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BRIEF REPORT

Children's *5-HTTLPR* genotype moderates the link between maternal criticism and attentional biases specifically for facial displays of anger

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Theorists have proposed that negative experiences in childhood may contribute to the development of experience-specific information-processing biases, including attentional biases. There are also clear genetic influences on cognitive processes, with evidence that polymorphisms in specific candidate genes may moderate the impact of environmental stress on attentional biases (e.g., a functional polymorphism in the serotonin transporter gene; *5-HTTLPR*). In the current study, we tested a gene × environment (G × E) model of risk for attentional biases. We hypothesised that children whose mothers exhibit high levels of expressed emotion criticism (EE-Crit) would display attentional biases specifically for angry, but not happy or sad, faces, and that this link would be stronger among children carrying one or two copies of the *5-HTTLPR* short allele than among those homozygous for the long allele. Results generally supported these hypotheses, though we found that carriers of the *5-HTTLPR* short allele who also had a critical mother exhibited attentional avoidance of angry faces rather than preferential attention.

Keywords: Attentional bias; *5-HTTLPR*; Expressed emotion; Criticism; G × E.

The ability to accurately process facial displays of emotion is essential to adaptive interpersonal functioning (Alley, 1988; Marsh, Adams, & Kleck, 2005) and biased attention to facial displays of different emotions has been linked to various forms of psychopathology (Bar-Haim,

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Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Dodge & Pettit, 2003; Joormann, 2009; Mathews & MacLeod, 2005). Given this, researchers have begun to explore potential developmental origins of these attentional biases.

Theorists have proposed that negative experiences in childhood may contribute to the development of information-processing biases specific to those experiences (i.e., experience-specific information-processing biases; see Cicchetti, Toth, & Maughan, 2000; Pollak, 2003; Rose & Abramson, 1992), including biases in attention, interpretation, and memory for affectively salient stimuli. For example, theorists have suggested that childhood physical abuse may contribute to the development of information-processing biases specifically to facial displays of anger (see Pollak, 2003). It may be adaptive for children to develop increased sensitivity to signals of anger, as this may facilitate attempts to avoid the abuse (Cicchetti et al., 2000; Pollak, 2003). Consistent with these hypotheses, studies have suggested that children with a history of physical abuse do exhibit attentional biases for angry faces (e.g., Pine et al., 2005; Pollak & Tolley-Schell, 2003). Also, consistent with the idea of experience-specific information-processing biases, attentional biases among physically abused children appear to be specific to angry faces rather than other emotions (e.g., sad or happy). Although results are somewhat mixed as to whether a history of abuse is related to preferential attention to (Pollak & Tolley-Schell, 2003) versus attentional avoidance of (see Pine et al., 2005) angry faces, Pollak and his colleagues have suggested, based on both behavioural and event-related potential (ERP) data, that a history of physical abuse is related to difficulty disengaging attention from angry faces (see Pollak, 2003, for a review).

One question that remains unclear from this research is whether similar attentional biases would be observed for other, less severe, forms of negative parental behaviour. Specifically, to the extent that the attentional biases observed among abused children resulted from conditioning to the angry expressions of the abuser (cf. Lee, Lim, Lee, Kim, & Choi, 2009; Pischek-Simpson, Boschen, Neumann, & Waters, 2009), which then generalised to facial displays of anger from other individuals, one would predict that exposure to potentially milder forms of anger expression should also contribute to the development of these attentional biases. In the current study, therefore, we focused on the potential role of maternal criticism.

Although previous research has supported the link between maternal criticism and children's negative cognitions (e.g., Jaenicke, Hammen, Zupan, & Hiroto, 1987; Murray, Woolgar, Cooper, & Hipwell, 2001), a limitation of these studies is that they focus on cognitive content rather than cognitive processes. In addition, these studies have relied upon participants' awareness of their cognitions, many of which are hypothesised to operate outside of one's awareness (Gotlib & Neubauer, 2000). It remains unclear, therefore, whether maternal criticism would also be related to measures of cognitive processes such as attentional biases. Another limitation of much of the work examining the impact of verbal victimisation more generally on children's cognitions (e.g., Gibb & Abela, 2008; Gibb et al., 2006) is that it has relied on children's reports of their experiences, which may be subject to recall or response bias. Seeking to address these limitations in the current study, we focused on interviewer-coded ratings of maternal criticism assessed using the Five Minute Speech Sample (FMSS; Magaña, Goldstein, Karno, & Miklowitz, 1986). For the FMSS, mothers are asked to speak about their child for five minutes and these comments are then coded for levels of criticism. Supporting the validity of expressed emotion-criticism (EE-Crit) ratings, previous research has shown that levels of EE-Crit coded from the FMSS are significantly related to levels of criticism and anger coded by independent raters observing parent–child interactions (Hermanns, Florin, Dietrich, Rieger, & Hahlweg, 1989; McCarty, Lau, Valeri, & Weisz, 2004). In the current study, we predicted that the relation previously observed between physical abuse and children's attentional biases for angry faces would generalise to experiences of maternal criticism. Specifically, we predicted that children

of mothers displaying high levels of EE-Crit would exhibit preferential attention specifically for angry, but not sad or happy, facial expressions.

In addition to potential environmental influences, there is growing interest in identifying specific genetic influences on cognitive processes (e.g., Beck, 2008; Bellgrove & Mattingley, 2008; Bishop, 2008; Canli, 2008). Genetic influences could be observed in terms of main effects, in which specific candidate polymorphisms are directly associated with the presence of attentional biases. These main effects could be mediated by environmental influences, reflecting a gene–environment correlation (rGE). For example, children at genetic risk may be exposed to more environmental stress (e.g., EE-Crit) that is either independent of (passive rGE) or dependent on (active or evocative rGE) the child's influence, which then contributes to the development of attentional biases. Genetic influences could be also observed in terms of gene–environment interactions ($G \times E$), in which certain genetic risk factors increase the impact of environmental stressors ($G \times E$). For example, experiences such as maternal criticism may be more likely to contribute to the development of attentional biases for angry faces among children carrying specific "high risk" genotypes.

