Cogn Ther Res (2010) 34:380–387
DOI 10.1007/s10608-009-9269-3

ORIGINAL ARTICLE

Reports of Childhood Physical Abuse, 5-HTTLPR Genotype, and Women's Attentional Biases for Angry Faces

Ashley L. Johnson · Brandon E. Gibb · John McGeary

Published online: 16 September 2009

Abstract The goal of this study was to examine environmental (childhood physical abuse) and genetic (*5-HTTLPR* genotype) correlates of adult women's attentional biases for facial displays of emotion. Supporting a gene × environment model of risk, women's reports of childhood physical abuse were related to their attentional biases for angry faces among carriers of the *5-HTTLPR* short allele, but not among those homozygous for the long allele. Specifically, women reporting a history of moderate to severe physical abuse who also carried at least one copy of the *5-HTTLPR* short allele exhibited attentional avoidance of angry faces. These results were specific to angry faces and were not observed for happy or sad faces. Supporting the robustness of these findings, they were maintained even after statistically controlling for the influence of women's lifetime diagnoses of major depression and anxiety disorders as well as their current symptoms of depression and anxiety, suggesting that the results were not due simply to current or past depression or anxiety.

Keywords Attention bias · Child abuse · 5-HTTLPR · Gene × environment

Introduction

Theorists (e.g., Clark et al. 1999; Williams et al. 1997) have proposed that biases in the processing of emotional stimuli (e.g., attentional biases) may contribute to the development and maintenance of psychopathology. Supporting these theories, a number of studies have found evidence of attentional biases in various forms of psychopathology (for reviews, see Bar-Haim et al. 2007; Joormann 2008; Mathews and MacLeod 2005). There is evidence, however, that the focus of these attentional biases differs across disorders. For example, whereas attentional biases in anxiety appear to be specific to threat-relevant stimuli (e.g., angry faces), attentional biases in depression appear to be specific to stimuli reflecting themes of sadness or loss (e.g., sad faces) (for reviews, see Joormann 2008; Mathews and MacLeod 2005). In addition to these cross-sectional findings, there is preliminary evidence that these attentional biases may contribute risk for the development and maintenance of psychopathology (e.g., Beevers and Carver 2003; Gibb et al. 2009a; MacLeod et al. 2002; Mathews and MacLeod 2002).

Given this, it becomes important to understand potential developmental origins of these attentional biases. In addition to the theoretical importance of this question, there are also clear clinical implications. Specifically, a better understanding of how attentional biases develop would facilitate the development of more targeted prevention and early intervention programs (cf. Clarke et al. 2008; Pollak 2003; MacLeod et al. 2009). This line of research may also help to identify specific subgroups of individuals particularly vulnerable to the development of biased information processing so that treatment resources could be targeted to those at greatest risk.

Theorists (e.g., Cicchetti et al. 2000; Pollak 2003; Rose and Abramson 1992) have proposed that negative experiences in childhood, particularly child abuse, may contribute to the development of experience-specific information-processing biases. Supporting this hypothesis, there is evidence that children with a history of physical abuse exhibit attentional

A. L. Johnson (✉) · B. E. Gibb
Department of Psychology, Binghamton University (SUNY), Binghamton, NY 13902-6000, USA
e-mail: ajohns11@binghamton.edu

J. McGeary
Providence Veterans Affairs Medical Center and Center for Alcohol and Addiction Studies, Brown University, Providence, RI, USA

biases specifically towards angry faces (e.g., Pollak and Tolley-Schell 2003; Shackman et al. 2007). It may initially be adaptive for children in abusive situations to selectively process signals of anger as this may facilitate attempts to avoid the abuse. However, theorists have suggested that these attentional biases may become maladaptive if they develop into relatively trait-like processing styles that are applied rigidly to a broader range of (objectively safer) interpersonal contexts (see Cicchetti et al. 2000; Crick and Dodge 1994; Gotlib and MacLeod 1997; Pollak 2003; Rose and Abramson 1992). To the extent that childhood abuse contributes to the development of relatively stable attentional biases, one would hypothesize that these biases would also be observed in adults with a history of childhood abuse. Supporting this hypothesis, there is evidence that young adults reporting a history of childhood abuse exhibit preferential attention specifically for angry, but not happy or sad, faces (Gibb et al. 2009c).

