HHS Public Access

Author manuscript

Clin Psychol Sci. Author manuscript; available in PMC 2024 July 22.

Published in final edited form as:

Clin Psychol Sci. 2024 May ; 12(3): 447–467. doi:10.1177/21677026231170563.

Multiple Adaptive Attention-Bias-Modification Programs to Alter Normative Increase in the Error-Related Negativity in Adolescents

Nader Amir[1], **Amanda Holbrook**[1], **Alex Kallen**[2], **Nicholas Santopetro**[2], **Julia Klawohn**[2], **Shaan McGhie**[1], **Alec Bruchnak**[2], **Magen Lowe**[2], **William Taboas**[1], **C. J. Brush**[2], **Greg Hajcak**[2]

[1]Department of Psychology, San Diego State University

[2]Department of Psychology, Florida State University

Abstract

In the current article, we examined the impact of two home-delivered attentional-bias-modification (ABM) programs on a biomarker of anxiety (i.e., the error-related negativity [ERN]). The ERN is sensitivity to ABM-related changes; however, it is unclear whether ABM exerts its influence on the ERN and anxiety by increasing general attentional control or by disengaging spatial allocation of attention. In this study, we measured the ERN, anxiety, attention bias, and attention control before and after two versions of ABM training and a waitlist control group in 546 adolescents. An ABM designed to increase attention control modulated the ERN but had no impact on anxiety. An ABM designed to reduce attentional bias changed bias and self-reported anxiety in youths but had no impact on the ERN or parent-reported anxiety. These results suggest that the ERN and normative anxiety may be modified using attention training.

Keywords

anxiety; ERN; ABM; developmental psychopathology

Corresponding Author: Nader Amir, Department of Psychology, San Diego State University namir@sdsu.edu.

Author Contribution(s)

Nader Amir: Conceptualization; Data curation; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Resources; Software; Supervision; Validation; Visualization; Writing – review & editing. **Amanda Holbrook:** Data curation; Formal analysis. **Alex Kallen:** Data curation; Investigation; Project administration; Validation.
Nicholas Santopetro: Data curation; Investigation; Project administration.
Julia Klawohn: Conceptualization; Investigation; Methodology; Project administration.
Shaan McGhie: Data curation; Investigation; Project administration.
Alec Bruchnak: Data curation; Investigation; Project administration.
Magen Lowe: Investigation; Project administration. **William Taboas:** Data curation; Investigation; Project administration.
C. J. Brush: Data curation; Investigation; Project administration.
Greg Hajcak: Conceptualization; Data curation; Funding acquisition; Investigation; Methodology; Project administration; Resources; Supervision; Validation; Writing – original draft; Writing – review & editing.

Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/21677026231170563

Clinical anxiety affects 15% to 20% of children and adolescents, making anxiety the most frequently diagnosed form of psychopathology among youths (Beesdo et al., 2009). Longitudinal studies have suggested that anxiety disorders are stable over time and predict future anxiety and depressive disorders in adolescence and adulthood (Bittner et al., 2007; Pine et al., 1998; Wittchen et al., 2000). These studies indicate that anxiety disorders follow trajectories that begin early in development and often result in chronic impairment, although the specific pathways are not fully understood. Given the chronic and impairing nature of anxiety, there is a critical need to identify core neural systems and measures implicated in the development of anxious symptoms and attempt to alter them. In the current study, we focus on an early-emerging biomarker of risk for anxiety, the error-related negativity (ERN).

The ERN is a negative deflection in the event-related-potential (ERP) that can be recorded at the scalp via electroencephalogram (EEG) within 100 ms of errors of commission (Falkenstein et al., 2000; Gehring et al., 1993). The ERN, correct-response negativity (CRN), and their difference (i.e., ΔERN) are commonly measured using a speeded-response Flanker task designed to elicit mistakes (Eriksen & Eriksen, 1974). The ERN is likely generated in the anterior cingulate cortex (ACC), a region of the brain where information about pain, threat, and punishment is integrated to change behavior (Shackman et al., 2011). Errors are motivationally salient events that could threaten an individual's safety thus requiring increased attention and corrective action (Moser et al., 2013). Unlike other aversive stimuli, errors are *internally* generated sources of threat. Errors are more salient for some individuals, and the magnitude of the ERN reflects individual differences in reactivity following mistakes (for a review, see Meyer & Hajcak, 2019).

Critically, the magnitude of the ERN is related to variation in psychopathology. Consistent with the hypothesis that anxious individuals are more sensitive to internal threat (Weinberg et al., 2016), the ERN is consistently larger among anxious individuals (for reviews, see Moser et al., 2013; Olvet & Hajcak, 2008; Simons, 2010; Vaidyanathan et al., 2012; Weinberg et al., 2012). That is, anxious individuals whose focus of threat is physical symptoms of danger (e.g., specific phobias or panic disorder) are often characterized by external sources of threat and therefore are less likely to show ERN differences compared with nonanxious control subjects. However, individuals with internal sources of danger (e.g., worry or obsessions) show larger ERN compared with control subjects. Indeed, our recent data (C. S. Brown & Amir, 2022) showed that even for scales that are traditionally thought of as related to external sources of threat (i.e., the Anxiety Sensitivity Index), the cognitive component of this scale is related to the ERN.

Consistently, disorders characterized by anxious apprehension (i.e., cognitive symptoms of anxiety), as opposed to anxious arousal (i.e., acute fear response), are related to an increased ERN, including obsessive compulsive disorder (OCD; for a review, see Weinberg, Dieterich, & Riesel, 2015), generalized anxiety disorder (GAD; Weinberg et al., 2012; Weinberg et al., 2010; Weinberg, Kotov, & Proudfit, 2014; Xiao et al., 2011), and social anxiety disorder (Endrass et al., 2014), whereas individuals with disorders characterized by sensitivity to external threat (e.g., posttraumatic stress disorder, simple phobia) display an ERN comparable with control subjects (Moser et al., 2005; Rabinak et al., 2013).

The ERN can be elicited early in development, and research suggests an elevated ERN in children and adolescents is related to clinical (Ladouceur et al., 2006; Meyer et al., 2013) and subclinical (Meyer et al., 2012) anxiety symptoms. However, some evidence suggests the relationship between anxiety and elevated ERN does not emerge until adolescence (Meyer et al., 2012, 2013; Weinberg et al., 2016), whereas heightened trait anxiety in younger children may instead relate to a blunted ERN (Meyer et al., 2012). This pattern of increasing ERN across childhood and adolescence has been attributed to the changing expression of normative fear from externally focused stimuli in early childhood (e.g., separation anxiety, simple phobia) to self-consciousness and worry about behavioral competence and social evaluation in middle childhood and adolescence (i.e., internal threat; Meyer, 2017; Weinberg et al., 2016).

The ERN has also been proposed as a neurobehavioral risk marker for anxiety across development, evident among individuals without an anxiety disorder (for reviews, see Meyer, 2017; Olvet & Hajcak, 2008). For example, Meyer et al. (2015) measured the ERN in 236 healthy 6-year-olds and found that increased ERN predicted new onset of anxiety disorders 3 years later while controlling for baseline anxiety symptoms and maternal history of depression. Likewise, two studies (Lahat et al., 2014; McDermott et al., 2009) found that early behavioral inhibition interacts with the ERN to predict later anxiety. In addition, Meyer et al. (2018) found an increased ERN in adolescents predicted first onset of GAD over 1.5 years independent of other prominent risk factors, including baseline anxiety and depression and parental lifetime psychiatric history. The ERN also predicted increases in anxiety in a sample of clinically anxious female children and adolescents over 2 years even when accounting for baseline symptoms (Meyer et al., 2021).

The ERN demonstrates excellent psychometric properties; it had high test-retest reliability over the course of 2 weeks (Olvet & Hajcak, 2009a) and up to 2 years (Weinberg & Hajcak, 2011). In addition, the ERN has high internal consistency even after relatively few (i.e., six) trials (Olvet & Hajcak, 2009b). Collectively, these findings suggest that the ERN is a potential vulnerability marker for the subsequent increases in anxiety. Given that treatment earlier in the course of development of anxiety disorders results in better long-term functioning (Mancebo et al., 2014), early identification and modification of the ERN may prevent increases in symptoms of anxiety. However, few studies to date have attempted to modulate the ERN.

Several researchers have examined the effect of attention-bias modification (ABM) on the ERN (Carlson et al., 2021; Klawohn et al., 2020; Nelson et al., 2015, 2017; Tan et al., 2021). ABM is a computerized intervention that trains attention away from negative stimuli and targets a core mechanism of dysfunction in anxiety (i.e., attention bias toward threat; Mathews & MacLeod, 2005). Previous studies have found that ABM decreases attentional bias and anxiety, but results are, at times, mixed (for reviews, see Bar-Haim, 2010; Beard, 2011; Browning et al., 2010; Martinelli et al., 2022). Moreover, functional-MRI research suggests that ABM produces changes in two key neural systems: (a) a bottom-up amygdala-based system that produces a signal reflecting the perceived salience of stimuli and directs attention toward salient stimuli and (b) a top-down system composed of the ACC and prefrontal cortex that produces a signal when conflicting demands are made on attention, for

example, when two or more stimuli compete for attentional resources (Bishop, 2008; Taylor et al., 2014). Given that ABM appears to target the ACC and anxiety, that the ERN may be localized to the ACC, and that elevated anxiety is linked to increased ERN amplitude, one possibility is that ABM may be effective in reducing the ERN.

Indeed, previous studies have found that ABM can modulate the ERN in the short term. Nelson et al., (2015) randomly assigned 59 undergraduates to complete a single session of an adaptive variant of ABM (AABM) either before or after the ERN was measured using a Flanker task (i.e., AB/BA design). The AABM program was a modified version of a Posner spatial-cuing task (Posner, 1980) that contained several modifications that differed from traditional versions of ABM (Amir et al., 2008; MacLeod et al., 2002). The AABM program was first introduced in Amir et al. (2016) and contains several task features designed to ensure successful manipulation of attention bias occurs. Specifically, participants are shown only one word above or below a fixation cross that cues them to either disengage (negative words) or sustain (positive words) attention, and this is followed by a probe (the letter “E” or “F”) presented in the opposite location of negative words and the same location as positive words. This program also progressively introduces training components designed to increase the attentional demand of the task (e.g., flanked probes, “EEFEE”) and explicitly informs participants of the goal of the task (i.e., to disengage attention from negative words and sustain attention toward positive words) to optimize change in bias. Using this version of the AABM, Nelson et al. found that the ERN was smaller in participants who completed AABM before the ERN was measured relative to participants who completed AABM after the ERN was measured. Furthermore, changes in attentional bias occurred on a continuum such that some participants showed change in their biases away from negative stimuli and toward positive stimuli. Greater attentional disengagement from negative stimuli during AABM was associated with a smaller ERN across both groups, suggesting that ability to disengage attention from threat may be a mechanism and predictor of reduction in the ERN.

Although this study provided support for the hypotheses that the ERN is modulated by AABM, several design features (e.g., lack of a pretest/posttest design; lack of analogous control task) limited the causal conclusions about this relationship. In the second study, Nelson et al. (2017) examined the impact of a single session of the same AABM on the ERN using a pretest/posttest design. Specifically, these researchers measured the ERN in 64 undergraduates before and after they completed either the AABM task from Nelson et al. (2015) or a control task. In the control task, participants did not receive instructions to train attention toward or away from any stimuli; the fixation cross was immediately followed by two words (one neutral, one negative), one presented above and one presented below the fixation cross. Participants were instructed to respond to the probe (the letter “E” or “F”) that appeared behind the negative and neutral words with equal frequency. Participants who completed AABM showed a decrease in their ERN, CRN, and ΔERN (ERN – CRN) from the pre- to posttraining assessment, whereas participants who completed the control task did not show a change in any of the ERP measures from pre- to posttraining assessment. However, several design limitations limit these findings. First, participants completed only a single session of AABM, and it is unclear whether additional administrations would produce greater reductions in the neural correlates of response monitoring. Second, the AABM task included neutral, positive, and negative words, whereas the control task included

only neutral and negative words. It is possible that the presence of positive words may have rendered the AABM task more cognitively demanding than the control task, thereby possibly contributing to the reduced ERN, CRN, and ΔERN. Third, the control task did not include an attention-training aspect. Therefore, it is unclear whether changes in the ERN reflect increased attention toward positive information and/or away from negative information or, rather, a general improvement in attentional control was responsible for the results.

