

Neural Responses to Emotional Stimuli Are Associated with Childhood Family Stress

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Background: An early family environment marked by harsh parenting has been related to risk for multiple mental disorders in adulthood, risks that may be mediated, in part, by deficits in emotion regulation skills. This study examined neural mechanisms underlying these consequences of "risky" families (RF) by exploring neural activity to tasks involving responses to emotional stimuli.

Methods: Participants completed an assessment of RF and participated in a functional magnetic resonance imaging (fMRI) investigation that examined 1) amygdala reactivity to observation of fearful/angry faces; 2) amygdala and right ventrolateral prefrontal cortex (RVLPFC) reactivity to labeling emotions displayed in these faces; and 3) the relation between RVLPFC and amygdala activity during the labeling task.

Results: Offspring from nonrisky families showed expected amygdala reactivity to observing fearful/angry faces and expected activation of RVLPFC while labeling the emotions, which was significantly negatively correlated ($-.44$) with amygdala activation. Offspring from risky families showed little amygdala activation during the observation task and a strong positive correlation ($+.66$) between RVLPFC and amygdala activation in the labeling task, suggesting a possible dysregulation in the neural systems involved in responses to emotional stimuli.

Conclusions: Offspring from risky families exhibit atypical responses to emotional stimuli that are evident at the neural level.

Key Words: Stress, family, emotion, fmri, amygdala, right ventrolateral prefrontal cortex

Early family environments marked by harsh, conflict-ridden, or chaotic parenting are reliably associated with mental and physical health disorders in offspring across the life span (Repetti et al 2002; Lundberg 1993; Felitti et al 1998; Walker et al 1999). By exposing offspring to chronic or recurrent familial stress, these "risky" families (RF) contribute to the accumulation of factors predictive of poor health in adulthood. Specifically, a risky family environment has been tied to enhanced physiological stress reactivity in offspring, including elevated autonomic and cortisol responses to challenge (Taylor et al, unpublished data); stress-related states predictive of chronic illness, including compromised metabolic functioning (Lehman et al 2005) and elevated C-reactive protein (Taylor et al 2005); major physical health disorders (Felitti et al 1998); and major mental health disorders including depression (Felitti et al 1998). Understanding the mechanisms that link early family environments to these mental and physical health outcomes is, thus, an important research priority.

Research suggests that a risky family upbringing is associated with deficits in offspring emotion regulation skills (Repetti et al 2002). These deficits include difficulty in identifying and labeling emotions in self and others (e.g., Camras et al 1988; Dunn and Brown 1994), as well as difficulty in managing emotions in challenging circumstances (e.g., Brody and Flor 1998; Dishion 1990; see Repetti et al 2002 for a review). Research indicates that offspring from risky families may overreact to stressful circumstances, responding aggressively to moderately stressful circumstances (Reid and Crisafulli 1990; see Repetti et al 2002 for a review), but may also respond with efforts to tune out or avoid

stressful circumstances (O'Brien et al 1991; Valentiner et al 1994) through the use of coping strategies marked by behavioral escape or avoidance (Johnson and Pandina 1991). Difficulty regulating emotional responses to challenging or stressful circumstances has been tied to heightened biological stress responses including evidence of stronger hypothalamic-pituitary-adrenocortical (HPA) responses to stress (e.g., Flinn and England 1997; Chorpita and Barlow 1998). Intense, chronic, and/or reoccurring biological responses to stress may, thus, represent one pathway by which risky families exert adverse effects on mental and physical health outcomes (Repetti et al 2002; McEwen 1998).

Mechanisms linking a risky family upbringing to adult mental and physical health outcomes nonetheless remain only partly understood, and the neural mechanisms relating risky family upbringing to emotion regulation deficits remain largely unexplored. The present study sought to investigate these neural mechanisms by examining the relationship between a risky family environment and neural responses to emotional stimuli, as assessed by functional magnetic resonance imaging (fMRI). Because we hypothesized that stress reactivity and poor emotional regulation skills are central mechanisms that may help to explain the relation between risky family environment and mental and physical health outcomes later in life, we focused on brain regions implicated in threat detection and in regulatory processes related to responses to threatening stimuli.

