5-HTTLPR moderates the association between attention away from angry faces and prospective depression among youth

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Abstract

Attention bias to emotion has been studied as a risk factor associated with depression. No study has examined whether attention bias within the context of measured genetic risk leads to increased risk for clinical depressive episodes over time. The current study investigated whether genetic risk, as indexed by the serotonin-transporter-linked polymorphic region (5-HTTLPR), moderated the relationship between attention bias to emotional faces and clinical depression onset prospectively across 18-months in a community sample of youth (n = 428; mean age = 11.97, SD = 2.28; 59% girls). Youth who attended away from angry emotional faces and were homozygous for the S allele of the 5-HTTLPR polymorphism were at greater risk for prospective depressive episode onset. The current study's findings highlight the importance of examining risk for depression across multiple levels of analysis and demonstrate attention away from threat as a possible point of intervention related to attention bias modification and depression treatment among youth.

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1. Introduction

Rates of depression increase markedly across adolescence (Hankin et al., 1998) and depression onset in adolescence predicts emotional and behavioral difficulties into adulthood (Rutter et al., 2006). Cognitive theories of depression posit that information processing of affective information (i.e., attention bias to emotion) may contribute to the development, maintenance, and recurrence of depression (Beck, 2008). There is a growing body of literature suggesting certain biologically plausible candidate genes may contribute to the association between attention bias and depression (Gibb et al., 2013). However, previous research has been primarily cross-sectional, so it is unknown whether attention bias to emotion functions as a cause, correlate, or consequence of clinical depression. The current study addressed this gap by examining attention bias to emotional faces and molecular genetic risk as prospective predictors of depression onset among youth.

Previous research demonstrates that currently depressed adults exhibit cognitive biases for negative material (Mathews and Macleod, 2005), and there is evidence supporting similar cognitive theories of depression among youth (Gibb et al., 2013). Many of the theorized underlying cognitive mechanisms (e.g., encoding, attention, memory) are thought to function outside of one's awareness and are best measured by information processing tasks that are less susceptible to reporting biases when compared to self-report measures (Gotlib and Neubauer, 2000). Several cross-sectional studies in adults have utilized computerized attention to emotion tasks, such as the dot-probe task (MacLeod et al., 1986), and shown those diagnosed with current or past major depression demonstrate attentional biases toward negatively valenced stimuli (Gotlib et al., 2004; Joormann and Gotlib, 2007). Specifically, meta-analytic findings among depressed adults show a medium effect size of attention bias to negative stimuli (i.e., sad faces, negatively valenced photos) and a trend toward greater attention bias toward socially threatening stimuli among depressed adults (Peckham et al., 2010). However, far less is known about the association between depression and attention bias among youth.

There have been mixed findings within the youth literature with some studies showing attention away from negative emotion and others finding attention toward negative emotion relates to depression. Cross-sectional studies utilizing similar dot-probe tasks and stimuli presentation times (1,000 ms, Hankin et al., 2010;
1,500 ms, Joormann et al., 2007) found at-risk (e.g., youth whose mothers have a history of depression) and currently depressed youth demonstrated biased attention toward negative emotion, particularly sad faces, as compared to control participants. However, two recent studies examining attention bias to emotion with a more precise eye-tracking methodology found attention away from negative emotion related most strongly with depression. Specifically, a cross-sectional study (Harrison and Gibb, 2014) found currently depressed youth attended away from sadness when passively viewing emotional faces for 20s, while a longitudinal examination of attention bias using a standard dot-probe task (2,000 ms presentation time) found attention away from fearful faces predicted greater depression symptom severity across 2 years (Price et al., 2015).

It is possible the discrepancies within the youth literature stem from variation across tasks (i.e., passive viewing versus dot-probe) and method of assessing bias (i.e., behavioral versus eye-tracking). Alternatively, developmental theory suggests that young children attend away from negative stimuli as a means of emotion regulation (Gross and Thompson, 2007). While adaptive in young childhood, this method of regulation may become maladaptive throughout development leading to greater risk for depression onset. Indeed, research has shown avoidance of engagement-based coping strategies are associated with internalizing psychopathology (Connor-Smith et al., 2000). Although aspects of emotion regulation may play a role in the association between attention bias and depression, there is a need for prospective designs that incorporate theoretically relevant moderators in order to draw temporal inferences regarding the role of attention bias in the development of clinical depression.