In another study, Carlson et al. (2021) examined the effect of multisession ABM on the ERN in 51 adults ages 18 to 38 (M = 22.08 years, SD = 5.33) with high trait anxiety as measured by the Spielberg State-trait anxiety inventory (Spielberg et al., 1983). Participants completed the Flanker task before and after a 6-week cellphone-delivered ABM or control training. The ABM task contained only trials in which the probe followed the neutral image (i.e., training attention toward neutral stimuli), whereas the control task included the standard dot-probe task (i.e., dot probe follows the neutral and negative images equally). To keep participants engaged across multiple sessions, the program gradually increased in difficulty. During the second week, face and word stimuli appeared alternatively every other session. Stimuli were originally presented for 500 ms in Weeks 1 and 2 and then for 300 ms in subsequent weeks. Starting in Week 4, distractor targets (i.e., other shapes) were also displayed. Results revealed that both the ABM and control groups experienced a significant decrease in attentional bias from pre- to posttraining. However, neither group experienced a significant change in anxiety symptoms or any ERP measures.

Tan et al. (2021) investigated the degree to which attention-bias (ABM) training modulated the ERN and symptom change in 36 youths with OCD ages 8 to 17. Participants completed either a 12-session computerized ABM program or an attentional control protocol (CON) over the course of 4 weeks. Both ABM and CON were dot-probe detection tasks. In the ABM task, the probe always followed neutral words, thereby encouraging youths to disengage from threat cues. In the CON task, the probe followed neutral and negative words with equal frequency, thereby not training spatial attention. These researchers measured the ERN during both a cognitive arrow version of the Flanker task and an emotional Flanker task before and after training. Results revealed that unlike participants who received CON, participants who received ABM showed significantly attenuated neural activity following error and correct responses in an emotional Flanker task. The ERN amplitude during the cognitive Flanker task was unchanged in both ABM and CON groups. Attenuations in the ERN from the emotional Flanker task were correlated with decreases in parent-reported social anxiety and depressive symptoms.

Finally, Klawohn et al. (2020) hypothesized that because individuals with OCD have reliably shown an increased ERN, ABM may be one promising strategy to reduce the ERN in this population. To this end, these researchers asked 32 participants with OCD and 24 control participants to perform a single session of a probe-detection task in a condition that trained attention toward neutral stimuli and away from negative stimuli; another group of 24 control participants performed a sham version of training that did not train spatial attention. The ERN was assessed before and after training. Results revealed significant reduction of initially increased ERN amplitudes in the OCD group after the ABM training but not in

the ABM or sham-control subgroups. Although these studies provide initial support for the hypotheses that ABM modulates the ERN, the lack of a control group in Nelson et al. (2015) and the noncontingent nature of the control condition in the other four studies (Carlson et al., 2021; Klawohn et al., 2020; Nelson et al., 2017; Tan et al., 2021) limit conclusions. For example, it is not clear whether ABM exerts its influence on the ERN by requiring participants to spatially engage or disengage attention from emotional stimuli or whether the critical ingredient is to perform an attention-training task more broadly. Thus, it is possible that ABM exerts its influence by increasing attentional control rather than by decreasing negative attentional bias. Attentional control is considered an important aspect of effortful control, which refers to the ability to inhibit a dominant (habitual) response to perform a subdominant one (Rothbart et al., 2003). This includes flexibility in attention that permits voluntary shifts from one stimulus to another (Derryberry & Reed, 2002; Posner & Rothbart, 2000). In the context of ABM, attentional control may be required to override automatic attention toward negative stimuli to instead direct attention toward a probe in the opposite location. Recent studies have found inverse associations between negative affect and attentional control. For example, several studies have demonstrated that performance on cognitive tasks (e.g., slower reaction times, increased error rates) is altered when subjects are presented with threat-related stimuli (Britton & Anderson, 2021; Vasey & MacLeod, 2001). Moreover, an increased attentional bias to threat-related stimuli has been associated with reduced attentional control in both children (Lonigan et al., 2004; Muris & Ollendick, 2005) and adults (Derryberry & Reed, 2002). Thus, training to reduce attentional bias to threat stimuli may also train general attentional control. Because the typical study has confounded these two factors, it is important to examine a condition in which these factors are examined separately. To this end, it is necessary to devise an ABM condition in which the emotional meaning of the stimuli is irrelevant to the task and instead actively train participants' attention using other aspects of the stimuli to improve their performance. Thus, one possible mechanism involved in the reduction of the ERN following ABM may be the enhancement of general attentional control.

In the current study, we compared two versions of ABM. One was the version used in three previous studies (Amir et al., 2016; Nelson et al., 2015, 2017), which was designed to train attention away from negative stimuli and toward positive stimuli (i.e., the meaning/valence of the word predicted the location of the probe). The second ABM was an active control condition in which participants performed a variation of the emotional Stroop task and ignored the meaning of words and instead used the color of a fixation cue and the location of a word to predict the location of a probe. We compared these two conditions with a waitlist-control condition to examine the effect of each type of training on the ERN and anxiety in a large sample of youths.

The primary aim of the study was to modulate the ERN and examine effects on anxiety. Consistent with this approach, we recruited 647 participants who were 11 to 14 years old and characterized by a robust[1] baseline ERN but varying in levels of anxiety symptoms. We

[1]·Our rational for including only participants with robust ERN was that if we were to target the ERN and the ERN was not robust (i.e., evident, scorable) in a participant, we could not examine the effect of the intervention on this construct. For example, in a study of the effect of a medication on cholesterol levels, participants whose cholesterol cannot be measured would be excluded.

focus on early adolescence—the developmental window consistent with increases in anxiety (Beesdo et al., 2007; Kessler et al., 2005) and when both ERN and anxious symptoms appear to increase in parallel (Meyer et al., 2012; Weinberg et al., 2016). Participants were randomly assigned to a twice-weekly 8-week (i.e., 16 sessions) at-home version of one of two AABM conditions or a waitlist control. In line with previous research showing AABM-related reduction in the ERN, we hypothesized that the ERN would be reduced from pre- to postassessments in AABM groups compared with the waitlist-control group. Consistent with previous research showing the effect of AABM or anxiety, we hypothesized AABM groups would demonstrate reduced anxiety symptoms compared with the waitlist-control group. We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study.

Method

Participants

We recruited a sample of 647 adolescents between the ages 11 and 14 in San Diego (N = 315) and Tallahassee (N = 332) as part of a multisite grant (https://clinicaltrials.gov/ct2/show/NCT03176004) with the preregistered primary outcome as change in child anxiety symptoms. We excluded participants with thought disorder, pervasive developmental disorder, intellectual disability, neurological diseases that impaired cognition, or significant head injuries in the past 3 months based on parent report during a phone-based screener. Because the goal of the current study was to modify the ERN, we excluded a total of 101 participants (15.6%)[2] who did not have a robust ERN. Participants characterized by a present and scorable ERN at baseline (N = 546;[3] San Diego: N = 269; Tallahassee: N = 277) were randomly assigned to one of the two AABM training conditions: emotion-contingent AABM (eAABM; N = 180), color-contingent AABM (cAABM; N = 191), or a waitlist-control condition (N = 175). See Figure 1 for CONSORT chart.

To characterize the sample, we collected age, sex, race, and ethnicity variables from participants. Two participants did not provide demographic data. Participant demographic data for the entire sample and by group (eAABM, cAABM, waitlist) are presented in Table 1. Demographic data by site are presented in the Supplemental Material available online. We also collected parent-reported household income for the full sample[4] (Mdn = $100,000) and by site (San Diego: Mdn = $100,000; Tallahassee: Mdn = $87,500) to estimate the socioeconomic characteristics of the sample.

[2]This is generally in line with the average percentage of participants excluded because of poor data quality or too few errors (7.3%) in previous ERN studies (Meyer et al., 2020; Nelson et al., 2015, 2017; Schroder et al., 2018). In the current study, and in line with prevention trials, individuals with an unidentifiable ERN were excluded.

[3]We performed a priori power analysis to determine a sample of 600 is sufficient to detect small to medium main effects and interactions. All power calculations were based on a final sample of 540, assuming 10% attrition; α was set to .05 for all power analyses. For all correlational analyses, with a final sample of 540, we have adequate power (greater than 99%) to detect a medium effect and adequate power (80%) to detect a small effect. Correlations between ERN and anxiety in both cross-sectional and prospective studies are associated with small to medium effects; thus, the current study is adequately powered.

[4]Sixty-seven participants did not provide income data.

Procedure

At the baseline laboratory visit, participants completed a Flanker task designed to elicit the ERN while we recorded their brain activity with EEG. We then scored the ERN immediately. Adolescents with a robust and identifiable ERN[5] (N = 546) were randomly assigned to either an 8-week (i.e., 16 session) home-training version of eAABM or cAABM or a waitlist condition. All participants completed the same initial practice session of AABM at the baseline visit. At the end of the practice session, study personnel installed the training program on the participants' personal laptop. If they did not have one, a laptop was provided to them for the 8-week training period. Participants were instructed on how to use the at-home program and given the opportunity to ask questions. At the end of the 8 weeks, participants returned to lab and completed the Flanker task again while we recorded EEG. We asked adolescents and their parents to complete self-report measures of anxiety and depression at the baseline and 8-week visits. We compensated participants at a rate of $20 per hour for their participation at pre- and postlab assessments and a $40 bonus for returning for the postassessment. Participants in the eAABM and cAABM groups received an additional $5 per session for completing the training sessions, an extra $5 per 100 levels reached, and a $20 bonus if all 16 training sessions were completed within the 8-week training period. On average, participants completed approximately 11.24 training sessions (eAABM = 11.01; cAABM = 11.46). Groups did not differ in training sessions completed, $t(367.82) = -0.61$, $p = .54$.[6] Descriptive statistics for AABM sessions by group and site are available in the Supplemental Material. Participants provided written informed consent. All procedures were approved by the Institutional Review Board.

Measures

Screen for Child Anxiety Related Emotional Disorders.—The Screen for Child Anxiety Related Emotional Disorders (SCARED) is a 41-item self-report questionnaire that assesses severity of anxiety symptoms in youths between ages 8 and 18. Items are rated on a 3-point scale (0 = *not true or hardly true*, 2 = *very true or often true*). A total score of 25 on the SCARED results in the optimal cutoff point maximizing both sensitivity and specificity when discriminating between anxiety and nonanxiety disorders in clinical populations (Birmaher et al., 1999). In the current sample, 191 (35%) participants met this criterion according to child report, and 87 (16%) met this criterion according to parent

5. We developed a training manual and example waveforms to accomplish this task. The manual outlined three steps to determine whether participants had an identifiable ERN. First, participants must have good overall EEG quality (i.e., no excessive sweat or muscle artifacts; drifting data; excessive eye noise). Second, participants must have committed at least six errors (Olvet & Hajcak, 2009b). Third, visual inspection was used to identify whether participants had a visible ERN at FCz or Cz (a negative deflection peaking within 100 ms of response that is distinguishable from the rest of the ERP). The large majority of the participants were excluded because they lacked a visible ERN. Two had fewer than six errors, and two did not have scorable EEG. To determine whether these steps successfully selected participants based on differences in error-related brain activity, we performed t tests between groups using baseline ERP amplitudes. Participants included in random assignment (N = 546) had a significantly more negative ERN, $t(120.49) = 2.92$, $p < .01$, and ΔERN, $t(111.58) = 8.35$, $p < .001$, than the 101 participants excluded from randomization. These groups did not differ on child-reported, $t(148.04) = -1.13$, $p = .26$, or parent-reported baseline anxiety symptoms, $t(144.56) = -1.46$, $p = .15$. Participants excluded from random assignment made significantly more errors than participants who were randomly assigned (excluded: $M = 57.5$, $SD = 41.2$; included: $M = 43.0$, $SD = 18.6$), $t(104) = 3.44$, $p = .01$. However, the three randomly assigned groups did not differ in their number of errors significantly (eAABM: $M = 44.3$, $SD = 17.9$; cAABM: $M = 42.1$, $SD = 18.7$; waitlist: $M = 42.5$, $SD = 19.2$), $F(2, 581) = 0.45$, $p = .63$.

6. Although, groups did not differ in number of sessions completed. We repeated all analyses with number of sessions included as a covariate. This did not change any of the results reported here.

report.[7] In the current sample, this scale showed excellent internal consistency for child report (α = .94) and parent report (α = .93) at baseline.

