Variations in the Promoter Region of the Serotonin Transporter Gene and Biased Attention for Emotional Information: A Meta-Analysis

Lee Pergamin-Hight, Marian J. Bakermans-Kranenburg, Marinus H. van IJzendoorn, and Yair Bar-Haim

Background: Selective attention to negative information has been strongly implicated in the etiology and maintenance of anxiety and offered as a potential intermediate phenotype for anxiety disorders. Attention biases have been studied in relation to a polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR) offering equivocal findings. The present meta-analysis tested whether the extent published data support the notion that variation in the 5-HTTLPR genotype modulates selective attention to negative information.

Methods: Eleven relevant samples from 10 published articles were identified through a systematic literature search (total n = 807). Relevant attention bias and 5-HTTLPR data were extracted based on specific coding rules, and Cohen’s d effect size index was used to calculate all outcome measures. Publication bias was assessed using various methods.

Results: Carriers of the low (SS, SL/L, L/L) transmission efficacy genotype display attentional vigilance toward negatively valenced stimuli, a pattern not found in the intermediate (SL/L, L/L) and high (L/L/L) efficacy genotypes. This phenomenon emerges as of medium effect size.

Conclusions: The meta-analysis supports the notion that allele variants of the 5-HTTLPR are associated with selective attention to negative stimuli. More studies are needed to fully establish the consistency of this effect. Future studies applying systematic attention bias modification may shed further light on the role of 5-HTTLPR in the development of anxiety disorders and in the prediction of clinical response to attention bias modification treatments.

Key Words: Anxiety, attention bias, gene, 5-HTTLPR, serotonin, threat

Identifying the impact of specific genetic variation on the development of discrete psychiatric disorders can considerably advance the understanding of disease-related etiology and inform the development of potential new treatments. However, because psychiatric disorders typically involve complex biobehavioral phenotypes that also interact with environmental factors (1), the identification of specific gene variants that are associated with a distinct psychiatric disorder remains an elusive target. One possible approach to this problem is to focus on hypothesis-driven candidate gene variants and their association with intermediate phenotypes presumed to lie between a moderating allele and a more complex disease phenotype. While still controversial (2,3), this research strategy holds promise for bridging the conceptual gap between specific genetic variation and complex psychiatric phenotypes.

Synaptic availability of serotonin has been widely implicated in the modulation of mood states and anxiety disorders and thus became a primary target for candidate gene research in psychiatry. A common repeat polymorphism was identified in the promoter region of the gene coding for the serotonin transporter protein (5-HTTLPR). The polymorphism in this gene was found to modulate synaptic availability of serotonin. A common 43-base pair deletion (short form S-allele) has been associated with less effective serotonergic function relative to the long allelic form (L-allele) (4). Importantly, the L-allele has further been shown to encompass a single nucleotide polymorphism (rs25531). The A/G single nucleotide polymorphism insertion within the 5-HTTLPR renders only the A variant and not the G variant of the L-allele, yielding high synaptic serotonin transporter (5-HT) messenger RNA levels. Thus, L-allele variants apparently functions equivalent to the low-expressing S-allele. Because both S- and L- show low transcriptional efficacy, they are considered similar to S-alleles in function (5).

Lesch et al. (6) first reported that the short form of the 5-HTTLPR polymorphism (S-allele) is associated with increased neuroticism (an anxiety-related construct). Additional studies have shown that S-allele carriers exhibit heightened amygdala reactivity to threat, altered functional connectivity between the amygdala and prefrontal cortical structures, decreased gray matter volume of the amygdala and medial prefrontal cortex, and exaggerated hypothalamic-pituitary-adrenal axis response to stress (7). These data suggest that the S-allele may act to predispose individuals to heightened neural and endocrine responses to stress, which, in turn, may elevate their risk of anxiety-related disorders (8).