In examining genetic influences on attentional biases, researchers have focused on genes known to influence functioning in brain regions implicated in these biases. There is growing evidence for the role of the amygdala and prefrontal cortex in attentional biases for threatening information (see Bishop, 2008, for a review). Specifically, these attentional biases appear to be driven by heightened amygdala reactivity to threatening stimuli, coupled with decreased activation in specific subregions of the prefrontal cortex (e.g., rostral anterior cingulate cortex and lateral prefrontal cortex). Although a number of genes are likely to modulate activity in these regions, the strongest evidence to date for a specific candidate gene has been obtained for a putatively functional polymorphism in the serotonin transporter gene (*5-HTTLPR*). There are two common variants in *5-HTTLPR*, a short allele (s) and a long allele (l), and carriers of the short allele have been shown to exhibit stronger amygdala reactivity to emotional stimuli (see Munafò, Brown, & Hariri, 2008, for a review) and decreased functional connectivity between the amygdala and subregions of the prefrontal cortex (Pacheco et al., 2009; Pezawas et al., 2005).

Based on these findings, researchers have recently begun examining the link between *5-HTTLPR* genotype and attentional biases. This research has supported the hypothesis that carriers of the *5-HTTLPR* short allele exhibit attentional biases for emotional stimuli (Beevers, Gibb, McGeary, & Miller, 2007; Beevers, Wells, Ellis, & McGeary, 2009; Osinsky et al., 2008; Pérez-Edgar et al., 2010; but see also Fox, Ridgewell, & Ashwin, 2009), which may be due to difficulty disengaging attention from these stimuli (Beevers et al., 2009; Osinsky et al., 2008). Importantly, however, there is initial evidence that *5-HTTLPR* genotype may be associated with attentional biases for facial expressions of emotion generally rather than any specific emotion type (Beevers et al., 2009), mirroring the results obtained for *5-HTTLPR* in fMRI studies of amygdala reactivity (e.g., Dannlowski et al., 2007; see also Munafò et al., 2008). Therefore, *5-HTTLPR* genotype may increase risk for the presence of attentional biases to a broad range of emotionally salient stimuli and we predict that the specific focus of the attentional biases exhibited among carriers of the *5-HTTLPR* short allele (e.g., attentional biases to angry vs. sad faces) will depend on the specific type of environmental stress encountered (cf. Fox, Hane, & Pine, 2007; Kalin et al., 2008). More specifically, integrating these findings with those reviewed above regarding environmental influences, we predict that *5-HTTLPR* genotype may heighten neurobiological reactivity to a broad range of environmental cues, strengthening the salience and subjective impact of these experiences, thereby magnifying associative learning and increasing the risk of developing experience-specific attentional biases.

In an initial test of this hypothesis, we recently examined the link between maternal history of

depression and children's attentional biases for facial expressions of emotion as a function of children's *5-HTTLPR* genotype (Gibb, Benas, Grassia, & McGeary, 2009a).

Consistent with our hypothesis, we found that children of mothers with a history of major depression during their children's lives exhibited attentional biases for sad faces, with some evidence that this relation was stronger among children carrying the *5-HTTLPR* short allele. Importantly, children's attentional biases were specific to sad, rather than happy or angry, faces, supporting the hypothesis that salient environmental stressors may contribute to the development of experience-specific attentional biases, particularly among children carrying the *5-HTTLPR* short allele.

Our goal in the current study was to extend previous research in two ways. First, we examined whether the relation previously reported between physical abuse and children's attentional biases for angry faces would generalise to experiences of maternal criticism (EE-Crit). We predicted that children of critical mothers would exhibit preferential attention specifically for angry, but not happy or sad, faces. Second, testing a gene × environment (G × E) model of risk, we hypothesised that this relation would be stronger among children carrying the *5-HTTLPR* short allele than among those homozygous for the long allele. Third, we predicted that the results would be maintained even when statistically controlling for the potential influence of child or mother psychopathology.

METHOD

Participants

Participants in this study were a subset of 74 mother–child pairs drawn from the community participating in a larger study of the intergenerational transmission of depression. Mothers were required to either meet criteria for a DSM-IV (American Psychiatric Association, 1994) major depressive disorder (MDD) during the child's lifetime ($n = 40$) or have no lifetime diagnosis of any DSM-IV mood disorder ($n = 34$). Exclusion criteria for both groups included symptoms of schizophrenia, organic mental disorder, alcohol or substance abuse within the last six months, or history of bipolar I disorder. Children's participation was limited such that no more than one child per mother could participate. The only inclusion criterion for children was that they be 8–12 years old. If more than one child was available within this age range, one child was chosen at random for participation. The average age of mothers in our sample was 39.04 years ($SD = 6.92$, Range = 26–53). In terms of race, 89.2% of the mothers were Caucasian, 5.4% were African American, 4.1% were Asian American, and 1 mother (1.3%) was multiracial. The median annual family income was $50,000–55,000. For the children in our sample, the average age was 9.96 years ($SD = 1.27$, Range = 8–12) and 51.4% were girls. In terms of race, 79.7% of the children were Caucasian, 6.8% were African American, 1.4% were Asian American, and 12.2% were multiracial. Maternal history of MDD was not significantly related to children's age, sex, or race (Caucasian vs. non-Caucasian).