Child Depression Inventory.—We also administered the child and parent versions of the Child Depression Inventory (CDI) at baseline and 8-week assessments. The CDI is a 27-item child-report depression-symptom questionnaire with good reliability, good convergent and construct validity, and moderate discriminant validity. Items are presented as three statements of varying symptom severity (i.e., "I am sad once in a while," "I am sad many times," "I am sad all the time"; Finch et al., 1985). Cutoff scores range from 11 to 19 (M = 14.5, SD = 2.67; Stockings et al., 2015). In the current sample, this scale showed excellent internal consistency for child (α = .90) and parent report (α = .86) at baseline.

Flanker task.—The Flanker task was administered using Presentation software (Neurobehavioral Systems Inc., Albany, CA). During the task, participants see five horizontally aligned white arrowheads and are asked to press the left or right mouse button as quickly as possible depending on the direction of the central arrowhead. In this task, there are two flanker "compatible" conditions ("<<<<<" and ">>>>>") and two flanker "incompatible" conditions ("<<><<" and ">><>>"). The stimuli are presented randomly such that 50% are incompatible and 50% are compatible. Each stimulus is presented for 200 ms, and the interval between the offset of one stimulus and the onset of the subsequent stimulus varies randomly between 2300 ms and 2800 ms. Participants completed a practice block of 30 trials during which they were instructed to be both as accurate and as fast as they can. The actual task comprises 11 blocks of 30 trials (330 trials total), and each block is initiated by the participant. To encourage both fast and accurate responding, participants received feedback based on their performance at the end of each block. If performance was 75% correct or lower, the message "Please try to be more accurate" was displayed; performance above 90% correct was followed by "Please try to respond faster"; otherwise, the message "You're doing a great job" was displayed.

AABM programs.—The AABM task was identical to the task used in our previous research (Amir et al., 2016, Nelson et al., 2015, 2017). Specifically, both AABM programs (a) showed only one word above or below a fixation cross that cued participants to either disengage attention (negative words in the eAABM condition or words following a red fixation cross regardless of the word's emotional content in the cAABM condition) or sustain attention (positive words in the eAABM condition or words following a green fixation cross regardless of the word's emotional content in the cAABM condition), (b) used ideographic stimuli (i.e., five negative, five positive, and five neutral words) generated by the

[7]·In the current study, parent- and child-reported total SCARED scores were moderately correlated, $r(540) = .53$, $p < .001$. This is in line with a recent meta-analysis that found an average weighted correlation coefficient of .488 (SE = .014, 95% confidence interval [CI] = [0.466, 0.509]) for parent–child agreement of the SCARED (Runyon et al., 2018). Parents generally report less frequent symptoms than their child (Cosi et al., 2010), which may be partly explained by characteristics of internalizing disorders, such as anxiety, that are more difficult for parents to perceive than those of externalizing disorders (Klein, 1991; Muris et al., 1999). Given this evidence, we included analyses of both parent and child reports; however, it is not surprising that we see some discrepancy. Cosi et al. (2010) compared child- and parent-reported SCARED scores to symptoms of a structured diagnostic interview (the M.I.N.I. Kid; Sheehan et al., 1998) and found higher correlations with child report for all anxiety categories. Therefore, we used the child-reported anxiety as the preregistered outcome in the current study.

participant, and (c) contained multiple training components and adjusted the criteria, within persons, to improve their attentional bias over the course of training.

Before AABM, participants were asked to provide 25 ideographically selected words and rate their emotional valence on a 5-point scale. Participants were instructed to choose 10 words that made them feel good, happy, or excited; 10 words that made them feel scared, mad, or unhappy; and five words that made them feel neither happy nor upset. Experimenters then selected the first five positive words that were rated “very happy,” the first five negative words that were rated “very upset,” and all five neutral words. These 15 words were then entered into the AABM program for each participant. The same words were used for the at-home training and 8-week lab visit. The AABM comprised two phases. The practice phase was completed by all participants at their initial laboratory assessment and was a single session designed to familiarize participants with the program while gradually introducing more attentionally demanding elements (e.g., flanking letters). Next, we asked participants to complete a computerized home-based training comprising 16 AABM sessions completed over 8 weeks (i.e., two sessions per week).

Each session of the program comprised 240 trials. Each trial began with a fixation cross presented in the center of the screen for 500 ms. Immediately following termination of the fixation cross, a single word (neutral, positive, or negative) appeared either above or below the fixation cross for 500 ms. After presentation of the word, a probe (either the letter “E” or “F”) appeared above or below the fixation cross. In the practice phase (i.e., Levels 1–30), participants were instructed to simply click the left mouse button when they saw the letter “E” and the right mouse button when they saw the letter “F.” See Figure 2 for task diagram. Participants progressed in levels by consistently performing accurately during the task. We gradually increased the difficulty of the program at Level 5 by instructing all participants, in both the eAABM and cAABM conditions, to use the color of the fixation cross to predict the location of the probe. Participants were instructed that when the fixation cross was green, the probe letter would appear in the same location as the word and when the fixation cross was red, the letter probe would appear in the opposite location as the word. At Level 10, only participants in the eAABM condition were instructed to use the valence of the cue word to predict the location of the probe. Specifically, the negative words always served as invalid cues (i.e., the probe always appeared in the location opposite the location of the negative word), and the positive words always served as valid cues (i.e., the probe always appeared in the same location as the positive word). Furthermore, as participants in the eAABM condition progressed through Levels 11 through 20, the color of fixation cross faded to white, encouraging them to rely on the valence of the word to predict the location of the probe. For participants in the cAABM condition, the color of the fixation cross continued to predict the location of the probe (i.e., participants were never instructed to use the meaning of the word to predict probe location). Thus, the fixation cross always appeared in color in the cAABM condition, and the probe appeared in the location of a negative or positive word with equal frequency. At Level 20, the task became more attentionally demanding for both eAABM and cAABM: The probe (“E” or “F”) was flanked by either congruent (i.e., “EEEEE” or “FFFFF”) or incongruent (i.e., “EEFEE” or “FFEFF”) letters, and participants were told to respond only to the middle letter. This required participants to increase their focus on the center letter. Overall, the practice phase

was designed to familiarize participants with the procedure of AABM and prepare them for the training phase. Advancing through the practice phase depended accurate performance, not reaction-time measures. All participants completed the full practice phase at the baseline laboratory visit. For images of task instructions presented to participants during the practice phase, see the Supplemental Material.

All participants completed their training (Levels 30+ of the program) at home. Participants in the eAABM condition continued to complete the same type of ABM as the end of the practice phase (i.e., white fixation cross, using word valence to predict location of the probe, responding to the middle letter and not flanked letters). Participants in the cAABM condition continued to complete the same adaptive attention-control task (i.e., using red or green fixation cross to predict location of the probe in relation to the word, responding to the middle letter and not flanked letters). Participants in both groups increased their level in the program by improving their bias (i.e., increasing positive or reducing negative bias for eAABM group and improving color bias for cAABM group) relative to the cumulative bias of all preceding trials.

We calculated negative attention bias by subtracting the average response latency for neutral invalid trials (i.e., probes following neutral words in the opposite location as the word) from the average response latency for negative invalid trials (i.e., probes following negative words in the opposite location). A smaller negative bias indicated faster disengagement from negative cues compared with neutral cues. We calculated positive bias by subtracting the average response latency for neutral valid trials (i.e., probes following neutral words in the same location as the word) from the average response latency for positive valid trials (i.e., probes following positive words in the same location). Finally, we calculated color bias for participants in the cAABM group by subtracting the average response latency for invalid trials (i.e., trials with a red fixation cross) from the average response latency on valid trials (i.e., trials with a green fixation cross). Average response latency on each trial was calculated using the cumulative sum of response time on all previous trials divided by number of trials, but only for correct trials and for trials within 2 *SD* of the participants' mean response latency. As expected, we found the split-half reliability of participant attention bias scores to be very high ($r = .99$) because bias scores on a single trial always reflected the entire set of response latencies to that point. Thus, bias at each trial included all trials up to that point.

Because the program was adaptive and depended on participant performance to move up in levels, it was possible for the game to become frustrating if the intended change in bias did not occur. To ensure participants remained engaged in the task and increase the chance that bias would improve, the program included a recalibration feature that lowered the difficulty of the program if participants were not advancing in levels. Recalibration occurred if progress did not occur after 100 trials. During recalibration, participants were instructed to take a 5-min break, and the program used their current bias level as the criterion for improving after the break. At the end of each AABM session, the program saved the participant's data as a game file with information about their bias and last level reached. Thus, if a participant ended a session at Level 53, then they would simply reload their existing game at the next session and start at Level 53, progressing in level based on improvement on last saved bias scores. Thus, the goal of this AABM design is for

participants to continue to improve their attention bias by informing them of current level in the program and accounting for individual performance on the task (Amir et al., 2016; Nelson et al., 2015, 2017).

Finally, although the current AABM task has not been used in samples of youths, several ABM paradigms have been used in other studies of anxiety in youths and adolescents (Britton et al., 2013; Lowther & Newman, 2014; Ollendick et al., 2019; Pergamin-Hight et al., 2016; Pettit et al., 2020). The current AABM program contained additional features intended to make the program engaging to a young adolescent population. The program was presented as a game designed to train their brain. Age-friendly pop-ups were used throughout the task to inform participants of their within-sessions progress (i.e., every time they moved up in 10 levels; when to take a break). We developed a homepage for the program, and on it, participants could track their progress over 8 weeks by filling in puzzle pieces of a session tracker. Each time participants completed a session, they filled in one of the 16 pieces. Finally, the game included a leadership board that tracked the levels reached by each participant in the study. Thus, participants could compare their progress with the progress of their peers. For images of program features, see the Supplemental Material.

EEG recording and processing.—We collected EEG data from 32 active electrodes placed on an EasyCap (EasyCap GmbH, n.d.) that were amplified and digitized with an actiCHamp amplifier (Brain Vision ActiChamp System, 2016) at a sampling rate of 1000 HZ and referenced online to Cz with ground placed at Fpz. We located and placed mastoid electrodes independently of the cap and used their average as the offline reference. We placed two passive electrooculagram electrodes above and below the left eye to detect eyeblinks and two additional electrodes near the outer canthus of both eyes to detect lateral eye movement. We analyzed the EEG data using Brain Vision Analyzer (BVA; Brain Products, Gilching, Germany). Using BVA, we rereferenced the data offline to averaged mastoids, band-pass filtered (0.1–30 HZ), and corrected for blinks and horizontal eye movements (Gratton et al., 1983). Finally, we extracted response-locked epochs with a duration of 1,500 ms, including a 500-ms preresponse interval. Epochs containing a voltage greater than 50 μV between sample points, a voltage difference of 300 μV within a segment, or a maximum voltage difference of less than 0.50 μV within 100-ms intervals were rejected. Additional artifacts were identified and removed based on visual inspection. The −500 to −300ms preresponse interval was used as the baseline. Trials with response times below 200 ms and above 700 ms were excluded from averaging. Both the ERN and the negative deflection on correct trials (i.e., the CRN) were quantified as the mean amplitude between 0 ms and 100 ms after responses at electrode FCz, where the ERN was maximal. To isolate neural activity specific to errors, we also analyzed the difference between the ERN and CRN (i.e., ΔERN; Simons, 2010). The average number of errors committed was 42.96 (*SD* = 18.61). In our sample, the ERN (α = .88), the CRN (α = .97), and the ΔERN ($\rho DD'$ = .72)[8] showed good to excellent internal consistency. Figure 3 presents the grand-average response-locked ERPs at FCz for error, correct, and the error minus correct difference.

8.To calculate the internal consistency of the ΔERN, we used the equation from Clayson et al. (2021).

In addition, behavioral measures including the number of error and correct trials at each assessment and average reaction times on error and correct trials were calculated separately.

Statistical methods and data analytical plan—The primary registered outcome for this study was reduction of anxiety symptoms as measured by self-report measure (SCARED; https://clinicaltrials.gov/ct2/show/NCT03176004). Secondary outcomes included the ΔERN, CDI, and negative-attentional-bias scores. We used an intent-to-treat (ITT) approach with no imputation multilevel longitudinal data analytic strategy, modeling scores of each participant at each assessment as a Level 1 variable. We then used the slope and intercept of each subject's data in Level 2 and used group to predict the slopes and intercepts for each dependent measure (Singer & Willett, 2003).

