DO UNIQUE MECHANISMS UNDERLIE THE EXPRESSION OF ATTENTION PROBLEMS IN ANXIOUS AND INATTENTIVE-IMPULSIVE YOUTH? IMPLICATIONS FOR DIFFERENTIAL DIAGNOSIS AND TREATMENT

by

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ABSTRACT OF THE DISSERTATION

Do Unique Mechanisms Underlie the Expression of Attention Problems in Anxious and Inattentive-Impulsive Youth? Implications for Differential Diagnosis and Treatment

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Accumulating evidence suggests that unique mechanisms may underlie the expression of attention problems in youth Attention-Deficit/Hyperactivity Disorder (AD/HD) and anxiety disorders (e.g., AD/HD-Inattentive Type and Generalized Anxiety Disorder). Kendall (2000) proposed that anxiety may be associated predominantly with emotion-based “distortions” in cognitive processing (e.g., misappraisal of the social/interpersonal environment, attentional bias toward perceived threat/danger), while inattention in AD/HD youth may be linked to more global cognitive “deficiencies” (e.g., selective/sustained attention, inhibitory control; Barkley, 1997). The current study compared performance of anxious (ANX; n=21; 8-17 years), inattentive-impulsive (I-I; n=22, 9-16 years), and typically developing children (NC; n=22; 8-13 years) on neurocognitive tests of both general (Stroop Color-Word Test, SCW; Conners' Continuous Performance Test, CPT) and emotion-based attentional processes (Emotional Stroop, ES; Faces Dot Probe Task, FDP). As hypothesized, I-I demonstrated poorer sustained attention and inhibitory control, as evidenced by lower CPT commission error raw scores, relative to ANX and NC, and a non-significant trend toward higher CPT omission error T-scores, relative to ANX. In addition, ANX demonstrated superior selective attention, relative to I-I, as indicated by higher SCW raw scores (i.e., more items completed in 45 seconds), higher SCW T-scores, and fewer SCW errors. As predicted, ANX demonstrated greater attentional bias toward threat cues, relative to I-I, as indicated by greater FDP bias scores in response to angry faces. No significant group differences were found in bias scores on happy or sad trials. In addition, ANX showed a trend toward significant "absolute bias" scores (i.e., relative to zero) in response to angry faces alone, suggesting a potential emotion-specific attentional bias toward threat cues in anxious youth; I-I exhibited an "absolute bias" toward sad faces, alone. ES bias scores were not significant and did not distinguish between groups. The findings provide initial evidence for the neuropsychological differentiation of attention problems in anxious (i.e., threat-related attentional bias) and inattentive-impulsive children (i.e., general selective/sustained attention), suggesting the potential utility of cognitive assessment as an aid for differential diagnosis and subsequent treatment of youth anxiety and AD/HD.

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Introduction

In the evolving landscape of clinical-translation science, the clinical applications of neuropsychology remain largely unexplored. Few studies have established disorder-specific brain-behavior links, and studies examining the utility of neurocognitive techniques in the differential diagnosis and treatment of childhood psychopathology are virtually absent from the literature. Accumulating evidence suggests that unique mechanisms may underlie the expression of attention problems in children diagnosed with Attention-Deficit/Hyperactivity Disorder (AD/HD) and anxiety disorders, two clinical phenomena with considerable comorbidity and symptom overlap (e.g., Generalized Anxiety Disorder and AD/HD-Inattentive Type; American Psychiatric Association, 2000; Kendall, Aschenbrand, & Hudson, 2003; Rowland, Lesesne, & Abramowitz, 2002). AD/HD youth are characterized by general deficits in attention processing (APA, 2000; e.g., selective/sustained attention; Barkley, 1997) associated with abnormalities in the prefrontal cortex and related circuitry (Durston, 2003), whereas anxious children experience emotion-based cognitive (e.g., worry, negative interpretation bias, attentional bias toward perceived threat/danger), physiological (e.g., rapid heartbeat), and behavioral (e.g., avoidance) interference that may distract a child from attending to his/her environment (Beck & Clark; 1988; Beck, Emery, & Greenberg, 1985; Kendall, 2000; Puliafico & Kendall, 2006). Despite their potentially distinct etiologies, these attention difficulties may present similarly in anxious and AD/HD youth (e.g., difficulty concentrating in class, distractibility, restlessness) - particularly in younger children where other behavioral symptoms may be difficult to detect - posing a unique challenge to differential diagnosis and subsequent treatment planning (e.g., cognitive-behavioral therapy vs. stimulant treatment?).

A compelling literature supports the role of cognition in the etiology, development, and maintenance of anxiety disorders and AD/HD (APA, 2000; Barkley, 1997; Beck & Clark; 1988; Puliafico & Kendall, 2006). Current theorists posit that the nature and specificity of cognitive dysfunction may differentiate anxiety-related attentional symptoms from more general attention deficits in AD/HD. Kendall (2000) proposed a model distinguishing cognitive processes in internalizing (e.g., anxious, depressed) versus externalizing (e.g., inattentive, impulsive) children. Specifically, he suggested that the etiology and maintenance of internalizing symptoms (e.g., worry, fear, rumination, depressed mood) may be linked to fundamental emotion-based "distortions" in cognitive processing, leading to misperceptions of the social/interpersonal environment and an attentional bias toward perceived threat and danger (Kendall, 2000, Puliafico & Kendall, 2006). In contrast, Kendall proposed that more global "deficiencies" in cognitive processing, or the absence of careful information processing when it would be most adaptive, may better explain the etiology and expression of inattention and impulsivity in externalizing youth.

According to this model, in internalizing youth, active information processing is intact, but is biased or distorted (Beck & Clark; 1988; Kendall, 2000). For example, an anxious/depressed child who receives an "F" on a math test may blame him/herself for the failure, associate the poor grade with his/her overall academic ability (even if he/she is otherwise an "A" student), and overestimate the significance of the failure, thereby maintaining or increasing anxiety for the next exam. This distorted thought process and internal, global, and stable attributional style may then lead the internalizing youth to misperceive his/her own experiences and environmental demands, magnify and bias attention toward perceived threat and danger, and ultimately underestimate his/her own competencies and coping skills (Kendall, Chu, Pimentel, & Choudhury, 2000; Puliafico & Kendall, 2006). This maladaptive cognitive template may then trigger emotional (e.g., fear, low self-esteem) and behavioral distress (e.g., avoidance, poor performance, inattentive behavior), and serve to reinforce worry and distorted thinking.

In contrast to the cognitive processes of anxious and depressed youth, externalizing (e.g., inattentive/impulsive) children may never fully process and/or encode information, resulting in overall poor performance and rash decision-making (i.e., a lack of forethought and planning; Kendall, 2000). Using the above example, an inattentive-impulsive child may perform poorly on an exam due to general information processing deficits (e.g., difficulty maintaining focus, distractibility, impulsive responding), but instead of catastrophizing the source of failure and its implications, may more accurately attribute the poor grade to carelessness and inattention to detail. Thus, the child's difficulties can be better attributed to a general cognitive processing deficiency rather than intact, but distorted, information processing. Understanding and recognizing this distinction between cognitive distortion in anxious youth (i.e., catastrophic misappraisal of/attentional bias toward perceived environmental threat) and cognitive deficiency in AD/HD youth (e.g., global deficits in selective/sustained attention; Barkley, 1997) may ultimately provide a useful clinical guideline for the differential diagnosis, case conceptualization, and subsequent treatment of youth anxiety and AD/HD, two of the most common psychiatric problems currently affecting school-aged children.

Youth AD/HD

AD/HD is among the most prevalent neurodevelopmental disorders in childhood (Rowland et al., 2002). Prevalence estimates range anywhere from 3%-5% (American Psychiatric Association, 2000) up to 17.8% in current epidemiological reviews (Goldman, Genel, Bezman, & Slanetz, 1998; Elia, Ambrosini, & Rapoport, 1999; Rowland et al., 2002). According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition –Text Revision (DSM-IV-TR; American Psychiatric Association, 2000), diagnosis of AD/HD requires six symptoms of Inattention (IA) or Hyperactivity-Impulsivity (HI) for a duration of at least six months, onset before the age of seven, clinically significant impairment in two or more settings (e.g., home, school, work), and the occurrence of symptoms independent of the course of another disorder.

For children with AD/HD, comorbidity is typically the rule rather than the exception (Rowland et al., 2002). Recent epidemiological studies have reported a 10% comorbidity rate with reading disorders, 27% with anxiety disorders, and 25%-40% with disruptive behavior disorders (i.e., CD, ODD). In addition to high rates of comorbidity, AD/HD is also associated with poor long-term prognosis if left untreated, featuring elevated rates of peer and family problems, academic difficulties, problems with adult social relationships, and other negative life events (e.g., car accidents, substance abuse/dependence, divorce, crime, unemployment; Barkley, 1997). As high as 60% of children with AD/HD continue to experience clinically significant symptoms into adulthood, and prognosis may be worse for youth with symptoms of both inattention and hyperactivity-impulsivity (Kessler, Adler, Barkley, Biederman, Conners, Faraone, et al., 2005). The current study used an analog inattentive-impulsive sample (identified by the

Conners Parent Rating Scale-Revised AD/HD Index; Conners, 2001) that can be expected to show similar cognitive deficits to those found in DSM-diagnosed AD/HD youth. Research suggests that children with elevated inattentive-impulsive symptoms may experience significant cognitive impairment (Campbell & von Stauffenberg, 2009; Gorenstein, Mammato, & Sandy, 1989).

General Cognitive Deficiencies in AD/HD Youth

An extensive theoretical and empirical literature has demonstrated neurobiological deficits and general cognitive processing deficiencies in AD/HD children. These deficits are believed to play a central role in the diagnostic profile and pathophysiology of the disorder (i.e., subtypes based on the presence/absence of attentional/inhibitory dysfunction; American Psychiatric Association, 2000). Durston (2003) reviewed twenty-four functional MRI (fMRI) studies of children with AD/HD, revealing a global reduction in cerebral volume, perfusion, and metabolism, and striking abnormalities in the fronto-striatal circuitry of AD/HD youth (i.e., decreased cerebral metabolism and hypoperfusion in the prefrontal cortex and caudate nucleus). Although the specific clinical and pathogenetic implications of these deficits remain unclear, abnormalities in the prefrontal cortex (PFC) have been associated with poor sustained and selective attention, response inhibition, and working memory, while deficits in the basal ganglia (e.g., caudate nucleus) have been linked to poor response inhibition, externalizing symptoms, and emotional dysregulation (Durston, 2003; Iversen & Dunnett, 1990; Strakowski, DelBello, Adler, Cecil, & Sax, 2000).

General Attentional/Inhibitory Deficits

Accumulating evidence suggests that AD/HD may be associated with impairment in a multitude of attentional processing domains (American Psychiatric Association, 2000; Seidman, 2006; Tsal, Shalev, & Mevorach, 2005). Two attentional constructs that have received considerable attention in the youth AD/HD literature are selective and sustained attention. According to Tsal and colleagues (2005), selective attention refers to the ability to ignore distracting information when performing a perceptual act on relevant information, whereas sustained attention involves the ability to sustain attention to relevant information over time while withholding responses to irrelevant items.

It has also been proposed that attention deficits may be epiphenomenological in the expression of AD/HD, and that dysfunction in behavioral inhibition or impulse control may be central to the pathophysiology of disorder. Barkley (1997) proposed that AD/HD youth may demonstrate impairment in three interrelated inhibitory processes: response inhibition (i.e., inhibition of a “prepotent response” or an automatic inclination to select or implement one response option over another; Casbon, Curtain, Lang, & Patrick, 2003), stopping of an ongoing response (thereby permitting a delay in the decision to respond), and interference control (the protection of this period of delay and any self-directed responses occurring within it from interference by competing responses or stimuli). Barkley further suggested that impairment in this primary domain may be linked to the secondary impairment of four other executive functions that rely heavily on inhibition for their successful execution: non-verbal working memory, self regulation of affect-motivation-arousal, internalization of speech, and reconstitution (i.e., planning and generativity). Barkley concluded that the interaction between primary behavioral inhibition deficits and these other specified EFs may contribute to artifactual deficits in attentional processing.

Neuropsychological studies of AD/HD youth have demonstrated distinct attentional/inhibitory deficits using a compendium of neurocognitive tests including continuous performance and “go/no-go” paradigms (i.e., sustained attention and response inhibition) and Stroop and conjunctive visual search tasks (i.e., selective attention and response inhibition; e.g., Barkley, Grodzinsky, & DuPaul, 1992; Fischer, Barkley, Smallish, & Fletcher, 2005; Homack & Riccio, 2003, Savitz & Jansen, 2003). Neurophysiological and anatomical findings have attributed deficits in selective and sustained attention to the right frontal lobes in AD/HD youth (Tsal et al., 2005), whereas behavioral inhibition deficits have been ascribed to the orbital-frontal regions of the PFC and its reciprocal interconnections with the ventromedial region of the striatum (Iversen & Dunnet, 1990).

Although the literature does not support the use of neuropsychological tests, exclusively, in the clinical diagnosis of AD/HD (Seidman, 2006), the Stroop Color-Word Association Test (SCWT), a measure of selective attention, response inhibition, and cognitive interference, and the Continuous Performance Test (CPT; e.g., Gordon, 1983; Conners, 2004; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956), a measure of sustained attention, response inhibition, and cognitive flexibility, have been widely used with AD/HD children to assess deficiencies in attentional processing and inhibitory control. Savitz & Jansen (2003) compared the performance of 36 AD/HD boys (8-12 years) and 45 age-matched controls on the Stroop Word (i.e., word reading) and Color-Word (i.e., selective attention, response inhibition) subtests. The AD/HD proband was outperformed on both Stroop conditions, suggesting AD/HD-related attentional, inhibitory, and reading impairments. When children with comorbid AD/HD and reading disorders were excluded from the analyses, the authors still found significant differences between the AD/HD and control groups on the Stroop Color-Word subtest, suggesting that the observed differences were not merely an artifact of reading deficits. In a similar vein, Reeve & Schandler (2001) compared 10 AD/HD adolescents (12-17 years) and 10 age- and gender-matched non-clinical controls in performance on the SCWT. Findings revealed significant impairment in the AD/HD group on the Stroop Color (i.e., color naming) and Color-Word scores and the Stroop Interference score (i.e., a calculated variable reflecting performance cost resulting from the presentation of interfering or competing stimuli) relative to the control group, indicative of selective attention, response inhibition, and basic cognitive deficits, and increased vulnerability to cognitive interference.