Measures

The Schedule for Affective Disorders and Schizophrenia – Lifetime Version (SADS-L; Endicott & Spitzer, 1978) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-PL; Kaufman, Birmaher, Brent, & Rao, 1997) were used to assess for current DSM-IV Axis I disorders in mothers and their children, respectively. Both measures are widely used diagnostic interviews with well-established psychometric properties (Angold, 1989; Endicott & Spitzer, 1978; Kaufman et al., 1997). The SADS-L and K-SADS-PL were administered by separate interviewers. For the K-SADS-PL, mothers and children were interviewed separately. As noted above, 40 mothers met criteria for at least one episode of MDD during their child's lifetime (6 of whom met criteria for current MDD). Eight mothers met criteria for one or more current anxiety disorders (social phobia = 6; post-traumatic stress disorder = 2; generalised anxiety disorder = 4). In terms of children's current diagnoses, 3 met criteria

for MDD, 8 children met criteria for an anxiety disorder (separation anxiety disorder = 1; social phobia = 5; obsessive-compulsive disorder = 1; generalised anxiety disorder = 1), and 6 children met criteria for one or more behaviour disorders (attention deficit hyperactivity disorder, ADHD = 5; oppositional defiant disorder = 2). In terms of comorbidity, three children met criteria for current comorbid behaviour and anxiety disorders and one child met criteria for current comorbid MDD and ADHD. A subset of 20 SADS-L and 20 K-SADS-PL interviews from this project were coded by a second interviewer and kappa coefficients for current diagnoses were: mother MDD ($\kappa = 1.00$), mother any anxiety disorder ($\kappa = 0.83$), child MDD ($\kappa = 1.00$), child any anxiety disorder ($\kappa = 0.83$), and child any behaviour disorder ($\kappa = 1.00$).[1]

Mothers' and children's symptoms of depression were assessed using the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) and Children's Depression Inventory (CDI; Kovacs, 1981), respectively. Numerous studies have supported the reliability and validity of both measures (e.g., Beck et al., 1996; Kovacs, 1981; Smucker, Craighead, Craighead, & Green, 1986). In the current study, both the BDI-II and the CDI exhibited good internal consistency (αs = .93 and .85, respectively).

Mothers' and children's symptoms of anxiety were assessed using the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) and the Revised version of the Screen for Child Anxiety Related Emotional Disorders (SCARED-R; Muris & Steerneman, 2001), respectively. Previous studies have supported the psychometric properties of both questionnaires (e.g., Beck et al., 1988; Creamer, Foran, & Bell, 1995; Muris & Steerneman, 2001; Muris, Dreessen, Bögels, Weckx, & van Melick, 2004). In the current study, both the BAI and the SCARED-R exhibited good internal consistency (αs = .90 and .94, respectively).

The externalising subscale of the Child Behaviour Checklist for Ages 6–18 (CBCL; Achenbach & Rescorla, 2001) was used to assess mothers' reports of children's externalising symptoms. The CBCL externalising subscale exhibits good reliability and validity (see Achenbach & Rescorla, 2001) and it exhibited good internal consistency in the current study ($\alpha = .91$).

The Five Minute Speech Sample (FMSS; Magaña et al., 1986) was used to assess mothers' levels of Expressed Emotion-Criticism (EE-Crit). In administering the FMSS, the mother is asked to speak uninterrupted for five minutes about her child. The response is audiotaped and coded for EE-Crit. Mothers are rated as high on EE-Crit if any of the following three criteria are met: their initial statement about the child is negative, they report a negative relationship, or they report one or more criticisms as defined by the FMSS coding system. Mothers are rated as borderline critical if they express dissatisfaction with the child not severe enough to be rated as a criticism. Responses to the FMSS are coded to indicate whether they reflect high, borderline, or low EE-Crit. A number of studies have supported the reliability and validity of the FMSS EE-Crit subscale (e.g., Asarnow, Tompson, Woo, & Cantwell, 2001; Magaña et al., 1986; McCarty et al., 2004; Rogosch, Cicchetti, & Toth, 2004). Levels of EE-Crit coded from the FMSS also exhibit good convergent validity with levels of criticism and anger coded by independent raters observing parent–child interactions (Hermanns et al., 1989; McCarty et al., 2004). In this study, the FMSS was administered and coded by individuals blind to the other study variables. Coders were trained to reliability standards by the creator of the FMSS (Ana

[1] We should also note that, as part of the K-SADS-PL interview, children's history of physical and sexual abuse was assessed. A past history of physical abuse was reported for 5 children and a past history of sexual abuse was reported for 7 children (a history of both physical and sexual abuse was reported for 1 child). Reports of children's abuse history (alone or interacting with *5-HTTLPR* genotype) were not significantly related to children's attentional biases for angry, happy, or sad faces (lowest $p = .16$) and, importantly, all of the significant relations reported in this study were maintained even after statistically controlling for reports of childhood abuse. The discrepancy between these results and those of previous studies (e.g., Pine et al., 2005; Pollak & Tolley-Schell, 2003) may have been due to the low base rate of abuse in this sample or to the fact that we focused on mothers' reports rather than documented cases of abuse.

Magaña-Amato). All samples were coded by two raters. When discrepancies arose, a third rater was consulted and a consensus rating was reached. In this study, dichotomous classifications were created to indicate whether mothers were categorised as high EE-Crit ($n = 14$, 18.9%) or borderline or low EE-Crit ($n = 60$; 81.1%). A subsample of 20 FMSSs was also coded by the creator of the FMSS (Ana Magaña-Amato) and inter-rater agreement for the dichotomous EE-Crit classifications was excellent ($\kappa = 1.00$).

