stimulus valence of personal items \((p > 0.05)\). This was further confirmed by the significant interaction of Risk × Valence at 200–220 ms over frontal–central areas (F3–F4: \(F(1, 13) = 5.52, p = 0.05\); C3–C4: \(F(1, 13) = 6.74, p = 0.05\) and at 460–580 ms over central–parietal areas (CP3–CP4: \(F(1, 13) = 7.62, p < 0.05\); P3–P4: \(F(1, 13) = 5.37, p < 0.05\)).

To evaluate to what degree the ERP effects linked to identification of environmental risks could predict subjective ratings of risky events, we calculated the correlation between subjective ratings and the magnitudes of the ERP effect. We found marginally significant correlation between the emotional rating scores of risky environmental items and the differential ERP amplitudes to risky and safe environmental items recorded at the parietal electrodes at 540–580 ms \((P6: r = 0.530, p = 0.051; P4: r = 0.515, p = 0.06; PO6: r = 0.519, p = 0.057, Fig. 2d)\). In addition, the mean ERP amplitudes associated with the risky environmental items recorded at frontal–central electrodes at 200–240 ms positively correlated with the risk rating scores of risky environmental items \((FC5: r = 0.610, p < 0.05; FC3: r = 0.541, p < 0.05; FC2: r = 0.538, p < 0.05, Fig. 2e\). The mean ERP amplitudes linked to risky environmental items recorded at the parietal electrodes at 580–620 ms also positively correlated with the risk rating scores of risky environmental items.
Table 1

<table>
<thead>
<tr>
<th>Brain region</th>
<th>BA</th>
<th>X</th>
<th>Y</th>
<th>Z</th>
<th>Z-Value</th>
<th>Voxel no.</th>
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<tbody>
<tr>
<td>Environmental risky &gt; environmental safe</td>
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<tr>
<td>Posterior cingulate gyrus/precuneus</td>
<td>BA31/5/7</td>
<td>-4</td>
<td>-40</td>
<td>38</td>
<td>4.94</td>
<td>1889</td>
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<tr>
<td>Ventral anterior cingulate</td>
<td>BA10/32</td>
<td>-2</td>
<td>52</td>
<td>-2</td>
<td>3.44</td>
<td>176</td>
</tr>
<tr>
<td>Personal risky &gt; personal safe</td>
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<tr>
<td>Inferior frontal gyrus (L)/insula (L)</td>
<td>BA13/45</td>
<td>-40</td>
<td>24</td>
<td>8</td>
<td>3.43</td>
<td>166</td>
</tr>
<tr>
<td>Medial frontal gyrus (L)</td>
<td>BA5/10</td>
<td>-18</td>
<td>54</td>
<td>20</td>
<td>3.83</td>
<td>154</td>
</tr>
</tbody>
</table>

BA: Brodmann area; R: right hemisphere; L: left hemisphere; cluster survived under voxel-level uncorrected p-value of 0.0005, voxel number >50.

(P1: \(r = 0.615, p < 0.05\); PO3: \(r = 0.546, p < 0.05\); PZ: \(r = 0.545, p < 0.05\), Fig. 2f).

3.3. fMRI results

Our ERP results suggest that two neural structures, i.e., vACC and PCC, may be engaged in the identification of environmental risks. To further localize the neural substrates differentiating between risky and safe environmental events, we conducted a whole-brain statistical parametric mapping (SPM) analysis to contrast risky and safe items correctly identified by the subjects inside the scanner. Relative to safe environmental events, risky environmental events induced increased activations in the PCC and vACC (Table 1; Fig. 3a). The time courses (hemodynamic responses) within the PCC and vACC for risky and safe environmental items were computed and illustrated in Fig. 3b. Similar analysis of the fMRI data associated with risky and safe personal events showed increased activation in the left inferior frontal gyrus/insula and MPFC (Table 1; Fig. 3c).