In addition to environmental correlates of attentional biases, there are also clear genetic influences on cognitive processes. In terms of specific genetic influences, there has been considerable interest in a functional polymorphism in the serotonin transporter gene (*5-HTTLPR*). There is growing evidence that carriers of the *5-HTTLPR* short allele, compared to individuals homozygous for the long allele, exhibit increased amygdala reactivity to negative facial stimuli (for a review, see Munafó et al. 2008). Importantly, there is also evidence that carriers of the short allele exhibit attentional biases for anxiety-relevant words (Beevers et al. 2007). In addition to the potential main effect of *5-HTTLPR* genotype on attentional biases, there is growing evidence that *5-HTTLPR* genotype moderates reactivity to environmental stressors. For example carriers of the *5-HTTLPR* short allele, compared to those homozygous for the long allele, exhibit greater cortisol reactivity to a laboratory-based stressor (Gotlib et al. 2008) and increased risk for depression and posttraumatic stress disorder following negative life events (for reviews, see Koenen 2007; Rutter et al. 2006; Uher and McGuffin 2008). An important question, therefore, is whether the link between childhood abuse and attentional biases would be stronger among carriers of the *5-HTTLPR* short allele than among those homozygous for the long allele. Offering some support for this hypothesis, we have found that children's *5-HTTLPR* genotype moderates the link between maternal expressed emotion-criticism and children's attentional biases for angry faces (Gibb et al. 2009b). Specifically, the presence of high expressed emotion-criticism was related to children's attentional biases for angry, but not happy or sad, faces among carriers of the *5-HTTLPR* short allele, but not among those homozygous for the long allele.

In the current study, we predicted that women reporting a history of childhood physical abuse would exhibit preferential attention specifically for angry, but not happy or sad, faces and that this relation would be stronger among carriers of the *5-HTTLPR* short allele than among those homozygous for the long allele. Further, we predicted that these relations would be maintained even after statistically controlling for the influence of women's lifetime history of major depression and anxiety disorders and their current depressive and anxious symptoms.

Methods

Participants

Participants in this study were 86 adult women drawn from the community who were part of a larger study of the intergenerational transmission of depression. Participants were required to either meet lifetime criteria for at least one *DSM-IV* major depressive disorder (MDD) ($n = 46$) or have no lifetime history of any *DSM-IV* mood disorder ($n = 40$). Exclusion criteria for both groups included symptoms of schizophrenia, organic mental disorder, or history of bipolar I disorder. The average age of mothers in our sample was 38.86 years (SD = 6.77, range = 26–53) and the median family income was $50,000–$55,000 per year. In terms of race, 87.2% of the mothers were Caucasian, 5.8% were African American, 4.7% were Asian American, and two mothers (2.2%) were from other racial/ethnic groups.

Measures

The Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L; Endicott and Spitzer 1978) was used to assess for *DSM-IV* Axis I disorders in participants. This measure is a widely used diagnostic interview with well-established psychometric properties (Endicott and Spitzer 1978). Of the 46 women meeting criteria for at least one lifetime episode of MDD, seven met criteria for current MDD. Twenty-six women met criteria for at least one lifetime anxiety disorder, of whom ten met criteria for one or more current anxiety disorders (social phobia = 7; posttraumatic stress disorder = 4; generalized anxiety disorder = 3, specific phobia = 5; Panic Disorder = 1). A subset of 20 SADS-L interviews from this project was coded by a second interviewer and kappa coefficients for diagnoses included in this study were: current MDD ($\kappa = 1.00$), lifetime MDD ($\kappa = 1.00$), current anxiety disorder ($\kappa = 0.83$), lifetime anxiety disorder ($\kappa = 0.78$).

Symptoms of depression and anxiety were assessed using the Beck Depression Inventory-II (BDI-II; Beck et al. 1996) and the Beck Anxiety Inventory (BAI; Beck et al. 1988), respectively. Numerous studies have supported the reliability and validity of both measures (e.g., Beck et al. 1996, 1988; Creamer et al. 1995). Both measures exhibited good

internal consistency in this sample (BDI-II: $\alpha = 0.93$; BAI: $\alpha = 0.89$).

The Childhood Trauma Questionnaire (CTQ; Bernstein and Fink 1998; Bernstein et al. 2003) was used to assess participants' histories of childhood physical abuse. Each item is rated on a 5-point Likert-type scale, with response options ranging from "Never true" to "Very often true." Subscale scores are calculated by summing responses within each abuse type. Scores on each scale can range from 5 to 25, with higher scores indicating more severe abuse. The CTQ has demonstrated excellent psychometric properties in both clinical and nonclinical samples, including high levels of concurrent validity with therapists' ratings of abuse (Bernstein and Fink 1998; Bernstein et al. 2003). The physical abuse subscale exhibited good internal consistency in the current study ($\alpha = 0.90$). Because of the extreme skew of this variable in our sample ($z = 7.56$), a dichotomous classification was created for reported histories of abuse (abuse history vs. no abuse history). To be classified as having a history of abuse, the participant must have scored in the moderate to severe range on the CTQ-Physical Abuse subscale (i.e., score greater than nine; cf. Bernstein and Fink 1998). Using this criterion 13 (15.1%) participants were classified as having a history of moderate to severe physical abuse.