Ozonoff & Jensen (1999) examined performance on the three subtests of the SCWT across three youth clinical samples, an AD/HD group (n=24; 8-18 years), an autistic group (n=40; 6-18 years), a Tourette Syndrome group (n=30; 8-17 years), and a non-clinical control group (n=29; 8-17 years). Results indicated poorer performance on the Stroop Color-Word subtest in the AD/HD youth relative to typically developing children. No significant differences were found between the control group and the other clinical groups on this measure. Finally, a meta-analytic review of 33 empirical studies (1984-2002) examining Stroop performance in AD/HD youth revealed attentional and inhibitory impairment on all four Stroop scores: the Word, Color, Color-Word, and Interference scores (Homack & Riccio, 2003). However, these scores did not consistently differentiate AD/HD children from other clinical groups across studies, suggesting that attentional/inhibitory dysfunction may not be unique to AD/HD youth.

In addition to the Stroop literature, research has also demonstrated deficits in sustained attention, attentional shifting, and inhibitory control using the Continuous Performance Test (CPT). Richards and colleagues (1990) examined performance on the CPT (Rosvold et al., 1956) in 12 AD/HD and 38 non-AD/HD youths (9-13 years; Richards, Samuels, Turnure, & Isseldyke, 1990). As hypothesized, students with AD/HD committed significantly more errors of commission (i.e., response to a non-target), a measure of sustained attention and response inhibition, than non-AD/HD youth, and displayed faster response times on hits (i.e., response to a target). This attenuation in CPT performance was shown to be consistent over time.

In a similar vein, Perugini and colleagues (2000) investigated performance on a battery of neuropsychological tests in 21 AD/HD males (mean age = 9.8 years) and 22 community control participants (mean age = 9.1 years; Perugini, Harvey, Lovejoy, Sandstrom, & Webb, 2000). Relative to typically developing youth, the AD/HD group performed more poorly on the CPT Overall Index score (Conners, 2004), a composite index of eleven performance measures including omission errors (i.e., non-response to a target; sustained attention), commission errors, hit reaction time, and several measures of performance variability. Fischer and colleagues (2005) compared the performance of 147 hyperactive children and 73 community controls (4-12 years) on the classic Gordon CPT at young adult follow-up (19-25 years; Fischer et al., 2005). The hyperactive group was further subdivided into children with and without AD/HD at follow-up. The hyperactive +AD/HD group, alone, demonstrated a significantly higher number of omission and commission errors on the CPT when compared with control participants, as well as an increase in AD/HD behavior while performing the CPT.

In addition, Taranowski, Prinz & Nay (1986) compared children (7-9 years) with AD/HD (n=14), learning disorders (n=12), comorbid AD/HD and learning disorders (n=12), and non-clinical youth (n=13) in performance on the CPT (Rosvold et al., 1956). Both AD/HD groups (with and without learning disorders) performed significantly worse on the CPT attention (d') score, a sensitivity measure of how well the participant discriminates between targets (letters besides X) and non-targets (X), relative to typically developing children (i.e., poorer attentional shifting ability). Finally, Barkley and colleagues (1992) compared performance on several tests of frontal lobe functioning (e.g., CPT, Gordon, 1983; SCWT) across four diagnostic groups: AD/HD youth with hyperactivity (n=12; M=9.2 years), AD/HD youth without hyperactivity (n=12; M=9.1 years), learning disordered youth without AD/HD (n=11; M=9.9 years), and a non-clinical control group (n=12; M=9.1 years). Both AD/HD groups committed significantly more CPT errors of omission, a measure of sustained attention, than the control group. Additionally, all three clinical groups performed more poorly on the Stroop Word and Color-Word subtests relative to healthy controls.

In summary, general attentional deficiencies (i.e., selective/sustained attention, attentional shifting) have been found in AD/HD children relative to non-clinical controls. Absent from the literature, however, are studies supporting emotion-based cognitive distortions and attentional bias in AD/HD youth. The following section will review the extensive literature on these kinds of cognitive deficits in anxious children.

Youth Anxiety

Anxiety is a multidimensional construct featuring symptoms of cognitive (e.g., excessive/uncontrollable worry, negative interpretation bias/cognitive distortions, attentional bias toward perceived threat), physiological (e.g., rapid heart beat, shortness of breath, muscle tension), and behavioral (e.g., avoidance) distress (Beck & Clark; 1988; Beck, Emery, & Greenberg, 1985; Lang, 1977; Roblek & Piacentini, 2005; Kendall, Aschenbrand, & Hudson, 2003; Weissman, Antinoro, & Chu, 2008). Recent epidemiological reviews have identified anxiety as among the most prevalent childhood psychiatric problems, estimated to affect between 6% and 20% of school-aged youth (Costello et al., 1996; Costello et al., 2005). These same reports have emphasized the persistence of anxiety and related symptoms into adulthood and overall poor prognosis if left untreated. Anxious youth may experience difficulties in social (e.g., limited or unrewarding social networks), familial (e.g., sibling/parental conflict, accommodation around anxiety), and academic arenas (e.g., poor academic performance, school refusal/dropout, limited extracurricular activities), and may experience higher rates of mood and behavioral disorders, drug and alcohol abuse, suicidal behavior, and early parenthood relative to their non-anxious peers (Angold, Costello, & Erkanli, 1999; Kendall et al., 2003; Van Ameringen, Mancini, & Farvolden, 2003; Weissman et al., 2008; Woodward & Fergusson, 2001).

Emotion-Based Cognitive Bias in Anxious Youth

Contrary to the general cognitive deficiencies experienced by AD/HD youth, cognitive models of anxiety posit that biased, distorted, or selective processing of threat may precipitate and maintain anxiety disorders in children and adults (Beck, Emery, & Greenberg, 1985; Lonigan, Vasey, Phillips, & Hazen, 2004; Mogg & Bradley, 1998). Cognitive processing in anxious individuals is characterized by emotion-based misperceptions of environmental threat and/or social evaluation, aversive imagery, negative self-talk (e.g., “they aren’t going to like me”; “I didn’t study enough for the exam!”), negative interpretation bias (e.g., “why are they looking at me? I must have done something embarrassing”), and the selective allocation of attention toward threatening information (Beck et al., 1985; Kendall, 2000; Mogg & Bradley, 1998; Puliafico & Kendall, 2006). Together, these symptoms and processes contribute to the cognitive (e.g., worry), physiological (e.g., arousal), and behavioral (e.g., avoidance) distress unique to the pathology, function, and expression of youth anxiety (Derryberry & Reed, 2002; Puliafico & Kendall, 2006).

Therapeutically, cognitive-behavioral interventions aim to alter dysfunctional thought processes and facilitate safe and adaptive approach behaviors, so that anxious children will think, feel, and behave differently in the future, leading to a reduction in anxiety and a permanent shift in emotional state (Barrett & Shortt, 2003; Brewin, 1996, Kazdin, 2000; Weissman et al., 2008). The centrality of cognitive distortion in the etiology, maintenance, clinical presentation, and treatment conceptualization of anxious youth implicates the clinical significance of actively assessing cognitive content and attentional bias early on in treatment, and accurately distinguishing these symptoms from the more general cognitive processing deficiencies experienced by inattentive-impulsive youth. Although the clinical utility of cognitive assessments (e.g., CPT, Stroop, and dot probe paradigms) in aiding differential diagnosis and intervention planning is not well-established, such measures may serve as a valuable diagnostic aid, reconciling issues of symptom overlap, differential diagnosis, and comorbidity between anxiety and AD/HD, thereby leading to a more informed and idiographic case conceptualization.

Emotion-Based Attentional Bias

Current cognitive models of anxiety suggest that selective attentional processing of threat may causally influence anxiety vulnerability and pathogenesis (e.g., Amir, Beard, Burns, & Bomyea, 2009; Hirsch, Hayes, & Mathews, 2009; Koster, Fox, & MacLeod, 2009; MacLeod, Koster, & Fox, 2009; Schmidt, Richey, Buckner, & Timpano, 2009; See, MacLeod, & Bridle, 2009). Beck and colleagues (1985) posited that anxiety is associated with a bias in the initial stimulus registration phase of cognitive processing, thereby precipitating the rapid and automatic allocation of attention toward emotionally negative or threat-relevant stimuli, when such stimuli compete with other information for attentional resources. This selective attention bias is thought to initiate, maintain, and even intensify problematic anxiety reactions by inducing a state of hypervigilance. Anxious children who magnify or bias attention toward threatening information in their environment may have greater difficulty disengaging threat and engaging safety, or more simply stated, greater difficulty coping and withstanding a stressful situation. Of equal importance, this threat-based attentional bias may compromise a child’s ability to allocate and sustain attention toward activities that may be adaptive for academic and social development, such as taking a test, paying attention in class, or talking to peers.

Two experimental paradigms that have been widely used to examine the relationship between anxiety and attentional bias in children include emotional Stroop and visual dot probe paradigms (e.g., Dalgleish, Taghavi, Neshat-Doost, Moradi, Centerbury, & Yule, 2003; Roy et al., 2008; Taghavi, Dalgleish, Moradi, Neshat-Doost, & Yule; 2003; Waters, Mogg, Bradley, & Pine, 2008). Emotional Stroop tasks are based on the principle of cognitive interference, such that longer response latencies for color naming of threatening versus neutral words indicate an attentional bias toward threat, due to interference ostensibly cause by semantic processing of stimuli. The validity of the emotional Stroop task has been challenged in the literature, and it has been suggested that the task may reflect processes other than attention, including interference in response selection or execution (MacLeod, Mathews, & Tata, 1986; Algom, Chajut, & Lev, 2004). The dot probe task, on the other hand, provides a more direct measure of attention, requiring participants to respond to neutral target probes that follow both threatening and neutral stimuli. Faster reaction times to probes replacing threat stimuli relative to neutral stimuli indicate a threat-related attentional bias.

The adult literature supports an attentional bias toward threat across multiple anxiety disorders, including Generalized Anxiety Disorder (GAD; Bradley, Mogg, White, Groom, & de Bono, 1999; Mogg & Bradley, 2005), Social Phobia (SP; Mogg, Philippot, & Bradley, 2004), Post-Traumatic Stress Disorder (PTSD; McNally, Kaspi, Riemann, & Zeitlin, 1990), Obsessive-Compulsive Disorder (OCD; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996), and Panic Disorder (PD; McNally, Riemann, & Kim, 1990; McNally, Amir, Louro, Lukach, Riemann, & Calamari, 1994), using both emotional Stroop and visual dot probe paradigms. This selective processing bias typically occurs with brief stimulus presentations, supporting its automaticity and its manifestation at the early stages of information processing (Mogg et al., 2004; Mogg & Bradley, 1999).

Research on threat-based attentional bias in anxious youth is limited and mixed. Several studies using emotional Stroop tasks reported findings similar to adult studies, demonstrating greater Stroop interference for threat words in children with specific phobia, GAD, and PTSD, relative to typically developing youth, suggesting biased attention toward (and greater difficulty disengaging from) threatening verbal stimuli (Kindt, Bierman, & Brosschot, 1997; Kindt & Brosschot, 1999; Moradi, Taghavi, Neshat-Doost, Yule, & Dalgleish, 1999; Richards, Richards, & McGeeney, 2000; Taghavi et al., 2003). One study also demonstrated a relationship between interference on the threat-related Stroop trial and scores on the Beck Anxiety Inventory (BAI; Beck & Steer, 1990) in a group of adolescents, such that threat-based attentional bias increased in relation to anxiety symptom severity (Richards et al., 2000).

In addition to the emotional Stroop literature, several studies using visual probe tasks with verbal stimuli have demonstrated similar biases toward threat cues. Vasey and colleagues (1995) reported a significant attention bias toward emotionally threatening words in the lower probe position, only, in anxious youth relative to healthy controls, partially supporting an attentional bias toward threat-related stimuli (Vasey, Daleiden, Williams, & Brown, 1995). In a similar vein, Vasey, El-Hag, & Daleiden (1996) reported a significant attentional bias toward threat cues (combined physical and social threat), relative to neutral words, in high-test-anxious youth. Additional analyses revealed a significant bias away from threat words in the low-test-anxious children, although this effect was mediated by gender and only occurred in low-test-anxious males. Taghavi and colleagues (1999) reported an emotion-specific attention bias toward threat-related, but not depression-related words in GAD children, relative to neutral stimuli, suggesting that

GAD youth may feature an affect-congruent bias toward threat-relevant but not depression-related words. The mixed anxiety-depression group included in this study did not show any attentional bias toward threat or depression cues.

Finally, Dalgleish and colleagues (2003) combined and analyzed data from previous studies in an attempt to compare the utility of emotional Stroop and dot probe tasks in detecting attentional bias in anxious youth. Children and adolescents (7-18 years) diagnosed with GAD (n=24), PTSD (n=24), depression or dysthymia (n=19), and a non-clinical control group (n=26) were administered a verbal dot-probe task (Taghavi et al., 1999), an emotional Stroop paradigm, and a word memory task, both featuring 12 threat-related (e.g., horror), 12 trauma-related (e.g., accident), 12 depression-related (e.g., hopeless), 12 happy (e.g., pleasant), and 12 neutral (i.e., animal) words. The GAD group demonstrated a content-specific attentional bias toward threat-related material on the dot probe paradigm, whereas the PTSD, depressed, and control groups did not. Interestingly, the PTSD group showed an attentional bias *away* from (i.e., avoidance of) depression-related words, indicating unique information processing of depressive material in this anxious subgroup. Although inconsistent with previous findings, no significant effects were reported in this study on the emotional Stroop task.