Neural Correlates of Threat Detection and Emotion Regulation

One neural region consistently associated with threat detection is the amygdala. The amygdala has been shown to respond to a variety of stimuli indicating threat, including pictures depicting physical threats (Hariri et al 2002; Ochsner et al 2002) and fear and anger faces presented either supraliminally or subliminally (Hariri et al 2000; Whalen et al 1998). The amygdala is also sensitive to novel stimuli that possess potential threat value (Whalen 1999). Once activated, the amygdala sets in motion a cascade of responses to threat via projections to the hypothalamus and prefrontal cortex (Davis 1989; LeDoux 1987, 1996), acting to amplify or attenuate the threat signal and/or preparing to respond to the threat itself.

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A neural region that is critical for regulating these threat responses is the ventrolateral prefrontal cortex (VLPFC) (Hariri et al 2000; Lieberman et al 2005, Lieberman et al, unpublished data; Ochsner et al 2004). Previous studies have shown that the labeling of negative affective states activates the right VLPFC (RVLVPC) (Hariri et al 2000, 2002; Lieberman et al 2005, Lieberman et al, unpublished data) and that increased activity in the RVLVPC is associated with decreased activity in the amygdala (Hariri et al 2000, 2002; Lieberman et al 2005, Lieberman et al, unpublished data) as well as other affective neural regions (Eisenberger et al 2003; Lieberman et al 2004; Small et al 2001). This pattern of increased RVLVPC activity and decreased amygdala activity may be implicated in emotion regulation. That is, activity in RVLVPC is thought to be involved in verbalizing negative experiences, a process that typically occurs as people try to understand, cope with, or control their responses to those negative experiences.

As reviewed above, offspring from risky families have been found to experience problems in responding to threatening situations and in regulating responses to emotional stimuli. Thus, we predicted that offspring from risky families would reveal deficits in threat detection abilities and in the regulation of responses to potentially threatening stimuli, as evidenced in amygdala and RVLVPC responses to threat-relevant tasks.

Methods and Materials

Participants

Prospective participants responded to flyers placed around campus. In the initial screening telephone interview, following Institutional Review Board (IRB) regulations for nonclinical samples, the entire list of exclusion criteria was read to prospective participants; they were asked to indicate if any of the conditions was true of them but not to indicate which one. The list included having received a diagnosis of a serious physical or mental health problem; use of medications affecting cardiovascular, monoamine, or endocrine function; current treatment from a mental health professional; current pregnancy or lactation; and factors contraindicating fMRI participation (such as claustrophobia or metal in the body other than dental fillings). The final sample was 30 healthy right-handed people, aged 18 to 36 (12 men, 18 women). All were students or employees at the University of California, Los Angeles. All procedures were approved by the IRB, and all participants gave written informed consent.

Risky Families Assessment

The risky families questionnaire was adapted from an instrument originally developed by Felitti et al (1998) to assess the relation of family stress to mental and physical health outcomes in adulthood. In previous research, we validated this questionnaire against clinical interviews conducted and coded by trained clinical interviewers; the dual assessments (questionnaire and interview) demonstrated high agreement and reliability (Taylor et al 2004).¹

In the present study, participants rated aspects of their childhood family environment on 4-point scales ranging from 1 (rarely or none of the time) to 4 (most or all of the time), with items including whether the individual felt loved and cared for;

¹The clinical interviews revealed that exposure to family conflict, especially fighting between parents, was a common family stressor. This stressor did not appear in the original Felitti et al (1998) questionnaire and so items addressing this dimension of family life were added to the assessment.



Figure 1. Experimental condition types. The left panel is an example of the observe only condition in which participants observed the faces but made no response. The center panel is an example of the emotion label condition in which participants chose the correct emotion label to characterize the facial expression of the target. The right panel is an example of the gender label condition in which participants chose the correct name based on the gender of the target face.

was insulted, put down, sworn at, or made to feel threatened; was shown physical affection; was pushed, grabbed, shoved, or slapped; was verbally abused; was physically abused; observed quarreling or shouting between parents; observed violence or aggression between family members; lived with a substance abuser; lived in a well-organized, well-managed household; and whether family members knew what the child was up to. Because individual items differed in their variability, each item was z-scored before a composite measure was formed. Positively worded items were reverse-coded. Cronbach's alpha was .86. Average scores ranged from 1.08 to 3.54, with higher values representing a riskier family environment.