Behaviorally, the S-allele has been associated with biased processing of negative information. Specifically, biased attention toward negative stimuli represents the single most frequently noted correlate of individual differences in anxiety and has been suggested as a cognitive intermediate phenotype for anxiety disorders (9,10). Attentional threat bias has been implicated in the etiology and maintenance of anxiety (11), with studies demonstrating causal associations by experimentally manipulating threat bias to induce anxious states (12–14). Based on such findings, novel evidence-based attention bias modification (ABM) treatments for anxiety disorders have started to emerge (15–17). Attention bias modification trains attention away from threats and appears to reduce anxiety via functional modulation of key structures of the serotonergic fear circuitry (e.g., frontal cortical regions and amygdala) (18,19). Furthermore, a recent study showed that carriers of the low transmission efficacy genotype of the 5-HTTLPR are more sensitive to ABM (20).
Beever et al. (21) were the first to report that carriers of the S-allele showed a bias toward negative stimuli. A steady stream of studies followed, offering equivocal findings. For instance, Perez-Edgar et al. (22) reported that carriers of the low efficacy genotype (SS, SL<sub>L</sub> (L<sub>L</sub>)) were indeed vigilant toward threat, whereas Fox et al. (9) reported that carriers of the low efficacy genotype showed no threat bias. In addition, some studies aggregate bias scores of participants with low and intermediate 5-HTT transmission efficacy, contrasting it with the bias score of participants with high transmission efficacy. Such analyses might hinder potential effects if the L-allele in the intermediate efficacy group serves as a protective factor against undue vigilance toward negative information.

Thus, the goal of the present set of meta-analyses was to determine whether the extant data support the notion that the 5-HTTLPR polymorphism modulates selective attention to negative information. At this early stage of research, such preliminary analyses could shed significant light on the potential of attentional bias to become a valid intermediate phenotype for anxiety disorders, and they could point the way for future studies on the association between 5-HTT, threat bias, and anxiety.

Methods and Materials

Literature Search and Inclusion Criteria

Studies were selected through a search in the PsychInfo and Web of Science databases using the following key words: attention bias or selective attention or attention bias or biased attention or dot-probe or probe detection task or Posner or spatial cueing task or Stroop or visual search intersected with 5-HTT or 5-HTTLPR or serotonin transporter or 5-hydroxytryptamine. The reference lists of the articles obtained from this search were systematically reviewed for additional relevant studies.

The following inclusion criteria were applied: 1) publication was in the English language until July 2011; 2) the study reported data on an association between 5-HTTLPR polymorphism and emotion-related attention bias; 3) the study reported or allowed a comparison between responses to neutral and emotion-laden stimuli; and 4) the data provided allowed for the calculation of either within- and/or between-genotype group effect sizes. Authors of studies that did not present the needed contrasts were contacted via e-mail requesting the relevant data. The above criteria led to the identification of 18 studies. Seven potentially relevant studies had to be excluded. Two studies (23, 24) were excluded because the characteristics of the paradigms used were not comparable with the classic attention bias conceptualization considered here. Both studies involved conscious judgments of stimulus valence that were part of task demands and therefore reflected elaborate cognitive processes not necessarily related to attention. We also excluded two eye-tracking studies (25, 26) because these appeared to tap more into processes of effortful regulation of attention rather than the more basic attention mechanisms studied in the included papers. Attention bias is not typically calculated in eye-tracking studies and would reflect a considerably longer time scale relative to the attention biases derived from the studies selected for analyses here, thus rendering these two sets of data incomparable. Two studies (27, 28) reported data from overlapping samples; therefore, only one entry was used in the meta-analysis. One study (20) was excluded because it did not provide attention bias data reflecting the difference between emotional and neutral stimuli. Finally, the necessary data from one of the potentially relevant studies could not be located (29). The final set of studies included in the meta-analyses consisted of 11 samples from 10 published articles (9, 21, 22, 27, 30–34, 52), with a total of 807 participants, published between February 2007 and February 2010. Table 1 lists the included studies along with their general characteristics and the derived effect sizes.

Coding System

A standard coding system was applied. We coded general background characteristics of the samples (the country in which study was conducted, percentage of Caucasian participants, and male-to-female ratio). We also coded whether the participants were children under 18 years of age or adults. We noted the origin of the DNA sample (saliva, hair, or buccal cells), whether the genotype grouping was in Hardy-Weinberg equilibrium, and whether the variants of L<sub>C</sub> and L<sub>A</sub> were considered in forming the genotype groups. We also coded the type of employed attention paradigm, type of stimuli presented in the experiment (words or pictures), and valence of the emotional stimuli (threat, dysphoric, happy). Finally, we coded whether cue exposure was supraliminal or subliminal and noted the exact exposure time.