Research examining emotion-based attention bias in anxious youth using visual dot probe paradigms with pictorial and facial stimuli has been less consistent. Waters, Lipp, & Spence (2004) compared attentional bias in 23 anxious and 23 non-anxious children (9-12 years) using a pictorial dot probe task featuring threat-related (e.g., snakes, spiders, sharks, aimed guns, abduction scenes), pleasant (e.g., puppies, kittens, ice cream, smiling faces), and neutral (i.e., household items and appliances) pictures as the target stimuli. Although both groups displayed an attentional bias toward threatening pictures, the inter-group difference in bias scores was not significant. Hence, the authors concluded that selective attention to threat may be common to all children, not just those with clinical-level anxiety disorders.

Pine and colleagues (2005) compared 34 PTSD youth (i.e., severe maltreatment) and 21 healthy controls (7-13 years) in performance on a facial dot probe task comprising happy, angry, and neutral faces (Pine et al., 2005). Contrary to the initial hypothesis, results showed that PTSD youth exhibited an attentional bias *away* from angry faces, which was associated with severity of physical abuse, whereas controls did not. Prior studies have suggested that PTSD youth may feature distinct attentional processes relative to other youth anxiety groups, and may, in fact, demonstrate unique attentional biases *away* from emotion-based stimuli (Dalgleish et al., 2003; Dalgleish, Moradi, Taghavi, Neshat-Doost, & Yule, 2001). In a similar vein, Monk and colleagues (2006) compared 18 adolescents (9-17 years) with GAD and 15 matched non-clinical controls in performance on a facial dot probe task featuring angry and neutral faces (Pine et al., 2005), while undergoing event-related functional magnetic resonance imaging (fMRI; Monk et al., 2006). Consistent with Pine and colleagues (2005), GAD youth demonstrated a greater attentional bias *away* from angry faces, as well as greater right ventrolateral prefrontal cortex activation in response to the threatening facial stimuli. In addition, as ventrolateral PFC activation increased in the GAD group, anxiety symptom severity diminished, suggesting that this activation may serve as a compensatory response in GAD youth. The link between youth anxiety, threat bias, and right ventrolateral PFC activation has received support elsewhere in the literature (e.g., Telzer et al., 2008).

In contrast to these findings, recent facial dot probe studies support an attentional bias *toward* threat cues in anxious youth. Waters and colleagues (2008) administered the same visual probe paradigm used by Pine and colleagues to 23 GAD children and 25 non-anxious controls (9-12 years) in a standard research setting (i.e., not under fMRI conditions). No threat bias was found in GAD youth or healthy controls. However, when the GAD group was divided by parent-reported anxiety severity, severely anxious children with GAD demonstrated a significant attention bias *toward* both angry and happy faces, whereas low-level GAD youth and non-clinical controls showed no attention bias for emotional faces. Moreover, within the GAD group, attentional bias toward angry faces was associated with the presence of comorbid social phobia, and increased parent-reported anxiety symptom severity, as measured by the Spence Children's Anxiety Scale (Spence, 1998). In a similar vein, Roy and colleagues (2008) compared 101 youth with GAD, social phobia, and/or separation anxiety (7-18 years) enrolled in a multi-site anxiety treatment study (Child Anxiety Multi-Modal Treatment Study; CAMS; e.g., Walkup et al., 2008) and 51 non-anxious youth recruited separately (9-18 years), in performance on the same visual probe task (e.g., Pine et al.). Consistent with Waters et al. (2008), anxious participants demonstrated a greater attentional bias *toward* the threatening facial stimuli relative to healthy controls. Of note, threat bias in the anxious group did not vary significantly across anxiety disorders, and there were no group differences in attention in response to happy faces.

In summary, while adult studies have reported a consistent link between anxiety and attentional bias toward threat, similar associations have been documented in anxious youth, albeit with greater inconsistency. Differences in these childhood findings may be due to small sample sizes, variable testing conditions (e.g., fMRI), and potentially distinct attentional processes across different disorders (e.g., PTSD) and/or stages of development. A subset of the research reviewed also suggests that stimulus type (i.e., words vs. pictures) may influence a measure's perceived threat level, and subsequent sensitivity to detect inter-group differences (e.g., Taghavi et al., 1999, Waters et al., 2004). Puliafico & Kendall (2006) hypothesized that pictorial stimuli may be more "objectively threatening" than words, enough so that both anxious and non-anxious children selectively attend to them, minimizing inter-group effects. This theory aligns well with the cognitive-inhibition (CI) hypothesis put forth by Kindt and colleagues (1997), which posits that all young children selectively attend to threat in their environments, but as they undergo cognitive maturation (e.g., executive skills development), low-anxious youth learn to inhibit this attentional bias, while high fear levels prevent clinically anxious children from doing the same.

The current study tested hypotheses drawn from contemporary cognitive theories of youth anxiety and AD/HD, and explored possible implications for differential diagnosis, by comparing anxious youth (ANX), children with elevated inattention and impulsivity symptoms (I-I), and a non-clinical (NC) sample in performance on neurocognitive tests of both general and emotion-based attentional processes. Given the "deficiency vs. distortion" distinction described by Kendall (2000), it was hypothesized that I-I youth would demonstrate greater impairment than ANX and typically developing children on tests of general attentional processes, but less dysfunction than ANX on tasks measuring threat-related attentional bias.

Specifically, I-I was expected to demonstrate poorer performance on the SCWT (Golden, 1978; i.e., number of errors, number of items completed in 45 seconds; selective attention) relative to ANX, in conjunction with higher Conners CPT (2004) errors of omission and commission (i.e., sustained attention) and lower CPT Att (d') scores (i.e., attentional shifting). Conversely, ANX was predicted to show a greater attentional bias *toward* threatening words on an emotional Stroop paradigm (adapted from Taghavi et al., 2003) compared with I-I, and a greater bias *toward* angry faces on a novel emotional faces dot probe task. Assessment of attentional bias employed *both* verbal Stroop and pictorial dot probe methodology in order to help minimize effects related to age-related differences in verbal comprehension and allow for a comparison across populations and across methods. Consistent with the literature supporting an "emotion-specific" threat bias in anxious youth (e.g., Dalgleish et al., 2003; Taghavi and colleagues, 1999), and in the absence of empirical data comparing cognitive processes of ANX and I-I in response to emotional stimuli, no ANX and I-I differences were anticipated on non-threat trials of each task (i.e., happy, sad, and neutral words/faces). In addition, ANX was expected to show a significant "absolute bias" *toward* threat cues (relative to zero or neutral cues), alone, on both tasks (i.e., threat words, angry faces). No significant bias scores were anticipated in I-I on any emotion-based subtest.

With respect to the control group, in light of the SCWT and CPT research reviewed, I-I was hypothesized to show greater attentional impairment than non-clinical youth on all Stroop and CPT variables. Consistent with adult findings (e.g., Price & Mohlman, 2007), no differences were expected between anxious and typically developing youth on these measures.

Methods

Participants

Inattentive-impulsive group. Inattentive-impulsive youth (I-I; n=22; 9-16 years, M = 11.73, SD = 2.23) with a past AD/HD diagnosis, according to parent-report, were recruited from two sources: (a) youth seeking services at local outpatient psychological clinics (e.g., Child AD/HD and AD/HD-Related Disorders Clinic, a specialty program within the outpatient clinic of Rutgers University; Behavior Therapy Associates P.A., a regional practice of child/adolescent psychology) and (b) contacting families who had participated in previous research studies directed by Rutgers faculty. To increase awareness of the study, flyers were distributed by therapists to families of eligible clients and letters were mailed to past research participants describing the goals and purpose of the study and inviting each family to participate Interested families were instructed to contact the PI via phone or email to schedule a half-hour in-person assessment at the Graduate School of Applied and Professional Psychology at Rutgers University.

Ninety-one percent of inattentive-impulsive participants were Caucasian and the other 9% were African-American. Seventy-three percent of the sample was male. Children with a T-score $\geq$ 60 (Mildly Atypical; Conners, 2001) on the AD/HD Index of the Conners' Parent Rating Scale-Revised (Conners, 2001) were included in the study. Two children with an AD/HD Index T-score < 60 were included in the I-I sample as their Hyperactive-Impulsive T-scores were > 60 (T=61 and 74). Children who were taking stimulant medication, who had received any clinical diagnosis of an anxiety disorder, bipolar disorder, schizophrenia, mental retardation, or a pervasive developmental disorder, or had a T-score $\geq$ 60 on any RCADS anxiety or depression subscale were excluded from the study. Children were not excluded on the basis of gender or racial/ethnic origin.

Anxiety group. Youth (ANX; n=21; 8-17 years, M = 12.62, SD = 2.69) with a primary DSM-IV-TR (American Psychiatric Association, 2000) anxiety diagnosis were recruited from families seeking services for anxiety at a local outpatient psychological clinic at Rutgers University (Youth Anxiety and Depression Clinic; YAD-C). Primary anxiety diagnoses included GAD (n=10), Social Phobia (n=4), Specific Phobia (n=4), SAD (n=2), and Panic Disorder with Agoraphobia (n=1) based on a clinician-administered Anxiety Disorder Interview Schedule (ADIS-IV-C/P; Silverman & Albano, 1996). Secondary anxiety diagnoses included Social Phobia (n=8), Specific Phobia (n=6), SAD (n=4), GAD (n=2), and OCD (n=1). Five participants were diagnosed with two anxiety disorders and seven children met criteria for three or more. Seventy-six percent of anxious participants were Caucasian, 14% were Asian-American, and 10% were African-American. Two thirds of the group was female.

Sixteen of the 21 anxious youth were recruited at pre-treatment, four were included at post-treatment, and one child was recruited at 3-month follow-up. Three of the four children included in the study at post-treatment and one child at pre-treatment met only subthreshold criteria for an anxiety disorder, but were retained in the sample to increase statistical power. Seven anxious youth were diagnosed with a comorbid depressive spectrum disorder (i.e., MDD, dysthymia, or minor depression), two had a co-occurring disruptive behavior disorder (i.e., CD or ODD), and one child was diagnosed with a concurrent eating disorder (i.e., anorexia nervosa). Children with a comorbid DSM-IV diagnosis of AD/HD, bipolar disorder, schizophrenia, mental retardation, or a pervasive developmental disorder were excluded from the study. Children were not excluded on the basis of gender or racial/ethnic origin.

Control group. Twenty-two (n=22) youth who had participated in a previous study of family history, childhood psychopathology, brain structure volume, and neuropsychological functioning (e.g., Chiu et al., 2008, Voelbel, Bates, Buckman, Pandina, & Hendren, 2006; Weissman & Bates, 2009) comprised a non-clinical (NC) control group. The children (8-13 years, M = 10.64, SD = 1.56) were recruited between 1996 and 2002 from local pediatric offices and by word of mouth, and had no current or previous psychiatric disorder, learning disability, or serious medical illness. Eighty-two percent of control participants were Caucasian, 9% were African-American, and 9% were Hispanic-American. Seventy-three percent of the sample was male. Children were not excluded on the basis of gender or racial/ethnic origin and were not taking any prescription medications at the time of assessment.

Measures

Cognitive and executive function measures. The *Stroop Color-Word Association Test* (SCWT, Stroop, 1935), commonly used with children 6 years and older, assesses selective attention, response inhibition, and controlled cognitive processing. In its standard format (Golden, 1978), the SCWT features three subtests: Stroop Word (SW), Stroop Color (SC), and Stroop Color-Word (SCW). The SW prompts the youth to read a series of color words (e.g., "red", "blue", "green") written in black ink, and the SC requires the child to name the colors of a series of "x"s. In the third subtest (SCW), the participant is asked to selectively attend to and name the ink colors of a series of unmatched color words (e.g., the word, "red," printed in blue ink, the word, "blue," printed in green ink, etc.), while suppressing prepotent responding to the lexical feature of the words (i.e., reading the words). The SCW was administered in the current study as a measure of selective attention and response inhibition. SCW age-corrected raw scores (SCW-Raw; the age-corrected number of items completed in 45 seconds), SCW age-corrected T-scores (SCW-T), and the number of SCW errors (SCW-Err), were used for statistical analysis. The SW provides an indicator of simple attention and reading ability and was administered in the current study to prime participants for word reading to increase interference on the SCW. All three Stroop subtests comprise 100 items and scoring is based on the number of items read or named correctly within a 45-second time interval. All three subtests have demonstrated good test-retest reliability ($r > 0.80$; Connor, Franzen, & Sharp, 1988; Graf, Utte, & Tuokko, 1995).

