ANXIETY, EXECUTIVE FUNCTIONING, AND QUALITY OF LIFE IN A PEDIATRIC CLINICAL POPULATION

By

ROBERT SCOTT MERRELL

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To Lisa, for her boundless compassion

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LIST OF ABBREVIATIONS

ACT	Attentional Control Theory
BASC	Behavior Assessment System for Children, Second Edition
BR	Behavioral Regulation (factor)
BRI	Behavioral Regulation Index
BRIEF	Behavior Rating Inventory of Executive Function
D-KEFS	Delis-Kaplan Executive Function System
EF	Executive functioning
ER	Emotional Regulation (factor)
GEC	Global Executive Composite
MI	Metacognition Index
PedsQL	Pediatric Quality of Life Inventory Version 4.
PET	Processing Efficiency Theory
PRS	Parent Rating Scale
QOL	Quality of life
SRP	Self-Report of Personality

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By

Robert Scott Merrell

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Anxiety disorders are the most prevalent mental illnesses in the United States. Despite early age of onset, research in pediatric samples is limited. Available data suggest that anxiety significantly impairs children's quality of life, spanning behavioral, emotional, and social domains. Threats to pediatric wellness may be diminished by developing a fuller understanding of the cognitive processes through which anxiety affects quality of life. Prior research suggests that executive functioning may represent one such construct.

Based on the Attentional Control Theory prediction that anxiety disrupts executive functioning (Eysenck, Derakshan, Santos, & Calvo, 2007), the current study examined the role of executive functioning in relation to anxiety and quality of life in a mixed clinical sample (ages 4 to 18; *M* = 10.8, *SD* = 3.4). Measurement included parent assessment for the three constructs of interest (*N* = 108), with subsamples of child-performance on executive functioning tasks (*n* = 81) and child self-reports for anxiety and quality of life (*n* = 42). Bootstrapped, bias-corrected mediation analyses (5,000 resamples) provided evidence for executive functioning's role as a mediator in the parent-assessed models. Also consistent with theory, the inhibition and shifting

subdomains of executive functioning were stronger mediators than other subdomains. Effect size for the shift subcomponent fell in the large range (κ^2 = .273). Findings are discussed in relation to developing literature on pediatric anxiety, executive functioning, and quality of life, with emphasis on rater concordance and measurement considerations.

CHAPTER 1
INTRODUCTION

Background and Significance

Anxiety disorders are the most common mental illnesses in the United States (Quilty, Van Ameringen, Mancini, Oakman, & Farvolden, 2003). Their impact on wellness is perhaps evidenced by the enormous amount of expenditure aimed at diminishing their effect. In fact, a recent analysis reported that the annual "true societal costs of anxiety disorders in the U.S. [are]…more than $100 billion" (Kessler & Greenberg, 2002, p. 990). With a median onset prior to the age of 12, anxiety disorders tend to develop early in life (Saddock, Saddock, & Ruiz, 2009). Even though other mental disorders (e.g., Depression, Oppositional-Defiant Disorder, Conduct Disorder, Substance Use Disorders, and Attention-Deficit/Hyperactivity Disorder) also trace back to childhood, their onsets are often preceded by a diagnosable anxiety disorder (Kessler & Greenberg).

Despite the 26.1% annual prevalence rate for anxiety in children and young adults (Kim-Cohen et al., 2003), research on the impact of psychiatric disorders in pediatric populations is sparse at best. Available data suggest that mental disorders may impact younger individuals more adversely than older individuals (Castaneda, Henriksson, Marttunen, Suvisaari, & Lonnqvist, 2008). Thus, anxiety often significantly decreases children's quality of life (Bastiaansen, Koot, Ferdinand, & Verhulst, 2004; Clark & Kirisci, 1996; Sawyer et al., 2002).

Quality of life (QOL) is a multidimensional construct, including physical, behavioral, emotional, and social aspects of functioning (Ravens-Sieberer, Erhart, Wille, Wetzel, Nickel, & Bullinger, 2006). The Diagnostic and Statistical Manual of Mental Disorders,

Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000) enumerates a range of potential QOL distress areas related to mental illness. For instance, relational, educational, and occupational disturbances or "problems in living" (see Szasz, 1960) constitute Axis IV diagnostic considerations or "Psychosocial and Environmental Problems." Such concerns also figure prominently in the "V Codes" subset of "Other Conditions That May Be a Focus of Clinical Attention." Fittingly, the DSM-IV-TR and others (e.g., Frisch, 2006) emphasized that QOL impairments often pre-date mental disorders or are products of the disorder. Mogotsi and colleagues (2000) highlighted this bi-directional nature of effects, positing that QOL concerns are both causes and sequelae of anxiety disorders.

Prospective studies are clearly needed to better elucidate the relationship between anxiety and QOL, yet it is equally apparent that anxiety disorders by their very nature significantly undermine QOL (Olatunji et al., 2007). Research on QOL and anxiety disorders has reliably portrayed a "uniform picture of anxiety disorders as illnesses that markedly compromise QOL and psychosocial functioning" (Mendlowicz & Stein, 2000, p. 669). Given that anxiety adversely affects behavioral and psychosocial functioning, it stands to reason that threats to pediatric QOL may be diminished by developing a fuller understanding of the cognitive processes through which anxiety affects QOL. Executive functioning (EF) likely represents one such construct.

Executive functioning refers to the cognitive processes involved in self-regulation (Sarsour et al., 2011). Behavioral disturbances, interpersonal difficulties, and limited academic/vocational achievement have been associated with impaired EF (Baron, 2004; Lezak, Howieson, & Loring, 2004). An emerging literature demonstrates the links

between compromised EF and psychiatric disorders, including Schizophrenia, Bipolar Disorder, Attention-Deficit/Hyperactivity Disorder, Depression, and substance use disorders (Banich, 2009). Nonetheless, relatively little is understood about cognitive deficits related to anxiety disorders, though preliminary findings suggest that adults with anxiety disorders exhibit decreased EF performance (Castaneda et al., 2008). These findings are consistent with Processing Efficiency Theory (PET; Eysenck & Calvo, 1992) and Attentional Control Theory (ACT; Eysenck, Derakshan, Santos, & Calvo, 2007). Whereas PET contends that anxiety disrupts global EF, ACT capitalizes on advances in EF research and maintains that anxiety specifically impairs two EF subcomponents: the ability to inhibit a prepotent response and the ability to shift back and forth to meet situational demands. Thus, anxiety theoretically undermines EF, and a growing corpus of studies empirically supports this relationship (for a review, see Derakshan & Eysenck, 2009).