Data Extraction and Meta-Analytic Procedures

Relevant data were extracted from text, tables, and figures reporting t, F, or p values or (in the majority of cases) mean attention bias scores and standard deviations or standard errors. Cohen’s d effect size index was used for all outcome measures, reflecting the difference between the means of two conditions or groups divided by their pooled standard deviation. All analyses and computations were carried out using the Comprehensive Meta-Analysis Software, version 2.002 (Biostat, Englewood, New Jersey). Because most of our data sets were heterogeneous in their effect sizes and because random effects models are somewhat more conservative than fixed effects parameters in such cases (35), in the current meta-analysis combined effect sizes and their confidence intervals (CIs) are presented in the context of random effects models.

5-HTTLPR Groupings

Serotonin transporter-linked promoter region polymorphism was grouped based on presumed efficacy of serotonin neurotransmission: low (SS/SL<sub>L</sub>), intermediate (SL<sub>A</sub>/L<sub>A</sub>), and high (L<sub>A</sub>L<sub>A</sub>). The L<sub>C</sub> allele has a similar transmission efficacy profile to the S-allele (5), so this information was applied in the groupings when available. In addition, because some studies grouped the low and intermediate genotypes into a low transmission efficacy group, we also provide analyses for this combination.

Derivation of Attention Bias Effect Sizes

Within-group attention bias effect size was calculated separately for each genotype group in each study. The within-group effect reflects the difference between response times to neutral and emotion-laden stimuli contrasted against a zero bias score (i.e., no attention bias). Positive d values indicate attention bias toward the emotion stimuli. A negative d value indicates attention bias away from the emotion and toward the neutral stimuli.

A between-groups effect refers to the difference between genotype groups on attention bias. Two types of between-groups comparisons were calculated. In one type of analyses, we computed combined effect sizes for each of the genotypes separately and then contrasted them. In the other type of analyses, we used the generic between-groups comparison reported in each study to generate a between-groups combined effect size of differences between genotypes. For the between-groups analyses, a positive d value indicates that the lower efficacy 5-HTTLPR genotype had a greater threat bias score than the higher efficacy group.

When more than one type of emotion-related stimuli was used in a single experiment, attention bias effects were separately coded for each valence. Thus, we computed a combined attention bias
effect for all negatively valenced stimuli (general negative), for threat-related stimuli (threat, e.g., angry or fearful faces, anxiety-related words), dysphoric stimuli (e.g., sad faces or words), and positive stimuli (e.g., smiling faces), each tested in a separate meta-analysis. We estimated overall effect sizes across studies for each within-group and between-groups outcome. To examine publication bias, we conducted Egger’s tests for funnel plot asymmetry and computed fail-safe numbers (36). Egger’s test is based on linear regression of the effect sizes divided by their standard errors on their precision defined as the reciprocal of the standard errors. For each meta-analysis, we also calculated the fail-safe number, which is the number of studies with average sample size and nonsignificant outcomes that would be required to bring the combined effect size of the meta-analysis down to a nonsignificant level (37). The trim and fill method was used to test the influence of possible adjustments to the estimated effect size due to publication bias (36). Effects of moderator variables were estimated using the Q-test for between-group contrasts.

Results

5-HTTLPR Polymorphism and Attentional Bias to Negative Stimuli

Figure 1 presents a plot of effect sizes by genotype groups (low, intermediate, and high 5-HTT synaptic transmission efficacy) for the individual studies included in the meta-analysis.

Within-genotype analyses (Table 2) indicated that carriers of the low efficacy genotype (SS/SLG) show a significant attention bias toward negative stimuli, whereas no bias emerged for the intermediate (SLA/LALG) and high (LALA) genotypes. Combining the effects of the low and the intermediate genotype groups (the so-called S-allele carriers) also revealed a significant attention bias toward negative stimuli. Egger’s tests indicated that there was no publication bias for the low genotype effect, intercept 1.93, p = .05. Fail-safe numbers of 12 and 18 for the low and for the combined low and intermediate genotypes, respectively, indicated that these effects might not be considered very robust. Contrasts between the combined effect sizes indicated that the low efficacy genotype, as well as the combined low and intermediate genotype, differed significantly from the high efficacy genotype, for which a tendency to avoid negative stimuli was detected.