The instructions for the SW were as follows: *"This is a test of how fast you can read the words on this page. After I say begin, you are to read down the columns starting with the first one (point to the leftmost column) until you complete it (run hand down the leftmost column) and then continue without stopping down the remaining columns in order (run your hand down the second column, third column, fourth and fifth columns). If you finish all the columns before I say "Stop", then return to the first column and begin again (point to the first column). Remember, do not stop reading until I say "Stop" and read out loud as quickly as you can. If you make a mistake, I will say 'No" to you. Correct your error and continue without stopping. Are there any questions?" Instructions may be repeated or paraphrased as often as necessary so that the participant understands what is to be done. Then continue. "Ready?... Then begin." As the* *participant starts, begin the stopwatch. After 45 seconds, say: "Stop. Circle the item you are on. If you finished the entire page and began again, put a one by your circle."*

The instructions for the SCW were identical to the SW, except the script begins: *"This is a test of how fast you can name the colors on this page. I want you to name the colors of the ink the words are printed in, ignoring the word that is printed in each item. For example (point to the first item of the first column), this is the first item: what would you say?" If the subject is correct, go on with the instructions. If incorrect, say: "No, that is the word that is spelled there. I want you to say the color of the ink the word is printed in. Now (pointing to the same item) what would you say to this item?" If correct, proceed; if incorrect, repeat above as many times as necessary until the participant understands or it becomes clear that it is impossible to go on. "Good. You will do this page just like the last one, starting with the first column (pointing) and then going on to as many columns as you can. Remember, if you make a mistake, just correct it and go on. Are there any questions?" Instructions may be repeated or paraphrased as often as necessary. "Then begin." After 45 seconds, say: "Stop. Circle the item you are on."*

The *Emotional Stroop* (ES; adapted from Taghavi et al., 2003) features four subtests each comprising twelve words categorized anxiety or threat-related (ES-Anx; i.e., terrified, dark, ghost, petrified, cold, horrible, worried, kidnapped, school, bully, failed, bomb), depression-related (ES-Dep; i.e., lonely, friendless, crying, miserable, helpless, lost, weak, funeral, alone, sick, bad, argument), happy (ES-Hap; i.e., pleased, kindness, smile, love, grateful, play, joke, friend, rich, holiday, excited, brilliant), or neutral (ES-Neu; i.e., sheep, donkey, gorilla, parrot, duck, bird, kangaroo, rabbit, sparrow, robin, butterfly, zebra). The majority of words were selected from a word corpus generated by 231 primary and secondary school students suitable for the age range of participants in the current study (Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 1999). Each word is presented eight times using three different ink colors: red, green, and blue. Four of the twelve words were randomly selected and repeated a ninth time bringing the total number of words to 100. The format, administration, and instructions for the four modified Stroop subtests match the standard SCW, and the order of the words were randomly generated for the present study. The order of administration of the four subtests was randomized for each participant. Similar to the SCW subtest, number of errors and number of items completed in 45 seconds were used for analysis. It has been suggested that the card format used in this investigation may facilitate inter-item semantic priming, and thus may be more sensitive to the detection of psychopathology and related deficits than a computerized single-trial Stroop (McNally, Amir, & Lipke, 1996). Studies employing a similar task have reported large effect sizes for anxiety-control group comparisons for threat versus neutral words (e.g., Taghavi et al., 2003; Cohen's d=.79).

The *Conners Continuous Performance Test* – II (CPT-II; Conners, 2004), a neurocognitive test of sustained attention, attentional shifting, and response inhibition was administered in its standard computerized format. The CPT features good test-retest reliability (omission errors: r=.84; commission errors: r=.65; Conners, 2004) and takes approximately 14 minutes to complete. The test features 6 blocks, each comprising 20-trial sub-blocks, and requires participants to press a specified key (i.e., spacebar) for any letter other than "X." Each letter is displayed for 250 milliseconds and the interstimulus intervals vary between 1, 2, and 4 seconds and are different for each block. Both raw scores and T-scores for CPT errors of omission (CPT-OmR/T; how many times a child fails to respond to a target; i.e., sustained attention, vigilance) and commission (CPT-ComR/T; how many times the child responds to a non-target; i.e., sustained attention, impulsivity) were used for statistical analysis. In addition, CPT Attention (d') T-scores (CPT-AttT), a sensitivity measure of the child's ability to distinguish between targets and non-targets (i.e., attentional shifting), were also examined across groups.

The *Emotional Faces Dot Probe* (FDP; Weissman, Chu, Reddy, Bates, Vietri & Mohlman, 2008) consists of 20 practice trials and 96 test trials (4 blocks, 24 trials per block) and takes approximately seven minutes to complete. Each trial begins with a fixation cross in the middle of the screen which appears for 500ms. Following the fixation cross, two faces (practice trials: both neutral; test trials: one neutral and the other happy, angry, or sad) are presented for 500ms. Once the pictures are presented, a small dot probe (i.e., an up or down arrow) appears in the location of one of the faces (i.e., either the right or left side of the screen). Children are prompted to press the up arrow key in response to an up arrow and the down arrow key in response to a down arrow. The probe appears for 10,000ms or until a response is made. After the dot probe is presented, there is a brief waiting period before the next trial. This inter-trial interval varies randomly between 750 and 1250ms. On the practice trials, a feedback display appears for 1,500ms following the probe indicating a correct or incorrect response or a failure to respond. This FDP paradigm was developed for the current study using E-Prime Version 1.0 (2002) and was presented with a refresh rate of 85 Hertz per minute. Participants were seated approximately two feet from the computer screen, and all stimuli (i.e., faces, arrows, fixation crosses, and instructions) were presented in black on a white background. Facial stimuli were approximately 2” high x 1.5” wide; arrows were approximately 1.5” high x 0.5” wide.

The FDP measures the time (in milliseconds) it takes each participant to respond to the dot probe. On trials where the arrow appears in the location of an emotion face, shorter response times (RTs) indicate an attentional bias toward emotionally valenced faces. Likewise, on trials where the probe appears in the location opposite an emotion face, longer response latencies indicate an attention bias toward the emotionally charged facial stimuli. The opposite pattern indicates a bias away from the emotional stimuli. Since children do not respond directly to the emotional faces, this paradigm minimizes the possibility that significant inter-group effects are attributed to a response bias (i.e., an interference in RT) rather than a selective attention processing bias. The dot probe task in the current study uses line drawings as stimuli, and thus may feature less ecological validity than photographic images (Mohlman, Carmin, & Price, 2007). However, research suggests that facial expression may be more easily recognized in cartoon than photographic depictions, as cartoon expressions may eliminate distracting idiosyncratic facial features (e.g., freckles, unusual hairline) and allow exaggeration of expression beyond what a human face is capable of (Calder, Rowland, Young, Nimmo-Smith, Keane, & Perrett, 2000; Mohlman et al., 2007).

Psychopathology measures. The *Schedule for Affective Disorders and Schizophrenia for School Age Children, Present State and Epidemiological Version* (K-SADS-IV-R; Ambrosini & Dixon, 1996) is a structured diagnostic interview featuring favorable psychometric properties (i.e., test-retest and inter-rater reliability, convergent/divergent validity) and sensitivity in diagnosing anxiety and mood disorders, externalizing disorders, and schizophrenia in youth 6-18 years (Ambrosini, 2000; Silverman & Ollendick, 2005). Threshold and subthreshold diagnoses are determined by a diagnosis-specific, criterion-based scoring algorithm (i.e., 0=no diagnosis, 1=subthreshold, 2=threshold).

The *Anxiety Disorders Interview Schedule-IV-Child/Parent version* (ADIS-IV-C/P; Silverman & Albano, 1996) features independent parent and child interviews that have shown good inter-rater (k=.98, parent interview; k=.93, child interview; Silverman & Nelles, 1988) and test-retest reliability (r=.76, parent interview; Silverman & Eisen, 1992), convergent/divergent validity, and sensitivity to treatment effects (Silverman & Ollendick, 2005; e.g., Kendall et al., 1997). The ADIS-C/P was designed specifically to assess presence/absence and clinical interference of anxiety and other childhood disorders in youth ages 6-18 years. A composite diagnosis or Clinician Severity Rating (CSR; 0=none to 8=incapacitating) of 4 or higher constitutes a clinical diagnosis. In the present study, diagnosticians were trained to reliability (K≥.8) on both the ADIS and the K-SADS by coding videotaped interviews and matching gold-standard ratings of clinical diagnoses.

The *Revised Children's Anxiety and Depression Scale-Parent Version* (RCADS-P; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000) is a 47-item parent-report scale that closely corresponds to DSM-IV anxiety and depressive disorders. Factor analysis has yielded subscales strongly associated with the diagnoses of interest in the current study. The subscales feature good factorial validity, internal consistency (SAD: α=.78; Social Phobia: α=.87; OCD: α=.82; PD: α=.88; GAD: α=.84; MDD α=.87), one-week test-retest reliability, and convergent/discriminant validity (Chorpita et al., 2000; Chorpita et al., 2004). Additionally, the RCADS has demonstrated good convergent validity with other leading anxiety measures (e.g., RCMAS).

The *Conners Parent Rating Scale-Revised* (CPRS-R; Conners, 2001) features 80 questions assessing childhood psychopathology (3-17 years) and AD/HD-related problem behaviors and takes approximately 15 minutes to complete. The CPRS-R comprises 7 DSM-IV-derived symptom subscales (i.e., oppositional, cognitive problems/inattention, hyperactivity, anxious-shy, perfectionism, social problems, psychosomatic) and an overall AD/HD index which contains a set of items for distinguishing AD/HD children from non-clinical youth. Children scoring a T-score ≥ 60 on the AD/HD Index (Mildly Atypical; Conners, 2001) were included in the present analog I-I sample. The CPRS-R has shown strong internal consistency (α=.75-.90) and 6-8-week test-retest reliability (r=.60-.90), as well as good convergent, divergent, and discriminant validity.

Procedure

Inattentive-impulsive group. Interested families of children previously diagnosed with AD/HD responded to study flyers and recruitment letters by contacting the principal investigator to schedule a half-hour in-person assessment. During this time, a cognitive assessment battery (i.e., CPT, SCWT, ES, FDP) was administered to the child and parent-report measures of child psychopathology (i.e., RCADS-P, CPRS-R) were administered to the parents. Each family was given a thorough explanation of the study and written informed consent was obtained from the parents and written assent from the child. All families received $20 and a $5 Blockbuster or Target gift card for their participation. In addition, a copy of the computerized testing report and verbal feedback about the child's task performance were shared with each family.

Anxiety group. Families of anxious youth contacted the YAD-C for treatment, completed a brief telephone-screening interview, and then both parent and child completed a comprehensive in-person intake assessment, including measures for the current study (i.e., CPT, SCWT, ES, FDP) and the usual assessment instruments for the clinic (e.g., ADIS-C/P, RCADS-P). The complete assessment battery took approximately 3 hours per family including breaks. Each family was given a thorough explanation of the study and written informed consent was obtained from the parents and written assent from the child.

Control group. Families recruited for the original study of family history, childhood psychopathology, brain structure volume, and neuropsychological functioning, completed an in-person interview following a telephone-screening questionnaire. During this time, functional magnetic resonance imaging (fMRI) and a comprehensive psychological (e.g., K-SADS-IV-R) and neuropsychological (e.g., CPT, SCWT) assessment battery were conducted with the child, and parent-report measures of child psychopathology were administered. The control group was not administered the ES or FDP, as these measures were not available during the original study. In addition, SCW-Err was not included in the initial dataset. The complete assessment battery took approximately 7 hours per child including breaks. If the child showed signs of fatigue, the assessment was completed at a second session scheduled on a different day of the same week. All families were compensated $100.00 for their participation. Each family was given a thorough explanation of the initial research project, written informed consent was obtained from the parents, and verbal assent was obtained from the children. The study was approved by the Institutional Review Boards of Rutgers University and UMDNJ-Robert Wood Johnson Medical School.

Results

All data were analyzed using SPSS Version 16.0 for Windows. Skew, kurtosis, outliers, and normality were examined for all dependent variables. Distributional properties for most dependent measures reasonably approximated the normal distribution and represented ranges consistent with the literature. For one variable, CPT omission errors, an outlier more than three standard deviations above the mean in the NC group may have reflected invalid responding and/or administration, significantly influencing the group SD. As a result, all CPT scores for this participant were excluded from the analysis of variance across groups. There were no missing data with the exception of one anxious child who chose not to complete the SCW and ES. In addition, initial correlations of age, gender, and ethnicity with all cognitive variables were conducted. Age and gender were found to significantly correlate with several dependent measures, and thus were included as covariates in all subsequent analyses. Age correlated with FDP happy bias scores, $r = -.44$, and number of items completed in 45 seconds on the ES-Anx, $r = .75$, ES-Dep, $r = .77$, ES-Hap, $r = .74$, and ES-Neu, $r = .78$. Gender correlated with CPT-ComR, $r = -.29$. Due to the differences in gender composition across groups, ANCOVAs, with age included as a covariate, were conducted to examine the effect of gender on all cognitive outcome measures across groups. No significant gender effects were found. All following analyses were also completed separately for boys and girls; the same pattern of results was found within each sex group.

Analysis of General Attention Measures: SCW and CPT

Distributional properties (i.e., n, M, and SD) of all general attention measures (i.e., SCW and CPT variables) were examined across the three groups and are summarized in Table 1 (raw scores) and Table 2 (T-scores). Omnibus analyses of covariance (ANCOVA), with age and gender included as covariates, revealed significant between-subjects effects across the three groups for SCW raw and T-scores (i.e., age-corrected number of items completed in 45 seconds), CPT commission error raw scores, and CPT omission error T-scores. However, CPT-OmR (Effect Size (ES) = .07), CPT-ComT (ES = .08), and CPT-AttT (ES = .06) did not differ significantly across the three groups.

I-I-Anxiety Comparisons. Follow-up ANCOVA was conducted to evaluate pairwise differences between ANX and I-I on all general attention measures (see Tables 1 and 2). As predicted, I-I youth demonstrated greater deficits in sustained attention relative to the ANX group, as evidenced by greater CPT-ComR scores, $F(1,42) = 8.94$, $p<.005$, ES = .19, and a non-significant trend toward higher CPT-OmT scores $F(1,42) = 3.56$, $p<.07$, ES = .08. In addition, anxious youth demonstrated superior selective attention as indicated by higher SCW-Raw scores (i.e., greater number of items completed in 45 seconds), $F(1,41) = 9.48$, $p<.005$, ES = .20, higher SCW-T scores, $F(1,41) = 5.35$, $p<.017$, ES = .12, and lower SCW-Err scores, $F(1,41) = 10.62$, $p<.005$, ES = .22. Bonferroni corrections were applied for CPT (i.e., errors of omission and commission raw and T-scores; alpha=.025) and SCW variables (i.e., age-corrected raw and T-scores, number of errors; alpha=.017) to control for Type I errors within pairwise comparisons involving conceptually similar dependent variables.