Children's attentional biases for facial displays of emotion were assessed using a modified dot-probe task (cf. MacLeod, Mathews, & Tata, 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 (sad, happy, or angry) and one neutral photograph from the same actor taken from a standardised stimulus set (Tottenham et al., 2009). 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 in each of 2 blocks, with a rest in between blocks (192 trials total). Stimuli were presented for 1000 ms, followed by a dot replacing one of the pictures. Following 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 inter-trial interval was 1000 ms. Trials with response errors were excluded (2.63%) as were trials with response times less than 150 ms or greater than 1500 ms (2.38%). Mean bias scores (Mogg, Bradley, & Williams, 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, children and their mothers 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, Stanier, & Williamson, 1988). The *5-HTTLPR* alleles were assayed using previously reported methods (Pooley, Houston, Hawton, & Harrison, 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′. Children and mothers were classified according to their *5-HTTLPR* genotype: those with either 1 or 2 copies of the short allele (ss/sl; $n = 46$ children; $n = 46$ mothers) and those homozygous for the long allele (ll; $n = 28$ children; $n = 28$ mothers). These frequencies did not differ from Hardy–Weinberg Equilibrium. We should note that although the number of children and mothers in each genotype group was identical, not every child's genotype matched that of their mother. Specifically, of the 46 children carrying the ss/sl genotype, 36 had mothers who also carried the ss/sl genotype while 10 carried the ll genotype. Similarly, of the 28 children carrying the ll genotype, 18 had mothers who carried the ll genotype while 10 had mothers who carried the ss/sl genotype.

Procedure

Potential participants were recruited from the community through a variety of means (e.g., newspaper ads, bus ads, and flyers). Mothers responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Those reporting either significant depressive symptoms during the child's life or no significant lifetime symptoms of depression were invited to participate in the study. Upon arrival at the laboratory, mothers

were asked to provide informed consent and children were asked to provide assent to be in the study. Next, the mother was administered the FMSS and K-SADS-PL interview. During this time, the child completed questionnaires, including the CDI and SCARED-R, as well as the attentional bias and DNA assessments in a separate room. After completing the K-SADS-PL with the mother, the same interviewer then administered the K-SADS-PL to the child. While the child was being administered the K-SADS-PL, the mother completed the DNA assessment, a series of questionnaires including the BDI-II, BAI, and CBCL, and was then administered the SADS-L by a separate interviewer. Participation in this initial assessment took approximately 3 hours, which included frequent breaks for children to minimise fatigue effects. Families were compensated $50 for their participation.

RESULTS

Descriptive statistics for the study variables are presented in Table 1. Preliminary analyses were also conducted to determine whether any of the study variables were significantly related to children's age or sex. The only significant finding was that girls were more likely than boys to possess the *5-HTTLPR* ll genotype, $\chi^2(1, N = 74) = 4.91, p = .03$, $r_{\text{effect size}} = .26$, though we should note that gender differences for *5-HTTLPR* genotype have not been observed in previous studies (e.g., Caspi et al., 2003). We also examined whether children's age or sex moderated any of the relations examined in this study; however, none of these analyses was significant. Therefore, all analyses were conducted collapsing across sex and age groups. As a means of investigating potential rGE effects, we examined whether children's or mothers' *5-HTTLPR* genotype was related to levels of EE-Crit and neither of

Table 1. *Descriptive statistics*

	Depressed moms (n = 40)	*Non-depressed moms (n = 34)*	$r_{\text{effect size}}$
Mom age	38.68 (6.73)	39.47 (7.23)	−.06
Mom race (% Caucasian)	82.50	97.06	−.23*
Child age (years)	10.05 (1.32)	9.85 (1.21)	.08
Child sex (% female)	52.50	50.00	.03
Child race (% Caucasian)	72.50	88.24	−.20
Mom current MDD (% yes)	15.00	0.00	.27*
Mom anxiety disorder (% yes)	40.00	0.00	.32**
Child MDD (% yes)	7.50	0.00	.19
Child anxiety disorder (% yes)	20.00	0.00	.32**
Child behaviour disorder (% yes)	15.00	0.00	.27*
BDI-II	13.55 (9.60)	2.79 (2.77)	.64**
BAI	9.46 (8.18)	2.91 (2.48)	.50**
CBCL-Externalising	9.00 (8.67)	4.00 (4.37)	.31**
CDI	8.76 (6.73)	5.22 (5.21)	.29*
SCARED-R	42.48 (21.47)	36.37 (15.86)	.17
Child *5-HTTLPR* (% ss/sl)	55.00	70.59	−.16
Mother *5-HTTLPR* (% ss/sl)	60.00	64.71	−.05
EE-Crit (% high criticism)	22.50	14.71	.10
Attent. bias: Angry faces (ms)	11.61 (48.70)	1.40 (56.66)	.10
Attent. bias: Happy faces (ms)	4.47 (57.92)	−6.78 (50.34)	.10
Attent. bias: Sad faces (ms)	−10.64 (44.45)	16.90 (46.53)	−.29*

Notes: MDD = Major Depressive Disorder. BDI-II = Beck Depression Inventory-II. BAI = Beck Anxiety Inventory. CBCL-Externalising = Child Behaviour Checklist – Externalising subscale. CDI = Children's Depression Inventory. SCARED-R = Revised Screen for Child Anxiety Related Emotional Disorders. *5-HTTLPR* = functional polymorphism in the serotonin transporter gene. EE-Crit = Expressed Emotion-Criticism. *$p < .05$; **$p < .01$.

these analyses was significant. Children's or mothers' *5-HTTLPR* genotype was also not significantly related to any of the symptom or diagnostic variables.