Experimental Paradigm

Two to 6 weeks after participants rated aspects of their family environment, they completed neuroimaging tasks designed to assess amygdala activity during the observation of negative faces and RVLVPC activity while labeling the emotional character of those faces²; the correlation between RVLVPC and amygdala activity during the labeling task was also assessed (Hariri et al 2000; Lieberman et al 2005, Lieberman et al, unpublished data). Specifically, in a block design, participants viewed target faces displaying negative emotional expressions and were asked to perform one of three tasks (Figure 1). In the observe only task, participants observed a single emotionally evocative face without making a response. During the emotion-labeling task, participants chose the correct emotion label ("fear," "anger") from a pair of words shown at the bottom of the screen. During the gender-labeling task, participants chose the gender-appropriate name from a pair of names shown at the bottom of the screen. This gender-labeling task is a comparison condition that controls for the general processing demands required for the emotion-labeling task. Emotion labeling, thus, differs from gender labeling solely in the affective nature of the verbal processing.

Each task block began with a 3-second instruction cue indicating the task type (observe only, emotion label, gender label) followed by 10 randomized trials of the specified task, each 5 seconds in length, resulting in task blocks that were 50 seconds in length. Blocks were separated from one another by a crosshair fixation, which remained on the screen for 10 seconds. Participants completed a total of two runs, each consisting of six blocks that included two observe only, two emotion label, and

²The fMRI stimuli consist of whole-face, full-color emotional expressions taken from the MacBrain Face Stimulus Set (Tottenham et al 2002) (development of the MacBrain Face Stimulus Set was overseen by Nim Tottenham and supported by the John D. and Catherine T. MacArthur Foundation Research Network on Early Experience and Brain Development. Please contact Nim Tottenham at tott0006@tc.umn.edu for more information).

two gender label blocks in a randomized order. Participants responded via button box and were told to respond as soon as they were sure of the correct answer. The stimuli remained on the screen for the entire 5-second trial.

Image Acquisition

Data were acquired on a Siemens Allegra 3T full-body scanner (Siemens, Erlangen, Germany). Head movements were restrained with foam padding and surgical tape placed across each participant's forehead. For each participant, a high-resolution, structural T2-weighted echo-planar imaging volume (spin-echo; repetition time [TR] = 5000 milliseconds; echo time [TE] = 33 milliseconds; matrix size 128 × 128; 36 axial slices; field of view [FOV] = 20 cm; 3 mm thick, skip 1 mm) was acquired coplanar with the functional scans. Two functional scans were acquired (echo-planar T2*-weighted gradient-echo; TR = 3000 milliseconds, TE = 25 milliseconds, flip angle = 90°, matrix size 64 × 64, 36 axial slices, FOV = 20 cm; 3 mm thick, skip 1 mm), each lasting 6 minutes and 15 seconds.

Data Analysis

The imaging data were analyzed using statistical parametric mapping (SPM99) (Wellcome Department of Cognitive Neurology, Institute of Neurology, London, United Kingdom). Images for each participant were realigned to correct for head motion, normalized into a standard stereotactic space as defined by the Montreal Neurological Institute (MNI), and smoothed with an 8 mm Gaussian kernel, full-width at half maximum. For each participant, observe only, emotion label, and gender label blocks were modeled as epochs. After the task was modeled for each participant, planned comparisons were computed as linear contrasts to investigate neural activity during the observe only condition compared with the crosshair fixation trials and during the emotion label condition compared with the gender label condition. Random effects analyses of the group were computed using the contrast images generated for each participant. The correction for multiple comparisons was carried out using an uncorrected *p*-value of .005 combined with a cluster size threshold of 10 voxels. All coordinates are reported in MNI format.