Attentional biases for facial displays of emotion were assessed using a modified dot-probe task (cf. MacLeod et al. 1986) administered using E-Prime (Psychological Software Tools 2002). Stimuli for the dot-probe task consisted of pairs of facial expressions that contained one emotional (angry, happy, sad) and one neutral photograph from the same actor taken from a standardized stimulus set (Tottenham et al., in press). Photographs from each actor (16 males and 16 females) were used to create angry-neutral, happy-neutral, and sad-neutral stimulus pairs (96 pairs total). Each stimulus pair was presented in random order over the course of two blocks, with a rest in between blocks. Stimuli were presented for 1,000 ms, followed by a dot replacing one of the pictures. A 1,000 ms stimulus duration was chosen because a history of childhood abuse is hypothesized to be related to difficulty disengaging attention from angry faces (Pollak and Tolley-Schell 2003; see also Gibb et al. 2009c) and studies examining this aspect of attentional bias have found stronger evidence at relatively longer presentation durations (i.e., 1,000 ms; see Mathews and MacLeod 2005). Following the presentation of the dot probe on the screen, participants were asked to indicate the location of the dot (left vs. right side of the screen) as quickly as possible using a response box. In each pair, the emotional face was presented with equal frequency on the left and right side of the screen and the probe occurred with equal frequency in the location of the emotional and neutral faces. The intertrial interval was 1,000 ms. Trials with response errors were excluded (0.16%) as were trials with response times less than 150 ms or greater than 1,500 ms (0.20%). Mean bias scores (Mogg et al. 1995) were then calculated separately for each emotion type (angry, happy, sad) by subtracting the mean response time for cases in which the probe replaced the emotional face from mean response times for cases in which the probe replaced the neutral face. Positive bias scores represent preferential attention toward the emotional faces, whereas negative scores indicate attentional avoidance of the emotional faces.

Finally, participants provided buccal cells by rubbing swabs along their cheeks and gums and rinsing out their mouths with 10 ml of distilled water. DNA was collected and isolated using published procedures (Freeman et al. 1997; Lench et al. 1988). The *5-HTTLPR* alleles were assayed using previously reported methods (Pooley et al. 2003). The primer sequences are forward, 5′-GCG TTG CCG CTC TGA ATG C-3′ and reverse 5′-GGA CTG AGC TGG ACA ACC AC-3′. Although our current sample size of physically abused women was too small to focus on trichotomous classifications of *5-HTTLPR* genotype (ss, sl, ll), we have found in our previous research (Beevers et al. 2007) that carriers of one versus two short alleles exhibited similar attentional biases, with both differing from individuals homozygous for the long allele. In the current study, therefore, two groups of participants were formed based on their *5-HTTLPR* genotype: individuals with either one or two copies of the short allele (ss/sl; $n = 50$) and those homozygous for the long allele (ll; $n = 36$). These frequencies did not differ from Hardy–Weinberg Equilibrium.

Procedure

Potential participants were recruited from the community through a variety of means (e.g., newspaper ads, bus ads, and flyers). Individuals responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Those reporting either significant depressive symptoms or no significant lifetime symptoms of depression were invited to participate in the study. Upon arrival at the laboratory, participants were asked to provide informed consent. The participant then completed the interview, questionnaire, computer, and DNA assessments. Participants were compensated $50 for their participation.

Results

Preliminary results revealed that women in the depressed group reported higher levels of depressive ($M = 13.54$, SD = 9.90 vs. $M = 2.68$, SD = 2.67; $t[84] = 6.73$, $p < 0.001$, $r_{\text{effect size}} = 0.59$) and anxious ($M = 9.18$, SD = 7.99 vs. $M = 2.85$, SD = 2.59; $t[84] = 4.80$, $p < 0.001$, $r_{\text{effect size}} = 0.46$) symptoms. This said, however, women in both

groups exhibited, on average, minimal to mild levels of depressive and anxious symptoms. Women in the depressed group were not significantly more likely to report a history of childhood physical abuse than were women in the control group (21.7% vs. 7.5%; $\chi^2[1, N = 86] = 3.38$, $p = 0.07$, $r_{\text{effect size}} = 0.20$). Finally, women's *5-HTTLPR* genotype was not significantly related to lifetime diagnoses of MDD ($\chi^2[1, N = 86] = 0.11, p = 0.74, r_{\text{effect size}} = -0.04$) or anxiety disorders ($\chi^2[1, N = 86] = 2.20, p = 0.14, r_{\text{effect size}} = -0.16$) or to women's reported histories of childhood physical abuse ($\chi^2[1, N = 86] = 2.44, p = 0.12, r_{\text{effect size}} = -0.17$).