Focusing next on our primary analyses, we tested the hypothesis that children's *5-HTTLPR* genotype would moderate the link between EE-Crit and children's attentional biases specifically for angry faces. We conducted a 2 (Child *5-HTTLPR* Genotype: ss/sl, ll) × 2 (EE-Crit: high, low) × 3 (Facial Expression: angry, happy, sad) repeated-measures analysis of variance (ANOVA) with attentional bias scores serving as the dependent variable. Because participants were chosen based on mothers' history of MDD, mother MDD history (yes, no) was included as an additional between-subjects factor in these analyses. None of the main effects in this analysis were significant, including the main effects of Child *5-HTTLPR* Genotype, $F(1, 66) = 1.29$, $p = .26$, $\eta_p^2 = .02$, or EE-Crit, $F(1, 66) = 0.37$, $p = .55$, $\eta_p^2 = .01$. Of the two-way interactions, the only significant finding was the Mom MDD × Facial Expression interaction reported previously (Gibb et al., 2009a), which was maintained in this analysis, $F(2, 132) = 3.04$, $p = .05$, $\eta_p^2 = .04$.[2] Importantly, the three-way Child *5-HTTLPR* × EE-Crit × Facial Expression interaction was significant, $F(2, 132) = 4.57$, $p = .01$, $\eta_p^2 = .07$. None of the other three-way interactions was significant, nor was the four-way Mom MDD × Child *5-HTTLPR* × EE-Crit × Facial Expression interaction, $F(2, 132) = 1.49$, $p = .23$, $\eta_p^2 = .02$. Exploring the form of the significant Child *5-HTTLPR* × EE-Crit × Facial Expression interaction, we conducted analyses separately in the two child *5-HTTLPR* genotype groups. Mother MDD history was included as a covariate in each of these follow-up analyses to statistically control for its effects. The EE-Crit × Facial Expression interaction was significant among children carrying the *5-HTTLPR* short allele, $F(2, 86) = 3.04$, $p = .05$, $\eta_p^2 = .07$, but not among those homozygous for the long allele, $F(2, 50) = 2.05$, $p = .14$, $\eta_p^2 = .08$.[3] Among children carrying the *5-HTTLPR* short allele, tests of simple main effects within Facial Expression type revealed significant EE-Crit differences for angry faces, $F(1, 43) = 6.67$, $p = .01$, $\eta_p^2 = .13$, but not happy, $F(1, 43) = 1.08$, $p = .30$, $\eta_p^2 = .03$, or sad, $F(1, 43) = 0.00$, $p = .96$, $\eta_p^2 = .00$, faces. Next, we conducted two one-sample *t*-tests to determine whether the two EE-Crit groups' bias scores for angry faces differed significantly from zero. This test was significant among *5-HTTLPR* short allele carriers who experienced high levels of EE-Crit, $t(5) = -2.52$, $p = .05$, indicating a significant attentional bias away from angry faces. In contrast, the attentional bias scores for angry faces did not differ significantly from zero among *5-HTTLPR* short allele carriers who experienced low levels of EE-Crit, $t(39) = 1.28$, $p = .21$. As hypothesised, therefore, high levels of maternal criticism were related to children's attentional biases specifically for angry faces, but only among children carrying the *5-HTTLPR* short allele. Contrary to hypothesis, however, the bias reflected attentional avoidance of angry faces rather than preferential attention toward angry faces (see Figure 1).

A series of analyses was then conducted to test the robustness and specificity of these results. First,

[2] As detailed in our previous article (Gibb et al., 2009a), this interaction was driven by maternal depression group differences in attentional biases for sad, but not happy or angry faces, such that children of mothers with a history of MDD, compared to control children, exhibited attentional avoidance specifically of sad faces.

[3] The similarity in effect size (η_p^2) in these analyses across the two genotype groups suggests that the non-significant result among children 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 EE-Crit group differences in attentional biases for the three facial expressions among these children. Among children homozygous for the *5-HTTLPR* long allele, tests of simple main effects within Facial Expression type revealed no significant EE-Crit differences for happy, $F(1, 25) = 0.47$, $p = .50$, $\eta_p^2 = .02$, or sad, $F(1, 25) = 0.07$, $p = .80$, $\eta_p^2 = .00$, faces, though results approached significance for angry faces, $F(1, 25) = 3.46$, $p = .08$, $\eta_p^2 = .12$, with children of critical mothers showing a tendency to exhibit greater vigilance of angry faces than children of non-critical mothers. Although not meeting formal criteria for significance, the current results suggest that these relations may warrant further investigation.