Diagnostic Group-Control Comparisons. Follow-up ANCOVA was conducted to evaluate pairwise differences between NC and the two clinical groups on all general attention measures (see Tables 1 and 2). As hypothesized, I-I youth committed a greater number of CPT-ComR relative to the control group, F (1,42) = 10.35, p<.005, ES = .21, indicative of impaired sustained attention and vigilance in the I-I sample. Also as expected, no significant differences were found between NC and ANX in CPT-ComR, CPT-ComT, CPT-OmR, or CPT-AttT (see Tables 1 and 2).

Contrary to our initial hypotheses, NC was outperformed by ANX on the SCW-Raw, F(1,41) = 6.65, p<.017, ES = .15. In addition, the NC group was outperformed by both the anxiety group, F(1,41) = 45.44, p<.001, ES = .55, and the inattentive-impulsive group, F(1,42) = 18.27, p<.001, ES = .32, on the CPT-OmT. Also unexpected, no significant differences were found between NC and I-I with respect to SCW-Raw or SCW-T scores.

Within-Group Comparisons

Post-hoc within-group comparisons were conducted across the three CPT T-scores (Table 2) to assess relative strengths and weaknesses within each group. Notably, the I-I group, the group with the shortest assessment duration, demonstrated lower CPTOmT relative to CPTComT, t(21) = 2.50, p<.05, and relative to CPT-AttT at a level of trend, t(21) = 1.75, p<.10. Conversely, the NC group, the group with the longest assessment procedure, showed the opposite pattern, exhibiting significantly higher CPTOmT relative to CPTComT, t(20) = 2.80, p<.05, and CPTAttT, t(20) = 4.31, p<.001. The difference between CPTComT and CPTAttT in this group was also significant, t(20) = 4.94, p<.001. Finally, no significant differences emerged in ANX, the group with the median assessment duration.

Analysis of Emotion-Based Attention Bias: FDP and ES

The distributional properties (i.e., n, M, and SD) of all attentional bias measures (i.e., FDP and ES variables) were examined across the two diagnostic groups and are summarized in Table 3. Consistent with the dot probe literature reviewed (e.g., Mogg, Philippot, & Bradley, 2004; Pine et al., 2005; Roy et al., 2008), only FDP data from trials with correct responses and response latencies > 200ms (and < 2000 ms) from the onset of the target were included in the analyses. Attentional bias scores on the FDP were derived from a standard algorithm used in previous research (e.g., Mogg & Bradley, 1999; Monk et al., 2006; Pine et al., 2005; Roy et al., 2008):

Bias Score = Avg. RT Emotion Opposite Probe – Avg. RT Emotion Following Probe

That is, for each participant, bias scores for angry, sad, and happy faces, respectively, were calculated by subtracting the mean RT on trials where the emotional face and visual probe appeared on the same side of the computer screen from the mean RT on trials where the emotional face and dot probe appeared on opposite sides of the display. Positive (+) values reflect a bias *toward* the emotionally valenced face, relative to neutral stimuli, whereas negative (-) values reflect a bias *away* from the emotional face. Bonferroni corrections were applied for each set of ES variables (i.e., number of errors and number of items completed in 45 seconds; alpha=.025) to control for family-wise error. Bonferroni corrections were not used for FDP scores wherein each statistical analysis represented a distinct research question and involved a unique dependent variable (i.e., threat bias, depression bias, happy bias).

As hypothesized, ANX demonstrated a greater attentional bias toward threatening facial stimuli, relative to I-I youth, as indicated by greater bias scores on the FDP in response to angry faces, $F(1,41) = 10.60$, $p<.005$, ES = .21; ANX M = 22.78, SD = 52.39; I-I M = -4.83, SD = 42.57 (see Table 3). There were no significant inter-group differences in bias scores on the other emotion-based trials (i.e., happy and sad faces).

In addition, one-sample T-tests were conducted to examine absolute differences in bias scores from zero (on the FDP) and neutral cues (on the ES) for each respective emotion in both clinical groups. Anxious children exhibited a trend toward significant "absolute bias" scores on the FDP in response to angry face trials only, $t(20) = 1.99$, $p<.06$; ANX M = 22.78, SD = 52.39, suggesting a potential emotion- or content-specific attentional bias toward threat cues in this group. In addition, the I-I sample demonstrated a significant "absolute bias" toward sad faces, alone, $t(21) = 2.58$, $p<.05$; I-I M = 16.58, SD = 30.17. ES bias scores were not significant and did not distinguish the two groups.

Discussion

The present study aimed to characterize and distinguish the underlying mechanisms of attention problems in anxious and inattentive-impulsive youth. Consistent with Kendall's (2000) "deficiency vs. distortion theory", the study findings provide initial support that AD/HD children may experience relative weaknesses in general attention processes compared with anxious and typically developing children, whereas anxious youth may experience a unique threat-based attention processing bias. Specifically, the I-I group's composite of lower CPT-ComR, relative to anxious and non-clinical youth, coupled with poorer SCW performance (i.e., lower SCW raw/T-scores, more errors) and a trend toward higher CPT-OmT, relative to anxious children, suggests that general attention difficulties (e.g., selective/sustained attention, vigilance) may underlie the behavioral manifestation of inattention in AD/HD youth. The inattentive-impulsive group also demonstrated marginally poorer performance on CPT commission T-scores, CPT omission raw scores, and calculated sensitivity T-scores (i.e., CPTAttT; attentional shifting), relative to anxious children (Tables 1 and 2), although these trends were not statistically significant. Further research with larger sample sizes and greater statistical power may help determine if these trends are clinically meaningful, and if, perhaps, inattentive-impulsive youth show even broader, more consistent relative weaknesses in general attention processes.

In contrast to these findings, the anxious group's greater attentional bias toward threatening faces on the FDP, relative to the inattentive-impulsive group, indicates a greater selective processing bias/attentional allocation toward threat cues in anxious youth, relative to inattentive-impulsive children. In addition, the unique trend toward significant “absolute bias” scores (i.e., relative to zero) in the anxious group on angry trials suggests the potential contribution of a content- or emotion-specific threat-related attention bias (and associated cognitive distortions) to the attentional challenges experienced by anxious youth.

Considered together, this pattern of findings is consistent with previous studies conducted with independent samples of anxious and AD/HD children and provides initial empirical support for the neuropsychological differentiation of attention problems in these two clinical groups. Further, this data supports theories positing that anxious youth manifest a unique selective processing bias toward threat due to “a perturbation in neural mechanisms controlling vigilance” (Beck, Emery, & Greenberg, 1985), which may be causally linked to anxiety vulnerability and pathogenesis and associated cognitive (e.g., interpretation bias), physiological (e.g., arousal), and behavioral (e.g., avoidance) sequelae (Amir, Beard, Burns, & Bomyea, 2009; Beck, Emery, & Greenberg, 1985; Hirsch, Hayes, & Mathews, 2009; Mogg & Bradley, 1998, Puliafico & Kendall, 2006; Roy et al., 2009; See, MacLeod, & Bridle, 2009).

It is important to highlight that Kendall’s theoretical model provides only a preliminary scientific basis for the current study design and rationale, and that the fit between theory and measurement may be less than perfect**.** Kendall’s model initially focused on broader, higher level interpretation biases in anxious youth, whereas the current emotional Stroop and dot probe paradigms measure specific, automatic attentional processes. Thus, one could speculate that unexpected findings in the present study might simply reflect a departure between Kendall’s preliminary theoretical model and our experimental methodology. However, in support of our methods, numerous studies have provided empirical evidence for a causal and reciprocal link between the higher level processing underpinning negative threat interpretation and the automatic processes inherent in threat bias (e.g., Amir et al., 2009; Koster et al., 2009; MacLeod et al., 2009; Mathews, Mogg, Kentish, & Eysenck, 1995; Schmidt et al., 2009; See et al., 2009). The Kendall group has recently acknowledged and discussed this important connection in their 2006 review article (Puliafico & Kendall). Considered together, these recent findings provide initial evidence that the experimental methods selected for the present study do, in fact, offer a practical and empirically-based heuristic for identifying unique cognitive processes in anxious youth. Applied clinically, results from the current study may suggest the potential discriminative validity and clinical utility of two classic neuropsychological measures (i.e., SCW, CPT) and an experimental facial dot probe paradigm, in diagnostically and theoretically differentiating youth anxiety from AD/HD. With continued research, these findings could have important implications for early identification and safe, timely, and effective treatment of childhood disorders.

Although consistent with data from recent visual-probe studies of anxious youth (e.g., Brotman et al., 2007; Waters et al., 2008; Roy et al., 2008; Telzer et al., 2008), the present findings differ from results of earlier studies, which reported an attentional bias away from angry faces in children and adolescents with either PTSD (Pine et al., 2005) or GAD (Monk et al., 2006). Discrepancies between findings of the current investigation and these previous studies may reflect the differing circumstances across the three studies. For example, Pine et al. studied children who experienced severe maltreatment and subsequent PTSD. It is possible, if not likely, that severely maltreated children might develop a strong, adaptive, automatic bias to avoid threat, leading to an attentional bias *away* from angry faces. This discrepancy lends support to distinct information-processing profiles in PTSD vs. other youth anxiety disorders that may reflect differences in underlying pathophysiology (Etkin & Wagner, 2007). Similarly, Monk et al. required participants to complete the visual-probe task under fMRI scanning conditions. Research with anxious adults suggests that under certain anxiogenic conditions, high levels of state anxiety may elicit cognitive suppression or trigger avoidance of threat (Amir, McNally, Riemann, Burns, Lorenz, & Mullen, 1996; Mogg, Bradley, Miles, & Dixon, 2004). Hence, results of the current study do not necessarily contradict these data, but may, instead, underscore the variability and sensitivity of threat bias in anxious children across varying contexts (Roy et al., 2008). Additional studies are needed to examine the effect of specific emotional, biological, and environmental variables on the valence and intensity of attentional bias in anxious youth.

Contrary to our initial hypotheses, the Emotional Stroop did not yield significant bias scores and did not differentiate between groups. Although accumulating research has challenged the validity of the emotional Stroop (e.g., MacLeod, Mathews, & Tata, 1986; Algom, Chajut, & Lev, 2004), there may be alternative explanations for these finding. First, it is possible that the animal words selected for the neutral subtest had a strong emotional valence for certain children (e.g., children with either an affinity for or fear of particular animals), thereby limiting the task's sensitivity to emotion-based biases in attention. Perhaps use of more objectively-neutral verbal stimuli (e.g., desk, chair, etc.) would have yielded greater sensitivity to biases across subtests and greater clinical utility in differentiating biases across groups. It is also likely that the ES may simultaneously tap emotion-based attentional bias *and* general executive attention skills (e.g., selective attention; like the SCW), thereby compromising performance in both clinical groups and minimizing inter-group differences. Further, it is possible that the inter-item semantic priming afforded by the ES card format used in the current study may have precipitated an affect-congruent mood induction (i.e., sadness in response to depression words, fear/anxiety in response to threat words, happiness in response to happy words), which could have interfered with any inter-group effects due to differences in attention. Finally, evidence suggests that the ES may not sufficiently detect threat bias in subclinically anxious youth (e.g., Kindt & Brosschot, 1999); perhaps a replication study using more stringently defined diagnostic groups might yield the hypothesized inter-group effects. Further comparison of the present ES results with data from a non-clinical control group may provide additional support for these prospective hurdles and help clarify limitations of the current findings.

In addition to our non-significant ES findings, an interesting and unexpected inverse trend emerged in attentional bias scores on the Faces Dot Probe task across the ANX and I-I groups. As expected, ANX demonstrated greater “absolute bias” scores toward angry faces relative to sad or happy faces (Table 3). However, I-I demonstrated greater “absolute bias” scores toward sad and happy faces, relative to threat cues. These sad “absolute bias” scores in the I-I group were statistically significant. With continued research and replication, this polarized effect might indicate a possible emotion-based contribution to the attention difficulties experienced by AD/HD youth that may be both cognitively and affectively distinct from threat bias in anxious children. Dodge (1985) hypothesized that certain externalizing youth (i.e., aggressive children) may, in fact, experience emotion-based “distortions” in cognitive processing (i.e., a misinterpretation of the intentions of others' behavior), which, in theory, might contribute to a negative attention processing bias (e.g., depression bias). However, there is a dearth of empirical research supporting this theory and little reason to speculate that the cognitive processes of children in the present analog I-I sample would mirror those of clinically aggressive youth. Nonetheless, in light of these preliminary findings, additional studies examining the nature and role of emotion-based attentional bias in the clinical and cognitive impairment of AD/HD youth are certainly warranted.

In addition to these findings, three unexpected results were also found on the CPT and SCW, with respect to the control group. First, our non-clinical youth demonstrated *higher* CPT omission error T-scores, relative to both clinical groups, indicative of *poorer* sustained attention and vigilance. Second, our I-I group did *not* show the hypothesized deficit in selective attention on the SCW, relative to NC (see Homack & Riccio, 2003 for review). Third, the ANX group demonstrated *elevated* SCW performance relative to NC, suggesting *superior* selective attention in anxious children relative to typically developing youth.

There are several possible explanations for these unexpected findings. First, it is reasonable to speculate that the large discrepancies in assessment duration across groups (i.e., NC = 7 hours; ANX = 3 hours; I-I = 0.5 hours) may have compromised or confounded the projected inter-group effects. In fact, our within-group CPT analyses provide some empirical support for a "fatigue hypothesis", such that the group with the *longest* assessment procedure, NC, performed respectively worse on variables with increased contingency on task duration (i.e., variables requiring greater sustained attention and vigilance). More specifically, the NC group demonstrated the poorest performance on CPT omission error T-scores, a primary measure of sustained attention/vigilance, significantly better performance on CPT commission error T-scores, a measure of sustained attention *and* inhibitory control, and still better on CPTAtt T-scores, a measure of attentional shifting ability. Conversely, the opposite pattern was observed in the I-I sample, the group with the *shortest* assessment procedure, such that I-I exhibited a relative *strength* on CPT-OmT. These within-group trends suggest that burnout or fatigue in the NC group may be a significant study limitation, perhaps contributing to: a) NC's relatively impaired performance on CPT-OmT, b) I-I's intact SCW performance relative to NC, and c) ANX's superior SCW performance, relative to NC. In this context, findings with respect to the NC group, in particular, should be interpreted with caution.