Next, we tested our hypotheses regarding women's attentional biases. Specifically, we conducted a 2 (Abuse History: Yes, No) × 2 (*5-HTTLPR* genotype: ss/sl, ll) × 3 (Facial expression: Angry, Happy, Sad) repeated measures analysis of variance (ANOVA). Because participants were selected for this study based on the presence vs. absence of lifetime MDD, women's MDD history was included as an additional between-subjects factor in these analyses. Analyses revealed a significant main effect for facial expression, $F(2, 158) = 4.08$, $p = 0.02$, $\eta_p^2 = 0.05$ and a significant *5-HTTLPR* × facial expression interaction, $F(2, 158) = 3.57$, $p = 0.03$, $\eta_p^2 = 0.04$. Importantly, the predicted *5-HTTLPR* × physical abuse × facial expression interaction was also significant, $F(2, 158) = 5.10$, $p < 0.007$, $\eta_p^2 = 0.06$. In these analyses, none of the main or interactive effects of mother MDD history were significant.

To determine the form of the significant *5-HTTLPR* × physical abuse × facial expression interaction, we examined the two-way physical abuse × facial expression interaction separately within each of the two *5-HTTLPR* genotype groups. In each of these analyses, MDD history was included as a covariate. Consistent with our hypothesis, the physical abuse × facial expression interaction was significant among carriers of a *5-HTTLPR* short allele, $F(2, 94) = 3.04$, $p = 0.05$, $\eta_p^2 = 0.06$, but not among individuals homozygous for the *5-HTTLPR* long allele, $F(2, 66) = 1.49$, $p = 0.23, \eta_p^2 = 0.04$.[1] Follow-up analyses among carriers of the *5-HTTLPR* short allele revealed that a reported history of childhood physical abuse was significantly related to women's attentional biases for angry faces, $F(1, 47) = 3.99$, $p = 0.05$, $\eta_p^2 = 0.08$. Specifically, among carriers of the *5-HTTLPR* short allele, a history of childhood physical abuse was related to an attentional avoidance of angry faces. In contrast, reports of childhood physical abuse were not significantly related to attentional biases for happy $F(1, 47) = 1.72$, $p = 0.20$, $\eta_p^2 = 0.04$, or sad, $F(1, 47) = 0.66$, $p = 0.42, \eta_p^2 = 0.01$, faces among these women. The results of these analyses are presented in Fig. 1.

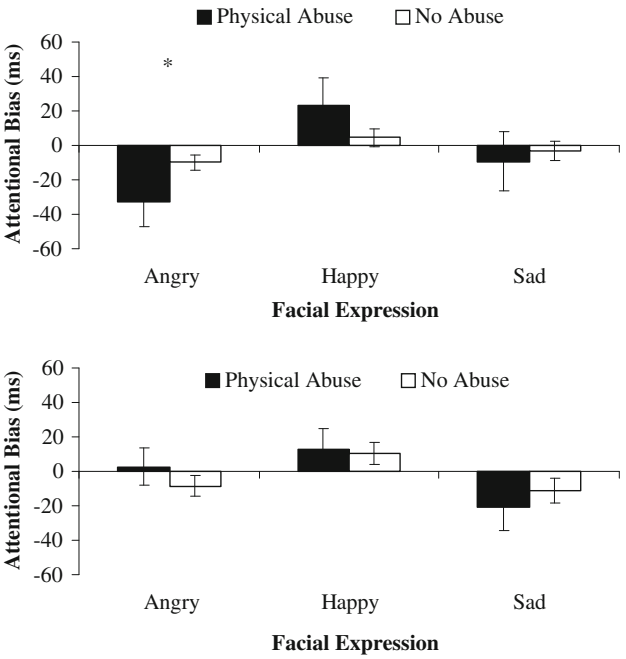

Fig. 1 Women's mean attentional bias scores (in milliseconds) across the three facial expression types as a function of reported childhood physical abuse among carriers of the *5-HTTLPR* short allele (*top panel*) versus women homozygous for the *5-HTTLPR* long allele (*bottom panel*). *Error bars* represent one standard error. * $p = 0.05$