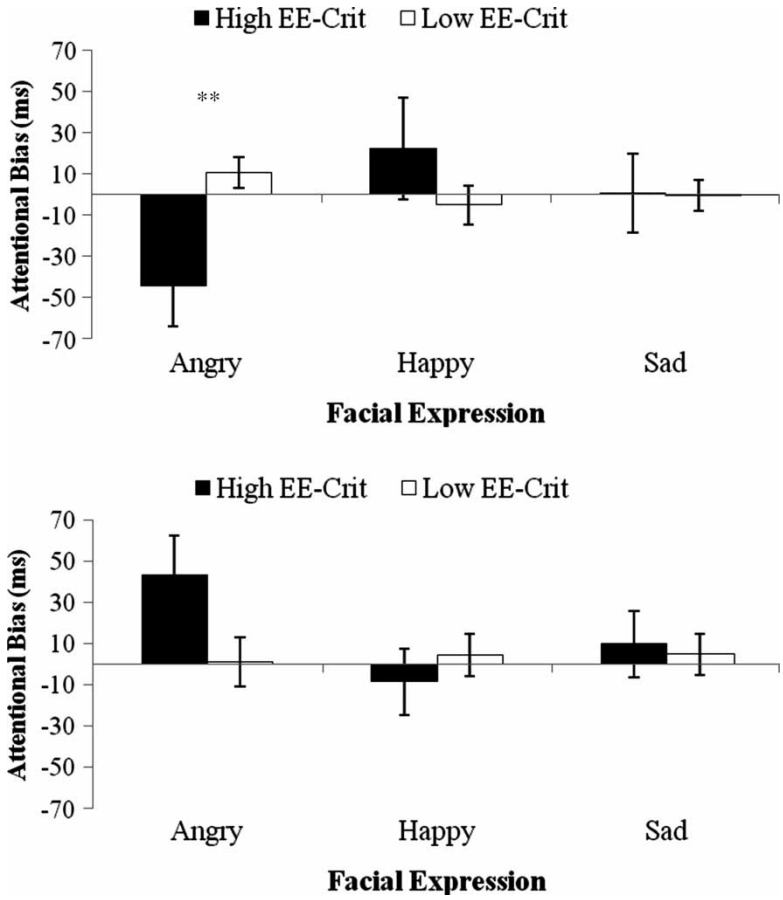

Figure 1. *Children's mean attentional bias scores (in milliseconds) across the three facial expression types as a function of maternal criticism (EE-Crit) among carriers of the 5-HTTLPR short allele (top panel) versus children homozygous for the 5-HTTLPR long allele (bottom panel). Error bars represent one standard error.* $^{**}p = .01$.

as noted above, neither children's nor mother's *5-HTTLPR* genotype was significantly related to levels of EE-Crit, suggesting that the effects are not due to rGE with *5-HTTLPR*. Second, to determine whether any significant G × E effects were better accounted for by mothers' *5-HTTLPR* genotype rather than children's, we re-conducted the analyses focusing on mother genotype, rather than child genotype. None of the effects including mother *5-HTTLPR* genotype was significant. Third, we examined whether effects based on children's genotype would be maintained even after statistically controlling for the influence of mother genotype, which is a very conservative test since child genotype is based, in part, on mother genotype. All of the significant relations were maintained. Fourth, we examined whether the observed relation between EE-Crit and attentional avoidance of angry faces among children carrying 1 or 2 copies of the *5-HTTLPR* short allele was due to children's or mothers' current diagnoses or symptom levels. In two separate analyses of covariance (ANCOVAs), we examined the relation between EE-Crit and children's attentional biases for angry faces among children carrying at least one copy of the *5-HTTLPR* short allele statistically controlling for (i) children's and mothers' current diagnoses of MDD, any anxiety

disorder, or any behaviour disorder (children only), and (ii) children's and mothers' current symptoms of depression, anxiety, and externalising disorders (children only). In each of these analyses, the relation between EE-Crit and attentional biases for angry faces among *5-HTTLPR* ss/sl children was maintained (both $ps < .05$), supporting the robustness of this finding.[4]

DISCUSSION

The results of this study add to a growing body of research suggesting that negative experiences in childhood may contribute to the development of experience-specific information-processing biases (e.g., attentional biases for angry faces; see Pollak, 2003). Further, they suggest that these effects may not be limited to childhood physical abuse, but may also be evident for other forms of negative parental behaviour (i.e., maternal criticism). The current results also extend previous research demonstrating the role of *5-HTTLPR* genotype in moderating the impact of salient environmental stressors (see Caspi, Hariri, Holmes, Uher, & Moffitt, 2010, for a review). Specifically, we found that maternal criticism (EE-Crit) was related to children's attentional biases specifically for angry, but not happy or sad, faces. This relation was observed among children carrying at least one copy of the *5-HTTLPR* short allele, but not among those homozygous for the long allele. If anything, carriers of the *5-HTTLPR* short allele versus children homozygous for the long allele exhibited opposite patterns of attention to angry faces. Importantly, these results did not seem to be due to rGE effects of child or mother *5-HTTLPR* genotype on levels of EE-Crit; children's current symptoms or diagnoses of depressive, anxiety, or externalising disorders; or mothers' current symptoms or diagnoses of MDD or anxiety disorders.

Although the exact mechanisms by which *5-HTTLPR* genotype influences reactivity to environmental stressors remain unknown, research has suggested that carriers of the *5-HTTLPR* short allele exhibit stronger amygdala reactivity to facial expressions of emotion (Munafò et al., 2008) and greater cortisol reactivity to stress (Gotlib, Joormann, Minor, & Hallmayer, 2008). Therefore, the presence of the *5-HTTLPR* short allele appears to be related to stronger neurobiological reactivity to salient environmental stressors. Importantly, the presence of the *5-HTTLPR* short allele may be a relatively non-specific risk factor (or marker of plasticity; see Belsky et al., 2009) in that it is associated with heightened amygdala reactivity, as well as attentional biases, to a variety of affective stimuli (see Beevers et al., 2009; Dannlowski et al., 2007). In contrast, there is increasing evidence for experience-specific attentional biases in children (Pollak, 2003) such that there is a specific match between the type of environmental stressor encountered (e.g., exposure to angry faces via physical abuse or maternal criticism) and attentional biases for specific facial displays of emotion (e.g., anger). Therefore, whereas *5-HTTLPR* genotype may increase risk for the development of attentional biases broadly, the specific focus of the attentional bias exhibited among carriers of the *5-HTTLPR* short allele appears to be specific to the types of negative life events experienced.