Research on the role of EF in pediatric anxiety with regard to QOL is limited. The present study sought to expand the empirical knowledge base by addressing this critical gap in current literature. Using PET (Eysenck & Calvo, 1992) and ACT (Eysenck et al., 2007) as a conceptual framework, the study tested the hypothesis that EF mediates the relationship between anxiety and QOL. Specifically, it was hypothesized that increased anxiety symptoms would be associated with increased executive dysfunction, which in turn would be associated with poorer QOL. Design implications notwithstanding, results provided supporting evidence that EF may serve as part of the mechanism by which anxiety affects QOL.

To set the context for this study, the ensuing literature review will demonstrate the following key points: (a) anxiety disorders stand out relative to other psychopathology due to the significant personal and societal burdens they engender; (b) children and adolescents are particularly at risk to be adversely impacted by anxiety; (c) the effects of anxiety extend well beyond the symptoms of the disorder itself and threaten QOL; and (d) in consonance with PET- and ACT-based predictions regarding how anxiety impacts cognitive functioning, EF may play a critical role in how anxiety influences QOL.

Anxiety

Anxiety disorders are the most frequently occurring psychiatric disorders in the United States (Mendlowicz & Stein, 2000; Quilty et al., 2003). They have high chronicity and early age of onset (Kessler & Greenberg, 2002). With lifetime prevalence for the constellation of disorders estimated as high as 28.8%, they are associated with significant personal (i.e., role impairment) and societal strain (Kessler, Berglund, Demler, Jin, & Walters, 2005). A staggering economic reality also underscores anxiety's pernicious effects. In their exhaustive review of psychiatric epidemiologic surveys, Kessler and Greenberg reported that a 1996 estimate listed the annual societal cost of anxiety disorders at $47 billion, although a 1999 estimate reduced the figure to $42 billion. As indicated earlier, they argued that the true social costs exceed these numbers (surpassing $100 billion annually) because long-term opportunity costs (i.e., extended unemployment or underemployment) and costs linked with comorbidity were excluded from prior analyses. Add to the financial burden factors such as severe distress, excessive worry, restlessness—in a word, misery—and a more complete picture of anxiety begins to emerge.

Pediatric anxiety. "Worry" figures prominently among the chief reasons for referral to children's healthcare providers (March et al., 1999). It is perhaps not surprising that, similar to community studies of adults, anxiety disorders are also the most prevalent mental disorders in children and adolescents (Saddock et al., 2009). Consider those who suffer from Obsessive-Compulsive Disorder (OCD), an often debilitating anxiety disorder and one of the most common childhood psychiatric illnesses (Stewart et al., 2004). Epidemiologic studies estimate prevalence rates of approximately 1-4% among children and adolescents (Douglass, Moffit, Dar, McGee, & Silva, 1995; Zohar, 1999). As delineated by DSM-IV-TR diagnostic criteria, the disorder is characterized by recurrent, time-consuming obsessions or compulsions that cause marked distress or significantly interfere with normal functioning (American Psychiatric Association, 2000). In children, such disturbances often manifest in familial, social, and academic domains (Flament et al., 1988; Piacentini, Bergman, Keller, & McCracken, 2003). Thus, this multidimensional turbulence extends into various domains of functioning, and it also reaches out temporally. Flament, Koby, Rapoport, and Berg (1990) tracked clinically referred youth with OCD and found that 68% of those seen again still had the disorder 7 years later. Thomsen and Mikkelsen (1995) found remission and reemergence of OCD in their pediatric participants, and approximately one half of their sample met diagnostic criteria at 5 year follow-up. Taking OCD as illustrative of pathological anxiety's significant and potentially durable-if-not-treated effects on QOL for both patients and their families/peers (Barlow, 2000), it stands to reason that recent decades have seen a dramatic increase in researchers' attention to

the link between anxiety and QOL (Hansson, 2002; Olatunji, Cisler, & Tolin, 2007; Quilty et al., 2003).

Quality of Life

Mendlowicz and Stein (2000) questioned the current validity of the idea that the cost of human suffering cannot be measured, contending that various aspects of human suffering—as well as its absence—can be reliably assessed via the concept of QOL. Increased research focus on QOL originated from the shift in criteria for evaluating medical outcomes that has occurred in recent decades, stemming in part from the World Health Organization's (1948) invitation to look beyond symptom-reduction/increased survival and toward a more patient-centered consideration of wellness, including physical, emotional, and social well-being. More phenomenological in nature, QOL has biopsychosocial underpinnings that emphasize healthy living and health outcomes as products of the interplay between physical and psychological factors (Engel, 1977). Differences in QOL have been detected between adult racial/ethnic groups (e.g., Utsey, Chae, Brown, & Kelly, 2002). As applied to children, QOL refers to a multidimensional construct involving physical, behavioral, psychological, and social aspects of functioning as perceived by either children themselves or parents/other observers (e.g., primary caregivers, teachers) (Bullinger, 2002; Ravens-Sieberer et al., 2006).

Quality of life was first applied in a medical context to assess how cancer treatments affected not only patients' survival time but also their subjective sense of well-being. Dissatisfied by the available measures' narrow focus on morbidity and mortality, Spitzer, Dobson, and Hall (1981) sought to develop an instrument more attuned to the social and emotional aspects of the patient's life. Interestingly, they

found very few low scores (i.e., indicative of poor QOL) during Australian field testing trials. After reasoning that a cultural bias toward under-reporting and methodological/psychometric shortcomings may plausibly explain their results, they alternatively questioned if they "may be underestimating the ability of the human mind and spirit to compensate for major infirmity" (p. 596). Accordingly, due to the inextricable links between one's experience of physical pain/disease, overall wellness, and psychological capacity to *compensate for major infirmity*, assessing QOL within clinical psychiatric populations is of vital importance. Examination of QOL in the mental health setting may reveal compensatory cognitive processes that could be targeted to help people overcome psychiatric challenges, such as anxiety.