Importantly, the magnitude of the relation between reports of childhood physical abuse and attentional biases for angry faces among carriers of the *5-HTTLPR* short allele remained virtually unchanged even after statistically controlling for the potential influence of women's current levels of depressive and anxious symptoms as well as their lifetime diagnoses of anxiety disorders and MDD, $F(1, 44) = 3.95$, $p = 0.05$, $\eta_p^2 = 0.08$, suggesting that the obtained results were not due simply to the presence of lifetime MDD or anxiety disorders, or current symptom levels.[2] Finally, given the link between childhood abuse and later traumatization (e.g., Widom et al. 2008), analyses were also conducted to determine if effects could be better explained by trauma experienced in

[1] The similarity in effect size $\left(\eta_p^2\right)$ in these analyses across the two genotype groups suggests the possibility that the nonsignificant result among women homozygous for the *5-HTTLPR* long allele was due, in part, to the smaller size of this subsample. Given this, exploratory analyses were conducted to test for potential physical abuse group differences in attentional biases for the three facial expressions among these women. Among women homozygous for the *5-HTTLPR* long allele, tests of simple main effects within Facial Expression type revealed no significant physical abuse differences for angry, $F(1, 29) = 0.69$, $p = 0.41$, $\eta_p^2 = 0.02$, happy, $F(1, 29) = 2.87$, $p = 0.10$, $\eta_p^2 = 0.09$, or sad, $F(1, 29) = 0.11$, $p = 0.75$, $\eta_p^2 = 0.004$ faces.

[2] Although not the primary focus of this study, we also examined whether women's current or lifetime diagnoses of MDD and/or anxiety disorders were related to attentional biases for any of the facial expressions. None of these analyses was significant, nor did diagnostic status significantly interact with abuse history of *5-HTTLPR* genotype to predict attentional biases.

adulthood (after age 18), assessed using the trauma screen from the SADS-L posttraumatic stress disorder section. According to this measure, nine women (10.5%) reported a physical assault in adulthood. The relation between reports of childhood physical abuse and attentional biases for angry faces among carriers of the *5-HTTLPR* short allele was maintained even after statistically controlling for the potential influence of physical assault in adulthood, $F(1, 46) = 4.13$, $p = 0.05$, $\eta_p^2 = 0.08$. Also, neither the adult assault × facial expression, $F(2,152) = 1.98$, $p = 0.14$, $\eta_p^2 = 0.03$, nor the 5-HTTLPR × adult assault × facial expression, $F(2,152) = 0.96$, $p = 0.39$, $\eta_p^2 = 0.01$, interactions were significant.

Discussion

The primary goal of this study was to examine environmental and genetic correlates of adult women's attentional biases for facial displays of emotion. Replicating previous research (e.g., Gibb et al. 2009c; Pine et al. 2005; Pollak and Tolley-Schell 2003), we found that women reporting a history of childhood physical abuse exhibited attentional biases specifically for angry, but not happy or sad, faces. Extending previous research, we found that this relation was specific to carriers of the *5-HTTLPR* short allele. Contrary to our predictions, however, women reporting a history of moderate to severe childhood physical abuse, who also carried at least one copy of the *5-HTTLPR* short allele, exhibited attentional avoidance of angry faces rather than preferential attention. Supporting the robustness of these findings, they were maintained even after statistically controlling for the potential influence of women's lifetime diagnoses of MDD and anxiety disorders and their current symptoms of depression and anxiety, suggesting that the biases are not simply a correlate of psychopathology. In addition, the relations were maintained even after statistically controlling for the influence of traumatic experiences (physical assault) in adulthood, suggesting some specificity to physical abuse in childhood.

These findings add to a growing body of research supporting the presence of attentional biases specifically for angry faces among individuals with documented or reported histories of abuse (see also Gibb et al. 2009c; Pine et al. 2005; Pollak and Tolley-Schell 2003; Shackman et al. 2007). This said, however, there is some mixed evidence regarding the direction of this bias. Specifically, whereas the majority of studies find evidence of preferential attention for (or difficulty disengaging attention from) angry faces (e.g., Gibb et al. 2009c; Pollak and Tolley-Schell 2003, Shackman et al. 2007), there is also evidence from previous research (e.g., Pine et al. 2005) and the current study of attentional avoidance. The exact reasons for this discrepancy are unclear. It is tempting to suggest that our finding of attentional avoidance was due to the relatively long stimulus presentation duration (1,000 ms) used in the current study and that preferential attention toward angry faces would be observed with shorter stimulus presentations (e.g., 500 ms). This would be consistent with theorists who have suggested the possibility of vigilance-avoidance patterns of attention (Mathews and MacLeod 2005) in which individuals may initially allocate their attention to threatening stimuli, but then avert their attention from the stimuli. However, we used the same experimental procedure, including a 1,000 ms stimulus presentation duration, for our previous study (Gibb et al. 2009c) in which we found evidence for preferential attention. Also, other studies utilizing a shorter stimulus presentation duration (500 ms) also yield mixed results regarding the direction of the attentional bias (Pine et al. 2005; Pollak and Tolley-Schell 2003). Therefore, although we can have some confidence in the specificity of abuse history to attentional biases for angry rather than happy or sad faces, we must remain tentative regarding the direction of the attentional bias. A limitation of this and other studies utilizing the dot probe as a measure of attentional biases is that it only allows a determination of where participants were allocating their attention at a specific point in time (e.g., 1,000 or 500 ms after stimulus onset). Future research utilizing procedures that allow for a more fine-grained analysis of participants' attentional patterns across an entire stimulus trial (e.g., eye-tracking) are necessary to more precisely define the potentially dynamic patterns of attention in response to angry faces observed among individuals with a history of childhood abuse.