Notwithstanding this significant methodological caveat, there may be alternative explanations for unexpected findings in the current study. For instance, with regard to II and NC's comparable SCW performance, it is equally plausible that the subthreshold inclusion criteria in our analog I-I sample might have yielded less clinical impairment than would a more stringently-defined threshold AD/HD group. This, in turn, might have attenuated the hypothesized Stroop effect in the I-I group, thereby minimizing inter-group differences.

In addition, with respect to ANX's superior SCW performance, relative to NC, the Yerkes-Dodson Law (Yerkes & Dodson, 1908) offers a plausible theoretical rationale. Yerkes and Dodson posited an inverted U-shaped correlation between arousal (on the X-axis) and performance (on the Y-axis), suggesting that a moderate level of arousal - or related concepts, e.g., anxiety - may be adaptive in achieving optimal performance or functioning. Clinical experience also suggests that anxious youth may be particularly motivated to perform well on timed tests, particularly those of short duration. Extending the Yerkes-Dodson model to the current study, one might speculate that ANX, which includes four subclinical youth, might exhibit a moderate level of anxiety, perhaps providing an unanticipated "boost" in selective attention relative to typically developing youth. Notwithstanding this hypothesis, ANX's superior SCW performance in this study is inconsistent with prior adult studies reporting no SCW differences between anxious and non-clinical participants (e.g., Price & Mohlman, 2007).

Findings from the current investigation should be viewed in light of several additional methodological limitations. First, the modest sample sizes ($n \leq 22$ in each group) limited statistical power and may have compromised the ability to detect significant inter-group differences on certain measures (e.g., ES subtests, CPT-OmR, CPTComT, CPT-AttT). Small samples may have also contributed to some discrepancies in results for comparisons using CPT raw versus T-scores. Larger sample sizes could potentially reduce these inconsistencies. However, despite the small groups in the current study, our power analysis indicates sufficient power to detect small to medium effect sizes.

Second, the three comparison groups employed different assessment and recruitment procedures. Specifically, the clinical instruments (e.g., ADIS vs. KSADS vs. CPRS-P) and assessment duration varied across groups; the ANX group, alone, included treatment seekers and three recruitment time points (i.e., pre-treatment, post-treatment, and follow-up); and the NC group was recruited several years earlier than both clinical groups and was not initially intended for use in the current study. Considered together, these methodological inconsistencies across groups may have contributed to additional variance in sample characteristics and how children subsequently responded to study procedures.

Third, the two clinical groups were poorly matched for diagnostic severity level, which may have led to greater clinical severity in the ANX group relative to I-I. Specifically, the ANX group included children meeting full DSM-IV criteria for an anxiety disorder (along with four subclinically anxious youth), whereas the analog I-I group, although previously diagnosed with AD/HD according to parent-report, required elevated, but not clinically significant inattentive-impulsive symptoms. Moreover, since the procedure and timeline for I-I's past AD/HD diagnoses are unknown, it is unclear to what extent these children might show similar cognitive differences when compared with anxious youth, relative to a current, more stringently defined, threshold AD/HD sample. In addition to these diagnostic inconsistencies, the variable rates of comorbidity across both groups may have further confounded our projected contrasts. The I-I group excluded children with comorbid psychiatric diagnoses or clinically significant CPRS-P T-scores. In contrast, seven anxious youth were diagnosed with a comorbid depressive spectrum disorder, two had a co-occurring disruptive behavior disorder, and one child was diagnosed with anorexia nervosa. In addition, twelve anxious children received a secondary anxiety diagnosis, seven of whom met criteria for at least a third. These high rates of comorbidity in the anxious group, relative to I-I, again suggest a clear discrepancy in clinical severity level and diagnostic complexity across the two groups, potentially obscuring inter-group effects due to differences in attention.

Considered together, these methodological caveats may have limited the consistency, homogeneity, and/or level of clinical contrast across groups, confounding certain hypothesized inter-group effects. It is likely that more consistent methodology across groups (i.e., uniform recruitment procedures, assessment instruments/duration, and clinical inclusion criteria) and a demographically and diagnostically homogeneous control group (i.e., matched for age and gender, assessed via the ADIS) may have yielded results more concurrent with previous research.

Despite these limitations, the current study is the first to compare anxious, inattentive-impulsive, and non-clinical youth on a standardized cognitive battery of both general and emotional-based attentional processes, in an effort to better understand, operationalize, and differentiate inattention in anxious and inattentive-impulsive youth. In light of the preliminary research reviewed, the present findings do offer *initial* support for the "cognitive deficiency vs. distortion theory" put forth by Kendall (2000), and further, provide some preliminary evidence for distinct attentional challenges in youth anxiety disorders and AD/HD**.**

Treatment Implications and Future Directions

The present study addresses a timely and important issue as the field continues to gain awareness of the overlap between youth anxiety and AD/HD, two of the most prevalent childhood psychiatric problems (Costello et al., 2005; Rowland, Lesesne, & Abramowitz, 2002), particularly between disorders in which the symptom phenotype looks virtually identical (e.g., AD/HD-Inattentive Type and GAD). Both anxiety and AD/HD are associated with high rates of comorbidity and poor long-term prognosis if left untreated, rendering early and accurate detection critical to maximizing clinical outcomes and quality of life (Barkley, 1997; Weissman et al., 2008). With continued research, cognitive assessment may prove useful in the differential diagnosis of anxiety, AD/HD, and comorbid youth, particularly in younger children where other behavioral symptoms may be equivocal or difficult to detect.

The current findings may also have implications for effective treatment and treatment matching. No child studies have directly examined the impact of CBT on attentional bias to threat cues in anxious youth, but adult researchers have begun to demonstrate the successful elimination of threat bias following a course of CBT (e.g., Mathews, Mogg, Kentish, & Eysenck, 1995). Several investigations of AD/HD youth have also reported significant improvements on cognitive indices of selective and sustained attention as a result of medication treatment (e.g., methylphenidate; Lengleben, Monterosso, Elman, Ash, Krikorian, & Austin, 2006; Monastra, Monastra, & George, 2002; atomoxetine; Faraone, Biederman, Spencer, Michelson, Adler, Reimherr, & Seidman, 2005). Continued research may identify such cognitive processes as important maintaining mechanisms to target in treatment, and focused attention on these mechanisms may improve therapy outcomes.

Such research may also support novel neurocognitive treatment approaches, such as cognitive bias modification (CBM; see Koster et al., 2009 and MacLeod et al., 2009 for reviews). CBM is designed to target and modify the underlying cognitive constructs thought to play a causal role in the pathogenesis and maintenance of anxiety, and has demonstrated initial efficacy as a time- and cost-efficient intervention for anxious adults. Recent studies have successfully trained attention *away* from threat cues using visual dot probe methodology, and have shown initial promise in reducing state and trait anxiety symptom severity and threat-based attentional bias in GAD (Amir et al., 2009), socially anxious (Schmidt et al., 2009) and non-clinical adults (See et al., 2009). No such studies have been completed with anxious youth.

The adult CBM findings provide initial evidence that selective processing of threat cues may, in fact, be malleable and amenable to treatment, and further suggest that attentional selectivity may not only play a causal role in anxiety pathology, but may also serve as an important mechanism of therapeutic change. The comparable effect sizes of CBM and traditional anxiolytic treatments (e.g., CBT and/or pharmacotherapy; e.g., Clark, Ehlers, McManus, Hackmann, Fennell, Campbell, et al., 2003; Gelenberg, Lydiard, Rudolph, Aguiar, Haskins, & Salinas, 2000; Gould, Safren, Washington, & Otto, 2004), suggest the potential utility, and plausible downward extension, of attention training procedures in mitigating both clinical and cognitive symptoms in anxious and comorbid anxious-AD/HD youth. Considered together, these early findings illustrate how the translation of basic psychopathology research may prove useful in developing new treatment approaches, and how visual probe methodology may further elucidate the unique attentional processes underlying anxiety vulnerability and pathogenesis (Schmidt et al., 2009).

In summary, the present study investigated specific threat-based attentional biases common in anxious youth versus more general attention processing deficiencies experienced by inattentive-impulsive children, in an effort to better understand, differentiate, and operationalize the unique attentional mechanisms underpinning both disorders. Our findings provide initial evidence for the underlying neuropsychological distinction of relative weaknesses in general selective/sustained attention in I-I children versus an emotion-specific, threat-based attention processing bias in anxious youth.

Future research examining these distinct attentional challenges, their moderating effect on treatment process (e.g., child engagement, therapeutic alliance) and outcome, and the additive and/or interactive effects of novel and traditional clinical interventions, may have potentially important implications for improving diagnostic assessment and informing the use of adjunctive techniques to enhance clinical and cognitive outcomes in anxious, AD/HD, and comorbid youth. Future studies might also explore the psychometric properties of novel cognitive tasks (e.g., emotional Stroop and visual dot probe paradigms) and compare their potential utility (e.g., verbal vs. pictorial stimuli, Stroop vs. dot probe methodology) in identifying unique attentional deficits and informing subsequent intervention planning. Continued research and standardization of these experimental cognitive assessment protocols may be an important next step in the development of a new operational approach for the clinical assessment, differential diagnosis, and subsequent treatment of childhood psychopathology.

References

Algom, D., Chajut, E., & Lev, S. (2004). A rational look at the emotional Stroop phenomenon: A generic slowdown, not a Stroop effect. *Journal of Experimental Psychology: General, 133*, 323-338.

Ambrosini, P. J. (2000). Historical development and present status of the Schedule for Affective Disorders and Schizophrenia for School Age Children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 49-58.

Ambrosini, P. J., & Dixon, J. F. (1996). *Schedule for Affective Disorders and Schizophrenia for School Age Children, Present State and Epidemiological Version*. New York: New York State Psychiatric Association.

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders, 4th Ed., Text Revision.* Arlington, VA.

Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with Generalized Anxiety Disorder. *Journal of Abnormal Psychology, 118*, 28-33.

Amir, N., McNally, R. J., Riemann, B. C., Burns, J., Lorenz, M., & Mullen, J. T. (1996). Suppression of the emotional Stroop effect by increased anxiety in participants with social phobia. *Behavior Research and Therapy, 34*, 945-948.

Angold, A., Costello, J., & Erkanli, E. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40*, 57-87.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*, 65-94.

Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology, 20*, 163-188.

Barrett, P. M., & Shortt, A. L. (2003). Parental involvement in the treatment of anxious children. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 101-119). New York: Guilford Press.

Beck, A. T., & Clark, D. A. (1988). Anxiety and depression: An information processing perspective. *Anxiety Research, 1*, 23-36.

Beck, A. T., Emery, G., & Greenberg, R. C. (1985). *Anxiety Disorders and Phobias: A Cognitive Perspective*. New York: Basic Books.

Beck, A. T., & Steer, R. A. (1990). Manual for the Beck Anxiety Inventory. San Antonio, TX: Psychological Corporation.

Bradley, B. P., Mogg, K., White, J., Groom, C., & de Bono, J. (1999). Attentional bias for emotional faces in generalized anxiety disorder. *British Journal of Clinical Psychology, 38*, 267-278.

Brewin, C. R. (1996). Theoretical foundations of cognitive-behavioral therapy for anxiety and depression. *Annual Review of Psychology*, 47, 33-57.

Brotman, M. A., Rich, B. A., Schmajuk, M., Reising, M., Monk, C. S., Dickstein, D. P., et al. (2007). Attention bias to threat faces in children with bipolar disorder and comorbid lifetime anxiety disorders. *Biological Psychiatry, 61*, 819-821.

Calder, A. J., Rowland, D., Young, A. W., Nimmo-Smith, I., Keane, J., & Perrett, D. I. (2000). Caricaturing facial expressions. *Cognition, 76*, 105-146.

Campbell, S. B., & von Stauffenberg, C. (2009). Delay and inhibition as early predictors of ADHD symptoms in third grade. *Journal of Abnormal Child Psychology, 37*, 1-15.

Casbon, T. S., Curtain, J. J., Lang, A. R., & Patrick, C. J. (2003). Deleterious effects of alcohol intoxication: diminished cognitive control and its behavioral consequences. *Journal of Abnormal Psychology, 112,* 476-487.

Chiu, S., Widjaja, F., Bates, M.E., Voelbel, G.T., Pandina, G., Marble, J., et al. (2008). Anterior cingulate volume in pediatric bipolar disorder and autism. *Journal of Affective Disorders, 105,* 93-99.

Chorpita, B. F., Yim, L., Moffitt, C., Umemoto, L. A., & Francis, S. E. (2000). Assessment of symptoms of DSM-IV anxiety and depression in children: a revised child anxiety and depression scale. *Behaviour Research and Therapy, 38,* 835-855.

Chorpita, B. F., Moffitt, C. E., & Gray, J. (2005). Psychometric properties of the Revised Child Anxiety and Depression Scale in a clinical sample. *Behaviour Research and Therapy, 43*, 309-322.

Clark, D., Ehlers, A., McManus, F., Hackmann, A., Fennell, Campbell, H., et al. (2003). Cognitive therapy versus fluoxetine in generalized social phobia: A randomized placebo-controlled trial. *Journal of Consulting and Clinical Psychology, 71*, 1058-1967.

Conners, C. K. (2001). *Conners' Rating Scales – Revised*. Toronto, Canada: Multi-Health Systems Inc.

Conners, C. K. (2004). *Conners' Continuous Performance Test II (CPT-II)*. Toronoto, Canada: Multi-Health Systems Inc.

Connor, A., Franzen, M. D., & Sharp, B. (1988). Effects of practice and differential on Stroop performance. *International Journal of Clinical Neuropsychology, 10*, 1-4.

Costello, E. J., Egger, H. L., & Angold, A. (2005). Prevalence and comorbidity. Child and Adolescent Psychiatric Clinics of North America, 14, 631-648.