The ability to effectively process and regulate emotion may be an important activator in the pathway from attention bias to depression. The serotonin–transporter-linked polymorphic region (5-HTTLPR) in the serotonin transporter gene (SLC6A4) has been of interest when studying attention bias and stress-related psychopathology (Lesch et al., 1996; Sen et al., 2004), including depression (Gibb et al., 2013), given its role in influencing neurotransmitters involved in the processing and regulation of emotion. The serotonin transporter (5-HTT) is a protein critical to the regulation of serotonin function in the brain because it terminates the action of serotonin in the synapse via reuptake. This is a well-studied protein that has a functional number of tandem repeats (VNTR) polymorphism in the promoter region referred to as 5-HTTLPR. Variants of 5-HTTLPR are the long allele (L), consisting of 16 copies of an approximately 22 base pair (bp) repeat unit, and a short allele (S), comprised of 14 copies (Hariri and Holmes, 2006). The S allele is associated with decreased transcriptional efficiency resulting in approximately 50% less serotonin being recaptured in the presynaptic neuron when compared to the L allele (Lesch et al., 1996). There is a large body of research showing S carriers of 5-HTTLPR demonstrate disruptions in emotion processing and reactivity including increased amygdala activation and reduced connectivity between regions of the prefrontal cortex and amygdala when viewing negative stimuli (Heinz et al., 2004) and greater cortisol reactivity to a laboratory stressor (Hankin et al., 2015a).

Specific to attention bias, SS carriers have shown attention bias for negatively valenced material (Jennens et al., 2016; Pergamin-Hight et al., 2012). Given that 5-HTTLPR, particularly SS carriers, has been implicated in emotion processing and reactivity, the current research posits this polymorphism may act as an important moderator to the relationship between attention bias to emotion and depression onset. Building upon the 5-HTTLPR and emotion processing literature, Gibb and colleagues (2009) found an association between mothers’ symptoms of depression and increases in child depressive symptoms among children at high genetic risk who attended away from negative emotion. Among youth, attention away from negative emotion may confer risk for depression given the prospective maladaptive psychological outcomes associated with behavioral avoidance and disengagement (Seiffge-Krenke and Klessinger, 2000); however, once in a current negative mood state, there may be a shift in biases toward self-referential attentional preference toward negative emotion (Mogg and Bradley, 2005). Further prospective data is necessary to better understand whether attentional preference or avoidance of negative emotion is associated with risk for depression onset.

The current study aimed to investigate whether genetic risk (i.e., 5-HTTLPR) moderates the association between attention bias to emotional faces and clinical depression onset prospectively across 18-months in a large community sample of youth. Of note, findings have generally shown attention toward negative emotion during a current negative mood state, but attention away from negative emotion when prospectively predicting increases in depressive symptoms (Gibb et al., 2009; Price et al., 2015). Therefore, we hypothesized that youth at highest genetic risk (i.e., SS carriers of the 5-HTTLPR polymorphism; Pergamin-Hight et al., 2012) who avoided negative emotion, such as sadness or anger, would be at increased risk for clinical depression over time. This study was designed to fill several notable gaps within the attention bias and depression literature including, 1) examining clinical depression onset via diagnostic interview rather than self-reported depression symptomatology, 2) utilizing a prospective design in order to establish a temporal precedence between attention bias and the development of depression, and 3) studying the relationship among attention bias, genetic risk, and depression in a sample of youth, which provides a developmentally significant time frame to identify risk factors for depression onset (Rutter et al., 2006).

2. Method and materials

2.1. Participants

Participants included a subsample of 428 children and adolescents drawn from a larger project utilizing a community sample of youth recruited from schools within urban and suburban Colorado and New Jersey (Hankin et al., 2015b) who completed the attention bias task. The larger study’s primary goal was to understand genetic, cognitive, and affective factors contributing to youth depression. Parents of youth who were in 3rd (n = 115; 27%), 6th (n = 175; 41%), or 9th (n = 138; 32%) grade completed a brief screening to determine their child’s eligibility. Youth were excluded if they had a severe learning or psychiatric problem (e.g., autism, psychosis) that was likely to interfere with completion of an extensive laboratory protocol. The sample was approximately evenly divided by sex, was of mixed ethnic origin representative of their geographic region, and ranged in age from 7 to 16 years old (Table 1). Parents of youth were primarily mothers (91%). Median annual parental income was $90,000 and 16% of the youth received free/reduced lunch at school.