The analyses using linear mixed effects accounts for missing values at all time points (Little & Yau, 1996). The mixed-model and ITT approach has several strengths: (a) It can accommodate missing data points in longitudinal data, (b) the approach does not need to have the same number of observations per subject, and (c) time can be continuous rather than a fixed set of points. To analyze change in each measure, we contrast-coded the grouping variable comparing participants receiving the eAABM condition or cAABM with participants in the waitlist-control condition (Rosnow & Rosenthal, 1996). For all models, we treated time as a random factor and compared the same models with time as a fixed factor. In each model, we included time as either a fixed factor or a random factor (Singer & Willet, 2003). The models with time as a random factor had a better fit to the data than the models with time as a fixed factor. Thus, we modeled time as a random factor and group as a fixed factor in all analyses. We assessed the effect of condition on primary and secondary outcome scores via hierarchical linear mixed-effects regression using the *lme4* package in R (Version 3.4.1; Bates et al., 2014).

Next, we performed completers analysis by calculating the residual score between baseline and 8 weeks for each of our primary and secondary outcomes measures. Using the residual, we submitted each of our primary and secondary outcomes measures to a one-way (group: eAABM, cAABM, waitlist) analysis of variance (ANOVA). The analyses of completers include only participants who returned for the 8-week visit. The completers analysis provides an estimate of the true efficacy of an intervention (i.e., among participants who completed the treatment as planned); however, effects can be exaggerated relative to ITT.

At baseline, the eAABM, cAABM, and waitlist groups did not differ significantly on our primary outcome measure, child-reported SCARED, $F(2, 540) = 0.42$, $p = .66$, $\eta_p^2 = .002$; parent-reported SCARED, $F(2, 542) = 0.38$, $p = .69$, $\eta_p^2 = .001$, or any of our secondary outcomes measures, child-reported CDI, $F(2, 540) = 0.50$, $p = .61$, $\eta_p^2 = .002$; parent-reported CDI, $F(2, 542) = 1.22$, $p = .30$, $\eta_p^2 = .004$; negative bias, $F(2, 519) = 0.91$, $p = .40$, $\eta_p^2 = .003$; ERN, $F(2, 540) = 1.19$, $p = .30$, $\eta_p^2 = .004$; CRN, $F(2, 540) = 0.78$, $p = .46$, $\eta_p^2 = .003$; ΔERN, $F(2, 540) = 0.24$, $p = .79$, $\eta_p^2 = .001$. Descriptive statistics for each measure at baseline for the full sample are presented in Table 2. Descriptive statistics for participants that completed both the baseline and 8-week assessments are presented in Table 3. Descriptive statistics for additional measures (e.g., behavioral data from the Flanker task) for baseline and 8 weeks are available in the Supplemental Material.

We conducted site analyses by comparing participants on our primary and secondary outcome measures across sites. To account for any baseline difference between sites, we conducted "change from baseline" analyses using the residual between participants' scores at baseline and 8 weeks for each measure (European Medicines Agency, 2015, p. 7). Descriptive statistics reported by site for each measure at baseline and 8 weeks are presented in the Supplemental Material.

Transparency and openness—We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study, and we follow JARS (Appelbaum et al., 2018). All data and analysis code are available on OSF at https://osf.io/r9xp8/.

Results

Change in bias

Because one of the goals of AABM was to promote learning through change in bias, we calculated the slope of change in bias (emotional bias in the case of eAABM and color bias in the case of cAABM) across levels of the game, showing that both groups showed the expected learning. For the eAABM group, mean of the slope was −0.41 (SD = 1.48) and differed significantly from zero, $t(179) = 3.74$, $p < .001$, $d = 0.56$. For the cAABM group, the mean of the slope was −0.55 (SD = 1.36) and differed significantly from zero, $t(190) = 5.56$, $p < .001$, $d = 0.81$. For the graphical representation of the slopes of change, see the Supplemental Material.

ΔERN

To isolate neural activity specific to errors, we examined the ΔERN (ERN – CRN).[9] Using the mixed-linear-model analysis and ITT approach with the ΔERN as the dependent variable comparing the cAABM group versus the waitlist, we found that the analysis did not reveal a main effect of time ($b = 0.06$, $SE = 0.27$, $df = 450$, $t = 0.23$, $p = .82$, $d = -0.02$) or group ($b = 0.05$, $SE = 0.61$, $df = 544$, $t = 0.08$, $p = .93$, $d = 0.01$). However, we did find a significant interaction of time and group ($b = 1.61$, $SE = 0.64$, $df = 450$, $t = 2.53$, $p = .01$, $d = 0.24$) such that the cAABM had a significantly greater reduction in ΔERN than the waitlist from baseline to 8 weeks. For the analysis comparing eAABM with cAABM, we did not find a main effect of time ($b = -0.09$, $SE = 0.27$, $df = 450$, $t = -0.34$, $p = .73$, $d = -0.03$) or group ($b = -0.35$, $SE = 0.61$, $df = 544$, $t = -0.58$, $p = .56$, $d = -0.05$). However, there was a significant interaction of time and group ($b = 1.48$, $SE = 0.65$, $df = 450$, $t = 2.28$, $p = .02$, $d = 0.21$) such that the cAABM had a significantly greater reduction in ΔERN than the eAABM group from baseline to 8 weeks. For the analysis comparing eAABM with waitlist, analyses did not show a significant main effect of time ($b = -0.02$, $SE = 0.27$, $df = 450$, $t = -0.06$, $p = .95$, $d = 0.01$), main effect of group ($b = 0.44$, $SE = 0.63$, $df = 544$, $t = 0.70$, $p = .49$, $d = 0.06$), or interaction of time and group ($b = 0.26$, $SE = 0.67$, $df = 450$, $t = 0.40$, $p = .69$, $d = 0.04$).

9. We report results separately for the ERN and CRN in the Supplemental Material.

To conduct completer analysis, we submitted residual ΔERN change scores to a one-way ANOVA and found a significant main effect of group, $F(2, 449) = 4.53$, $p = .01$, $\eta^2 = .02$. Pairwise t tests between groups revealed that the cAABM group showed a significantly larger drop in ΔERN than the eAABM group, $t(293.11) = -2.33$, $p = .02$, $d = -0.273$, and the waitlist, $t(305.74) = 2.74$, $p < .01$, $d = 0.314$.[10] The difference between the eAABM group and waitlist, $t(286.29) = 0.54$, $p = .59$, $d = 0.064$, was not significant. See Figure 4.

SCARED

Child version.—Comparing the eAABM group with the cAABM group, we found that the main effect of group was not significant ($b = 1.41$, $SE = 1.44$, $df = 544$, $t = -0.98$, $p = .33$, $d = -0.08$). However, we found a significant main effect of time ($b = -4.46$, $SE = 0.41$, $df = 459$, $t = -10.94$, $p < .001$, $d = -1.02$) that was modified by an interaction of time and group ($b = 2.20$, $SE = 1.00$, $df = 459$, $t = 2.21$, $p = .03$, $d = 0.21$) such that the eAABM group had a significantly steeper reduction in SCARED than the cAABM group between baseline and 8 weeks.[11] When we compared the eAABM group and cAABM group with the waitlist group, analyses revealed a significant main effect of time (eAABM vs. waitlist: $b = -4.41$, $SE = 0.41$, $df = 459$, $t = -10.82$, $p < .001$, $d = -1.01$; cAABM vs. waitlist: $b = -4.39$, $SE = 0.41$, $df = 459$, $t = -10.75$, $p < .001$, $d = 1.00$) but not a significant main effect of group (eAABM vs. waitlist: $b = -0.46$, $SE = 1.47$, $df = 544$, $t = 0.31$, $p = .75$, $d = 0.03$; cAABM vs. waitlist: $b = -0.97$, $SE = 1.44$, $df = 544$, $t = -0.67$, $p = .50$, $d = 0.06$) or interaction of time and group (eAABM vs. waitlist: $b = -1.56$, $SE = 1.01$, $df = 459$, $t = -1.54$, $p = .12$, $d = 0.14$; cAABM vs. waitlist: $b = 0.68$, $SE = 0.98$, $df = 459$, $t = 0.70$, $p = .49$, $d = 0.07$).

To conduct completer analysis, we submitted residual SCARED change scores to a one-way ANOVA. These analyses revealed a significant main effect of group, $F(2, 458) = 3.13$, $p = .045$, $\eta^2 = .013$). Pairwise t tests between groups revealed that the eAABM group showed a significantly larger drop in SCARED scores than the cAABM group, $t(301.14) = 2.55$, $p = .01$, $d = 0.294$.[12] The differences between the eAABM group and waitlist group, $t(296.96) = 1.50$, $p = .14$, $d = 0.174$, and cAABM group and waitlist group, $t(316.44) = -1.01$, $p = .31$, $d = -0.114$, were not significant. See Figure 5.

Parent version.—We repeated the same ITT and completers analyses using the parent-reported SCARED scores. We did not find any significant main effect for group or interaction of time and group. Results are reported in the Supplemental Material.

[10] Although the requirement of time was missing from a formal mediation model, we examined the impact of condition on ERN via attention-control reduction (Tingley et al., 2014). The effect of group (cAABM vs. eAABM or cAABM vs. waitlist) on change in ERN was not mediated via the change in attention control.

[11] When running the mixed-linear-model analysis including site (Tallahassee, San Diego) with SCARED as the dependent variable, we found a significant main effect of site at baseline such that San Diego participants were more anxious overall, which interacted with time such that overall, San Diego participants experienced a greater reduction in anxiety over 8 weeks. However, we did not find any interaction between site and group; thus, we excluded site from further analysis.

[12] Although the requirement of time was missing from a formal mediation model, we examined the impact of condition on anxiety via ERN reduction (Tingley et al., 2014). The effect of group (eAABM, cAABM) on change in SCARED was partially mediated via the change in ERN. The indirect effect was 0.24. We tested the significance of this indirect effect using bootstrapping procedures (95% CI = [–0.02, 0.64], $p = .08$). Similar analysis using negative bias score ($p = .54$) and attention control ($p = .62$) also failed to find evidence of mediation. Finally, we examined the moderation of the impact of condition on anxiety by age. Age did not moderate this relationship ($p = .17$).

CDI—We repeated the same ITT and completers analyses using the child- and parent-reported CDI scores. We did not find any significant main effect for group or interaction of time and group. Results are reported in the Supplemental Material.

Negative bias—We used the above-described mixed-linear-model analysis and ITT approach with negative attention bias as the dependent variable comparing the eAABM and waitlist groups. This analysis did not reveal a main effect of time ($b = 1.20$, $SE = 2.40$, $df = 390$, $t = 0.50$, $p = .62$, $d = 0.05$) or group ($b = 1.85$, $SE = 5.20$, $df = 533$, $t = 0.36$, $p = .72$, $d = 0.03$), but it did reveal a significant interaction of time and group ($b = -12.71$, $SE = 6.10$, $df = 390$, $t = -2.08$, $p = .04$, $d = -0.21$) such that the eAABM group had a larger reduction in negative bias than the waitlist between baseline and 8 weeks. For the analysis comparing cAABM and waitlist groups, we did not find a main effect of time ($b = 0.81$, $SE = 2.44$, $df = 390$, $t = 0.33$, $p = .74$, $d = 0.03$) or group ($b = -4.76$, $SE = 5.08$, $df = 533$, $t = -0.94$, $p = .35$, $d = -0.08$) or interaction of time and group ($b = 1.60$, $SE = 5.98$, $df = 390$, $t = 0.27$, $p = .79$, $d = 0.03$). For the analysis comparing eAABM and cAABM groups, we did not find a main effect of time ($b = 0.28$, $SE = 2.42$, $df = 390$, $t = 0.12$, $p = .91$, $d = 0.01$) or group ($b = -6.35$, $SE = 4.99$, $df = 533$, $t = -1.27$, $p = .20$, $d = -0.11$), but it did reveal a significant interaction of time and group ($b = 11.83$, $SE = 5.70$, $df = 390$, $t = 2.07$, $p = .04$, $d = 0.21$) such that the eAABM group had a greater reduction in negative bias than the cAABM group between baseline and 8 weeks.