The generic between-groups comparisons (Table 3) also indicated a significant overall difference between the low and high efficacy genotypes. Although our analyses showed publication bias for this central effect size estimate (Table 3), applying the trim and fill (36) method, we still found a highly significant overall combined effect size of d = .44 after three studies were trimmed and filled. In addition, the intermediate genotype differed significantly from the high genotype. In both cases, the lower efficacy genotype had
Moderator Analyses

Table 4 summarizes the results of a set of analyses conducted to shed light on potential moderators of the association between 5-HTTLPR polymorphism and attention bias. Most moderators did not procure a sufficient number of studies (k < 4) (38) to allow meaningful analyses. Due to the same constraint, moderator analyses were viable only for the combined low + intermediate efficacy genotype group and the high efficacy genotype group, thereby constraining the scope of these analyses relative to the analyses combining all the available data. Table 4 provides contrasts, combined effect sizes, and confidence intervals for moderators with enough studies for valid analysis (k ≥ 4) in at least two categories of the moderator. Below, we describe the results of analyses showing significant moderators.

Age. As with the general analysis, adults with low + intermediate efficacy genotype showed vigilance toward negative material (d = .23), whereas those with the high efficacy genotype showed attentional avoidance of such materials (d = −.52). This difference was significant, Q = 8.46, p < .01. However, unlike the general analyses that included studies on children, attentional avoidance of negative stimuli by adults with the high efficacy genotype appeared to carry a more significant weight in this effect. For children, the contrast between the high versus low + intermediate efficacy genotype groups could not be tested (k < 4).

Paradigm. Analyses based on studies using the dot-probe task revealed the same pattern of findings obtained for the general analysis that also included studies relying on the Posner paradigm. Attentional bias toward threats was found for the low + intermediate efficacy genotype (d = .37), whereas no threat bias was noted for the high efficacy genotype (d = −.22). A contrast between these combined effect sizes indicates that the low + intermediate efficacy genotype differed significantly from the high efficacy genotype, Q = 3.96, p < .05. Differences related to the Posner task could not be tested (k < 4).

Discussion

The present meta-analysis provides support for the notion that allele variants of the 5-HTTLPR are associated with selective atten-
tion to negative stimuli. Carriers of the low transmission efficacy genotypes display enhanced attentional vigilance toward negatively valenced stimuli, a pattern not observed in carriers of the intermediate and high efficacy genotypes. This phenomenon is of medium effect size, and given the modest number of studies included in the analyses, more studies are needed to establish its consistency.

The current findings indicate that negative attention bias holds some promise as an intermediate phenotype for anxiety disorders (9,31). Negative attention bias as measured with the dot-probe task has been established as a reliable correlate of anxiety disorders (11), and based on the present analyses, its operation appears to be supported by a 5-HTTLPR genetic architecture that may be closer to the level at which the serotonin transporter gene operates than the more remote phenotypes of clinical anxiety disorders (39). However, to our knowledge, a study testing the full mediation model bridging 5-HTTLPR, attention bias, and anxiety is yet lacking. Such a study would constitute a logical and much needed next step of research patterns in the 5-HTTLPR genotypes among children and adults. In adults, the high efficacy (LL) 5-HTTLPR genotype shows response to threat and biased information processing, creating biological mechanisms underlying emotional vigilance and arousal (43) and are differentially activated in anxious patients relative to healthy control subjects during performance on the dot-probe task (44,45).

Taken together, these findings along with the current results suggest a potential mechanistic association between serotonin reuptake efficacy and specific neural circuits that mediate emotional reactivity (9). Thomason et al. (32) suggested a neural gain mechanism in the processing of negative emotion stimuli that is boosted in carriers of the S-allele. This neurobiological mechanism may correspond to the behaviorally observed attention bias found in this genotype group. A more transactional view of these processes may see the S-allele as a factor that primes both neurobiological response to threat and biased information processing, creating biological and cognitive processes that work in tandem to increase vulnerability to anxiety (22).

Informal inspection of our data hint at different selective attention patterns in the 5-HTTLPR genotypes among children and adults. In adults, the high efficacy (LL) 5-HTTLPR genotype shows attentional avoidance of negative stimuli; in children, this genotype

### Table 3. Between-Genotype Groups Comparisons of Combined Effect Sizes of Attention Bias to Negative Stimuli

<table>
<thead>
<tr>
<th>Low vs. High</th>
<th>k</th>
<th>n₁</th>
<th>n₂</th>
<th>d</th>
<th>p</th>
<th>95% CI</th>
<th>Fail-Safe Number</th>
<th>Egger Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low vs. Intermediate</td>
<td>6</td>
<td>133</td>
<td>136</td>
<td>.65</td>
<td>.000</td>
<td>.33, .97</td>
<td>41</td>
<td>2.82, .03</td>
</tr>
<tr>
<td>Intermediate vs. High</td>
<td>5</td>
<td>197</td>
<td>99</td>
<td>.46</td>
<td>.007</td>
<td>.13, .80</td>
<td>14</td>
<td>3.71, .06</td>
</tr>
<tr>
<td>Low vs. Intermediate vs. High</td>
<td>5</td>
<td>97</td>
<td>149</td>
<td>.24</td>
<td>.16</td>
<td>.05, .63</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Cl, confidence interval.