Quality of Life and Anxiety

The DSM-IV-TR (American Psychiatric Association, 2000) notes that impaired QOL is frequently a common cause or consequence of mental illness, and thus should figure prominently in treatment planning. Even though anxiety disorders are the most prevalent mental disorders, it would be erroneous to assume that they represent mild psychopathology (Barlow, 2000). On the contrary, research on QOL and anxiety disorders has reliably demonstrated anxiety's association with compromised physical, behavioral, psychological, and social functioning (Hansson, 2002; Mendlowicz & Stein, 2000; Mogotsi, Kaminer, & Stein, 2000; Olatunji et al., 2007; Quilty et al., 2003).

Reflective of anxiety disorders' high occurrence and pervasive influence on well-being, research on the impact of anxiety on QOL has increased considerably in recent years yet is still in its nascence (Hansson, 2002). Principally, researchers have employed epidemiologic and clinical studies. Epidemiological surveys have been utilized to infer QOL from various indicators such as subjective assessment of physical

and emotional health, psychosocial functioning, and financial solvency (Markowitz et al., 1989). Clinical studies have relied on QOL-specific measures, often assessing aspects of physical, social, emotional, and vocational well-being. Each methodological approach has its merits and drawbacks. What community-based samples gain in descriptive value for the larger population, they sacrifice in terms of relevance to clinical practice.

Turning now to the relationship between QOL and particular anxiety disorders, some evidence suggests that patients with panic disorder (PD) and post-traumatic stress disorder (PTSD) report poorer QOL than individuals with other anxiety conditions (Hansson, 2002). For instance, community and clinical samples of individuals with PD report a high frequency of suicide attempts, severe vocational impairment, significant psychological distress/constraints, and impaired emotional wellness (Quilty et al., 2003; Sherbourne, Wells, & Judd, 1996). Post-Traumatic Stress Disorder research has generally focused on community samples consisting of veterans (Jordan et al., 1992; Stein, Walker, Hazen, & Forde, 1997). Familial discord (ranging from nonspecific marital distress to increased violence) was more prevalent in families of veterans with PTSD than in families of veterans not suffering from the disorder. Additionally, children of individuals with PTSD were more likely than their counterparts to demonstrate behavioral problems, findings that highlight how the impact of psychopathology extends beyond symptoms, transcending the intrapersonal and into the interpersonal (Mendlowicz & Stein, 2000).

Nevertheless, despite preliminary findings that PD and PTSD were associated with compromised QOL, a meta-analysis of 23 separate studies (N = 2892) indicated that no

particular anxiety disorder was associated with significantly poorer QOL than any other particular anxiety disorder (Olatunji et al., 2007). Broadly stated, individuals with social phobia exhibited substantial deficits in educational, occupational, social, and romantic functioning (Stein & Kean, 2000). Despite scores indicative of robust physical health, individuals with obsessive-compulsive disorder (OCD) also demonstrated severe impairments in social functioning and role limitation due to emotional problems and mental health (Koran, Thienemann, & Davenport, 1996). Commonly proffering nosological concerns and comorbid disorders as an explanation, researchers noted that the paucity of data for QOL in individuals with non-comorbid generalized anxiety disorder (GAD) indicated substantial overall life impairment (Massion, Warshaw, & Keller, 1993).

In sum, Olatunji and colleagues' (2007) meta-analysis demonstrated that anxiety disorders are associated with significant QOL impairment. Mogotsi and colleagues (2000) noted this earlier: "Increasingly, the impact of anxiety disorders on QOL is being recognized and empirically documented, and current data indicate that both objective and subjective dimensions of QOL are significantly reduced in all of the anxiety disorders" (p. 278). Taken collectively, these data bolster Mendlowicz and Steins' (2000) call to action:

> Anxiety disorders…markedly compromise quality-of-life and psychosocial functioning in several domains…[and] it is hoped that these findings will translate into a more accurate public (and health care policy) view of anxiety disorders as serious mental disorders worthy of future research and appropriate health care expenditures. (p. 680)

Pediatric Quality of Life and Mental Illness

Childhood and adolescence are regarded as periods of optimum health (Millstein, 1989), yet youth are clearly not immune from the ravages of illness. The sobering

reality is that children and adolescents are among the most medically underserved in the United States (McManus, Shejavali, & Fox, 2003). Because “adolescents represent 15% of our nation’s population and 100% of our nation’s future” (McManus et al., p. 1), recent literature suggests that researchers allocate energy toward better elucidating the psychological factors involved in the pediatric health promotion (Lerner, 2000; March et al., 1999; Tucker, 2002). Compared to otherwise psychologically healthy counterparts, children with psychiatric illness have a greater likelihood of developing another disorder post-remission and they are more likely to experience social, educational, and occupational impairment as they age (Costello et al., 2003).

Consistent with the dearth of research in this age group, measurement of pediatric QOL represents a long-disregarded topic of study (Bastiaansen et al., 2004; Kazdin, 2001; Sawyer et al., 2002). Important methodological considerations for this line of research regard how to best obtain meaningful child data. Researchers have offered mixed views on the advantages and disadvantage of child-self report, parent-proxy report, and informed-other report (e.g., health care provider and teacher). Methodological issues such as age-appropriateness of language, syntax, and response options have led some to view structured interviews and interviewer-administered instruments as optimal (Matza, Swensen, Flood, Secnik, & Leidy, 2004) Whereas inherent costs may be prohibitive (Ravens-Sieberer et al., 2006), issues regarding potential limits of children’s comprehension of more abstract psychological constructs appear to represent a more foundational constraint. For instance, given that low concordance rates between adults and significant others have been detected in adults with psychiatric disorders (Saintfort, Becker, & Diamond, 1996), it is not surprising that

such disparities emerge in pediatric research examining complex psychological constructs such as anxiety. In one study, findings revealed that all 8-year-old participants understood the word “nervous” compared to only 57% of 5-year-olds (Rebok et al., 2001). Developmental comprehension concerns notwithstanding, some researchers concluded that parent-proxy report may offer greater reliability (Matza et al.), yet this benefit may come with a loss of validity in age groups better suited for self-assessment. General consensus appears to point toward incorporating disparate points of view (Ravens-Sieberer et al.), with design decisions ultimately deriving from the particular aims of a given study and the intended use of the data.