To conduct completer analysis, we submitted residual negative-bias-change scores to a one-way ANOVA and found a significant main effect of group, $F(2, 389) = 3.83$, $p = .02$, $\eta^2 = .02$. Pairwise t tests between groups revealed that the eAABM group showed a significantly larger drop in negative bias than the cAABM group, $t(290.48) = 2.27$, $p = .02$, $d = 0.266$, and the waitlist group, $t(227.61) = 2.67$, $p < .01$, $d = 0.354$. The difference between the cAABM group and waitlist group, $t(246.87) = 0.33$, $p = .74$, $d = 0.042$, was not significant. See Figure 6.

Attention control—The flanker task can also provide a measure of executive attention control (Fan et al., 2002), calculated by subtracting mean response latency for congruent trials (center arrow in the same direction as the flanking arrows) from mean response latency for incongruent trials (center arrow in the opposite direction as the flanking arrows) for correct trials. We used the above-described mixed-linear-model analysis and ITT approach with attention control as the dependent variable comparing the cAABM and waitlist groups. This analysis did not reveal a main effect of group ($b = -4.33$, $SE = 3.53$, $df = 544$, $t = -1.23$, $p = .22$, $d = -0.10$). However, we found a main effect of time ($b = -18.92$, $SE = 1.26$, $df = 450$, $t = -14.96$, $p < .001$, $d = -1.41$) that was modified by a significant interaction of time and group ($b = -8.72$, $SE = 3.04$, $df = 450$, $t = -2.86$, $p < .01$, $d = -0.27$) such that the cAABM group had a larger increase in attention control than the waitlist group between baseline and 8 weeks. For the analysis comparing cAABM and eAABM, we did not find a significant main effect of group ($b = 1.55$, $SE = 3.51$, $df = 544$, $t = 0.44$, $p = .66$, $d = 0.04$). However, we found a main effect of time ($b = -18.73$, $SE = 1.27$, $df = 450$, $t = -14.73$, $p < .001$, $d = -1.39$) that was modified by a significant Time × Group interaction ($b = -8.50$, $SE = 3.10$, $df = 450$, $t = -2.74$, $p < .01$, $d = -0.26$) such that the cAABM group had a larger

increase in attention control than the eAABM group between baseline and 8 weeks. For the analyses comparing eAABM and waitlist, we found a significant main effect of time (b = −19.20, SE = 1.28, df = 450, t = −15.05, p < .001, d = −1.42). However, we did not find a main effect of group (b = −6.36, SE = 3.68, df = 544, t = −1.77, p = .08, d = −0.15) or a significant Time × Group interaction (b = −1.05, SE = 3.18, df = 450, t = −0.33, p = .74, d = −0.03).

To conduct completer analysis, we submitted residual attention-control-change scores to a one-way ANOVA and found a marginally significant main effect of group, $F(2, 449)$ = 2.43, p = .09, η^2 = .011). Pairwise t tests between groups revealed that the cAABM group showed a significantly larger increase in attention control than the eAABM group, $t(281.4)$ = −1.97, p = .05, d = −0.235). The difference between the cAABM and waitlist was marginally significant, $t(313.93)$ = 1.73, p = .08, d = 0.196. The difference between the eAABM group and waitlist group, $t(270.34)$ = −0.41, p = .68, d = −0.050, was not significant. See Figure 7.

Discussion

In the current study, we aimed to modify normative changes in the ERN—a neural measure of risk for subsequent increases in anxiety. Because previous studies have found that an elevated ERN in childhood and adolescence predicts future increase in anxiety symptoms, we aimed to modify the ERN in a sample of adolescents characterized by a robust and measurable ERN. Because previous studies have shown that ABM successfully modifies the ERN (Klawohn et al., 2020; Nelson et al., 2015, 2017; Tan et al., 2021), we compared two AABM procedures: one focused on modulating spatial allocation away from negative cues and toward positive cues (eAABM) and the other focused on modulating attention using nonemotional features (i.e., color of the cue; cAABM); these were both compared with a waitlist-control group in terms of their impact on ERN, anxiety symptoms, and levels of negative attentional bias and attention control. Finally, the AABM was delivered at participants' home. This is a considerable strength of the study because one of the promises of ABM has been its scalability potential, but many past studies were ineffective once tried remotely.

We found that cAABM reduced ΔERN compared with the waitlist control and eAABM groups. This finding suggests that the ERN may be modified by attention-control training broadly, as opposed to training specifically designed to direct spatial attention away from negative stimuli and toward positive stimuli. During cAABM, participants must use the color of the fixation cross to determine where to shift attention following presentation of a negative, positive, or neutral word. It is possible that requiring participants to train their attention on a speeded reaction-time task while ignoring potentially distracting task-irrelevant stimuli allowed participants to train top-down attentional-control processes (i.e., cognitive flexibility, goal-directed inhibitory control; Mogg et al., 2017), in turn reducing the ERN. Consistent with this hypothesis, we found that the cAABM condition was associated with an increase in an independent behavioral index of attention control obtained from the Flanker task. Future research should test whether attention training in the presence of emotional stimuli modifies ERN in clinical anxiety. Finally, our examination of the mediating effect of attention control on the ERN showed only marginally significant results.

There are at least two explanations for this finding. First, our mediator, change in attention control, may have lacked sufficient psychometric properties to assess the construct of interest. Second, the mechanism thought which group assignment may have affected the ERN may be different than attention control.

On the other hand, we found that eAABM reduced the severity of child-reported anxiety symptoms across 8 weeks compared with cAABM. However, change in anxiety symptoms did not differ between the eAABM and waitlist groups; indeed, the strongest effects on anxiety were found over time—such that all three groups experienced a reduction in child-reported anxiety symptoms. Moreover, no group effects were found in parent-reported anxiety symptoms. Again, our examination of the mediating effect of attention bias, ERN, and attention control on the anxiety did not result in significant mediation for any of these variables. Similar explanations may have resulted in this lack of mediation, lack of sufficient psychometric properties of mediations, or different mechanism thought that group assignment may have affected anxiety.

Although we obtained cAABM-related changes in ERN, we did not observe corresponding changes in anxiety. It is possible that changes in anxiety would follow in a longer timescale than changes in the ERN. That is, modifying the ERN using the cAABM may reduce subsequent risk for increases in anxiety—a possibility consistent with longitudinal studies that have found that a potentiated ERN predicts increases in anxiety (Meyer, 2017). A longer-term follow-up is needed to measure whether reduction of the ERN is a protective factor against increases in anxiety symptoms. Indeed, we are currently collecting follow-up data 2 years after the baseline visit to address this possibility.

There are key differences between eAABM and cAABM when interpreting this pattern of results. In the more traditional eAABM, negative versus positive words inform that the probe will occur in either the opposite or same position as the word, respectively; participants learn to quickly shift attention when the cue is negative or maintain attention when the cue is positive. Thus, participants must attend to the valence of the cue words on each trial to improve their performance on the eAABM task. On the other hand, the valence of the word cue is completely irrelevant in the cAABM task. Rather, participants must focus on the fixation cue color and the spatial location of the word to improve performance while ignoring whether the word is negative, positive, or neutral in emotional content. It is possible that cAABM requires more cognitive control as far as it requires individuals to override their prepotent tendency to attend to emotional content regardless of task relevance (Bretherton et al., 2020; Mikhael et al., 2021; Sawaki & Luck, 2010). In fact, attending to the emotional valence of the word is counterproductive during cAABM because doing so would reduce participant performance (i.e., slower reaction time, reduced accuracy). If this is the case, it is possible that ERN can be manipulated by more demanding cognitive-control interventions. Future studies are required to fully test this possibility.

Researchers have attempted to modulate the ERN using different methods, and studies have shown that traditional cognitive-behavioral-therapy (CBT) approaches do not seem to affect the ERN (Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). For example, Hajcak et al. (2008) found that participants who completed exposure

therapy for OCD and responded to this “gold-standard” treatment continued to show an increased ERN following treatment. Ladouceur et al. (2018) found similar results showing that CBT decreased symptoms of anxiety but did not affect the ERN. Thus, the ERN does not seem to be affected by CBT.

Other more recent attempts have used a brief, computerized intervention directly targeting error sensitivity to reduce the ERN. Meyer et al. (2020) examined the extent to which a brief, computerized intervention (“Treating the ERN” [TERN]) might affect the ERN by reducing error sensitivity in 39 undergraduates. These researchers found that TERN reduced the ΔERN with an effect size of .48. Likewise, Schroder et al. (2018) used a tailored intervention—expressive writing—in an attempt to reduce the ERN among a sample of individuals with chronic worry. These researchers found that the ERN was reduced in the expressive-writing group compared with an unrelated writing-control group with an effect size of .33. However, this latter effect size was for an uncontrolled effect because this study did not employ a pre-post design. In the current study, we demonstrate that an attention-control training was effective in reducing the ΔERN with an effect size of .24. The extent to which these procedures affect the same population or can be targeted to individuals awaits further study.

Our study has limitations. First, we did not report longer-term follow-up to examine the longevity of any of the obtained results. Second, we did not examine the moderating effect of all demographic variables (e.g., sex) on the obtained results. We plan to assess these moderators of outcomes in subsequent studies and results from our 2-year follow-up assessment. Third, our examination of the moderating effect of attention control provided only partial support for the effect of group on ERN and none for the effects on anxiety. Therefore, the examinations of causal mechanisms that may be responsible for our effects await future research using different or more robust mediators. Fourth, our registered primary outcome measures were ERN and anxiety. Therefore, the results regarding other measures (CDI, bias, attention control) should be considered preliminary and in need of replication. Fifth, the aim of the current project was to examine the impact of AABM on a biomarker for anxiety and not anxiety itself. This is a weakness from a clinical perspective because we did not use anxiety as an inclusion criteria.

However, following a reviewer’s suggestion, we conducted a one-way ANOVA on residual SCARED change scores in youths who report SCARED scores over 25. As expected, this group showed a larger decrease in anxiety (eAABM: $M = 9.82$, $SD = 9.55$; cAABM: $M = 7.0$, $SD = 10.7$; waitlist: $M = 6.78$, $SD = 11.5$) compared with the entire sample (eAABM: $M = 5.74$, $SD = 7.91$; cAABM: $M = 3.24$, $SD = 9.08$; waitlist: $M = 4.04$, $SD = 9.20$). However, when we submitted residual SCARED change scores for this subsample to a one-way ANOVA, the group differences were not statistically significant, $F(2, 153) = 1.54$, $p = .218$ $\eta^2 = .02$. Nevertheless, the effect size was larger for this subsample. We are happy to include this analysis at the editor’s discretion. Finally, the current study compared two versions of an adaptive ABM that contain several design features absent in other, more traditional versions of ABM. Thus, the current findings may not generalize to more traditional ABM programs.

These limitations notwithstanding, this study provided initial support for the utility of two distinct forms of AABM in modulating the ERN, which has been linked to vulnerability to increases in later anxiety.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We thank Alessandro D'Amico and Miguel Montero for their contribution to data collection and preliminary data analysis.

Funding

This project was funded by National Institute Mental Health Grant R01MH106477 awarded to G. Hajcak and N. Amir.

References

Amir N, Kuckertz JM, & Strege MV (2016). A pilot study of an adaptive, idiographic, and multi-component attention bias modification program for social anxiety disorder. Cognitive Therapy and Research, 40(5), 661–671. 10.1007/s10608-016-9781-1 [PubMed: 27795598]

Amir N, Weber G, Beard C, Bomyea J, & Taylor CT (2008). The effect of a single-session attention modification program on response to a public-speaking challenge in socially anxious individuals. Journal of Abnormal Psychology, 117(4), 860–868. 10.1037/a0013445 [PubMed: 19025232]

Appelbaum M, Cooper H, Kline RB, Mayo-Wilson E, Nezu AM, & Rao SM (2018). Journal article reporting standards for quantitative research in psychology: The APA Publications and Communications Board task force report. The American Psychologist, 73(1), 3–25. 10.1037/amp0000191 [PubMed: 29345484]

Bar-Haim Y. (2010). Research review: Attention bias modification (ABM): A novel treatment for anxiety disorders. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 51(8), 859–870. 10.1111/j.1469-7610.2010.02251.x [PubMed: 20456540]

Bates D, Machler M, & Walker S. (2014) Fitting linear mixed-effects models using Ime4. Journal of Statistical Software, 67(1), 1–48.