![Fig. 3. fMRI results in the environmental and personal risk identification tasks. (a) Increased brain activations associated with risky environmental events relative to safe environmental events; (b) time courses (hemodynamic responses) were computed for each condition within PCC and vACC identified from the contrast of risky vs. safe environmental events, bars indicate standard error of the mean; (c) increased brain activations associated with risky personal events relative to safe personal events; (d) percent signal changes in the PCC differentiating identification of risky environmental (or personal) items relative to safe environmental (or personal) items, bars indicate standard error of the mean; (e) correlation between the percent signal changes observed within the PCC related to risky environmental events and the corresponding subjective rating scores of emotional impact; (f) correlation between the percent signal changes observed within the PCC related to risky environmental events and the corresponding subjective rating scores of risk degree. The mean rating score and fMRI signal change of each subject are indicated by a single disk. The lines represent the linear best fit; r refers to the correlation coefficient. PCC: posterior cingulate cortex; vACC: ventral anterior cingulate cortex; IFG: inferior frontal cortex; MPFC: medial prefrontal cortex.](image-url)
In addition, we exclusively masked the contrast of risky vs. safe environmental items with the contrast of environmental vs. personal items and found increased PCC/precuneus activation ($x = -4; y = -32; z = 52$, $Z = 4.50$, cluster size = 1018 voxel). However, masking the contrast of risky vs. safe personal items with the contrast of personal vs. environmental items failed to show any activation. Moreover, we conducted ROI analysis by calculating percent signal changes in the PCC and vACC (defined by the mean percent signal changes of two successive time points around the peak of the BOLD signals extracted from the PCC and vACC clusters). We found that a marginally significant interaction of Risk × Valence for the PCC activity ($F(1, 13) = 4.303$, $p = 0.058$, Fig. 3d) and the vACC activity ($F(1, 13) = 3.302$, $p = 0.092$).

Similarly, we calculated the correlation between subjective rating scores of the risky environmental events and the magnitude of the neural activities associated with identification of risky environmental events (defined by the mean percent signal changes of two successive time points around the peak of the BOLD signals extracted from the PCC and vACC clusters). Interestingly, we found that the difference in PCC activity between risky and safe environmental events positively correlated with the emotional rating scores of the risky environmental events ($r = 0.665$, $p < 0.05$, Fig. 3e). In addition, the PCC activity elicited by risky environmental events positively correlated with the risk rating scores of the risky environmental events ($r = 0.601$, $p < 0.05$, Fig. 3f).

4. Discussion

The current work combined ERP and fMRI to assess neurocognitive mechanisms underlying the identification of environmental and personal risks depicted in words or phrases. Our ERP results first showed evidence that the identification of environmental risks induced modulation of the neural activities at two successive time windows. Both the early P200 with maximum amplitudes over the frontal–central cortex and the following LPP with a central–parietal scalp distribution were enlarged by the identification of risky relative to safe environmental events. In addition, the P200 and LPP amplitudes positively correlated with subjective ratings of risky environmental events. Consistent with the ERP results, our FMRI results showed evidence that two distinct brain areas subserved the identification of environmental risks because risky environmental events generated increased brain activations in the vACC and PCC relative to safe environmental events. In addition, the PCC activity elicited by risky environmental events was positively correlated with subjective ratings of risky environmental events. Interestingly, all the neural activities associated with the identification of environmental risks were not observed in the identification of personal risks.