In part due to the challenging methodological considerations, the scarcity of pediatric QOL research is unsettling in light of Costello and colleagues’ (2003) longitudinal community study (*N* = 1420) that investigated the prevalence and development of psychiatric disorders in children from ages 9 through 16 years. Findings suggested that at least 1 in 3 children will suffer from 1 or more psychiatric disorders by the age of 16. Furthermore, children with significant emotional/behavior disorders have a higher likelihood of developing another disorder after remission than do their unaffected peers. The authors of this epidemiologic study reported that affected children are also more likely to experience functional impairment (i.e., interpersonal, educational, vocational) as they grow older.

This enduring cascade of effects beyond the symptoms seems especially relevant for the QOL implications for pediatric anxiety disorders, which frequently last for decades or an entire lifetime if left untreated (Barlow, 2000). Yet there is nearly a complete lack of research on QOL and pediatric anxiety. Matza and researchers (2004)

emphasized that current pediatric QOL literature examines almost exclusively medical diseases. This fact is surprising in light of observations that children with psychiatric disorders report more compromised QOL in many areas in comparison to children with physical disorders (Landgraf, Abetz, & Ware, 1996; Sawyer et al., 2002). Only a few studies have focused on QOL and pediatric psychopathology (Bastiaansen et al., 2004, 2005; Clark & Kirisci, 1996; Sawyer et al.). The limited data suggest that children with psychiatric disorders have a substantially poorer overall QOL relative to other children.

Bastiaansen and colleagues (2005) investigated the context in which the effects of pediatric psychopathology on QOL differ. They found that girls experienced a greater detrimental impact of mental illness on QOL than boys. They argued that one possible explanation might be that boys tend to exhibit externalizing behavior, and thus may not experience their symptoms as adversely as girls, who may be less prone to externalizing. The authors also found that the negative impact of mental disorders on children's QOL increased with age. Thus, beyond their demographic value, sex and age appear to be of interest with regard to studies of pediatric QOL.

Clark and Kirisci (1996) found that the effects on QOL differed by diagnosis for adolescents in their mixed clinical and community sample. Specifically, self-report data on youth between the ages of 12 and 18 with Major Depressive Disorder (MDD) indicated significant adverse effects on social and academic functioning. Whereas youth with PTSD exhibited a similar pattern, those who endorsed substance use had impoverished role functioning. Sawyer and researchers (2002) found that youth aged 6 to 17 years with MDD had more impaired QOL relative to children with ADHD and

Conduct Disorder. Compared to youth with purely physical disorders, youth with psychiatric disorders had substantially poorer QOL in multiple domains.

In what appears to be the first study on QOL with respect to pediatric anxiety, Bastiaansen and colleagues (2004) examined QOL in a clinical sample of 310 children between the ages of 6 and18. They employed the *Pediatric Quality of Life Inventory Version 4.0* (PedsQL; Varni, Seid, & Kurtin, 2001) to measure QOL within four subdomains: physical, emotional, social, and school functioning. Assessing QOL differences across six diagnostic categories, they concluded that specific disorders were associated with different impacts across QOL subdomains. The authors cautioned that "clinicians may consider anxiety disorders less severe than other psychiatric disorders" (p. 228), which may reflect a clinical bias to underestimate the effects of pediatric anxiety. Nonetheless, their results indicated that youth with anxiety disorders had poorer emotional functioning in comparison to those with other disorders on both parent and clinician reports. These findings align with those of Mogotsi and colleagues (2000) who also detected compromised QOL in anxious adults.

Thus, there are many compelling reasons to examine QOL and pediatric anxiety. Of course there is value in the treatment of symptoms, and outcomes may improve if interventions also target other factors that may affect QOL (Bastiaansen et al., 2004). Given that QOL encompasses physical, emotional, and social well-being (Bullinger, 2002; Ravens-Sieberer et al., 2006), it is conceivable that threats to pediatric QOL may be affected by developing a richer understanding of the cognitive processes related to mental disorders. Executive functioning may represent one such construct. This construct refers to cognitive processes that govern self-regulation (Sarsour et al., 2011).

Psychiatric disorders have been associated with compromised EF (Airaksinen et al., 2005; Boonstra, Oosterlaan, Sargeant, & Buitelaar, 2005; Emerson, Mollet, & Harrison, 2005; Francis, 1988; Julian & Arnett, 2009; Kendall & Chansky, 1991; Micco et al., 2009; Toren, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Furthermore, deficits in EF have been reliably associated with behavioral disturbance, restricted academic/occupational achievement, and social dysfunction (Baron, 2004; Lezak et al., 2004). Noting that these domains are integral to QOL, we now turn to relevant EF literature.

Executive Functioning

Although a neurobiological examination of EF exceeds the scope of this study, a brief explanation of terminology is appropriate. As indicated in Elliot's (2003) review, EFs are commonly thought of as higher order cognitive processes carried out in neural networks including the prefrontal cortex, thalamus, and basal ganglia. Miyake and researchers (2000) explained that the terms "frontal lobe tasks" and "executive tasks/functions" are often employed synonymously in spite of their practical dissimilarity: studies on patients with frontal lobe lesions or other physical insults have revealed variable degrees of functional impairment or no impairment whatsoever (Reitan & Wolfson, 1994; Shallice & Burgess, 1991). Such contradictory findings suggest the imprecision of interchangeably using the anatomical term "frontal lobe" and the functional term "executive." Usage of functional terminology (including the plural "executive functions," or EFs) better fits the purpose of the present study.