We first examined amygdala activity for the entire sample during the observe only condition relative to the crosshair fixation trials and extracted parameter estimates of activity (i.e., betas) from significantly active regions of the amygdala. We then performed standard *t* tests on the parameter estimates to investigate whether there were differences between individuals who scored low versus high on the risky family assessment (based on

Table 1. A Priori Brain Regions Differential Activity

Region	Talaraich Coordinate			Voxels	t-stat
Observe > Crosshair Fixation ^a					
Left Amygdala	-22	-8	-18	162	4.44
Right Amygdala	16	-8	-20	40	3.93
Affect Labeling > Gender Labeling ^b					
RVL PFC	54	24	-10	44	3.37
RVL PFC	48	46	-6	18	3.28
Gender Labeling > Affect Labeling ^c					
Left Amygdala	-24	0	-24	56	3.39

N = 30.

RVL PFC, right ventrolateral prefrontal cortex.

^aObserve Condition relative to the crosshair fixation.

^bAffect labeling condition relative to the gender labeling condition.

^cGender labeling condition relative to the affect labeling condition.

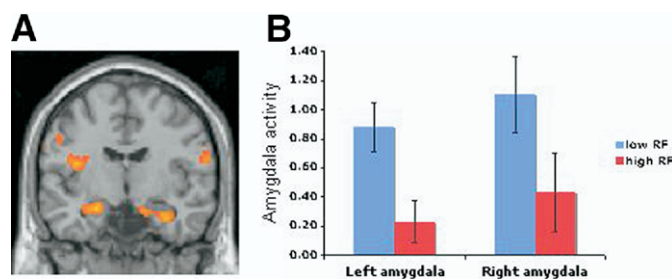


Figure 2. (A) Amygdala activity during observe only compared with the crosshair fixation. (B) Neural activity in the left and right amygdala for those low and high on the risky family assessment.

a median split). Similarly, we examined RVL PFC and amygdala activity during the emotion label relative to the gender label condition, extracted data from relatively activated or deactivated regions of the RVL PFC and amygdala, and then performed standard *t* tests to investigate whether there were differences between individuals who scored low versus high on the risky family assessment.

We also conducted similar analyses using a between-subjects approach in whole-brain analyses to investigate regions of the amygdala and RVL PFC that were significantly different in activity for those who scored low versus high on the risky family assessment. This was done using a one-way analysis of variance (ANOVA) with two groups (low vs. high risky family) in whole-brain analyses with a *p*-value of .005 combined with a cluster size threshold of 10 voxels. Lastly, we examined the correlations between RVL PFC and amygdala separately for those low and high on the risky family assessment by investigating the correlation between the parameter estimates from RVL PFC and the amygdala for each group.

Results

Behavioral Data

Reaction time data from the gender- and emotion-labeling conditions revealed that there were no reaction time differences between those low or high on the risky family assessment (*p*'s > .9). Moreover, there were no significant between-group differences in the number of errors made during the gender- or emotion-labeling conditions (*p*'s > .48).

Neuroimaging Data: Observe Condition

As noted, the observation of fearful or angry faces typically produces activation of the amygdala. Replicating previous findings, two regions of the amygdala that extended into the anterior hippocampus were significantly active for the full sample during the observe only condition compared with the crosshair fixation condition (see Table 1 and Figure 2A).³ Because we were primarily interested in amygdala responses to the emotional faces, we focused on the amygdala rather than the anterior hippocampal portion of this activation. We extracted data from these two amygdala regions and compared the amount of amygdala activation for those low versus high on the risky family assessment. Results indicated that individuals from a risky family background had significantly less activity in the left amygdala (*p* < .01) and marginally less activity in the right amygdala (*p* = .09; see Figure 2B). Similarly, in between-groups whole brain

³For overall group analyses (observe compared with crosshair fixation; emotion label compared with gender label), independent of the role of risky families, see Lieberman et al (2005a).

analyses, we found that the left amygdala was significantly less active during the observe condition compared with the crosshair fixation condition for those from a risky family background ($-20, -8, -18, t = 2.91, p < .005$). There were no between-group differences in right amygdala activation during the observe condition relative to baseline.