2.2. Procedures

Each eligible parent and youth participated in four points of data collection including the baseline assessment and 6-month, 12-month, and 18-month follow-up assessments. Parents provided informed written consent for their own and their child’s participation, and youth provided written assent at the baseline laboratory visit. The baseline assessment consisted of youth completing the dot-probe task, collecting youth DNA via saliva collection, and diagnostic interviewing with youth and parents about their child, in that order. The 6- and 12-month follow up assessments consisted of
diagnostic interviews with youth and parents about their child by phone. Families returned to the laboratory for the 18-month follow-up assessment where updated diagnostic interviewing took place. The retention rate from baseline to 18-month follow-up was 89%. Attrition did not differ across primary variables of interest (p > 0.06). Those with complete genetic data did not differ from the total sample on age, gender, or ethnicity/race (ps > 0.13).

### Measures

#### 2.3.1. Attention bias

Youth's attentional biases for facial displays of emotion were assessed using a modified dot-probe task (MacLeod et al., 1986) administered using E-Prime. Stimuli for the dot-probe task consisted of pairs of facial expressions that contained one affective (angry, sad, or happy), and one neutral photograph from the same actor taken from a standardized stimulus set (Tottenham et al., 2009). Photographs from each actor (16 men and 16 women) were used to create sad–neutral, happy–neutral, and angry–neutral stimulus pairs (96 pairs). Each stimulus pair was presented in random order across two blocks, with a rest in between blocks, for a total of 192 trials. Each trial began with a white fixation cross in the middle of the screen for 1000 ms. Then, a pair of pictures was presented for 1000 ms, followed by a dot presented for 1000 ms that replaced either the affective or neutral picture. Youth were instructed to indicate as quickly as possible the location of the dot (left versus right side of the screen) using the computer keyboard (“z” labeled “left”; “/” labeled “right”). The computer recorded the accuracy and response time for each response. Consistent with prior research (Gotlib et al., 2004), trials with response errors were excluded as were trials with response times less than 150 ms or greater than 1,500 ms. Error rates were low (less than 1.5%), and a small portion (1.8%) were excluded for being out of response time range. Of the 428 children who completed the dot-probe task, 378 had complete genetic data. Those with complete genetic data did not differ from the total sample on age, gender, or ethnicity/race (ps > 0.13).

#### 2.3.2. Genotyping

Saliva samples were obtained from participants with Oragene™ (DNA Genotek, Ontario, Canada) collection kits, and DNA was extracted using standard salting-out and solvent precipitation methods. The method for 5-HTTLPR and SNP rs25531 (n = 378) is detailed elsewhere (Whisman et al., 2011). The rs25531 SNP genotypes (LA vs. LC) were obtained by incubating the PCR products with MspI (Wendland et al., 2006). Based on meta-analytic findings showing those homozygous for the S allele demonstrate biased attention for negative material as compared to L allele carriers (Pergamin-Hight et al., 2012), two groups of participants were formed based on their 5-HTTLPR genotyping: youth homozygous for the lower expressing S or LC alleles (i.e., SS) and those heterozygous or homozygous for the higher expressing LA allele (i.e., SL/LL). The successful call-rate for the overall project was 97.5% for 5-HTTLPR. 5-HTTLPR was in Hardy-Weinberg Equilibrium.

#### 2.3.3. Diagnostic status

Trained interviewers administered the mood disorders and psychosis subsections of the Schedule for Affective Disorders and Schizophrenia for School Age Children (K-SADS-PL) (Kaufman et al., 1997) to youth and parents about their child to assess for current and past lifetime episodes of depression and mania at baseline and current and past (i.e., last 6-months) episodes of depression and mania at each follow-up. No youth was diagnosed with a bipolar spectrum disorder or psychosis. Interviewers then utilized both youth report and parent report about youth to determine symptom count, depression severity, and diagnostic status using best estimate procedures. Interviewers were trained and supervised by Ph.D. level, licensed psychologists. Interviews (20%) were randomly selected to conduct reliability analyses (κ = 0.91). Furthermore, all interviews containing a threshold or threshold criterion symptom of depression were reviewed for accuracy by another interviewer; disagreements were resolved by consensus.