The P200 modulation by environmental risk identification reflected an initial neural differentiation between risky and safe environmental events. The P200 was localized to the vACC in the current source analysis, which was reinforced by our FMRI results. Previous ERP studies have shown that the P200 was associated with detection of threats such as fearful faces (Correll, Urland, & Ito, 2006; Thomas, Johnstone, & Gonçalvez, 2007). FMRI research also found that vACC was associated with the processing of emotional distractors and involved in resolving emotionally laden conflict (Vuilleumier, Armony, Driver, & Dolan, 2001; Whalen et al., 1998) and was activated by the presence of infrequent threat-related distractors such as fearful faces (Bishop, Duncan, Brett, & Lawrence, 2004). These findings suggest that vACC plays a key role in monitoring emotion-related conflict between the functional state of the organism and any new information that has potential affective or motivational consequences (Dalgleish, 2004). Although the environmental risks were depicted in words or phrases in our study, the detection of threat generated by risky environmental events may occur as early as the detection of threat shown in images. We suggest that the P200 and vACC activation observed in our study subserve an early detection of environmental risks by monitoring of the conflict between affective consequences of risky environmental events and the survival of a population. In addition, because vACC activity is involved in the processing of all types of emotional stimuli (Bush, Luu, & Posner, 2000; Phan, Wager, Taylor, & Liberzon, 2002) and can be enhanced when anticipating painful stimuli (Ploghaus, Becerra, Borras, & Borsook, 2003) or aversive pictures (Nitschke, Sarinopoulos, Mackiewicz, Schaefer, & Davidson, 2006), it may be proposed that early detection of environmental risks is accompanied with emotional response. These neural activities, to a certain degree, contributed to the subjective ratings of environmental risk degree because the P200 amplitude correlated with the risk rating scores of risky environmental events.

In a latter time window, we found that identification of risky environmental events invoked enlarged centro-parietal LPP. The current source analysis indicates that the LPP originated from the PCC. The PCC activation was further supported by the FMRI results. Previous research has shown evidence that the LPP is an index of evaluation process of emotional stimuli (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Schupp et al., 2000; Schupp, Junghöfer, Weike, & Hamm, 2003, 2004). It has been suggested that increased PCC activity mediates the evaluation of emotional stimuli and reflects the interaction between memory retrieval and emotion (Maddock, 1999; Maddock, Garrett, & Buonocore, 2003). Comparing with safe environmental events, identification of risky environmental events may induce enhanced retrieval of previous self-related emotional experience. The correlation analysis indicates that the LPP and PCC activity also contributed to subjective ratings of risky environmental events. The more emotional experiences are retrieved, the greater the risk degree and the emotional impact would be given to the risky environmental event. Taken these with the earlier activities, our ERP and FMRI results consistently support that the identification of environmental risks is underpinned by an early detection process and a late process of emotional experiences retrieval. There has been increasing evidence that the cortical midline structures such as PCC and vACC are involved in self-referential processing such as self-experience based memory retrieval (Fossati et al., 2003; Johnson et al., 2002) and self trait judgment (Han et al., 2008; Kelley et al., 2002; Zhu, Zhang, Fan, & Han, 2007; also see Northoff et al., 2006 for review). As most evaluative judgments are self-referential (Zysset, Huber, Ferstl, & von Cramon, 2002), it is not surprising that the neural activations observed in our current work overlapped with the cortical midline structures that were demonstrated to engage in self-referential processing.

In addition, our results indicate that the identification of dreadful environmental risks depicted in words is different from the detection of evolutionary prepared threats to individuals (e.g., angry fearful faces, snakes, or spiders), because threats to individuals usually induce activation in subcortical structure such as amygdala (Carlsson et al., 2004; Carretié, Hinojosa, Mercado, & Tapia, 2005; Morris et al., 1996; Pissiotta et al., 2003). This further suggests that cognitive processing of consequences associated with risky environmental events rather than salient emotional responses such as fear subserves identification of environmental risks depicted in words or phrases. A recent FMRI study investigated the neurobiological substrates of dread using delay-conditioning paradigm and found the subjective experience of dread comes from the attention devoted to the expected physical response (SI, SII, the caudal ACC, and the posterior insula) and not simply a fear or anxiety response (Berns et al., 2006). Similar to this, our results suggest that the identification of the dread depicted in words may require
cognitive processes such as detection and retrieval rather than pure emotional response. Are the neurocognitive processes of environmental risks different from the identification of signals that indicate negative utility? Utility is computed as the product of the value and probability of each potential outcome (Kahneman & Tversky, 1979; von Neumann & Morgenstern, 1947), and the neural mechanisms underlying the processing of utility has been studied extensively (Sanfey et al., 2006). Specifically, negative utility results in increases in ACC activity that correlates with the magnitude of anticipated consequences (Gehring & Willoughby, 2002; Yeung & Sanfey, 2004). The vACC activation associated with environmental risks observed in the current work suggests an important role of ACC in detection of negative utility in different domains such as environmental and financial. However, the identification of environmental risks is also characterized with increased PCC activity, which has not been observed in association with negative utility in the previous neuroeconomic studies. The PCC activity reveals the unique function of retrieval of previous emotional experiences in the process of environmental risks depicted in words, which may not be required for evaluation of instantaneous outcome when making economic decisions. Moreover, our results suggest that the probability of risky events might be neglected during the identification of environmental risks, because the neural activities associated with processing of probability, such as prefrontal cortex (Casey et al., 2001; Huettel et al., 2005; Longe et al., 2001), were not observed in our results.