To ensure that the reduced amygdala activity seen among individuals from a risky family background was not driven by a lack of attention to the task, we investigated activity in a region of the fusiform face area (FFA), previously shown to be activated by attending to faces (Kanwisher et al 1997). After extracting data from a region of the FFA for the full sample ($-44, -52, -12, t = 4.48, p < .005$) during the observe only task relative to the crosshair fixation and comparing the magnitude of FFA activation for those low versus high on the risky family variable, there was no significant difference in FFA activation between the two groups ($p = .60$). Similarly, in between-group whole-brain analyses, there was no significant difference in FFA activity between those low and high on the risky family assessment. Thus, individuals from risky families showed less amygdala reactivity to negative emotional faces, a task that reliably evokes amygdala activation, and this reduced activity was not driven by inattention to the task, as evidenced by similar amounts of FFA activity in both groups.

Neuroimaging Data: Labeling Condition

The labeling of affect in fearful and angry faces typically leads to activation of the RVLPPFC, coupled with corresponding decreases in amygdala activation. Two regions of RVLPPFC were significantly activated during the emotion label compared with the gender label task for the full sample (Table 1 and Figure 3A). There were no significant differences in RVLPPFC activity related to risky family background. Also, a region of the left amygdala was relatively deactivated during the emotion label compared with the gender label condition (Table 1 and Figure 3B); however, there were no differences in the magnitude of this activation for those low or high on the risky family variable. Similarly, in whole-brain between-groups analyses, there were no significant differences between those low and high on the risky family variable in either amygdala or RVLPPFC activity.

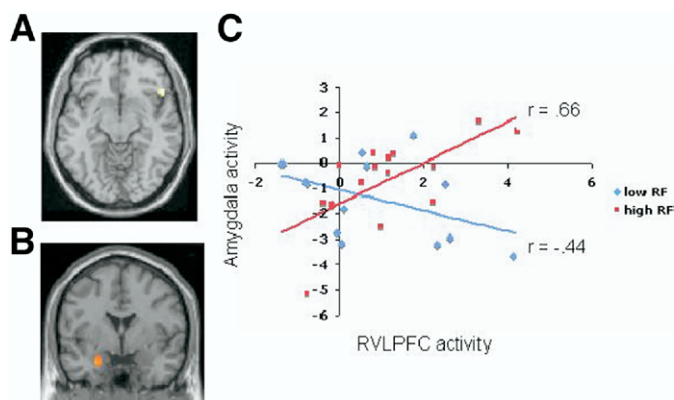


Figure 3. (A) Right ventrolateral prefrontal cortex (RVLPPFC) activity during the emotion label, relative to the gender label, condition for the full sample. (B) Relative amygdala deactivation during the emotion label, relative to the gender label, condition for the full sample. (C) Scatterplot showing the relationship between RVLPPFC and amygdala activity during the emotion label, relative to gender label, condition for individuals who scored low and high on the risky family assessment. Each point represents the data from a single participant. RVLPPFC, right ventrolateral prefrontal cortex.

We next examined connectivity between RVLPPFC and amygdala during the emotion label compared with the gender label task. Previous studies have reported negative associations between RVLPPFC and amygdala activity during emotion-labeling tasks (Hariri et al 2000; Lieberman et al 2005, Lieberman et al, unpublished data), consistent with the hypothesized role of the RVLPPFC in regulating negative affect. To assess this, we examined the correlation between RVLPPFC and amygdala activity for those low and high on the risky family variable separately. Individuals who scored low on the risky family assessment showed the previously reported pattern with greater RVLPPFC activity ($52, 24, -10$) correlating with reduced amygdala activity [$-24, 0, -24; r(30) = -.44, p < .05$] (Figure 3C). Conversely, individuals who scored high on the risky family assessment showed the opposite pattern, such that greater RVLPPFC activity ($52, 24, -10$) was associated with greater amygdala activity [$-24, 0, -24; r(30) = +.66$] (Figure 3C).