Seventy youth (16%) met DSM-IV criteria for a clinically significant depressive episode (i.e., Major Depressive Disorder or Depressive Disorder– Not Otherwise Specified) between the baseline and 18-month follow-up assessments. Rates of depressive episode onset found in the sample are similar to those found in other community samples (Avenevoli et al., 2008).
for anxiety disorders among community samples of youth (Merikangas et al., 2009), 6% of youth were above clinical cutoff (T scores >65).

2.4. Data analytic plan

Logistic regression analyses were used to test the Genetic (5-HTTLPR) x Attention Bias (sad, angry, or happy facial expressions) interaction as a predictor of depression group status (depression onset coded 1 and no depression onset coded 0) across 18-months using the SPSS macro PROCESS (Hayes, 2013). Attention bias scores were centered prior to analyses. History of depression (i.e., episodes of clinical depression occurring concurrent or prior to the baseline assessment) and baseline anxiety symptoms (i.e., MASC scores) were entered to control for covariance between past depression and anxiety and future depression episode onset. All main effects and interactions were entered simultaneously and unstandardized regression coefficients are reported (Hayes, 2013) for each set of analyses.

Post hoc analyses of significant interactions were conducted (Aiken and West, 1991). New product terms were computed at different levels (i.e., genotype groups) of the moderator variable. Separate regressions were conducted for each of these product terms. This enabled examination of the significance of simple slopes across genotype. Significant interactions were further explored following Hayes (2013) guidelines for testing regions of significance to the present study, the J-N technique indicated at what degree of genotype differences at higher or lower genetic risk differ significantly. Applied to the present study, the J-N technique indicated at what degree of attention bias those at higher or lower genetic risk differ significantly in their risk for depression disorder onset.

Sensitivity analyses were conducted to examine whether significant results remained once controlling for other key demographic variables known to influence depression onset, including age and biological sex. Additionally, we re-ran significant models only within the largest ethnic group (i.e., Caucasian participants) to address concerns regarding population stratification (Jorm and Easteal, 2000). Finally, we tested whether significant findings remained among participants without a history of depression at the baseline assessment. These analyses provide a more stringent test of hypotheses by examining whether the relationship between attention bias and 5-HTTLPR genotype applies specifically to first onset of depression across the 18-months of the study.

3. Results

3.1. Preliminary analyses

Demographic information and descriptive statistics for all primary variables separated by diagnostic status are presented (Table 1). Consistent with epidemiological studies of youth depression (Avenevoli et al., 2008) older youths, girls, and those with a past episode of depression were more likely to experience an onset of depression across 18-months, while attention bias scores, ethnicity/race, and genotype did not significantly differ across diagnostic groups. There were no significant differences among genotypes across age (p = 0.28), race (p = 0.17), gender (p = 0.94), depression onset across 18-months (p = 0.66), or past history of depression (p = 0.71). Zero-order correlations between primary variables of interest are presented in Table 2.

### Table 2: Correlations between primary variables of interest.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>1</th>
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<tr>
<td>Intercept</td>
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<td>-0.21</td>
<td>0.05</td>
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<tr>
<td>Sad Bias</td>
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<td>-0.04</td>
<td>0.03</td>
<td>1.31</td>
<td>0.19</td>
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<tr>
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<td>0.02</td>
<td>0.60</td>
<td>0.55</td>
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<tr>
<td>Past MDD</td>
<td>1.37</td>
<td>0.76</td>
<td>1.58</td>
<td>4.38</td>
<td>&lt;0.001</td>
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Note: MASC = Multidimensional Anxiety Scale for Children; Past MDD = History of clinical depression as measured at the baseline assessment by using a best estimate of parent and child K-SADS interviews.

### Table 3: Prediction of depressive episode onset across 18-months by 5-HTTLPR and attention bias to emotional faces (sad, angry, happy).

#### Angry Emotion

<table>
<thead>
<tr>
<th>Predictor</th>
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Note. 18-Month MDD refers to onset of a clinically significant depressive episode between the baseline and 18-month follow-up assessments; Past MDD refers to onset of a clinically significant depressive episode prior to or concurrent with the baseline assessment.