Beard C. (2011). Cognitive bias modification for anxiety: Current evidence and future directions. Expert Review of Neurotherapeutics, 11(2), 299–311. 10.1586/ern.10.194 [PubMed: 21306216]

Beesdo K, Bittner A, Pine DS, Stein MB, Höfler M, Lieb R, & Wittchen HU (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. Archives of General Psychiatry, 64(8), 903–912. 10.1001/archpsyc.64.8.903 [PubMed: 17679635]

Beesdo K, Knappe S, & Pine DS (2009). Anxiety and anxiety disorders in children and adolescents: Developmental issues and implications for DSM-V. Psychiatric Clinics of North America, 32(3), 483–524. 10.1016/j.psc.2009.06.002 [PubMed: 19716988]

Birmaher B, Brent DA, Chiappetta L, Bridge J, Monga S, & Baugher M. (1999). Psychometric properties of the Screen for Child Anxiety Related Emotional Disorders (SCARED): A replication study. Journal of the American Academy of Child and Adolescent Psychiatry, 38(10), 1230–1236. 10.1097/00004583-199910000-00011 [PubMed: 10517055]

Bishop SJ (2008). Neural mechanisms underlying selective attention to threat. Annals of the New York Academy of Sciences, 1129, 141–152. 10.1196/annals.1417.016 [PubMed: 18591476]

Bittner A, Egger HL, Erkanli A, Jane Costello E, Foley DL, & Angold A. (2007). What do childhood anxiety disorders predict? Journal of Child Psychology and Psychiatry, and Allied Disciplines, 48, 1174–1183. 10.1111/j.1469-7610.2007.01812.x [PubMed: 18093022]

Brain Vision ActiChamp System. (2016). Brain Products GmbH.

Bretherton PM, Eysenck MW, Richards A, & Holmes A. (2020). Target and distractor processing and the influence of load on the allocation of attention to task-irrelevant threat. Neuropsychologia, 145, Article 106491. 10.1016/j.neuropsychologia.2017.09.009

Britton JC, Bar-Haim Y, Clementi MA, Sankin LS, Chen G, Shechner T, Norcross MA, Spiro CN, Lindstrom KM, & Pine DS (2013). Training-associated changes and stability of attention bias in youth: Implications for Attention Bias Modification Treatment for pediatric anxiety. Developmental Cognitive Neuroscience, 4, 52–64. 10.1016/j.dcn.2012.11.001 [PubMed: 23200784]

Britton MK, & Anderson BA (2021). Attentional avoidance of threatening stimuli. Psychological Research, 85(1), 82–90. 10.1007/s00426-019-01255-6 [PubMed: 31605204]

Brown CS, & Amir N. (2022). The moderating effect of anxiety diagnosis on the relationship between error-related negativity and anxiety sensitivity cognitive concerns. Biological Psychology, 175, Article 108443. 10.1016/j.biopsycho.2022.108443

Browning M, Holmes EA, & Harmer CJ (2010). The modification of attentional bias to emotional information: A review of the techniques, mechanisms, and relevance to emotional disorders. Cognitive, Affective & Behavioral Neuroscience, 10(1), 8–20. 10.3758/CABN.10.1.8

Carlson J, Fang L, & Andrzejewski J. (2021). No change in electrocortical measures of performance monitoring in high trait anxious individuals following multi-session attention bias modification training. NeuroImage: Reports, 1(4), Article 100067. 10.1016/j.ynirp.2021.100067

Clayson PE, Baldwin SA, & Larson MJ (2021). Evaluating the internal consistency of subtraction-based and residualized difference scores: Considerations for psychometric reliability analyses of event-related potentials. Psychophysiology, 58(4), Article 13762. 10.1111/psyp.13762

Cosi S, Canals J, Hernández-Martinez C, & Vigil-Colet A. (2010). Parent-child agreement in SCARED and its relationship to anxiety symptoms. Journal of Anxiety Disorders, 24(1), 129–133. 10.1016/j.janxdis.2009.09.008 [PubMed: 19864109]

Derryberry D, & Reed MA (2002). Anxiety-related attentional biases and their regulation by attentional control. Journal of Abnormal Psychology, 111(2), 225–236. 10.1037//0021-843x.111.2.225 [PubMed: 12003445]

EasyCap GmbH. (n.d.). https://www.easycap.de/products/

Endrass T, Riesel A, Kathmann N, & Buhlmann U. (2014). Performance monitoring in obsessive-compulsive disorder and social anxiety disorder. Journal of Abnormal Psychology, 123(4), 705–714. 10.1037/abn0000012 [PubMed: 25286372]

Eriksen B, & Eriksen C. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task;. Attention, Perception, & Psychophysics, 16(1), 143–149. 10.3758/bf03203267

European Medicines Agency. (2015). Adjustment for baseline covariates in clinical trials (Reference No. EMA/CHMP/295050/2013). https://www.ema.europa.eu/en/adjustment-baseline-covariates-clinical-trials

Falkenstein M, Hoormann J, Christ S, & Hohnsbein J. (2000). ERP components on reaction errors and their functional significance: A tutorial. Biological Psychology, 51(2–3), 87–107. 10.1016/s0301-0511(99)00031-9 [PubMed: 10686361]

Fan J, McCandliss BD, Sommer T, Raz A, & Posner MI (2002). Testing the efficiency and independence of attentional networks. Journal of Cognitive Neuroscience, 14(3), 340–347. 10.1162/089892902317361886 [PubMed: 11970796]

Finch AJ Jr., Saylor CF, & Edwards GL (1985). Children's depression inventory: Sex and grade norms for normal children. Journal of Consulting and Clinical Psychology, 53(3), 424–425. 10.1037//0022-006x.53.3.424 [PubMed: 4008727]

Gehring WJ, Goss B, Coles MGH, Meyer DE, & Donchin E. (1993). A neural system for error detection and compensation. Psychological Science, 4(6), 385–390. 10.1111/j.1467-9280.1993.tb00586.x

Gratton G, Coles MGH, & Donchin E. (1983). A new method for off-line removal of ocular artifact. Electroencephalography and Clinical Neurophysiology, 55(4), 468–484. 10.1016/0013-4694(83)90135-9 [PubMed: 6187540]

Hajcak G, Franklin ME, Foa EB, & Simons RF (2008). Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. American Journal of Psychiatry, 165(1), 116–123. 10.1176/appi.ajp.2007.07010143 [PubMed: 17986681]

Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, & Walters EE (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry, 62(6), 593–602. 10.1001/archpsyc.62.6.593 [PubMed: 15939837]

Klawohn J, Hajcak G, Amir N, Kathmann N, & Riesel A. (2020). Application of attentional bias modification training to modulate hyperactive error-monitoring in OCD. International Journal of Psychophysiology, 156, 79–86. 10.1016/j.ijpsycho.2020.07.005 [PubMed: 32711018]

Klein RG (1991). Parent–child agreement in clinical assessment of anxiety and other psychopathology: A review. Journal of Anxiety Disorders, 5, 187–198.

Kujawa A, Weinberg A, Bunford N, Fitzgerald KD, Hanna GL, Monk CS, Kennedy AE, Klumpp H, Hajcak G, & Phan KL (2016). Error-related brain activity in youth and young adults before and after treatment for generalized or social anxiety disorder. Progress in Neuro-Psychopharmacology & Biological Psychiatry, 71, 162–168. 10.1016/j.pnpbp.2016.07.010 [PubMed: 27495356]

Ladouceur CD, Dahl RE, Birmaher B, Axelson DA, & Ryan ND (2006). Increased error-related negativity (ERN) in childhood anxiety disorders: ERP and source localization. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 47(10), 1073–1082. 10.1111/j.1469-7610.2006.01654.x [PubMed: 17073986]

Ladouceur CD, Tan PZ, Sharma V, Bylsma LM, Silk JS, Siegle GJ, Forbes EE, McMakin DL, Dahl RE, Kendall PC, Mannarino A, & Ryan ND (2018). Error-related brain activity in pediatric anxiety disorders remains elevated following individual therapy: A randomized clinical trial. Journal of Child Psychology and Psychiatry, 59(11), 1152–1161. 10.1111/jcpp.12900 [PubMed: 29603219]

Lahat A, Lamm C, Chronis-Tuscano A, Pine DS, Henderson HA, & Fox NA (2014). Early behavioral inhibition and increased error monitoring predict later social phobia symptoms in childhood. Journal of the American Academy of Child and Adolescent Psychiatry, 53(4), 447–455. 10.1016/j.jaac.2013.12.019 [PubMed: 24655654]

Little R, & Yau L. (1996). Intent-to-treat analysis for longitudinal studies with drop-outs. Biometrics, 52(4), 1324–1333. [PubMed: 8962456]

Lonigan CJ, Vasey MW, Phillips BM, & Hazen RA (2004). Temperament, anxiety, and the processing of threat-relevant stimuli. Journal of Clinical Child and Adolescent Psychology, 33(1), 8–20. 10.1207/S15374424JCCP3301_2 [PubMed: 15028537]

Lowther H, & Newman E. (2014). Attention bias modification (ABM) as a treatment for child and adolescent anxiety: A systematic review. Journal of Affective Disorders, 168, 125–135. 10.1016/j.jad.2014.06.051 [PubMed: 25046738]

MacLeod C, Rutherford E, Campbell L, Ebsworthy G, & Holker L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. Journal of Abnormal Psychology, 111, 107–123. 10.1037/0021-843X.111.1.107 [PubMed: 11866165]

Mancebo MC, Boisseau CL, Garnaat SL, Eisen JL, Greenberg BD, Sibrava NJ, Stout RL, & Rasmussen SA (2014). Long-term course of pediatric obsessive-compulsive disorder: 3 years of prospective follow-up. Comprehensive Psychiatry, 55(7), 1498–1504. 10.1016/j.comppsych.2014.04.010 [PubMed: 24952937]

Martinelli A, Grüll J, & Baum C. (2022). Attention and interpretation cognitive bias change: A systematic review and meta-analysis of bias modification paradigms. Behaviour Research and Therapy, 157, Article 104180. 10.1016/j.brat.2022.104180

Mathews A, & MacLeod C. (2005). Cognitive vulnerability to emotional disorders. Annual Review of Clinical Psychology, 1, 167–195. 10.1146/annurev.clinpsy.1.102803.143916

McDermott JM, Perez-Edgar K, Henderson HA, Chronis-Tuscano A, Pine DS, & Fox NA (2009). A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. Biological Psychiatry, 65(5), 445–448. 10.1016/j.biopsych.2008.10.043 [PubMed: 19108817]

Meyer A. (2017). A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. Developmental Cognitive Neuroscience, 27, 58–68. 10.1016/j.dcn.2017.08.001 [PubMed: 28818707]

Meyer A, Gibby B, Wissemann K, Klawohn J, Hajcak G, & Schmidt NB (2020). A brief, computerized intervention targeting error sensitivity reduces the error-related negativity. Cognitive, Affective & Behavioral Neuroscience, 20(1), 172–180. 10.3758/s13415-019-00760-w

Meyer A, & Hajcak G. (2019). A review examining the relationship between individual differences in the error-related negativity and cognitive control. International Journal of Psychophysiology, 144, 7–13. 10.1016/j.ijpsycho.2019.07.005 [PubMed: 31362030]

Meyer A, Hajcak G, Torpey DC, Kujawa A, Kim J, Bufferd S, Carlson G, & Klein DN (2013). Increased error-related brain activity in six-year-old children with clinical anxiety. Journal of Abnormal Child Psychology, 41(8), 1257–1266. 10.1007/s10802-013-9762-8 [PubMed: 23700171]

Meyer A, Hajcak G, Torpey-Newman DC, Kujawa A, & Klein DN (2015). Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. Journal of Abnormal Psychology, 124(2), 266–274. 10.1037/abn0000044 [PubMed: 25643204]

Meyer A, Mehra L, & Hajcak G. (2021). Error-related negativity predicts increases in anxiety in a sample of clinically anxious female children and adolescents over 2 years. Journal of Psychiatry & Neuroscience, 46(4), E472–E479. 10.1503/jpn.200128 [PubMed: 34346200]

Meyer A, Nelson B, Perlman G, Klein DN, & Kotov R. (2018). A neural biomarker, the error-related negativity, predicts the first onset of generalized anxiety disorder in a large sample of adolescent females. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 59(11), 1162–1170. 10.1111/jcpp.12922 [PubMed: 29665048]