Costello, E. J., Angold, A., Burns, B. J., Stangl, D. K., Tweed, D. L., Erkanli, A., Worthman, C. M. (1996). The Great Smoky Mountains study of youth: goals, design, methods, and the prevalence of DSM-III-R disorders. *Archives of General Psychiatry, 53*, 1129-1136.

Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology, 111*, 225-236.

Durston, S. (2003). A review of the biological bases of ADHD: What have we learned from imaging studies? *Mental Retardation and Developmental Disabilities Research Reviews*, *9*, 184-195.

Elia, J., Ambrosini, P. J., & Rapaport, J. L. (1999). Treatment of attention-deficit-hyperactivity disorder. *The New England Journal of Medicine, 340*, 780-788.

Etkin, A., & Wagner, T. D. (2007). Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*, 1476-1488.

Faraone, S. V., Biederman, J., Spencer, T., Michelson, D., Adler, L., Reimherr, F., et al. (2005). Atomoxetine and Stroop task performance in adult Attention-

Deficit/Hyperactivity Disorder. *Journal of Child and Adolescent Psychopharmacology, 15*, 664-670.

Fischer, M., Barkley, R. A., Smallish, L., & Fletcher, K. (2005). Executive functioning in hyperactive children as young adults: Attention, inhibition, response perseveration, and the impact of comorbidity. *Developmental Neuropsychology, 27*, 107-133.

Gelenberg, A. J., Lydiard, R. B., Rudolph, R. L., Aguiar, L., Haskins, J. T., & Salinas, E. (2000). Efficacy of venlafaxine extended-release capsules in nondepressed outpatients with generalized anxiety disorder: A 6-month randomized controlled trial. *Journal of the American Medical Association, 283*, 3082-3088.

Gould, R. A., Safren, S. A., Washington, D. O., & Otto, M. W. (2004). A meta-analytic review of cognitive behavioral treatments. In R. Heimberg, C. Turk, & D. Mennin (Eds.), Generalized anxiety disorder: Advances in research and practice (pp. 248-264). New York, NY: Guilford Press.

Goldman, L. S., Genel, M., Bezman, R. J., & Slanetz, P. J. (1998). Diagnosis and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Journal of American Medical Association, 279*, 1100-1107.

Gordon (1983). *The Gordon Diagnostic System*. DeWitt, NY: Gordon Systems

Gorenstein, E. E., Mammato, C. A., & Sandy, J. M. (1989). Performance of inattentive-overactive children on selected measures of prefrontal-type function. *Journal of Clinical Psychology, 45*, 619-632.

Hirsch, C. R., Hayes, S., & Mathews, A. (2009). Looking on the bright side: Accessing benign meanings reduces worry. *Journal of Abnormal Psychology, 118*, 44-54.

Homack, S. & Riccio, C. A. (2003). A meta-analysis of the sensitivity and specificity of the Stroop Color and Word Test with children. *Archives of Clinical Neuropsychology*, *19*, 725-743.

Iversen S. D. & Dunnett, S. B. (1990). Functional organization of striatum as studied with neural grafts. *Neuropsychologia, 28*, 601-626.

Kazdin, A. E. (2000). *Psychotherapy for children and adolescents: Directions for research and practice.* New York: Oxford University Press.

Kendall, P. C. (2000). Guiding theory for therapy with children and adolescents. In P. C. Kendall (Ed.), *Child and adolescent therapy: Cognitive-behavioral procedures, 2nd ed.* (pp. 3-27). New York: Guilford Press.

Kendall, P. C. (1994). Treating anxiety disorders in children: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology, 62*, 100-110.

Kendall, P. C., Aschenbrand, S. G., & Hudson, J. L. (2003). Child-focused treatment of anxiety. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 81-100). New York: Guilford Press.

Kendall, P. C., Chu, B. C., Pimentel, S., & Choudhury, M. (2000). Treating anxiety disorders in youth. In P. C. Kendall (Ed.), *Child and adolescent therapy: Cognitive-behavioral procedures, 2nd ed.* (pp. 235-290). New York: Guilford Press.

Kendall, P. C., Flannery-Schroeder, E., Panichelli-Mindel, S. M., Southam-Gerow, M., Henin, A., & Warman, M. (1997). Therapy for youths with anxiety disorders: A second randomized clinical trial. *Journal of Consulting and Clinical Psychology, 65*, 366-380.

Kendall, P. C., Hudson, J. L., Gosch, E., Flannery-Schroeder, E., & Suveg, C. (2008). Cognitive-behavioral therapy for anxiety disordered youth: A randomized clinical trial evaluating child and family modalities. *Journal of Consulting and Clinical Psychology. 76*, 282-297.

Kessler, R. C., Adler, L. A., Barkley, R., Biederman, J., Conners, C. K., Faraone, S. V., et al. (2005). Patterns and predictors of attention-deficit/hyperactivity disorder persistence into adulthood: results from the national comorbidity survey replication. *Biological Psychiatry*, *57*, 1442-1451.

Kindt, M., Bierman, D., & Brosschot, J. F. (1997). Cognitive bias in spider fear and control children: Assessment of emotional interference by a card format and a single-trial format of the Stroop task. *Journal of Experimental Child Psychology, 66*, 163-179.

Kindt, M., & Brosschot, J. F. (1999). Cognitive bias in spider-phobic children: Comparison of a pictorial and a linguistic Spider Stroop. *Journal of Psychopathology and Behavioral Assessment, 21*, 207-220.

Kollins, S. H., & March, J. S. (2007). Advances in the pharmacotherapy of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 62*, 951-953.

Koster, E. H. W., Fox, E., & MacLeod, C. (2009). Introduction to the special section on cognitive bias modification in emotional disorders. *Journal of Abnormal Psychology, 118*, 1-4.

Lang, P. J. (1977). Psychophysiological assessment of anxiety and fear. In J. D. Cone, & R. P. Hawkins (Eds.), *Behavioral assessment: New directions in clinical psychology.* New York: Brunner-Mazel.

Langleben, D. D., Monterosso, J., Elman, I., Ash, B., Krikorian, G., & Austin, G. (2006). Effect of methylphenidate on Stroop Color-Word task performance in children with Attention Deficit Hyperactivity Disorder. *Psychiatry Research, 141*, 315-320.

Lonigan, C. J., Vasey, M. W., Phillips, B. M., & Hazen, R. A. (2004). Temperament, anxiety, and the processing of threat-related stimuli. *Journal of Clinical Child and Adolescent Psychology, 33*, 8-20.

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

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

MacLeod, C., Rutherford, E., Campbell, L., Ebsworth, 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.

Mathews, A., Mogg, K., Kentish, J., & Eysenck, M. (1995). Effect of psychological treatment on cognitive bias in generalized anxiety disorder. *Behaviour Research and Therapy, 33*, 293-303.

McNally, R. J., Amir, N., & Lipke, H. J. (1996). Subliminal processing of threat cues in posttraumatic stress disorder? *Journal of Anxiety Disorders, 10*, 115-128.

McNally, R. J., Amir, N., Louro, C. E., Lukach, B. M., Riemann, B. C., & Calamari, J. E. (1994). Cognitive processing of idiographic emotional information in panic disorder. *Behaviour Research and Therapy, 32*, 4119-122.

McNally, R. J., Kaspi, S. P., Riemann, B. C., & Zeitlin, S. B. (1990). Selective processing of threat cues in posttraumatic stress disorder. *Journal of Abnormal Psychology, 99*, 398-402.

McNally, R. J., Riemann, B. C., & Kim, E. (1990). Selective processing of threat cues in panic disorder. *Behaviour Research and Therapy, 28*, 407-412.

Mogg, K., & Bradley, B. P. (2005). Attentional bias in generalized anxiety disorder versus depressive disorder. *Cognitive Therapy Research, 29*, 29-45.

Mogg K., & Bradley, B. P. (1999). Some methodological issues in assessing attentional biases for threatening faces in anxiety: a replication study using a modified version of the probe detection task. *Behaviour Research and Therapy, 37*, 595-604.

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*, 809-848.

Mogg, K., Bradley, B. P., Miles, F., & Dixon, R. (2004). Time course of attentional bias for threat scenes: testing the vigilance-avoidance hypothesis. *Cognition and Emotion, 18*, 689-700.

Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology, 113*, 160-165.

Mohlman, J., Carmin, C. N., & Price, R. B. (2007). Jumping to interpretations: Social anxiety disorder and the identification of emotional facial expressions. *Behaviour Research and Therapy, 45*, 591-599.

Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG Feedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiological and Biofeedback, 27*, 231-249.

Monk, C. S., Nelson, E. E., Mogg, K., Bradley, B. P., Leibenluft, E., Blair, R. J. R., et al. (2006). Ventrolateral prefrontal cortex activation and attentional bias in response to angry faces in adolescents with generalized anxiety disorder. *American Journal of Psychiatry, 163*, 1091-1097.

Moradi, A. R., Taghavi, M. R., Neshat-Doost, H. T., Yule, W., & Dalgleish, T. (1999). Performance of children and adolescents with PTSD on the Stroop colour-naming task. *Psychological Medicine, 29*, 415-419.

Neshat-Doost, H. T., Moradi, A. R., Taghavi, M. R., Yule, W., & Dalgleish, T. (1999). The development of a corpus of emotional words produced by children and adolescents. *Personality and Individual Differences, 27*, 433-451.

Ozonoff, S., & Jensen, J. (1999). Brief report: Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders, 29*, 171-177.

Perugini, E. M., Harvey, Harvey, E. A., Lovejoy, D. W., Sandstrom, K. & Webb, A. H. (2000). The predictive power of combined neuropsychological measures for attention-deficit/hyperactivity disorder in children, *Child Neuropsychology, 6*, 101-114.

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

Price, R. B., & Mohlman, J. (2007). *Inhibitory control and symptom severity in late life generalized anxiety disorder*. Manuscript submitted for publication.

Puliafico, A. C., & Kendall, P. C. (2006). Threat-related attentional bias in anxious youth: A review. *Clinical Child and Family Psychology Review, 9*, 162-180.

Reeve, W. V., & Schandler, S. L. (2001). Frontal lobe functioning in adolescents with attention deficit hyperactivity disorder. *Adolescence, 36*, 749-765.

Richards, A., Richards, L. C., & McGeeney, A. (2000). Anxiety-related Stroop interference in adolescents. *The Journal of General Psychology, 127*, 327-333.

Richards, G. P., Samuels, S. J., Turnure, J. E., & Isseldyke, J. E. (1990). Sustained and selective attention in children with learning disabilities. *Journal of Learning Disabilities, 23*, 129-136.

Roblek, T., & Piacentini, J. (2005). Cognitive-behavioral therapy for childhood anxiety disorders. *Child and Adolescent Psychiatric Clinics of North America, 14*, 863-876.

Rosvold, H. E., Mirsky, A. F., Sarason, I., Bransome, E. D., & Beck, L. H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology, 20*, 343-352.

Rowland, A. S., Lesesne, C. A., & Abramowitz, A. J. (2002). The epidemiology of attention-deficit/hyperactivity disorder (ADHD): A public health view. *Mental Retardation and Developmental Disabilities Research Reviews*, 8, 162-170.

Roy, A. K., Vasa, R. A., Bruck, M., Mogg, K., Bradley, B. P., Sweeney, M., et al. (2008). Attention bias toward threat in pediatric anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 1189-1196.

Savitz, J. B. & Jansen, P. (2003). The Stroop Color-Word Interference Test as an indicator of ADHD in poor readers. *Journal of Genetic Psychology*, *164*, 319-333.

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for Generalized Social Anxiety Disorder. *Journal of Abnormal Psychology, 118*, 5-14.

See, J., MacLeod, C., & Bridle, R. (2009). The reduction of anxiety vulnerability through the modification of attention bias: A real-world study using a home-based cognitive bias modification procedure. *Journal of Abnormal Psychology, 118*, 65-75.

Seidman, L. J. (2006). Neuropsychological functioning in people with ADHD across the lifespan. *Clinical Psychology Review, 26*, 466-485.

Silverman, W. K., & Albano, A. M. (1997). *The Anxiety Disorders Interview Schedule for Children (DSM-IV)*. San Antonio, TX: Psychological Corporation.

Silverman, W. K, & Eisen, A. R. (1992). Age differences in the reliability of parent and child reports of child anxious symptomatology using a structured interview. *Journal of the American Academy of Child and Adolescent Psychiatry, 31,* 117-124.

Silverman, W. K., & Nelles, W. B. (1988). The Anxiety Disorders Interview Schedule for Children. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 772-778.

Silverman, W. K., & Ollendick, T. H. (2005). Evidence-based assessment of anxiety and its disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*, 380-411.

Strakowski, S. M., DelBello, M. P., Adler, C., Cecil, K. M., & Sax, K. W. (2000). Neuroimaging in bipolar disorder. *Bipolar Disorders, 2*, 148-164.

Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology,* 28, 643-662.

Taghavi, M. R., Neshat-Doost, H. T., Moradi, A. R., Yule, W., & Dalgleish, T. (1999). Biases in visual attention in children and adolescents with clinical anxiety and mixed anxiety-depression. *Journal of Abnormal Child Psychology, 27*, 215-223.

Taghavi, M. R., Dalgleish, T., Moradi, A. R., Neshat-Doost, H. T., & Yule, W. (2003). Selective processing of negative emotional information in children and adolescents with Generalized Anxiety Disorder. *British Journal of Clinical Psychology, 42*, 221-230.

Taranowski, K. J., Prinz, R. J., & Nay, S. M. (1986). Comparitive analysis of attentional deficits in hyperactive and learning-disabled children. *Journal of Abnormal Psychology, 95*, 341-345.

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in obsessive compulsive disorder. *Behaviour Research and Therapy, 34*, 53-60.

Telzer, E. H., Mogg, K., Bradley, B. P., Mai, X., Ernst, M., Pine, D. S., et al. (2008). Relationship between trait anxiety, prefrontal cortex, and attention bias to angry faces in children and adolescents. *Biological Psychiatry, 79*, 216-222.