#### Sad Emotion

<table>
<thead>
<tr>
<th>Predictor</th>
<th>1</th>
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#### Happy Emotion

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3.2. 5-HTTLPR and attention bias

There were no significant gene-environment correlations (rGEs) between 5-HTTLPR and attention bias to any emotion (ps > 0.07). Logistic regression analyses revealed no significant main effects and a significant interaction between 5-HTTLPR and attention bias to anger predicting depression group status across 18-months (Table 3). This effect is shown in Fig. 1 across the range of_simple slopes across genotype. Significant interactions were further explored following Hayes (2013) guidelines for testing regions of significance to the present study, the J-N technique indicated at what degree of attention bias those at higher or lower genetic risk differ significantly in their risk for depression disorder onset.

Sensitivity analyses were conducted to examine whether significant results remained once controlling for other key demographic variables known to influence depression onset, including age and biological sex. Additionally, we re-ran significant models only within the largest ethnic group (i.e., Caucasian participants) to address concerns regarding population stratification (Jorm and Easteal, 2000). Finally, we tested whether significant findings remained among participants without a history of depression at the baseline assessment. These analyses provide a more stringent test of hypotheses by examining whether the relationship between attention bias and 5-HTTLPR genotype applies specifically to first onset of depression across the 18-months of the study.

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Note. 18-Month MDD refers to onset of a clinically significant depressive episode between the baseline and 18-month follow-up assessments; Past MDD refers to onset of a clinically significant depressive episode prior to or concurrent with the baseline assessment.

*p < 0.001, *p < 0.05.
Fig. 1. Interaction between 5-HTTLPR and bias away from angry faces predicting depression onset across 18 months using observed attention bias scores within the study’s sample. Note. Negative attention bias scores indicate attention away from anger while positive scores indicate attention toward anger. Johnson-Neyman region of significance testing indicates the value of attention bias at and below (i.e., ≤ −50 ms) which the effect of 5-HTTLPR on depression onset is significant.

attention bias scores seen in our sample. Post-hoc analyses showed a significant slope for those with the SS genotype \((b = -1.43, SE = 0.66, t = -2.16, p = 0.03)\) indicating that youth homozygous for the S allele were more likely to experience an onset of clinical depression when avoiding compared to attending to angry facial expressions.\(^1\) The slope for the SL/LL genotype group \((b = 0.82, SE = 0.73, t = 1.13, p = 0.26)\) was not significant. The J-N tests confirmed that the effect of 5-HTTLPR emerged only at high levels of attention away from anger (i.e., ≤ −50 ms) as opposed to preferential attention to anger to predict depression onset (Fig. 1).

No significant interaction between attention bias to sad or happy faces and 5-HTTLPR predicting depression group status was found (Table 3). Non-significant interactions were removed from each regression model and no significant main effect of 5-HTTLPR or attention bias remained within sad or happy analyses \((ps > 0.38)\). As covariates, previous history of clinical depression was significantly associated with depression onset across 18-months among all analyses, while baseline anxiety symptoms were not significantly associated with prospective depression onset (Table 3).

3.2.1. Sensitivity analyses

Analyses revealed a continued significant interaction between attention bias to anger and 5-HTTLPR \((b = -0.03, SE = 0.009, p = 0.002)\) once controlling youths’ biological sex and age. Additionally, we re-ran significant models only within Caucasian participants to further address concerns regarding population stratification (Jorm and Easteal, 2000). Again, findings showed the attention bias to anger by 5-HTTLPR interaction was comparable to the whole sample and remained significant at a trend level \((b = -0.02, SE = 0.01, p = 0.07)\). Finally, the attention bias to anger by 5-HTTLPR interaction remained significant once removing youth with a history of depression, therefore predicting first onsets of depression \((b = -0.03, SE = 0.01, p = 0.03)\).

4. Discussion

The current study investigated whether biased attention to emotion was moderated by 5-HTTLPR to predict clinical depression over time. Results supported hypotheses and demonstrated that youth who attended away from angry faces and were homozygous for the S allele of the 5-HTTLPR polymorphism were more likely to experience a depressive episode onset across 18-months. Findings were robust to sensitivity analyses controlling for sex, age, ethnicity, anxiety symptoms, and examining only first onset of depression.