Given the dramatic differences in connectivity between those low versus high on the risky family assessment, it is surprising that no differences in amygdala activation were found during the emotion-labeling task. To further investigate possible amygdala differences in this condition, we performed a small-volume correction on a sphere centered on the point in the amygdala of maximal relative deactivation (during the emotion-labeling compared with the gender-labeling condition) for the full sample ($-24, 0, -24, 4$ mm radius) and observed a region of the amygdala ($-26, 2, -22$) that was more active for those from a risky family background in a between-subjects analysis ($p < .05$, false discovery rate [FDR] corrected). Overall, the findings suggest atypical neural responses during the labeling task for those from a risky family background.

Discussion

An early family environment marked by harsh, chaotic, or conflict-ridden parenting has been reliably related to mental and physical health risks across the life span (Felitti et al 1998; Repetti et al 2002). Deficits in emotion regulation skills have been posited to be one mechanism that may link childhood environment to these adverse outcomes (Repetti et al 2002). The present study investigated potential neural mechanisms that may underpin these relations and found evidence for potential deficits in threat detection and responses to emotional stimuli at the neural level.

Specifically, we had predicted that offspring from risky and nonrisky families would show different patterns of neural activation to tasks assessing reactivity and responses to emotional, potentially threatening stimuli. Consistent with these predictions, we found that offspring from risky families showed less amygdala activation when asked to observe negative and fearful faces. Usually this task reliably activates the amygdala, as it did for the participants in our study who were from nonrisky families. This pattern, then, suggests that offspring from risky families may not have been processing the angry/fearful faces as threatening stimuli to the same degree as offspring from more nurturant families. It is possible that offspring from risky families become sufficiently accustomed to fearful or angry faces that they habituate to them. Alternatively, the reduced amygdala activity may reflect an avoidance of potentially threatening stimuli that do not require active coping efforts. We return to this issue shortly.

When asked to label angry and fearful faces, a task that reliably elicits RVLPPFC activity and corresponding lower levels of amygdala activation, participants who were not from risky families showed this expected pattern. However, those from

risky families, instead, showed evidence of increased amygdala activation and a strongly positive correlation between amygdala activation and RVL PFC. This pattern suggests that offspring from risky families may not recruit RVL PFC effectively for regulating amygdala responses to threatening stimuli. The greater amygdala activation among participants from risky families during the labeling task also suggests that the habituation explanation for the lack of amygdala activation in the observation condition (i.e., that amygdala activity is not present because these individuals have habituated to negative emotional faces) may not hold, because habituation would be expected to prompt lower amygdala activation for those from risky families in both the observation and the labeling conditions.

The pattern of little activation of the amygdala in response to observing fearful and angry faces, coupled with evidence of increased amygdala activation and a positive relationship with RVL PFC activity when labeling these faces, is intriguing and hints at a multifaceted stress-responding signature developed by offspring from adverse family environments. Although the offspring of risky families often show greater arousal in distressing situations (Repetti et al 2002), studies have also found that they show avoidant coping responses when confronted with stress, possibly in an effort to mitigate this arousal. For example, studies of teens' coping styles found that those from higher-conflict families were more likely to demonstrate escape/avoidant coping (Johnson and Pandina 1991; Valentiner et al 1994). In addition, research on posttraumatic stress disorder (PTSD) has linked past traumatic experience with a tendency to dissociate, or "space out," in trying circumstances (Asmundson et al 2004; Johnson et al 2003). Taken together, these lines of research suggest that offspring from risky families may evidence both responses, that is, increased reliance on avoidant or escapist coping responses, as well as difficulty in regulating emotional responses to potentially threatening stimuli.

In the present study, the observation only and labeling tasks may have implicated different sides of this multifaceted response to stress. When participants were instructed simply to look at the negative faces (the observe only task), participants from riskier families responded less to the emotionality of the expressions being displayed (all individuals showed similar levels of face processing as evidence by FFA activity to the presentation of the faces). The significantly lower activation of the amygdala shown by participants from riskier families may reflect a propensity to detach from threatening stimuli that do not require an active response. In contrast, labeling the negative emotions shown on the faces presented an active task that requires engaging with emotional stimuli. This task's demands may have prevented an avoidant response. Forced to engage with the negative faces, participants from riskier families showed greater activation of the amygdala as well as activation of the RVL PFC; however, the relationship between these two regions was positive rather than negative, suggesting that RVL PFC activation was not succeeding in reducing amygdala activation.