Tsal, Y., Shalev, L., & Mevorach, C. (2005). The diversity of attention deficits in ADHD: The prevalence of four cognitive factors in ADHD versus controls. *Journal of Learning Disabilities*, *38*, 142-157.

Van Ameringen, M., Mancini, C., & Farvolden, P. (2003). The impact of anxiety disorders on educational achievement. *Anxiety Disorders, 17*, 561-571.

Vasey, M. W., Daleiden, E. L., Williams, L. L., & Brown, L. M. (1995). Biased attention in childhood anxiety disorders: A preliminary study. *Journal of Abnormal Child Psychology, 23*, 267-279.

Vasey, M. W., El-Hag, N., & Daleiden, E. L. (1996). Anxiety and the processing of emotionally threatening stimuli: Distinctive patterns of selective attention among high- and low-test-anxious children. *Child Development, 67*, 1173-1185.

Voelbel, G. T., Bates, M. E., Buckman, J. F., Pandina, G., & Hendren, R. L. (in press). Caudate nucleus volume and cognitive performance: Are they related in childhood psychopathology? *Biological Psychiatry*.

Walkup, J. T., Albano, A. M., Piacentini, J., Birmaher, B., Compton, S. N., Sherrill, J. T., et al. (2008). Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *New England Journal of Medicine, 359*, 2753-2766.

Waters, A. M., Lipp, O. V., & Spence, S. H. (2004). Attentional bias toward fear-related stimuli: an investigation with nonselected children and adults and children with anxiety disorders. *Journal of Experimental Child Psychology, 89*, 320-337.

Waters, A. M., Mogg, K., Bradley, B. P., & Pine, D. S. (2008). Attentional bias for emotional faces in children with Generalized Anxiety Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 435-442.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children-3rd Edition*. New York: Psychological Corporation.

Weissman, A. S., Antinoro, D., & Chu, B. C. (2008). Cognitive-behavioral therapy for anxiety in school settings: Advances and challenges. In M. Mayer, R. Van Acker, J. E. Lochman, & F. M. Gresham (Eds.), *Cognitive-behavioral interventions for students with emotional/behavioral disorders* (pp. 173-203). New York: Guilford Press.

Weissman, A. S., & Bates, M. E. *Increased psychological and neuropsychological impairment in children with autism spectrum disorders and comorbid bipolar disorder*. Manuscript *s*ubmitted for publication.

Weissman, A.S., Chu, B.C., Reddy, L.A., Bates, M.E., Vietri, J. T., & Mohlman, J. (2008, November). General vs. emotion-based attention processing deficits in anxious and inattentive-impulsive youth: Implications for differential diagnosis and CBT. In A. S. Weissman (Chair), *The expanding role of neuropsychology in clinical practice*. Symposium conducted at the Annual Meeting of the Association for Behavioral and Cognitive Therapies, Orlando, FL.

Woodward, J.L., & Fergusson, D.M. (2001). Life course outcomes of young people with anxiety disorders in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 1086-1093.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18*, 459-482.

Table 1

Raw scores of general attention measures and comparison across Anxious (ANX), Inattentive-Impulsive (I-I), and nonclinical (NC) youth: Sample sizes (n), means (M), standard deviations (SD), omnibus[1] F statistics (F), and effect sizes (ES)

	NC			ANX			I-I			F-test	ES
Score	N	M	SD	n	M	SD	n	M	SD		
CPT-OmR	21	12.24	10.55	21	3.14	4.37	22	10.68	12.18	3.06	.094
CPT-ComR	21	20.00[b]	6.33	21	17.00[b]	7.90	22	25.73[a]	4.72	7.57**	.204
SCW-Raw	22	44.73[b]	8.71	20	54.35[a]	13.17	22	42.77[b]	6.28	7.24*	.197
SCW-Err	--	--	--	20	0.85[b]	1.04	22	2.05[a]	1.81	10.15*	.218

Note. CPT-OmR = Conners' Continuous Performance Test Errors of Omission Raw Score; CPT-ComR = Conners' Continuous Performance Test Errors of Commission Raw Score; SCW-Raw = Stroop Color-Word Age-Corrected Raw Score; SCW-Err = Stroop Color-Word Number of Errors.

[1] F-tests represent omnibus ANCOVA assessing differences among ANX, I-I, and NC groups (or ANX and I-I for SCW-Err).

[b] indicates a value that is significantly lower than [a] in pairwise comparisons.

*$p < 0.005$. **$p < 0.001$.

Table 2

T-scores for general attention measures and comparison across Anxious (ANX), Inattentive-Impulsive (I-I), and nonclinical (NC) youth: Sample sizes (n), means (M), standard deviations (SD), omnibus[1] F statistics (F), and effect sizes (ES)

	NC			ANX			I-I			F-test	ES
Score	N	M	SD	n	M	SD	n	M	SD		
CPT-OmT	21	59.52[a]	8.08	21	44.09[b]	2.76	22	48.41[b]	9.30	21.57**	.422
CPT-ComT	21	51.67	9.04	21	45.87	10.55	22	53.62	7.42	2.90	.089
CPT-AttT	22	45.81	7.87	21	47.10	13.91	22	52.50	8.68	2.18	.068
SCW-T	22	49.70	8.73	20	57.30[a]	14.79	22	47.77[b]	6.28	4.46*	.131

Note. CPT-OmT = Conners' Continuous Performance Test Errors of Omission T-Score; CPT-ComT = Conners' Continuous Performance Test Errors of Commission T-Score; CPT-AttT = Conners' Continuous Performance Test Attention (d') T-Score; SCW-T = Stroop Color-Word Age-Corrected T-Score.

[1] F-tests represent omnibus ANCOVA assessing differences among ANX, I-I, and NC groups.

[b] indicates a value that is significantly lower than [a] in pairwise comparisons.

*p < .05. **p < 0.001.

Table 3

Raw scores of attentional bias measures and comparison across Anxious (ANX) and Inattentive-Impulsive (I-I) youth: Sample sizes (n), means (M), standard deviations (SD), omnibus F statistics (F), and effect sizes (ES)

Score	ANX			I-I			F-test	ES
	N	M	SD	N	M	SD		
ES-Anx Raw	20	50.85	13.37	22	45.36	10.98	0.67	.017
ES-Dep Raw	20	52.15	9.96	22	44.32	12.31	2.79	.068
ES-Hap Raw	20	51.40	11.34	22	46.14	10.02	0.52	.013
ES-Neu Raw	20	50.05	12.16	22	44.95	10.49	0.27	.007
ES-Anx Err	20	0.50	0.51	22	0.82	1.47	2.88	.070
ES-Dep Err	20	0.35	0.49	22	0.86	1.36	3.34	.081
ES-Hap Err	20	0.30	0.66	22	0.82	1.14	1.65	.042
ES-Neu Err	20	0.50	0.61	22	0.64	1.09	0.00	.000
FDP-Angry	21	22.78	52.39	22	-4.83	42.57	10.60*	.214
FDP-Sad	21	-3.32	21.78	22	16.58	30.17	3.35	.079
FDP-Happy	21	4.64	33.46	22	15.07	43.65	0.02	.0.00

Note. ES-Anx Raw = Emotional Stroop Anxiety Raw Score; ES-Anx Err = Emotional Stroop Anxiety Errors; ES-Dep Raw = Emotional Stroop Depression Raw Score; ES-Dep Err = Emotional Stroop Depression Errors; ES-Hap Raw = Emotional Stroop Happy Raw Score; ES-Hap Err = Emotional Stroop Happy Errors; ES-Neu Raw = Emotional Stroop Neutral Raw

Score; ES-Neu Err = Emotional Stroop Neutral Errors; FDP-Angry = Faces Dot Probe Threat Bias; FDP-Sad = Faces Dot Probe Sad Bias; FDP-Happy = Faces Dot Probe Happy Bias.

*$p < 0.005$.

Cirriculum Vita

Adam Scott Weissman

EDUCATION

September 1998 - May 2002	University of Pennsylvania B.A. in Psychology and French
September 2004 - October 2006	Rutgers, The State University of New Jersey M.S. in Clinical Psychology, GPA: 4.0
September 2004 - October 2009	Rutgers, The State University of New Jersey Ph.D. in Clinical Psychology, GPA: 4.0
July 2008 – June 2009	New York University Child Study Center/Bellevue Hospital Center Pre-Doctoral Fellow in Clinical Child & Adolescent Psychology
September 2009 – Present	Judge Baker Children's Center/Harvard Medical School/Harvard University Post-Doctoral Research Fellow in Clinical Psychology

HONORS AND AWARDS

2009 – Present	Treasurer/Secretary/Membership Coordinator, Association for Behavioral and Cognitive Therapies (ABCT) Neurocognitive Therapies & Translational Research Special Interest Group (SIG)
2009 – Present	Executive Committee Member, ABCT Child/Adolescent Anxiety SIG
February 2009	New Jersey Psychological Association (NJPA) Research into Treatment and/or Causes of Social Problems Graduate Student Dissertation Award

November 2008	ABCT Child/Adolescent Anxiety SIG Graduate Student Research Award
November 2007 & 2008	ABCT Neurocognitive Therapies/Translational Research SIG Student Poster Awards
July 2007 & 2008	Graduate Student Research Travel Awards, Rutgers University
April 2008	Sigma Xi, The Scientific Research Society, Rutgers University
2005-2008	Tuition Award and Teaching Assistantship, Rutgers University
2004-2005	Tuition Award and Graduate Assistantship, Rutgers University
May 2002	Honors Distinction in Psychology, University of Pennsylvania
1999-2002	Psi Chi National Honor Society, University of Pennsylvania

WORK EXPERIENCE

September 2002 – June 2004	Project Coordinator Treatment Research Center, University of Pennsylvania Medical School
September 2004 – August 2005	Graduate Research Fellow Center of Alcohol Studies, Rutgers University
September 2005 – June 2008	Graduate Teaching Fellow Department of Psychology, Rutgers University
September 2007-June 2008	Clinical Psychology Extern Institute for Anxiety and Mood Disorders, NYU Child Study Center
July 2008 - June 2009	Pre-Doctoral Fellow in Clinical Child/Adolescent Psychology New York University Child Study Center/Bellevue Hospital Center
June 2000 – Current	Owner/Director and Vice President of Public Relations Weissman Teen Tours, Inc.

September 2009 - Current Director, Child STEPs Clinic Treatment Project-Maine

MATCH-ADTC Trainer & Consultant

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PUBLICATIONS

Weissman, A. S., Reddy, L. A., & Hale, J. B. (in preparation). The integration of neuropsychological assessment and clinical intervention for emotional and behavior disordered youth: Advances and challenges. In L. A. Reddy, J. B. Hale, & **A. S. Weissman** (Eds.), *Neuropsychological Assessment and Clinical Intervention for Emotional and Behavior Disordered Youth: An Integrated Step-by-Step Evidence-Based Approach*. New York: Guilford Press.

Reddy, L. A., Hale, J. B., & **Weissman, A. S.** (in preparation). Integrated neuropsychological assessment and clinical intervention for youth with Attention-Deficit/Hyperactivity Disorder–Inattentive Type. In L. A. Reddy, J. B. Hale, & **A. S. Weissman** (Eds.), *Neuropsychological Assessment and Clinical Intervention for Emotional and Behavior Disordered Youth: An Integrated Step-by-Step Evidence-Based Approach*. New York: Guilford Press.

Reddy, L. A., Hale, J. B., & **Weissman, A. S.** (in preparation). *Neuropsychological Assessment and Clinical Intervention for Emotional and Behavior Disordered Youth: An Integrated Step-by-Step Evidence-Based Approach*. New York: Guilford Press.

Weissman, A. S., Chu, B. C., Reddy, L. A., Bates, M. E., Vietri, J. T., & Mohlman, J. (in preparation). Do unique mechanisms underlie the expression of attention problems in anxious and inattentive-impulsive youth? Implications for differential diagnosis and treatment.

Weissman, A. S., & Bates, M. E. Increased psychological and neuropsychological impairment in children with autism spectrum disorders and comorbid bipolar disorder. Submitted to the *Journal of Autism and Developmental Disorders*.

Goldman, M., Ehrman, R. N., MacDougall, M., Marquez, K., Vietri, J. T., **Weissman, A. S.**, Hakun, J. K., Langleben, D., Hurt, H., O'Brien, C. P., & Childress, A. R. Development of a novel affect-congruent Go-NoGo task for use with inner city adolescents at risk for drug dependence. Submitted to *Drug and Alcohol Dependence*.

Chu, B. C., Colognori, D., **Weissman, A. S.**, & Bannon, K. (in press). An initial description and pilot of group behavioral activation therapy for anxious and depressed youth. *Cognitive and Behavior Practice*.

Weissman, A. S., Antinoro, D., & Chu, B. C. (2008). Cognitive-behavioral therapy for anxiety in school settings: Advances and challenges. In M. Mayer, R. Van Acker, J. E. Lochman, & F. M. Gresham (Eds.), *Cognitive-behavioral interventions for students with emotional/behavioral disorders*. New York: Guilford Press.

Weissman, A. S., Chu, B. C., Reddy, L. A., Vietri, J. T., & Mohlman, J. (2008). General versus

emotion-based attention processing deficits in anxious and inattentive-impulsive youth: Implications for differential diagnosis and CBT. Lay article for the *Association for Behavioral and Cognitive Therapies Child/Adolescent Anxiety SIG Newsletter*.

Bates, M. E., Lehrer, P., Vaschillo, E., Udo, T., Ray, S., Vaschillo, B., Pandina, R., & **Weissman, A.** (2005). Alcohol and self-regulation [Abstract]. *Alcoholism: Clinical and Experimental Research, 29*, 66A.

Franklin, T. R., Ehrman, R., Sciortino, N., **Weissman, A.**, Gariti, P., & Childress, A. (2003, November). Menstrual cycle at onset of smoking cessation predicts treatment outcome. Lay article for the *Society for Neuroscience Press Book*.