Meyer A, Weinberg A, Klein DN, & Hajcak G. (2012). The development of the error related negativity (ERN) and its relationship with anxiety: Evidence from 8 to 13 year-olds. Developmental Cognitive Neuroscience, 2(1), 152–161. 10.1016/j.dcn [PubMed: 22308177]

Mikhael S, Watson P, Anderson BA, & Le Pelley ME (2021). You do it to yourself: Attentional capture by threat-signaling stimuli persists even when entirely counterproductive. Emotion, 21(8), 1691–1698. 10.1037/emo0001003 [PubMed: 34843309]

Mogg K, Waters AM, & Bradley BP (2017). Attention Bias Modification (ABM): Review of effects of multisession ABM training on anxiety and threat-related attention in high-anxious individuals. Clinical Psychological Science, 5(4), 698–717. 10.1177/2167702617696359 [PubMed: 28752017]

Moser JS, Hajcak G, & Simons RF (2005). The effects of fear on performance monitoring and attentional allocation. Psychophysiology, 42(3), 261–268. 10.1111/j.1469-8986.2005.00290.x [PubMed: 15943679]

Moser JS, Moran TP, Schroder HS, Donnellan MB, & Yeung N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. Frontiers in Human Neuroscience, 7, Article 466. 10.3389/fnhum.2013.00466

Muris P, Merckelbach H, Van Brakel A, & Mayer B. (1999). The revised version of the screen for child anxiety related emotional disorders (SCARED-R): Further evidence for its reliability and validity. Anxiety, Stress, & Coping, 12, 411–425. [PubMed: 21777069]

Muris P, & Ollendick TH (2005). The role of temperament in the etiology of child psychopathology. Clinical Child and Family Psychology Review, 8(4), 271–289. 10.1007/s10567-005-8809-y [PubMed: 16362256]

Nelson BD, Jackson F, Amir N, & Hajcak G. (2015). Single-session attention bias modification and error-related brain activity. Cognitive, Affective & Behavioral Neuroscience, 15(4), 776–786. 10.3758/s13415-015-0365-4

Nelson BD, Jackson F, Amir N, & Hajcak G. (2017). Attention bias modification reduces neural correlates of response monitoring. Biological Psychology, 129, 103–110. 10.1016/j.biopsycho.2017.08.059 [PubMed: 28867538]

Ollendick TH, White SW, Richey J, Kim-Spoon J, Ryan SM, Wieckowski AT, Coffman MC, Elias R, Strege MV, Capriola-Hall NN, & Smith M. (2019). Attention bias modification treatment for adolescents with social anxiety disorder. Behavior Therapy, 50(1), 126–139. 10.1016/j.beth.2018.04.002 [PubMed: 30661553]

Olvet DM, & Hajcak G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. Clinical Psychology Review, 28(8), 1343–1354. 10.1016/j.cpr.2008.07.003 [PubMed: 18694617]

Olvet DM, & Hajcak G. (2009a). Reliability of error-related brain activity. Brain Research, 1284, 89–99. 10.1016/j.brainres.2009.05.079 [PubMed: 19501071]

Olvet DM, & Hajcak G. (2009b). The stability of error-related brain activity with increasing trials. Psychophysiology, 46(5), 957–961. 10.1111/j.1469-8986.2009.00848.x [PubMed: 19558398]

Pergamin-Hight L, Pine DS, Fox NA, & Bar-Haim Y. (2016). Attention bias modification for youth with social anxiety disorder. Journal of Child Psychology and Psychiatry, and Allied Disciplines, 57(11), 1317–1325. 10.1111/jcpp.12599 [PubMed: 27435286]

Pettit JW, Bechor M, Rey Y, Vasey MW, Abend R, Pine DS, Bar-Haim Y, Jaccard J, & Silverman WK (2020). A randomized controlled trial of attention bias modification treatment in youth with treatment-resistant anxiety disorders. Journal of the American Academy of Child & Adolescent Psychiatry, 59, 157–165. [PubMed: 30877049]

Pine DS, Cohen P, Gurley D, Brook J, & Ma Y. (1998). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. Archives of General Psychiatry, 55, 56–64. 10.1001/archpsyc.55.1.56 [PubMed: 9435761]

Posner MI (1980). Orienting of attention. The Quarterly Journal of Experimental Psychology, 32(1), 3–25. 10.1080/00335558008248231 [PubMed: 7367577]

Posner MI, & Rothbart MK (2000). Developing mechanisms of self-regulation. Development and Psychopathology, 12(3), 427–441. 10.1017/s0954579400003096 [PubMed: 11014746]

Rabinak CA, Holman A, Angstadt M, Kennedy AE, Hajcak G, & Phan KL (2013). Neural response to errors in combat-exposed returning veterans with and without post-traumatic stress disorder: A preliminary event-related potential study. Psychiatry Research, 213(1), 71–78. 10.1016/j.pscychresns.2013.01.002 [PubMed: 23684979]

Riesel A, Endrass T, Auerbach LA, & Kathmann N. (2015). Overactive performance monitoring as an endophenotype for obsessive-compulsive disorder: Evidence from a treatment study. The American Journal of Psychiatry, 172(7), 665–673. 10.1176/appi.ajp.2014.14070886 [PubMed: 25783756]

Rosnow RL, & Rosenthal R. (1996). Computing contrasts, effect sizes, and counternulls on other people's published data: General procedures for research consumers. Psychological Methods, 1(4), 331–340. 10.1037/1082-989X.1.4.331

Rothbart MK, Ellis LK, Rueda MR, & Posner MI (2003). Developing mechanisms of temperamental effortful control. Journal of Personality, 71(6), 1113–1143. 10.1111/1467-6494.7106009 [PubMed: 14633060]

Runyon K, Chesnut SR, & Burley H. (2018). Screening for childhood anxiety: A meta-analysis of the screen for child anxiety related emotional disorders. Journal of Affective Disorders, 240, 220–229. 10.1016/j.jad.2018.07.049 [PubMed: 30081293]

Sawaki R, & Luck SJ (2010). Capture versus suppression of attention by salient singletons: Electrophysiological evidence for an automatic attend-to-me signal. Attention, Perception, & Psychophysics, 72(6), 1455–1470. 10.3758/APP.72.6.1455

Schroder HS, Moran TP, & Moser JS (2018). The effect of expressive writing on the error-related negativity among individuals with chronic worry. Psychophysiology, 55(2), Article 10.1111/psyp.12990. 10.1111/psyp.12990

Shackman AJ, Salomons TV, Slagter HA, Fox AS, Winter JJ, & Davidson RJ (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. Nature Reviews Neuroscience, 12, 154–167. 10.1038/nrn2994 [PubMed: 21331082]

Sheehan D, Lecrubier Y, Sheehan K, Amorim P, Janavs J, Weiller E, Hergueta T, Baker R, & Dunbar GC (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. The Journal of Clinical Psychiatry, 59(Suppl. 20), 22–33.

Simons RF (2010). The way of our errors: Theme and variations. Psychophysiology, 47(1), 1–14. 10.1111/j.1469-8986.2009.00929.x [PubMed: 19929897]

Singer JD, & Willett JB (2003). Applied longitudinal data analysis: Modeling change and event occurrence. Oxford University Press. 10.1093/acprof:oso/9780195152968.001.0001

Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, & Jacobs GA (1983). Manual for the state-trait anxiety inventory. Consulting Psychologists Press.

Stockings E, Degenhardt L, Lee YY, Mihalopoulos C, Liu A, Hobbs M, & Patton G. (2015). Symptom screening scales for detecting major depressive disorder in children and adolescents: A systematic review and meta-analysis of reliability, validity and diagnostic utility. Journal of Affective Disorders, 174, 447–463. 10.1016/j.jad.2014.11.061 [PubMed: 25553406]

Tan PZ, Rozenman M, Chang SW, Jurgiel J, Truong HV, Piacentini J, & Loo SK (2021). The ERN as a neural index of changes in performance monitoring following attention training in pediatric obsessive-compulsive disorder. Biological Psychology, 166, Article 108206. 10.1016/j.biopsycho.2021.108206

Taylor CT, Aupperle RL, Flagan T, Simmons AN, Amir N, Stein MB, & Paulus MP (2014). Neural correlates of a computerized attention modification program in anxious subjects. Social Cognitive and Affective Neuroscience, 9(9), 1379–1387. 10.1093/scan/nst128 [PubMed: 23934417]

Tingley D, Yamamoto T, Hirose K, Keele L, & Imai K. (2014). mediation: R package for causal mediation analysis. Journal of Statistical Software, 59(5), 1–38. 10.18637/jss.v059.i05 [PubMed: 26917999]

Vaidyanathan U, Nelson LD, & Patrick CJ (2012). Clarifying domains of internalizing psychopathology using neurophysiology. Psychological Medicine, 42(3), 447–459. 10.1017/S0033291711001528 [PubMed: 21854683]

Vasey MW, & MacLeod C. (2001). Information-processing factors in childhood anxiety: A review and developmental perspective. In Vasey MW & Dadds MR (Eds.), The developmental psychopathology of anxiety (pp. 253–277). Oxford University Press.

Weinberg A, Dieterich R, & Riesel A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. International Journal of Psychophysiology, 98(2, Pt. 2), 276–299. 10.1016/j.ijpsycho.2015.02.029 [PubMed: 25746725]

Weinberg A, & Hajcak G. (2011). Longer term test-retest reliability of error-related brain activity. Psychophysiology, 48(10), 1420–1425. 10.1111/j.1469-8986.2011.01206.x [PubMed: 21496055]

Weinberg A, Klein DN, & Hajcak G. (2012). Increased error-related brain activity distinguishes generalized anxiety disorder with and without comorbid major depressive disorder. Journal of Abnormal Psychology, 121(4), 885–896. 10.1037/a0028270 [PubMed: 22564180]

Weinberg A, Meyer A, Hale-Rude E, Perlman G, Kotov R, Klein DN, & Hajcak G. (2016). Error-related negativity (ERN) and sustained threat: Conceptual framework and empirical evaluation in an adolescent sample. Psychophysiology, 53(3), 372–385. 10.1111/psyp.12538 [PubMed: 26877129]

Weinberg A, Olvet DM, & Hajcak G. (2010). Increased error-related brain activity in generalized anxiety disorder. Biological Psychology, 85(3), 472–480. 10.1016/j.biopsycho.2010.09.011 [PubMed: 20883743]

Wittchen H, Lieb R, Pfister H, & Schuster P. (2000). The waxing and waning of mental disorders: Evaluating the stability of syndromes of mental disorders in the population. Comprehensive Psychiatry, 2, 122–132.

Xiao Z, Wang J, Zhang M, Li H, Tang Y, Wang Y, Fan Q, & Fromson JA (2011). Error-related negativity abnormalities in generalized anxiety disorder and obsessive-compulsive disorder. Progress in Neuro-Psychopharmacology & Biological Psychiatry, 35(1), 265–272. 10.1016/j.pnpbp.2010.11.022 [PubMed: 21111024]

Fig. 1.
CONSORT flow diagram.

Fig. 2.
Adaptive Attention-Bias-Modification (AABM) task diagram.