Even though a consensually agreed upon definition of the construct is lacking (Hughes & Ensor, 2008), EF generally encompasses planning, information updating and monitoring, mental set shifting, inhibition of prepotent responses, and

commencing/maintaining mental and physical activity (Lehto et al., 2003; Lezak et al., 2004; Mikaye et al., 2000; Smitherman et al., 2007). EF refers to cognitive skills used to "effortfully guide behavior toward a goal" (Banich, 2009, p. 89) or an individual's efforts to modify her or his inner state and responses to contextual demands (Graziano, McNamara, Geffken, & Reid, 2011).

Executive functioning is often personified as a business executive who lacks specialization in a specific domain and instead oversees and manages multiple sub-domains (Salthouse, Atkinson, & Berish, 2003). Optimal living depends on one's capacity to assess options and choose from them. These choices often but not always pit proximal versus distal fulfillment: our "actions are often directed toward achieving a positive outcome in a simulated future context and must therefore compete with alternative actions that might maximize initial benefits but have larger long term-costs" (Willcutt et al., 2005, p. 1336). In sum, EF refers to the neurocognitive processes that enable us to sustain an appropriate problem solving set in order to move toward future goals. Despite the apparent lack of an integrated account of EF to guide research (Banich, 2009), factor analytic studies have supported a three factor model: mental set shifting, information updating and monitoring, and inhibition (Lehto et al., 2003; Miyake et al., 2000).

Executive Functioning and Mental Health

It follows intuitively that researchers have emphasized EF's centrality to attaining success in school, work, and life in general (Diamond, Barnett, Thomas, & Munro, 2007). An emerging literature demonstrates EF's associations with various health processes and outcomes. For instance, adult studies have linked obesity to EF impairments (Ellis et al., 2004; Seeyave et al., 2009; Wang et al., 2001). Studies in

pediatric samples have detected similar results (Braet, Claus, Verbeken, & Van Vlierberghe, 2007; Cserjesi, Luminet, Molnar, & Lenard, 2007).

Regarding EF's nexus with psychopathology, there is a growing corpus of studies demonstrating the links between executive dysfunction and Schizophrenia, Bipolar Disorder, Attention-Deficit/Hyperactivity Disorder, Depression, and substance use disorders (Banich, 2009). The majority of studies have examined ADHD (for a review, see Boonstra et al., 2005). Prevailing theoretical and neurobiological explanations maintain that EF deficits, chiefly as evidenced in behavioral dis-inhibition, may be of etiological relevance for the disorder (Barkley, 1997; Durston, 2003). In their meta-analytic review, Wilcutt and colleagues (2005) concluded that although EF impairment alone is "neither necessary nor sufficient to cause all cases of ADHD" (p. 1343), significant executive dysfunction in key domains (e.g., response inhibition and planning) is associated with the disorder. Not only does this line of research illustrate EF's role within psychiatric disorders, it also raises questions of practical, theoretical, and clinical importance related to how EF affects and/or is affected by psychopathology. Due to the typically early onset of anxiety (Kessler & Greenberg, 2002) and Eysenck and colleagues' (2007) theoretical prediction that anxiety causes EF impairment, research on EF and anxiety appears warranted because of the plausible etiological relevance of executive dysfunction cited earlier in this paragraph.

In comparison to studies examining EF's relation to ADHD, research on EF and clinical anxiety is scarce (Castaneda et al., 2008). Consequently, researchers have highlighted the need for inquiry in this area. Julian and Arnett (2009) studied the independent contributions of anxiety and depression to EF in a sample of adults with

Multiple Sclerosis (MS). They found that each disorder predicted EF dysfunction. They concluded that the more traditional assessment of only depressive symptoms in MS patients is insufficient and argued for anxiety assessment as well because "the treatment of anxiety may benefit patients with MS not only by alleviating psychiatric distress, but also by increasing the availability of cognitive resources" (p. 802). Airaksinen and colleagues (2005) examined the EF/anxiety connection in their population-based study of adult neuropsychological functioning in Sweden. The authors concluded that adults with anxiety disorders exhibited significant EF impairment relative to healthy controls. Specifically, EF deficits were noted in participants with OCD and Panic Disorder with and without agoraphobia. The authors asserted that compromised EFs associated with anxiety disorders may have a deleterious influence on QOL, and, in particular, social and occupational functioning. Before returning to this critical point of contact between anxiety, EF, and QOL, a review of studies on pediatric anxiety and EF is in order.

Executive Functioning and Pediatric Mental Health

Adult evidence to the contrary notwithstanding (Smitherman et al., 2007), a small yet compelling body of literature attests to the link between pediatric anxiety and executive dysfunction (Emerson et al., 2005; Francis, 1988; Kendall & Chansky, 1991; Micco et al., 2009; Toren et al., 2000). Francis (1988) reported that children with higher anxiety levels also had significantly more task-inhibiting thoughts. Similarly, Kendall and Chansky (1991) found that children with anxiety disorders complained of frequent intrusive thoughts during cognitive tasks. Participants between the ages of 9 and 14 exhibited difficulties in shifting attention from internal to external stimuli. Toren and researchers (2000) studied neurocognitive correlates of anxiety disorders in a sample of

Hebrew youth ranging from 6 to 18 years in age. Compared to age-matched non-anxious controls, the clinical participants exhibited decreased cognitive flexibility. Emerson and colleagues (2005) assessed anxious-depressed boys between 9 and 11 years of age. They noted EF deficits in their clinical sample relative to their non-anxious, non-depressed peers. Specifically, the anxious-depressed participants demonstrated significantly poorer ability on set shifting, hypothesis testing, and problem solving.