These patterns were found in a nonclinical sample in which the "riskiness" of the early environments of participants was relatively modest. There was, for example, no evidence of physical or sexual abuse. No participant with a diagnosed major mental disorder or a PTSD diagnosis was included in the study. The findings, thus, suggest that even moderate family conflict and distress may be tied to deficits in threat detection and responses to emotional stimuli.

Limitations

There are several limitations to these findings. The risky family assessments and the fMRI component of the study were completed at different points in time. Previous research, however, indicates that risky family assessments are stable across time (Taylor et al 2004). Second, assessment of family environment involves reconstruction by these young adult participants and thus may engage certain biases. Most problematic is the potential for a negative emotional overlay to contribute to response bias and influence the reconstruction of early environment. Several factors suggest that this possibility does not account for risky family assessments. The instrument on which the risky family assessment is based (Felitti et al 1998) has demonstrated a dose-response relationship to a broad array of diagnosed mental and physical health outcomes (depression, cancer, coronary heart disease), and a response bias alone is highly unlikely to yield such effects. Moreover, in previous investigations, we have formally evaluated statistical models that give psychosocial functioning causal priority to see if it explains the reconstruction of childhood events (Taylor et al, unpublished data; Taylor et al 2005; Lehman et al 2005). In all cases, this alternative model is a weak fit to the data. Nonetheless, other factors, such as genetic contributions to risky family environments that were not assessed in the present investigation, may contribute to both the neural patterns of activation seen here and to risky family experiences and/or assessments.

Conclusions

In conclusion, this research suggests that growing up in a risky family environment marked by harsh parenting may have effects on processes involving threat detection and responses to emotional stimuli at the neural level. When faced with a passive observation task of threatening stimuli, offspring from risky families appeared to tune out the stimuli or at least the emotional aspects of the stimuli. When forced to actively engage with threatening stimuli in the emotion-labeling task, offspring from risky families not only showed signs of greater amygdala activation but showed a significantly positive relationship between RVL PFC and amygdala activation, suggesting that their efforts at this form of emotion regulation may have been counterproductive. This pattern is consistent with the idea that children from risky families do not have effective threat detection and emotion regulation skills for coping with threat. As such, the results point to the potential value of intervening with troubled families, even those not marked by severe family pathology. Whether therapeutic intervention might reverse or attenuate these neural patterns of activation in response to threat is an intriguing next question.

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Asmundson GJG, Stapleton JA, Taylor S (2004): Are avoidance and numbing distinct PTSD symptom clusters? *J Trauma Stress* 17:467–475.

Brody GH, Flor DL (1998): Maternal resources, parenting practices, and child competence in rural, single-parent African-American families. *Child Dev* 69:803–816.

Camras LA, Ribordy S, Hill J, Martino S, Spaccarelli S, Stefani R (1988): Recognition and posing of emotional expression by abused children and their mothers. *Dev Psychol* 24:776–781.