The current study is the first to examine prospective risk for depression conferred by attention bias to emotion and genetic risk. This is an important advancement as attention bias to emotion has been theorized to function as an intermediate trait associated with depression and other psychiatric conditions (Jorm and Easteal, 2000) whereas the present study and other prospective investigations among youth (Gibb et al., 2009; Price et al., 2015) show attention away from negative emotion among SS carriers to be associated with prospective depression symptoms and clinical onset. This leads to the question: Why would attention away from negative emotion function as a vulnerability factor for depression among youth? Attention away from negative emotion, particularly an interpersonally threatening emotion like anger, may be associated with maladaptive responses to interpersonal stress. Research shows that interpersonal difficulties and subsequent maladaptive responses (i.e., ineffective problem solving, disengagement) are prospectively related to depression in adolescence (Flynn and Rudolph, 2010). Therefore, transactional processes between interpersonal stress and attentional away from negative emotion may contribute to depression onset, whereas mood congruent attention bias, such as attention toward sad faces, may

\(^1\) A similar pattern of findings emerged when predicting depression symptom count \((b = -0.01, SE = 0.006, p = 0.08)\) and severity \((b = -0.05, SE = 0.03, p = 0.07)\) among SS carriers who attend away from angry faces.
manifest once the individual is currently experiencing a depressive episode.

Of note, while our findings showed specificity to attention away from angry faces when prospectively predicting clinical depression, findings from Gibb et al. (2009) and Price et al. (2015) were specific to sad and fearful faces, respectively. As Gibb et al. examined subclinical levels of depression among youth and Price et al. did not examine other emotional biases, it is possible the discrepancy in emotion specificity is related to methodological differences in sample characteristics and measures. Irrespective of emotion type, attention away from as opposed to towards negatively valenced stimuli among youth is an important distinction that may have clinical implications regarding attention bias modification as a treatment target for depression. In order to more clearly understand the role of attention bias specificity in the development of depression, future research must investigate attention bias pre-to post-onset of depression and possible ecologically relevant consequences of attentional bias patterns.

Furthermore, the current study’s findings highlight the importance of examining risk for depression across multiple levels, such as combined genetic and cognitive risk (Hankin, 2012). Our results suggest that attention away from negative emotion alone did not predict future onset of depression; rather, only SS carriers who exhibited attention away from anger were at greater risk for prospective depression onset. The S allele of 5-HTTLPR has been associated with emotional reactivity (Munafò et al., 2008) and cortisol stress reactivity (Hankin et al., 2015a), so it is possible that those who are both more reactive to their environment and engage in maladaptive coping strategies, such as attending away from negative emotions, are at highest risk for experiencing depression over time. As the efficacy of attention bias modification in depressed adults is mixed (Hallon and Ruscio, 2011), the present study’s findings suggest targeting avoidance of threat among those with emotion regulation deficits observed among S allele carriers of 5-HTTLPR may improve treatment success.

Strengths of the present study include being the first to examine an integrated, prospective model examining genetic risk and attention bias to predict prospective clinical depression among either adults or youth. We employed a multi-informant, well-validated diagnostic interview to evaluate depression diagnoses using best estimate procedures over 18-months as opposed to cross-sectional or symptom level measurement of depression. A theory-based candidate gene approach versus atheoretical GWAS methodology allows for a more sensitive and powerful test of genetic effects (Caspi et al., 2010). Finally, the use of a community sample of youth provided generalizable results compared to clinically selected samples (Cohen and Cohen, 1984).

Limitations of the current study provide avenues for future research. While the current study’s measure of attention bias is more objective compared to self-report data, there are other, more precise tools available, such as eye-tracking methodology that allows for assessment of the time-course of attention bias. Finally, although 5-HTTLPR genotype status may relate to emotion dysregulation and stress reactivity, further studies should utilize self-report and objective measures (i.e., laboratory stressors) to investigate whether emotion regulation ability moderates or mediates the relationship between 5-HTTLPR, attention bias, and depression.

In sum, the current study demonstrated that youth who attended away from angry faces and were homozygous for the S allele of 5-HTTLPR were at higher risk for experiencing an episode of clinical depression over time. This finding may inform future depression intervention research involving modification of attention bias to emotion.

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Contributors

Jessica L. Jenness developed the study concept. All authors contributed to the study design. Data collection was performed in part by Jessica L. Jenness. Jessica L. Jenness performed the data analysis and interpretation under the supervision of Benjamin L. Hankin. Jessica L. Jenness drafted the paper, and Benjamin L. Hankin and Jami F. Young provided critical revisions. All authors approved the final version of the paper for submission.

Conflicts of interest

None.

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