*Egger’s regression intercept and p value, respectively.

---

### Table 4. Contrasts, Combined Effect Sizes, and Confidence Intervals for Moderator Subsets of Studies

<table>
<thead>
<tr>
<th>Stimulus Valence</th>
<th>Low + Intermediate</th>
<th>High</th>
<th>Q for Contrast</th>
</tr>
</thead>
<tbody>
<tr>
<td>k</td>
<td>n</td>
<td>d</td>
<td>p</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------</td>
<td>------</td>
<td>----------------</td>
</tr>
<tr>
<td>Threat</td>
<td>8</td>
<td>444</td>
<td>.34</td>
</tr>
<tr>
<td>Dysphoric</td>
<td>6</td>
<td>264</td>
<td>.08</td>
</tr>
<tr>
<td>Positive</td>
<td>7</td>
<td>404</td>
<td>.19</td>
</tr>
<tr>
<td>Age</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Adults</td>
<td>8</td>
<td>402</td>
<td>.23</td>
</tr>
<tr>
<td>Children</td>
<td>3</td>
<td>152</td>
<td>.37</td>
</tr>
<tr>
<td>Paradigm</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Dot-probe</td>
<td>8</td>
<td>402</td>
<td>.37</td>
</tr>
<tr>
<td>Posner</td>
<td>3</td>
<td>152</td>
<td>.01</td>
</tr>
<tr>
<td>L/L0</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Yes</td>
<td>4</td>
<td>340</td>
<td>.32</td>
</tr>
<tr>
<td>No</td>
<td>7</td>
<td>214</td>
<td>.30</td>
</tr>
<tr>
<td>DNA Sample</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Buccal cells</td>
<td>7</td>
<td>296</td>
<td>.15</td>
</tr>
<tr>
<td>Saliva</td>
<td>3</td>
<td>177</td>
<td>.50</td>
</tr>
<tr>
<td>Stimulus Type</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Pictures</td>
<td>9</td>
<td>467</td>
<td>.30</td>
</tr>
<tr>
<td>Words</td>
<td>2</td>
<td>87</td>
<td>.29</td>
</tr>
</tbody>
</table>

Cl, confidence interval; L, long allele.

*p < .01.

*No analysis (k < 4).

*p < .05.

*p < .10.
group shows a tendency to attend toward threat. Generally, non-anxious children and adults show no attention bias to negative stimuli (11). However, the exact mechanisms underlying the development and regulation of negative attentional bias are yet uncharted (46). It has been suggested that young children display a robust vigilance toward threats that is being regulated later in life with maturation of frontal control processes (47,48). Such a mechanism could explain bias toward threat in carriers of the high efficacy genotype among children. In light of the relation between 5-HTTLPR and anxiety and given the established association between anxiety and threat-related attention biases in children, it is particularly important to further investigate the association between 5-HTTLPR and selective attention in children.

Some limitations and suggestions for further research can be mentioned. First, the current meta-analysis is based on a relatively small number of studies performed by a limited number of research groups. Furthermore, the small fail-safe numbers of the reported meta-analytic effect sizes and the possibility of publication bias highlight the need for additional research consolidating the reported association between the low transmission efficacy genotype and negative attention bias. It may be noted that the combined effect size of $d = .65$ for the contrast between the low efficacy genotype and the high efficacy genotype implies that primary studies should involve samples of at least $n = 120$ to have enough power to find a significant contrast ($power > .80, \alpha = .05$, one-tailed), based on a distribution of low, intermediate, and high efficacy genotypes similar to the current set of studies.