We previously reported that children of mothers with a history of MDD during the children's lives exhibited attentional biases specifically for sad faces, with some evidence that this link was stronger among children carrying the *5-HTTLPR* short allele (Gibb et al., 2009a). In the current study, children of highly critical mothers who also carried one or two copies of the *5-HTTLPR* short allele exhibited attentional biases specifically for angry faces, a finding that appeared to be independent of mother and child psychopathology. Based on these findings, we would predict that children of mothers who

[4] Given potential concerns regarding population stratification, we should note that the relation between EE-Crit and attentional biases for angry faces among carriers of the *5-HTTLPR* short allele was maintained in analyses even when limiting the sample to Caucasian children.

exhibit high levels of both criticism and depression would exhibit attentional biases to both angry and sad faces. Although we did not find support for this hypothesis in the current study, the non-significant finding could have been due, in part, to the relatively small size of our sample. Future research with larger samples should continue to examine potential experience-specific information-processing biases to determine whether the presence of multiple forms of negative events in children's lives (e.g., maternal criticism and depression) may contribute to the development of attentional biases for multiple forms of affective stimuli. Given that different forms of psychopathology appear to be related to attentional biases for different types of affective stimuli (e.g., individuals with social phobia display attentional biases specifically for angry faces, while depressed individuals exhibit attentional biases specifically for sad faces; Garner, Mogg, & Bradley, 2006; Gotlib et al., 2004a; Gotlib, Krasnoperova, Yue, & Joormann, 2004b; Joormann & Gotlib, 2007; Mogg, Philippot, & Bradley, 2004), this line of research may ultimately contribute to a better understanding of developmental antecedents to the onset of comorbid disorders.

Although the current results are consistent with the hypothesis that maternal criticism contributes to the development of attentional biases for angry faces among children carrying the *5-HTTLPR* short allele, no causal conclusions can be drawn given the study's cross-sectional design. Key questions for future research, therefore, are precisely how and when these attentional biases may develop. In terms of *how*, there is a growing body of basic research suggesting that attentional biases for specific affective displays of emotion may develop through associative learning processes—repeated pairing of a specific stimulus (e.g., facial display of a specific emotion) with an aversive unconditioned stimulus (e.g., negative emotions such as fear in children; cf. Pischek-Simpson et al., 2009). Based on this, one would expect that repeated exposure to facial displays of any type of emotion (or potentially a single exposure if aversive enough) that causes negative affect in the child would contribute to the development of attentional biases specific to facial displays of that emotion. Initially, this attentional bias should be exhibited only for facial displays of the emotion in the person toward whom the conditioning developed. Over time, however, the child's attentional bias may generalise to other contexts (e.g., displays of the same emotion in other people) and stabilise into a relatively stable "style" of attentional processing. More longitudinal research is clearly needed to test this hypothesis and also to determine what factors may moderate the generalisation of these attentional biases across contexts.

Also in terms of how attentional biases may develop, research has demonstrated that associative learning of aversive stimuli is mediated by amygdala activation (Rodrigues, LeDoux, & Sapolsky, 2009), which is also a key brain region underlying attentional biases for emotional stimuli (Bishop, 2008). In the search for genetic risk factors that may moderate the impact of environmental experiences on the development of attentional biases, therefore, the most promising candidate polymorphisms may be those known to influence amygdala reactivity to affective stimuli. In the current study, we chose to focus specifically on *5-HTTLPR* for a variety of reasons. Specifically, short allele carriers, compared to those homozygous for the long allele, exhibit heightened amygdala reactivity to emotional stimuli (see Munafò et al., 2008, for a review), decreased functional connectivity between amygdala and prefrontal cortex (Pacheco et al., 2009; Pezawas et al., 2005), which is also involved in the modulation of attention (Bishop, 2008), heightened cortisol reactivity to environmental stress (Gotlib et al., 2008), and attentional biases for emotional stimuli (Beevers et al., 2007, 2009; Osinsky et al., 2008; Pérez-Edgar et al., 2010). Therefore, *5-HTTLPR* appeared to be the most promising candidate polymorphism for our initial examination of a G × E model of risk for attentional biases in children. This said, however, genetic influences are unlikely to be limited to any single gene. In addition to examining other candidate polymorphisms shown to impact amygdala reactivity, researchers may wish to

examine genes shown to affect hypothalamic pituitary axis (HPA) reactivity, given the impact of environmental stress on attention regulation (Liston, McEwen, & Casey, 2009) and the influence of stress hormones in fear conditioning (Rodrigues et al., 2009). Future studies, therefore, would benefit from the examination of other genes that have been shown to moderate reactivity to environmental stress including polymorphisms in the corticotrophin-releasing hormone receptor gene (*CHRH1*; e.g., Bradley et al., 2008) and the brain-derived neurotrophic factor gene (*BDNF*; e.g., Kaufman et al., 2006), as well as genes linked to basic attentional processes such as the dopamine D4 receptor gene (*DRD4*; see Bellgrove & Mattingley, 2008). Research is needed to determine whether polymorphisms in these genes, alone or interacting with *5-HTTLPR*, also moderate the link between negative events and children's information-processing biases.

Another important question is, *when* do attentional biases develop? And, related to this, are there critical windows of heightened risk for children in response to specific environmental stressors? To our knowledge, there are no published studies examining environmental influences on prospective changes in attentional biases. However, research shows that even infants may exhibit attentional biases to facial displays of emotion. For example, there is evidence that infants of depressed mothers spend less time looking at their mothers than do infants of non-depressed mothers (e.g., Boyd, Zayas, & McKee, 2006). Indeed, in our earlier study (Gibb et al., 2009a), we found that the degree of children's attentional avoidance of sad faces was significantly correlated with the child's age when his or her mother first experienced a major depressive episode, such that children who were younger when their mother first became depressed exhibited more attentional avoidance of sad faces than children who were older. These results suggest that the effect of environmental stimuli on children's attentional biases may be particularly pronounced early in life. To adequately address this question, multi-wave longitudinal studies are need in which environmental stressors and attentional biases are assessed repeatedly.

We believe that the current results are quite promising and provide preliminary support for a G × E model of risk for attentional biases in which a polymorphism in a specific gene (*5-HTTLPR*) may interact with a specific environmental influence (maternal criticism) to increase risk for attentional biases to a specific type of stimuli (angry faces). However, several caveats to the current findings should be noted. First, as noted above, no causal conclusions can be drawn given the study's cross-sectional design. Future longitudinal research is needed to determine whether maternal criticism does indeed contribute to prospective changes specifically in children's attentional biases for angry faces.