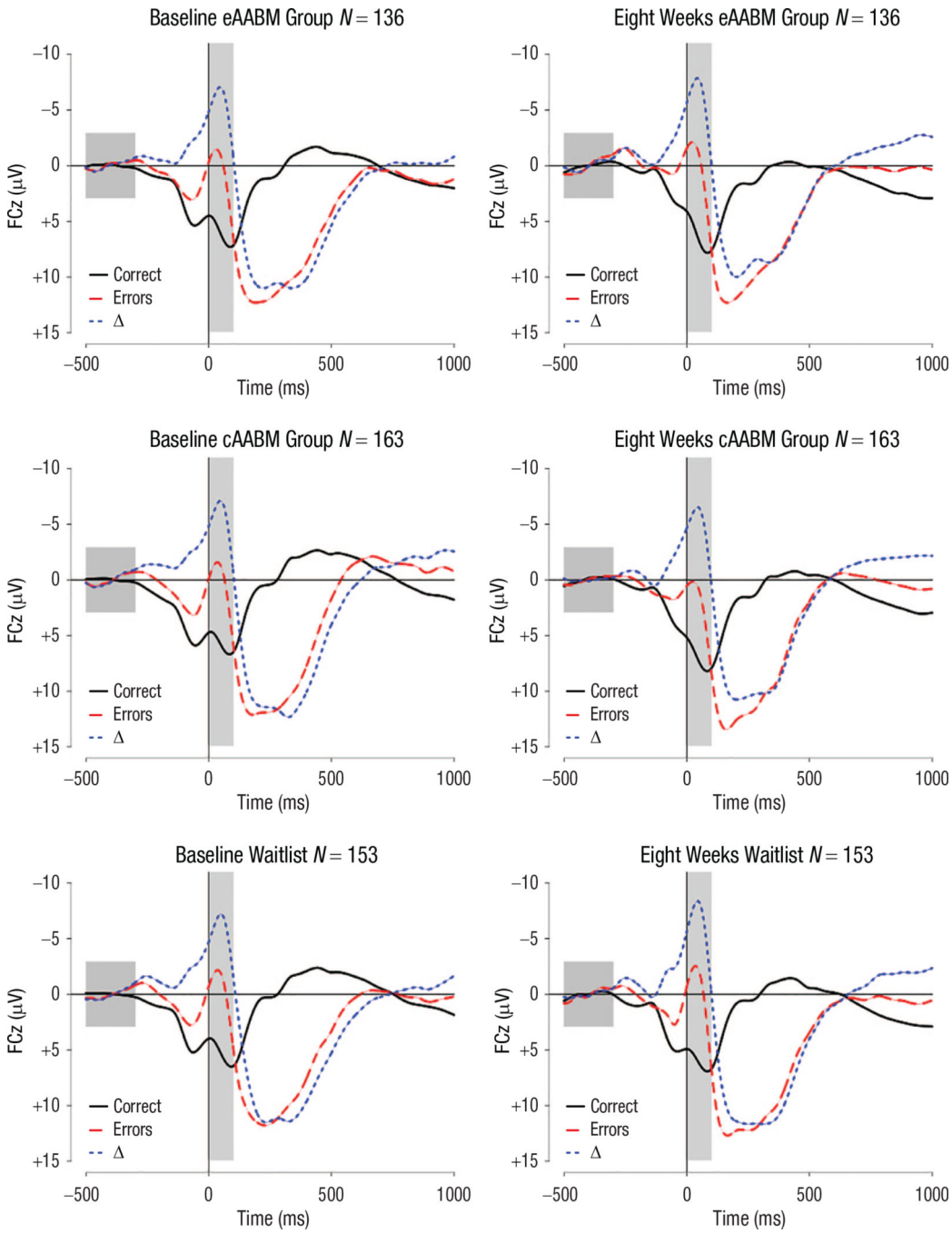

Fig. 3.
Grand average event-related-potential waveforms at FCz.

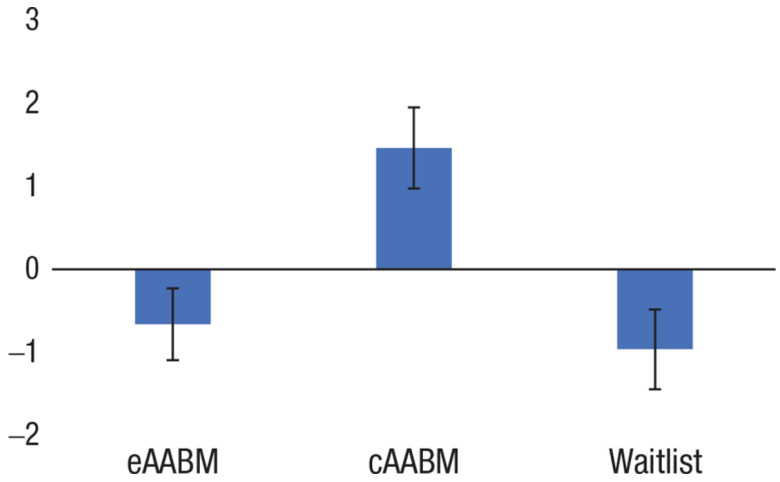

Fig. 4.

Residual change in ΔERN values (8 weeks – Baseline) for eAABM, cAABM, and waitlist groups. ERN = error-related negativity; eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification.

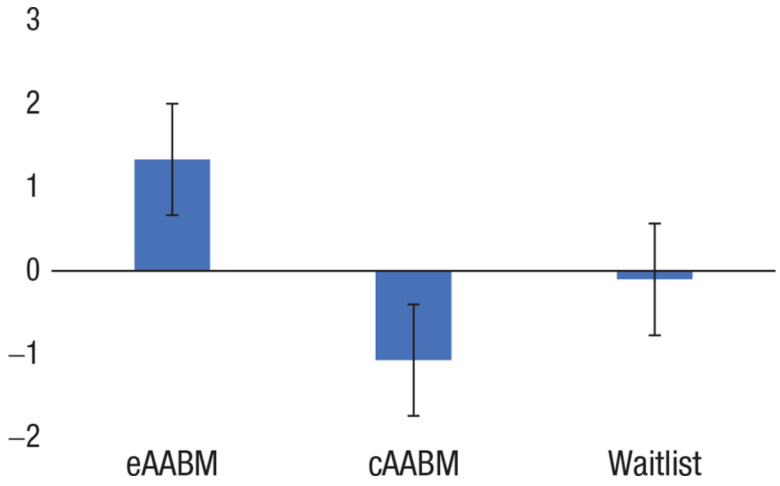

Fig. 5.

Residual change in child-reported SCARED scores (Baseline – 8 weeks) for eAABM, cAABM, and waitlist groups. SCARED = Screen for Child Anxiety Related Emotional Disorders; eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification.

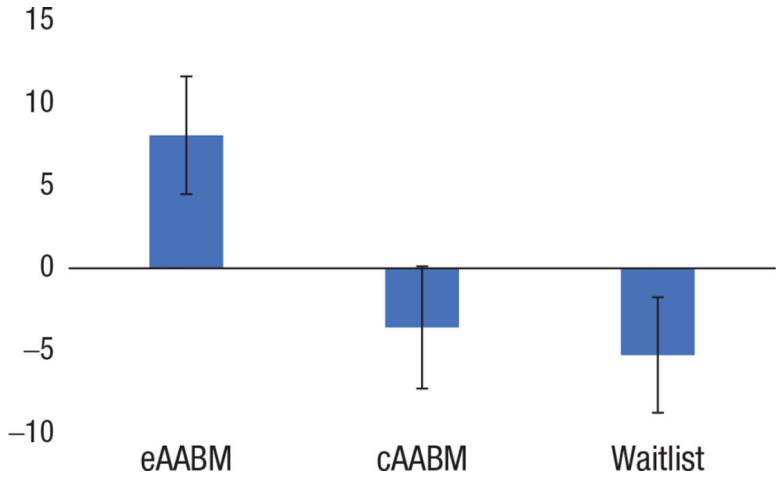

Fig. 6.

Residual change in negative-bias scores (Baseline – 8 weeks) for eAABM, cAABM, and waitlist groups. eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification.

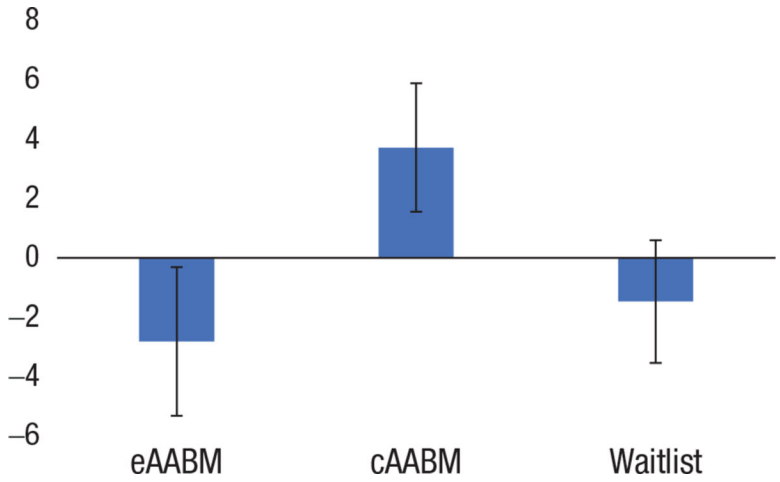

Fig. 7.

Residual change in attention control (Baseline – 8 weeks) for eAABM, cAABM, and waitlist groups. eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification.

Table 1.

Demographics

Characteristics	Participants			
	Total (*N* = 544)	eAABM (*n* = 179)	cAABM (*n* =190)	Waitlist (*n* = 175)
Age[a]	12.4 (1.1)	12.5 (1.1)	12. 4 (1.1)	12.5 (1.1)
Sex[b]				
Female	257 (47.2)	86 (48.0)	90 (47.4)	81 (46.3)
Male	287 (52.8)	93 (52.0)	100 (52.6)	94 (53.7)
Race[b]				
White	376 (69.1)	119 (66.5)	129 (67.9)	128 (73.1)
African American	38 (7.0)	15 (8.4)	12 (6.3)	11 (6.3)
Black	12 (2.2)	6 (3.4)	2 (1.1)	4 (2.3)
Asian	17 (3.1)	9 (5.0)	5 (2.6)	3 (1.7)
Hispanic	21 (3.9)	7 (3.9)	11 (5.8)	3 (1.7)
Native Hawaiian/Pacific Islander	4 (0.7)	0	3 (1.6)	1 (0.6)
Native American	1 (0.2)	0	0	1 (0.6)
Two or more	37 (6.8)	14 (7.8)	14 (7.4)	9 (5.1)
Other	13 (2.4)	2 (1.1)	7 (3.7)	4 (2.3)
Unknown	25 (4.6)	7 (3.9)	7 (3.7)	11 (6.3)
Ethnicity[b]				
Hispanic	115 (21.1)	36 (20.1)	48 (25.3)	31 (17.7)
Non-Hispanic	417 (76.7)	139 (77.7)	140 (73.7)	138 (78.9)
Unknown	12 (2.2)	4 (2.2)	2 (1.1)	6 (3.4)

Note: Demographic data were not available for two participants. eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification.

[a]Age (in years) is provided as an average with standard deviations in parentheses.

[b]Data are shown as number (percentage) of participants.

Table 2.

Descriptive Statistics at Baseline for Clinical Assessment Measures: Full Sample

	eAABM			cAABM			Waitlist		
Measure	***n***	***M***	***SD***	***n***	***M***	***SD***	***n***	***M***	***SD***
Child-reported SCARED	178	21.6	14.76	190	20.34	13.64	175	21.28	12.99
ΔERN[a]	178	–5.38	5.94	191	−5.7	6.03	174	−5.78	5.64
Attention control[b]	178	73.7	36.1	191	75.5	32.0	174	80.1	33.0
Negative bias[b]	172	–0.47	45.46	188	−7.08	52.18	162	−2.69	43.54

Note: eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification; SCARED = Screen for Child Anxiety Related Emotional Disorders; ΔERN = ERN minus correct-response negativity; attention control = incongruent trial reaction time minus congruent trial reaction time (correct trials only).

[a]Data show in μV.

[b]Data shown in milliseconds.

Table 3.

Descriptive Statistics for Clinical Assessment Measures: Completers Sample

	eAABM			cAABM			Waitlist		
Measure Time	***n***	***M***	***SD***	***n***	***M***	***SD***	***n***	***M***	***SD***
Child-reported SCARED									
Baseline	142	21.48	13.97	162	19.41	13.12	157	21.06	12.84
8 weeks	142	15.75	12.84	162	16.17	13.01	157	17.02	12.7
ΔERN[a]									
Baseline	136	−5.43	5.92	163	−5.47	5.91	153	−5.69	5.8
8 weeks	136	−5.92	5.2	163	−4.64	6.22	153	−6.39	6.46
Attention control[b]									
Baseline	136	74.17	36.45	163	−76.01	32.65	153	80.98	34.03
8 weeks	136	57.43	29.98	163	51.29	24.76	153	64.57	23.94
Negative bias[b]									
Baseline	135	5.22	42.08	158	−5.62	46.05	99	−6.54	38.0
8 weeks	135	−5.7	32.5	158	−1.74	29.49	99	1.87	32.22

Note: eAABM = emotion-contingent adaptive variant attention-bias modification; cAABM = color-contingent adaptive variant attention-bias modification; SCARED = Screen for Child Anxiety Related Emotional Disorders; ΔERN = ERN minus correct-response negativity; attention control = incongruent trial reaction time minus congruent trial reaction time (correct trials only).

[a]Data show in μV.

[b]Data shown in milliseconds.