Available research clearly demonstrates the association between anxiety and executive dysfunction. Methodological constraints, however, limit conclusions regarding causality. Although the results are tempered by the study's design, Micco and colleagues' (2009) research provided tentative data regarding directionality of effects. The authors examined 147 children of parents with Major Depressive Disorder (MDD), Panic Disorder (PD), the two comorbid disorders, and parents who did not meet diagnostic criteria for any mood or anxiety disorder. Their aim was to assess whether offspring (between the ages of 6 and 17) of parents with these mental disorders would show compromised EF relative to the children of the healthy control parents. Citing prior research supporting greater rates of psychopathology in the offspring of affected parents as compared to the offspring of healthy parents, the authors expected that if deficits in EF were markers for the development of MDD or PD, then they would detect more impaired EF in the children at risk for depression and anxiety. However, they found no association between offspring status and executive dysfunction. They concluded that compromised EF may not serve as a trait marker for developing anxiety or depression. Of additional value, their results indicated that the children with current

depressive and anxiety symptoms also exhibited EF impairment. Thus, executive dysfunction appeared to be symptomatic of the current disorder rather than a cause of the disorder. Caution must be exercised when interpreting these findings given the cross-sectional and correlational nature of the data. Nevertheless, Micco and colleagues' findings tentatively support the notion that anxiety leads to executive dysfunction rather than vice versa. These data coincide with Eysenck and colleagues' (2007) theoretical stance that anxiety disrupts EF, as explicated by ACT.

Attentional Control Theory: Anxiety Impairs Executive Functioning

Within the cognitive psychology literature, a substantial body of studies examines the relationship between anxiety and cognitive performance (for a review, see Eysenck, 1992). Anxiety is defined as "an aversive emotional and motivational state…[and] individuals frequently worry about the threat to a current goal and try to develop effective strategies to reduce anxiety and achieve the goal" (Eysenck et al., 2007, p. 336). Findings generally indicate that anxiety detrimentally influences cognitive performance, and its disruptive effects tend to be more damaging with increasingly complex/cognitively demanding tasks. According to Deraksan and Eysenck's (2009) review, empirical support for this generalization has been detected when anxiety is regarded as a temporary mood state (i.e., state anxiety) and a relatively stable aspect of personality (i.e., trait anxiety).

Theory as to how anxiety affects cognitive functioning is continually evolving. Eysenck and Calvo (1992) elaborated Processing Efficiency Theory (PET) to address gaps in extant theory (e.g., Sarason, 1988) on how anxiety diminishes cognitive performance. In brief, underlying the theory was the assumption that anxiety drained cognitive resources by impairing EF and overall processing efficiency. Although the

theory generated considerable research supporting its assumptions (for a review, see Eysenck et al., 2007), precision regarding the specific nature of EF impairment was lacking (Derakshan & Eysenck, 2009).

Spurred by this imprecision and advances in EF research (Friedman & Miyake, 2004; Miyake et al., 2000), Eysenck and colleagues (2007) developed Attentional Control Theory (ACT). In its short existence ACT has already gained substantial empirical backing (Derakshan & Eysenck, 2009), perhaps due to the fact that it further articulates the already well supported PET (Eysenck & Calvo, 1994). An in-depth account of ACT falls outside the scope of the present study. Of relevance is ACT's prediction that anxiety impairs two subcomponents of EF: the ability to inhibit a prepotent response and the ability to shift back and forth to meet situational demands. Whereas PET posited that anxiety compromises EF and "impairs processing efficiency because anxiety produces worry" (Eysenck et al., p. 339), ACT contends that anxiety specifically leads to executive dysfunction in set shifting and inhibition. As the authors emphasized, a consensually agreed upon definition of EF is not existent, yet factor analyses have supported three principal EF domains: mental set shifting, information updating and monitoring, and inhibition of prepotent responses (Lehto et al., 2003; Miyake et al., 2000).

Unlike most PET- and ACT-informed research, the present study did not focus on narrowly defined areas of performance (e.g., continuous motor, saccade, reading, visuospatial, and reaction time tasks). It instead relied on their theoretical predictions that anxiety negatively influences EF in a less context-specific manner. To date, PET and ACT have been utilized almost exclusively to predict the effects of anxiety on

EF/cognitive performance in non-clinical populations (Derakshan & Eysenck, 2009). The present study expands the literature by beginning to investigate anxiety's impact on EF in a psychiatric population, which addresses the theory's authors' call for increased research in "nonstressful conditions" (Eysenck et al., 2007, p. 349).

In conclusion, prior research has demonstrated the association between anxiety and impaired EF. Eysenck and colleagues' (2007) most recent theory of attentional control predicts that their relationship is more defined: anxiety disrupts EF, particularly in its shifting and inhibiting subcomponents. Executive dysfunction, in turn, has been linked with behavioral disturbance, constrained educational/vocational achievement, and interpersonal strife. These domains figure prominently in QOL. Consequently, it stands to reason that disorders linked with compromised EF—which is "critical for self-directed behavior"—might be at increased risk for QOL erosion (Banich, 2009, p. 90). Similar relationships have been detected in studies of ADHD (Klassen, Miller, & Fine, 2004), traumatic brain injuries (Horneman, Folkesson, Sintonen, Von Wendt, & Emanuelson, 2005), and epilepsy (Sherman, Slick, & Eyrl, 2006). Research on the role of EF in pediatric anxiety with regard to QOL is sparse. Following Airaksinen and colleagues' (2005) postulation that EF deficits would likely predict impaired QOL in anxious individuals, the present study addressed this vital gap in the literature using cognitive psychological theory as a conceptual framework from which to base its predictions.

Specific Aims and Hypotheses

Specific Aim 1

The first aim of the present study was to investigate the nature of the association between anxiety, EF as a unitary construct, and QOL in children and adolescents.

Hypothesis 1 (A and B)

In light of the reviewed literature (e.g., Airaksinen et al., 2005; Bastiaansen et al., 2004, 2005; Mendlowicz & Stein, 2000; Micco et al., 2009) and theory that anxiety disrupts EF (PET; Eysenck & Calvo, 1992), it was hypothesized that EF would mediate the relationship between anxiety and QOL. Specifically, increased scores on a measure of anxiety were hypothesized to be associated with poorer scores on a measure of EF, which in turn were hypothesized to be associated with reduced scores on a measure of QOL. Hypothesis 1A examined parent reports on anxiety and QOL mediated by parent-assessed EF. Hypothesis 1B evaluated children's self-reports of anxiety and QOL mediated by parent-assessed EF. Based on prior research demonstrating the associations between age and EF (Diamond, 2007; Zelazo et al., 2003), sex and QOL (Bastiaansen, Koot, & Ferdinand, 2005), and racial/ethnic status and QOL (Utsey et al., 2002), the model included age, sex, and ethnicity as covariates. A primary diagnosis of ADHD was also covaried due to its substantial representation in the total sample (56%) and documented links with executive dysfunction (Barkley, 1997).