Although not a primary focus of this study, we should also highlight the nonsignificant relations between women's attentional biases and their history of MDD and anxiety disorders. The nonsignificant findings with anxiety could be due to the 1,000 ms stimulus presentation duration used in our dot probe task. Supporting this hypothesis, a recent meta-analysis indicated that stronger effects for attentional bias towards anger or fear stimuli were found for shorter duration stimuli presentation (i.e., 500 ms; Bar-Haim et al. 2007). In the current study, we chose to use a 1,000 ms stimulus presentation duration in our dot probe task because we were primarily interested in prolonged attention/difficulty disengaging attention from angry faces among women reporting a history of childhood abuse rather than relations with anxiety. This said, future research employing eye tracking technology will allow the determination of attentional allocation patterns across an entire stimulus presentation and would likely yield stronger evidence for the relation between anxiety and attentional biases for angry faces. With regard to depression, previous studies finding evidence for attentional biases to sad faces have focused on

a 1,000 ms stimulus presentation duration; however, they have also utilized a mood induction to "prime" latent schema (e.g., Joormann and Gotlib 2007). Indeed, theorists (e.g., Clark et al. 1999; Persons and Miranda 1992) have suggested that depressive schema should be primed with a negative mood induction prior to assessing information-processing biases (e.g., attention biases). A mood induction was not used in this study because we were primarily interested in genetic and environmental correlates of attentional biases and studies investigating this aspect of attention have not used mood induction procedures (e.g., Beevers et al. 2007; Gibb et al. 2009c; Pine et al. 2005; Pollak and Tolley-Schell 2003).

Despite these caveats, the current results add to a growing body of research supporting the presence of experience-specific information processing biases (see also Gibb et al. 2009a, b, c; Pine et al. 2005; Pollak and Tolley-Schell 2003; Shackman et al. 2007). Specifically, reports of childhood physical abuse were related to attentional biases for angry, but not happy or sad, faces. Importantly, the current results suggest that this relation may be particularly likely among individuals carrying certain genotypes (e.g., one or two copies of the *5-HTTLPR* short allele) associated with heightened reactivity to environmental stimuli. Although we chose to focus on *5-HTTLPR* given prior evidence of its relation to attentional biases (Beevers et al. 2007) and evidence that it moderates the impact of environmental stress (for reviews, see Rutter et al. 2006; Uher and McGuffin 2008; see also Gotlib et al. 2008), it is unlikely that these effects are limited to any single gene. Therefore, future research would benefit from a more extensive investigation of candidate polymorphisms in addition to *5-HTTLPR* that are linked to heightened stress reactivity such as polymorphisms in the brain-derived neurotrophic factor (*BDNF*) gene (e.g., Kaufman et al. 2006) and the corticotrophin-releasing hormone type 1 receptor (*CHRH1*) gene (e.g., Bradley et al. 2008).

Although these results are consistent with the hypothesis that physical abuse may contribute to the development of attentional biases specifically for angry faces (Pollak 2003), particularly among carriers of the *5-HTTLPR* short allele, no causal conclusions can be drawn given the study's cross-sectional design. Ethical considerations preclude prospective studies of ongoing childhood abuse making it impossible to determine whether ongoing physical abuse contributes to the development of attentional biases. There is evidence, however, that milder environmental stressors like verbal victimization predict prospective changes in children's cognitions (see Gibb and Alloy 2006; Gibb and Abela 2008). Importantly, there is also evidence that a related construct, maternal criticism, is related to children's attentional biases specifically for angry faces, again only among carriers of the *5-HTTLPR* short allele (Gibb et al. 2009b). In combination, these results support the utility of additional research focused on G × E models of the development of attentional biases.