A second caveat regards the nature of our sample. Specifically, children were chosen for inclusion based on their mothers' lifetime histories of major depression (at least one major depressive episode during the child's life vs. no lifetime history of any mood disorder), which may limit the generalisability of our results to other samples. Importantly, we were able to show that the results were maintained even after statistically controlling for the influence of mother and child psychopathology. This said, future research would benefit from either more representative samples or samples that have no lifetime history of psychopathology to rule out this potential confound. Also, our sample size was fairly small. Therefore, although the size of our sample was sufficient to detect the hypothesised relation between maternal criticism and children's attentional biases specifically for angry, but not sad or happy, faces among carriers of the *5-HTTLPR* short allele, we had limited statistical power to detect other effects. For example, although not significant in the current study, it appears that children homozygous for the *5-HTTLPR* long allele may exhibit an opposite pattern of attentional allocation to angry faces in the context of maternal criticism as children carrying one or two copies of the short allele. Similarly, although our classification of *5-HTTLPR* genotype into carriers of the short allele (ss or sl) versus those homozygous for the long allele (ll), in which the short allele is

assumed to have a dominant effect, is consistent with previous research examining links between *5-HTTLPR* and attentional biases (e.g., Beevers et al., 2007; Osinsky et al., 2008), there is some evidence for a linear effect of *5-HTTLPR* genotype on attentional biases in terms of the number of short alleles present (e.g., Pérez-Edgar et al., 2010). In the current study, cell sizes for combinations of each genotype and EE-Crit category were too small to allow these more focused analyses, but they should be examined in future research testing G × E models or risk for attentional biases. Finally, the size of our sample limited our power to detect potential differences in the magnitude of the relations examined based on children's age or sex. Future studies with larger samples are necessary to more definitively examine these relations.

Third, contrary to our hypothesis, we found evidence for attentional avoidance of angry faces rather than preferential attention. Although this is consistent with some previous research among children with a history of physical abuse (Pine et al., 2005), we had designed our dot-probe task based on Pollak's (2003) assertion that physical abuse is associated with difficulty disengaging attention from angry faces rather than attentional avoidance. Specifically, we chose to use a relatively long stimulus presentation duration (1000 ms) in our dot-probe task in an effort to capture prolonged attention/difficulty disengaging attention from angry faces (cf. Mathews & MacLeod, 2005). Indeed, using this same paradigm in a previous study (Gibb, Schofield, & Coles, 2009b), we found that young adults reporting a history of childhood abuse, compared to those reporting no abuse history, exhibited preferential attention specifically for angry faces using a 1000 ms stimulus presentation duration. Although the precise reason for the discrepancy in findings across studies regarding the direction of the attentional biases (preferential attention vs. attentional avoidance) remains unclear, it does not appear to be due to either the stimulus presentation duration or to the age of the participants as both preferential attention and attentional avoidance have been observed in children and at both 500 ms and 1000 ms stimulus presentation durations. However, we should note that a limitation of the dot-probe task is that it only allows a determination of where children were allocating their attention at a specific point in time (e.g., 1000 ms after stimulus onset). Therefore, although we believe the data provide initial evidence for the specificity of maternal criticism to children's attentional biases for angry, rather than happy or sad, faces, future research would benefit from procedures that allow for a more fine-grained analysis of children's patterns of attentional allocation across an entire stimulus trial (e.g., eye-tracking). Future research would also benefit from the inclusion of experimental tasks designed to more specifically assess difficulty disengaging attention from emotional stimuli (cf. Beevers et al., 2009; Pollak & Tolley-Schell, 2003).

Fourth, as noted above, additional research is needed to determine whether the genetic moderation effects observed in this study are unique to *5-HTTLPR* or whether they would also be observed for other candidate polymorphisms. Finally, there is always the possibility in any genetic association study of an unmeasured genetic or non-genetic third variable accounting for the associations reported (e.g., population stratification or linkage disequilibrium between measured variant and actual functional variant). Future studies, therefore, would benefit from the inclusion of a genomic control.

In summary, the current results are consistent with previous research suggesting the presence of experience-specific information-processing biases (Gibb et al., 2009b; Pine et al., 2005; Pollak, 2003). They add to this literature by suggesting that milder forms of parental aggression may also contribute to the development of attentional biases for angry faces. Importantly, they also identify a specific candidate polymorphism (*5-HTTLPR*) that moderates the strength of this relation, in that maternal criticism was significantly related to children's attentional biases for angry faces only among children carrying the *5-HTTLPR* short allele. Future research is needed to determine whether maternal criticism or other forms of negative life events interact with specific candidate polymorphisms such as *5-HTTLPR* to predict the actual development of children's attentional

biases. These studies should also seek to determine whether these influences may occur across the lifespan or whether there are specific developmental windows during which children's attentional processes are particularly malleable and open to the influence of environmental stressors. Research is also needed that will allow a finer-grained analysis of children's attentional allocation patterns (e.g., eye tracking) to more definitively determine which aspects of attention may be dysregulated when these children are confronted with angry faces. Finally, given increasing evidence for the efficacy of computer-based attentional modification programmes in treating anxiety disorders (e.g., Amir, Beard, Burns, & Bomyea, 2009; Amir, Weber, Beard, Taylor, & Bomyea, 2008; Schmidt, Richey, Buckner, & Timpano, 2009), research is needed to determine whether similar approaches could be used for targeted prevention or early intervention programmes among at-risk youth to reduce the likelihood that these attentional biases would contribute to the development of psychopathology later in life.

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