Specific Aim 2

Whereas the first aim investigated a global EF construct as a mediator for the relation between pediatric anxiety and QOL, the second aim examined the mediating functions of specific EF subcomponents.

Hypothesis 2 (A through D)

Hypothesis 1 focused on EF as a unified construct. Capitalizing on theoretical advances in how anxiety impairs EF, Hypothesis 2 dismantled EF into various subcomponents. In keeping with ACT's prediction that anxiety specifically compromises inhibit and shift domains of EF (Eysenck et al., 2007), it was hypothesized that the

corresponding inhibit and shift scales of the utilized EF measure would demonstrate stronger mediation effects than the other non-theory relevant EF subcomponent scales (e.g., updating). Six mediation models using different raters (i.e., parent or child) and alternate EF measurement (i.e., parent assessment or child performance) were tested. Inhibition/shifting were tested as mediators of parent-assessed anxiety and QOL (Hypothesis 2A.1). Inhibition/shifting were also evaluated as mediators of child self-reported anxiety and QOL (Hypothesis 2B.1). Statistically significant results supporting the differing mediation roles of EF subdomains led to exploratory post-hoc analyses (Analyses 2A.2 and 2B.2) of slightly different EF subdomains derived from prior empirical findings for the related EF measure (Gioa, Isquith, Retzlaff, & Espy, 2002). Finally, inhibition and monitoring as measured on a child performance task were evaluated as mediators of parent-assessed anxiety and QOL (Hypothesis 2C) and child self-reported anxiety and QOL (Hypothesis 2D). These analyses also covaried age, sex, ethnicity, and ADHD status.

Post-hoc Analyses

Likely an artifact of the disparate emphases of the cited studies, the absence of analyses on pharmacological interventions is apparent in the reviewed literature. Medication effects are worthy of examination in the current sample due to the proportion of participants reporting use of prescription medication (60% of total sample). Whereas a comprehensive assessment of this critical topic merits dedicated studies, follow-up analyses were conducted to assess potential effects related to participants' medication use. Scores for the total sample parent-assessed anxiety and QOL were analyzed with the BRIEF total score as the mediator variable. Medication status (i.e., on medication versus medication-naïve) was covaried, and covariates from the prior best fitting models

were included. Results were aggregated across 20 multiply imputed data sets. Lastly, medication status was assessed as a potential moderator of anxiety's effects on EF, and then again as a moderator of EF's effects on QOL (Figure 2-7).

CHAPTER 2
METHOD

Participants

Data for participants in the present study was drawn from an archive of youth presenting for assessment at an outpatient psychiatric clinic during the period of January 2009 to May 2010. Participants were typically referred for assessment by pediatricians or child psychiatrists. The clinic was housed within the Department of Psychiatry in the College of Medicine at a large southeastern university. In addition to assessment services, the clinic provided ongoing cognitive-behavioral treatments for a variety of psychological disorders. A licensed psychologist with more than twenty years of clinical experience supervised the pre-doctoral level psychology interns who performed the assessments.

Table 2-1 summarizes the data handling and analyses steps to facilitate comprehension of the sequence used to construct the subsamples and perform the analyses. Table 2-2 presents demographic information, such as age, sex, and racial/ethnic distribution. The total sample included data for 108 individuals between the ages of 4 and 18, with a mean age of 10.8 years.[1] Some analyses utilized age-restricted subsamples, given their examination of age-specific measures as follows: Sample A, the total sample, 108 individuals, all ages (*M* = 10.8 years, *SD* = 3.4); Sample B, 42 individuals, participants ages 12 years and older (*M* = 14.4 years, *SD* = 1.4); and Sample C, 81 individuals, participants ages 8 years and older (*M* = 12.2 years, *SD* = 2.6). Individuals in the overall sample were 28.7% female and 29.6% of the

[1] Sample A contained two individuals (ages 4.3 and 4.6 years) that fell below the BASC age normative guideline of 5 years old.

participants reported a non-White racial/ethnic cultural background (70.4% White, 14.8% African American, 9.3% Hispanic, 5.5% Other). Table 2-3 presents the primary diagnoses for individuals in each sample, including Attention-Deficit/Hyperactivity Disorder, Pervasive Developmental Disorders, Disruptive Behavior Disorders, Learning Disorders, Mood Disorders, Anxiety Disorders, Language Disorders, and other disorders. Note that most individuals had comorbid diagnoses: 76% in Sample A, 74% in Sample B, and 77% in Sample C. Table 2-4 presents the individuals' primary classes of reported psychotropic medications, including stimulants, antidepressants, atypical antipsychotics, and other mood stabilizers. Note that participants endorsed using multiple types of prescription psychotropic medications: 28%, 43%, and 33% in Samples A, B, and C, respectively.

Procedure

Assessments were performed to evaluate the individuals' psychological and emotional functioning. Referral questions typically addressed whether behavioral, learning, and/or emotional problems were present. Assessments also commonly evaluated individuals' academic performance in relation to their behavioral/emotional disturbances and cognitive functioning in order to inform subsequent treatment and academic accommodations. Prior to assessment, participants were informed that, upon their consent, their de-identified data would become part of a large HIPAA-compliant clinical research database maintained by the college's Division of Medical Psychology. Research staff obtained a signed informed consent from the legal guardian of all participants who agreed to have their data enter the database.

Before completing the assessment battery, participants and their parents or primary caregivers engaged in a one hour semi-structured clinical interview conducted

by a pre-doctoral level psychology intern and the licensed psychologist. Evaluations lasted between three and six hours depending on the referral question, the length of the participant-specific testing battery, and the participant's and examiner's joint pacing. Evaluations generally began at 9:00 a.m., ended around 3:00 p.m., and included a one hour break at midday for lunch. After the evaluation, the psychology intern composed an individualized report under the supervision of the licensed psychologist. The participant and parent or primary caregiver were invited to a feedback session with the intern and licensed psychologist approximately two to three weeks later in order to review the findings, discuss recommendations, and address other questions.