Two other potential limitations should be noted. First, because we focused exclusively on women, additional research is needed to determine if similar G × E effects would be observed in men. Second, histories of childhood abuse were based exclusively on participants' self-report, which may be subject to recall or response bias. This said, studies have suggested that adults' recall of specific childhood events such as abuse is relatively accurate (for a review, see Brewin et al. 1993) and the measure of childhood abuse used in this study has shown good criterion-related validity with therapists' ratings of abuse (Bernstein et al. 2003). We should also note that the criteria used to define the abused group in this study were fairly liberal, and included participants who reported at least "moderate" levels of physical abuse (cf. Bernstein and Fink 1998). The consistency of our results with previous studies of children with documented histories of abuse (Pine et al. 2005; Pollak and Tolley-Schell 2003) adds confidence to the validity and robustness of the association between childhood physical abuse and attentional biases, and suggests that effects of childhood physical abuse may not be limited to the most severe examples of these experiences.

In conclusion, the current results add to the growing body of research supporting the link between childhood abuse and attentional biases specifically for angry faces and suggest that these biases may be maintained into adulthood. Importantly, the current results also suggest the presence of a specific subgroup of individuals—those with one or two copies of the *5-HTTLPR* short allele—who may be especially likely to exhibit attentional biases in relation to histories of abuse. Future research is needed to clarify the direction of the attentional bias in response to angry faces (vigilance vs. avoidance). Research is also is needed to examine G × E models for the actual development of attentional biases.

Acknowledgments This project was supported by National Institute of Child Health and Human Development grant HD048664 and by funding from the Center for Developmental Psychobiology, Binghamton University, awarded to the B. E. Gibb.

References

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & vn IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*, 1–24.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory manual* (2nd ed.). San Antonio, TX: Psychological Corporation.

Beevers, C. G., & Carver, C. S. (2003). Attentional bias and mood persistence as prospective predictors of dysphoria. *Cognitive Therapy and Research, 27*, 619–637.

Beevers, C. G., Gibb, B. E., McGeary, J. E., & Miller, I. W. (2007). Serotonin transporter genetic variation and biased attention for emotional word stimuli among psychiatric inpatients. *Journal of Abnormal Psychology, 116*, 208–212.

Bernstein, D. P., & Fink, L. (1998). *Childhood trauma questionnaire: A retrospective self-report*. San Antonio, TX: The Psychological Corporation.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., et al. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse and Neglect, 27*, 169–190.

Bradley, R. G., Binder, E. B., Epstein, M. P., Tang, Y., Nair, H. P., Liu, W., et al. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. *Archives of General Psychiatry, 65*, 190–200.

Brewin, C. R., Andrews, B., & Gotlib, I. H. (1993). Psychopathology and early experience: A reappraisal of retrospective reports. *Psychological Bulletin, 113*, 82–98.

Cicchetti, D., Toth, S. L., & Maughan, A. (2000). An ecological-transactional model of child maltreatment. In A. J. Sameroff, M. Lewis, & S. M. Miller (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 689–722). New York: Kluwer.

Clark, D. A., Beck, A. T., & Alford, B. A. (1999). *Scientific foundations of cognitive theory and therapy of depression*. New Jersey: Wiley.

Clarke, P., MacLeod, C., & Shirazee, N. (2008). Prepared for the worst: Readiness to acquire threat bias and susceptibility to elevate trait anxiety. *Emotion, 8*, 47–57.

Creamer, M., Foran, J., & Bell, R. (1995). The beck anxiety inventory in a non-clinical sample. *Behaviour Research and Therapy, 33*, 477–485.

Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin, 115*, 74–101.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The schedule for affective disorders and schizophrenia. *Archives of General Psychiatry, 35*, 837–844.

Freeman, B., Powell, J., Ball, D., Hill, L., Craig, I., & Plomin, R. (1997). DNA by mail: An inexpensive and noninvasive method for collecting DNA samples from widely dispersed populations. *Behavior Genetics, 27*, 251–257.

Gibb, B. E., & Abela, J. R. Z. (2008). Emotional abuse, verbal victimization, and the development of children's negative inferential styles and depressive symptoms. *Cognitive Therapy and Research, 32*, 161–176.

Gibb, B. E., & Alloy, L. B. (2006). A prospective test of the hopelessness theory of depression in children. *Journal of Clinical Child and Adolescent Psychology, 35*, 264–274.

Gibb, B. E., Benas, J. S., Grassia, M., & McGeary, J. (2009a). Children's attentional biases and 5-HTTLPR genotype: Potential mechanisms linking mother and child depression. *Journal of Clinical Child and Adolescent Psychology, 38*, 415–426.