Second, it is important to consider the possibility that the association of 5-HTTLPR with attention bias toward negative information is moderated by gene expression that does not involve a change in nucleotide sequence but may instead be affected by messenger RNA transcription (49,50). DNA methylation, for example, is an important determinant of gene expression, and it should therefore be taken into account when associations of gene sequences with psychological intermediate phenotypes are examined (49). Third, genetics research should also consider interaction between genes in predicting pathological states. There may be other risk or protective alleles that interact with 5-HTTLPR to influence selective attention (39). Fourth, interactions between 5-HTTLPR and stressful developmental life experiences have been suggested to impact later emotional and social behavior (1,51). Three studies have looked for such potential gene-by-environment interactions between 5-HTTLPR, attentional bias to negative stimuli, and stressors during development such as maternal depressive symptoms (27), maternal expressed criticism (28), and a history of child abuse (52). We were unable to investigate this factor in the present meta-analysis due to the small number of available studies. Finally, despite a wealth of research demonstrating attentional biases toward negative information in anxiety disorders, there is still a need for better understanding of the psychometric properties of the paradigms measuring attention bias (53). In particular, some concern has been voiced regarding the reliability of the dot-probe task (54), but see also promising reliability data (55,56). Although not all the studies in the present meta-analysis used the dot-probe task, reliability issues potentially increase the risk of biased results.

In sum, the present meta-analysis shows promising evidence for a relation between 5-HTTLPR and selective attention to negative information. This evidence marks attention bias as a potentially valid intermediate phenotype for anxiety disorders. That said, at this early stage of research, these conclusions should be taken cautiously as promising research leads rather than a bottom line. Additional research is particularly needed to test whether 5-HTTLPR indeed mediates the association between attention biases to negative information and anxiety. Also needed is a more thorough examination of the association between 5-HTTLPR and selective attention to negative information in children, taking into greater account neural, cognitive, and environmental factors. Such studies may pave the way for the development of more comprehensive etiological models of anxiety vulnerability and enhance our specificity in treatment development (15,48). In particular, future studies may examine whether 5-HTTLPR efficacy moderates the effect of ABM treatments for anxiety disorders (57,58). Indeed, a first study revealed that following ABM training, healthy individuals with the low expression 5-HTTLPR genotype developed stronger attention biases for both negative and positive affective pictures relative to individuals with the high expression genotype (20). These results suggest that variation in 5-HTTLPR may serve as a marker for therapeutic efficacy of cognitive treatments for anxiety.

Yair Bar-Haim was supported by the Israeli Science Foundation (Grant #964/08); Marian Bakermans-Kranenburg was supported by awards from the Netherlands Organization for Scientific Research (VIDI Grant 452-04-306; VICI Grant 453-09-003); and Marinus van Uzendoorn was supported by a Spinoza prize of the Netherlands Organization for Scientific Research.

The authors report no biomedical financial interests or potential conflicts of interest.

prefrontal cortex mediates the cognitive modification of at-
porter gene alters sensitivity to attention bias modification: Evidence
genetic variation and biased attention for emotional word stimulii
gene are associated with attention bias patterns to positive and nega-
23. Koizumi A, Kitagawa N, Kitamura MS, Kondo HM, Sato T, Kashino M
(2010): Serotonin transporter gene and inhibition of conflicting emo-
on mood, memory, and attention bias following acute tryptophan
gene promoter region polymorphism and selective processing of emo-
(2011): Associations between serotonin transporter gene promoter re-
gion (5-HTTLPR) polymorphism and gaze bias for emotional informa-
27. Beever CG, Wells TT, Ellis AJ, Stote DL, Kambara N, Kitamura MS, Kondo
HM, Sato T, Kashino M (2010): Serotonin transporter gene and inhibition of conflicting emo-
Children’s 5-HTTLPR genotype moderates the link between maternal
criticism and attentional biases specifically for facial displays of anger.
*Cogn Emot* 25:1104–1120.
Beyond affect: A role for genetic variation of the serotonin transporter
J (2008): Variation in the serotonin transporter gene modulates selec-
31. Kwang T, Wells TT, McCray JE, Swart WB, Beever CG (2010): Associa-
tion of the serotonin transporter promoter region polymorphism
with biased attention for negative word stimuli. *Depress Anxiety* 27:746–751.
al. (2010): Neural and behavioral responses to threatening emotion
faces in children as a function of the short allele of the serotonin trans-
serotonin transporter gene promoter region (5-HTTLPR) polymor-
to Meta-Analysis*. Chichester, UK: Wiley.