- Chorpita BF, Barlow DH (1998): The development of anxiety: The role of control in the early environment. *Psychol Bull* 124:3–21.
- Davis M (1989): The role of the amygdala and its efferent projections in fear and anxiety. In: Tyrer P, editor. *Psychopharmacology of Anxiety*. Oxford, United Kingdom: Oxford University Press, 52–79.
- Dishion TJ (1990): The family ecology of boys' peer relations in middle childhood. *Child Dev* 61:874–891.
- Dunn J, Brown J (1994): Affect expression in the family, children's understanding of emotions, and their interactions with others. *Merrill Palmer Q* 40:120–137.
- Eisenberger N, Lieberman MD, Williams KD (2003): Does rejection hurt? An fMRI study of social exclusion. *Science* 302:290–292.
- Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Aritz AM, Edwards V, et al (1998): Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *Am J Prev Med* 14:245–258.
- Flinn MV, England BG (1997): Social economics of childhood glucocorticoid stress responses and health. *Am J Phys Anthropol* 102:33–53.
- Hariri AR, Bookheimer SY, Mazziotta JC (2000): Modulating emotional responses: Effects of a neocortical network on the limbic system. *Neuroreport* 11:43–48.
- Hariri AR, Tessitore A, Mattay VS, Fera F, Weinberger DR (2002): The amygdala response to emotional stimuli: A comparison of faces and scenes. *Neuroimage* 17:317–323.
- Johnson DM, Sheahan TC, Chard KM (2003): Personality disorders, coping strategies, and posttraumatic stress disorder in women with histories of childhood sexual abuse. *J Child Sex Abus* 12:19–39.
- Johnson V, Pandina RJ (1991): Effects of family environment on adolescent substance use, delinquency, and coping styles. *Am J Drug Alcohol Abuse* 17:71–88.
- Kanwisher N, McDermott J, Chun M (1997): The fusiform face area: A module in human extrastriate cortex specialized for the perception of faces. *J Neurosci* 17:4302–4311.
- LeDoux J (1996): *The Emotional Brain: The Mysterious Underpinnings of Emotional Life*. New York: Simon & Schuster.
- LeDoux JE (1987): Emotion. In: Mountcastle V, Plum F, Geiger SR, editors. *Handbook of Physiology, vol. 5 (Section 1: The Nervous System)*. Bethesda, MD: American Physiological Association, 419–455.
- Lehman BJ, Taylor SE, Kiefe CI, Seeman TE (2005): Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosom Med* 67(6):846–854.
- Lieberman MD, Hariri A, Jarcho JM, Eisenberger NI, Bookheimer SY (2005): An fMRI investigation of race-related amygdala activity in African-American and Caucasian-American individuals. *Nat Neurosci* 8:720–722.
- Lieberman MD, Jarcho JM, Berman S, Naliboff B, Suyenobu BY, Mandelkern M, et al (2004): The neural correlates of placebo effects: A disruption account. *Neuroimage* 22:447–455.
- Lundberg O (1993): The impact of childhood living conditions on illness and mortality in adulthood. *Soc Sci Med* 36:1047–1052.
- McEwen BS (1998): Protective and damaging effects of stress mediators. *N Engl J Med* 338:171–179.
- O'Brien M, Margolin G, John RS, Krueger L (1991): Mothers' and sons' cognitive and emotional reactions to simulated marital and family conflict. *J Consult Clin Psychol* 59:692–703.
- Ochsner KN, Bunge SA, Gross JJ, Gabrieli JD (2002): Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *J Cogn Neurosci* 14:1215–1229.
- Ochsner KN, Ray RD, Cooper JC, Robertson ER, Chopra S, Gabrieli JDE, Gross JJ (2004): For better or for worse: neural systems supporting the cognitive down- and up-regulation of negative emotion. *Neuroimage* 23:483–499.
- Reid RJ, Crisafulli A (1990): Marital discord and child behavior problems: A meta-analysis. *J Abnorm Child Psychol* 18:105–117.
- Repetti RL, Taylor SE, Seeman TE (2002): Risky families: Family social environments and the mental and physical health of offspring. *Psychol Bull* 128:330–366.
- Small DM, Zatorre RJ, Dagher A, Evans AC, Jones-Gotman M (2001): Changes in brain activity related to eating chocolate: From pleasure to aversion. *Brain* 124:1720–1733.
- Taylor SE, Lerner JS, Sage RM, Lehman BJ, Seeman TE (2004): Early environment, emotions, responses to stress, and health. *J Pers* 72:1365–1393.
- Valentiner DP, Holahan CJ, Moos RH (1994): Social support, appraisals of event controllability, and coping: An integrative model. *J Pers Soc Psychol* 66:1094–1102.
- Walker EA, Gelfand A, Katon WJ, Koss MP, Von Korff M, Bernstein D, et al (1999): Adult health status of women with histories of childhood abuse and neglect. *Am J Med* 107:332–339.
- Whalen P (1999): Fear, vigilance, and ambiguity: Initial neuroimaging studies of the human amygdala. *Curr Dir Psychol Sci* 7:177–188.
- Whalen PJ, Rauch SL, Etcoff NL, McInerney SC, Lee MB, Jenike MA (1998): Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J Neurosci* 18:411–418.