Most importantly, our ERP and fMRI results failed to find evidence for modulations of the P200/LPP and vACC/PCC by stimulus valence of personal risks. The results of identification of personal risks rule out the possibility that ERP and fMRI results linked to identification of environmental risks arose from the specific task utilized in the current study. Moreover, the results indicate that the neural processes such as early detection and late emotional experiences retrieval may be specific to the identification of environmental risks, as indexed by the P200/vACC effects and the LPP/PCC effects. This could be due to that environmental risks can lead to more serious catastrophic consequences and stronger emotional reactions relative to personal risks. The enhanced PCC activation and LPP amplitudes may also reflect ethical considerations involved in environmental risk identification since more ethical concerns may be involved in identification of risky environmental than risky personal events (Böhm, 2003; Böhm & Pfister, 2000). This should be assessed in future work.

Together with our previous fMRI study (Qin & Han, in press), the current ERP and fMRI findings provide further evidence for domain specific neurocognitive processes in risk perception. Our previous fMRI study found distinct neural mechanisms underlying social and physical risk identifications and thus provided neural bases for the categorization of personal risks into social and physical domains (Qin & Han, in press). The findings of the current study indicate the existence of distinct neural and cognitive mechanisms underlying identification of risks in environmental and personal domains, providing neuroimaging evidence for the categorization of risks into environmental and personal risks (Gattig & Hendrickx, 2007; Schütz et al., 2000). Both our previous (Qin & Han, in press) and the current work found increased MPFC activation to risky than safe personal events, suggesting that the MPFC mediates intensive evaluation of stimulus valence in terms of the safety of human behaviors. However, the vACC and PCC activity was increased to risky than safe personal physical events in the previous work (Qin & Han, in press) but not the in the current study. A key difference between the two studies is that the personal physical risk identification task was intermixed with the identification of personal social risks assigned with lower rating scores in the previous work but with the identification of environmental risks assigned with higher rating scores in the current work. Apparently, the relative risk salience of personal physical events was lower in the current than previous studies although the risky and safe items were similar in the two studies. It appears the neural substrates underlying risk identification are not only domain specific but are modulated by the context in which the risks were identified as well.

In conclusion, our ERP and fMRI results provide consistent evidence that the identification of environmental risks consists of an early detection process mediated by vACC and a late process of retrieval of emotional experiences subserved by PCC. These neurocognitive processes are more salient for the identification of environmental risks in comparison with that of personal risks. These results indicate that the neural substrates of environmental risk identification are different from those of personal risk identification and possibly reflecting the consequences of evolution on human risk processing. It should be noted that ethnic cultural and socioeconomic background (Vaughan & Nordenstam, 1991) and personal variables such as profession (Barke, Jenkins-Smith, & Slovic, 1997; Slovic, 1987) affect risk perception. As our study only recruited college students, future research should investigate whether the neurocognitive processes identified in the current work could be modulated by individual knowledge of risks in specific fields.

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