Gibb, B. E., Johnson, A. L., Benas, J. S., Uhrlass, D. J., & McGeary, J. (2009b). *Children's 5-HTTLPR genotype moderates the link between maternal criticism and attentional biases specifically for facial displays of anger*. Manuscript submitted for publication.

Gibb, B. E., Schofield, C. A., & Coles, M. E. (2009c). Reported history of childhood abuse and young adults' information processing biases for facial displays of emotion. *Child Maltreatment, 14*, 148–156.

Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR stress, and depression. *Biological Psychiatry, 63*, 847–851.

Gotlib, I. H., & MacLeod, C. (1997). Information processing in anxiety and depression: A cognitive-developmental perspective. In J. A. Burack & J. T. Enns (Eds.), *Attention, development, and psychopathology* (pp. 350–378). New York: Guilford.

Joormann, J. (2008). Cognitive aspects of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 298–321). New York: Guilford.

Joormann, J., & Gotlib, I. (2007). Selective attention to emotional faces following recovery from depression. *Journal of Abnormal Psychology, 116*, 80–85.

Kaufman, J., Yang, B., Douglas-Palumberi, H., Grasso, D., Lipschitz, D., Houshyar, S., et al. (2006). Brain-derived neurotrophic factor-5-HHTLPR gene interactions and environmental modifiers of depression in children. *Biological Psychiatry, 59*, 673–680.

Koenen, K. C. (2007). Genetics of posttraumatic stress disorder: Review and recommendations for future studies. *Journal of Traumatic Stress, 20*, 737–750.

Lench, N., Stanier, P., & Williamson, R. (1988). Simple non-invasive method to obtain DNA for gene analysis. *Lancet, 1*, 1356–1358.

MacLeod, C., Koster, E. H., & Fox, E. (2009). Whither cognitive bias modification research? Commentary on the special section articles. *Journal of Abnormal Psychology, 118*(1), 89–99.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*, 15–20.

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology, 111*(1), 107–123.

Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. *Cognition & Emotion, 16*(3), 331–354.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*, 167–195.

Mogg, K., Bradley, B. P., & Williams, R. (1995). Attentional bias in anxiety and depression: The role of awareness. *British Journal of Clinical Psychology, 34*, 17–36.

Munafó, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry, 63*, 852–857.

Persons, J. J., & Miranda, J. (1992). Cognitive theories of vulnerability to depression: Reconciling negative evidence. *Cognitive Therapy and Research, 16*, 485–502.

Pine, D. S., Mogg, K., Bradley, B. P., Montgomery, L., Monk, C. S., McClure, E., et al. (2005). Attention bias to threat in maltreated children: Implications for vulnerability to stress-related psychopathology. *American Journal of Psychiatry, 162*, 291–296.

Pollak, S. D. (2003). Experience-dependent affective learning and risk for psychopathology in children. *Annals of the New York Academy of Sciences, 1008*, 102–111.

Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology, 112*, 323–338.

Pooley, E. C., Houston, K., Hawton, K., & Harrison, P. J. (2003). Deliberate self-harm is associated with allelic variation in the tryptophan hydroxylase gene (TPH A779C), but not with polymorphisms in five other sertonergic genes. *Psychological Medicine, 33*, 775–783.

Psychological Software Tools. (2002). *E-Prime* (Version 1.1). [Computer software]. Pittsburgh, PA: Author.

Rose, D. T., & Abramson, L. Y. (1992). Developmental predictors of depressive cognitive style: Research and theory. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium of developmental psychopathology* (Vol. IV, pp. 323–349). Rochester, NY: University of Rochester Press.

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47*, 226–261.

Shackman, J. E., Shackman, A. J., & Pollak, S. D. (2007). Physical abuse amplifies attention to threat and increases anxiety in children. *Emotion, 7*, 838–852.

Tottenham, N., Tanaka, J. W., Leon, A. C., McCarry, T., Nurse, M., Hare, T. A., et al. (2008). The NimStim set of facial expressions: Judgments from untrained research participants. *Psychiatry Research, 168*, 242–249.

Uher, R., & McGuffin, P. (2008). The moderation by the serotonin transporter gene of environmental adversity in the aetiology of mental illness: Review and methodology analysis. *Molecular Psychiatry, 13*, 131–146.

Widom, C. S., Czaja, S. J., & Dutton, M. A. (2008). Childhood victimization and lifetime revictimization. *Child Abuse and Neglect, 32*, 785–796.

Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders*. Oxford, England: Wiley.