Measures

Consistent with general consensus on responsibly conducting pediatric quality of life (QOL) research (Bastiaansen et al., 2005; Ravens-Sieberer et al., 2006), data in the present study regarding the child and adolescent participants were collected from both parents/primary caregivers and the pediatric participants themselves when feasible. Because of the dependent nature of the parent-child relationship and the centrality of the parent's perspective in determining whether a child will seek treatment (Matza et al., 2004), parent/primary caregiver data figured prominently in the study. Researchers have argued that the perspective of significant others is vital in psychiatric research because mental health symptoms may distort self-assessment (Saintfort et al., 1996). Younger children's limitations in self-assessment of complex psychological symptoms such as anxiety have also been documented (Rebok et al., 2001). Additionally, due to evidence that executive dysfunction may be detected better by family members who can assess children's performance in everyday life situations, the "most well-known" (Sherman et al., 2006, p. 1938) parent-proxy assessment of EF was administered.

Measures Completed by the Parents/Primary Caregivers

Demographics. Parents or primary caregivers completed a general demographic form, including child's age, sex, ethnicity, and medication status.

Quality of life. The parent-proxy version of the Pediatric Quality of Life Inventory Version 4.0 (PedsQL; Varni, Seid, & Kurtin, 2001) was administered to assess QOL. This measure was designed to capture the World Health Organization's (1948) three core dimensions of functioning: Physical, Emotional, and Social. The measure also includes items evaluating academic functioning. The 23-item inventory typically requires fewer than 4 minutes to complete. Parents or caregivers indicated how much of a problem their child has had on each item during the past month using a 5-point Likert-type scale ranging from *never* to *almost always* (e.g., "In the past *ONE month*, how much of a *problem* has your child had with walking more than one block/feeling sad or blue/getting teased by other children/keeping up with schoolwork?"). Raw scores per item are on a 0–4 scale and are reverse scored and linearly transformed to a 0–100 scale (0 = 100, 1 = 75, 2 = 50, 3 = 25, 4 = 0). The total score is the mean of the 23 items, with higher scores representing better QOL.

Age-appropriate PedsQL versions were administered based on the following children's age ranges: ages 2–4, 5–7, 8–12, and 13–18 years. The items for each version are essentially identical, differing only in the usage of developmentally appropriate language. Varni and colleagues (2001) reported a high internal consistency coefficient for the total score ($\alpha = 0.92$). Similarly, Cronbach's alpha for the current sample was .91. Correlations with other measures of disease burden and the significantly different scores for children with and without a chronic health condition provide evidence for the instrument's validity (Varni, Burwinkle, Seid, & Skarr, 2003).

Consistent with prior research (Varni, Seid, & Kurtin, 2001), construct validity was further evidenced by significant associations between scores indicative of poorer QOL and more school absences, inability to play, and increased overall sickness.

Anxiety. The Parent Rating Scale (PRS) of the Behavior Assessment System for Children, Second Edition (BASC; Reynolds & Kamphaus, 2004) was completed by parents or primary caregivers to assess children's anxiety. The measure typically requires 10 to 20 minutes to complete. A commonly employed behavior checklist that assesses emotional and behavioral domains of children's functioning, the measure provides scores on broad internalizing, externalizing, and behavior symptom domains as well as specific adaptive/social functioning skills scales. As illustrated in these examples, anxiety items generally assess fear, worry, and nervousness. Different versions of the PRS correspond to ages 2 through 5 (Preschool form, 134 items), 6 through 11 years (Child form, 160 items) and ages 12 through 21 (Adolescent form, 150 items). The present study examined the Anxiety scale of the PRS, which has 13 items on the Preschool form, 14 items on the Child form, and 11 items on the Adolescent form. Items are rated on a four-point Likert-type scale of Never, Sometimes, Often, and Almost Always (e.g., caregivers assess the frequency of how often their child worries about making mistakes/is nervous/is fearful). Responses are associated with point values that are summed to form a raw score, which is then converted to a *T* score for the appropriate gender and age range. *T* scores greater than 65 indicate a significantly elevated level of anxiety (i.e., scores 1.5 SD above the normative mean; Reynolds & Kamphaus, 2004).

According to a review by Tan (2007), the BASC is psychometrically sound, including high internal consistency reliability across domains and evidence of good construct validity. Factor analyses detected moderate to high loadings, and criterion-related validity has been demonstrated by examining correlations between BASC composites/scales and other widely used child assessment instruments. The authors reported test-retest reliabilities in the low .90's and internal consistencies in the range of .80–.87. Alphas for parent forms in the current sample ranged from .84 to .90.

Executive functioning. The Behavior Rating Inventory of Executive Function (BRIEF; Gioa, Isquith, Guy, & Kenworthy, 2000) was completed by parents or primary caregivers to assess children's EF behaviors in a real-world setting. The instrument contains 86 items that measure different aspects of EF in children between the ages of 5 and 18. It typically requires 10 to 15 minutes to complete the measure. There are eight theoretically and empirically derived scales in total. Inhibit, Shift, and Emotional Control constitute the Behavioral Regulation Index (BRI). Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor form the Metacognition Index (MI). Collectively, these indices form the Global Executive Composite (GEC). Within the Behavioral Regulation Index, the Inhibit scale assesses one's ability to control impulses and appropriately stop behavior. The Shift scale measures the ability to transition freely from one task to another or to shift from one aspect of a problem to another in accordance with contextual demands. The Emotional Control scale measures the ability to control emotional responses appropriately.

Within the Metacognition Index, the Initiate scale measures the ability to begin a task and independently produce problem-solving strategies. The Working Memory

scale assesses one's ability to retain information in order to complete a task. Whereas the Plan/Organize scale measures the ability to anticipate future events or perform tasks systematically, the Organization of Materials scale assesses the ability to maintain an orderly play area or workspace. Finally, the Monitor scale assesses one's ability to monitor progress on work or be attuned to one's behavioral impact on others. Items for the BRIEF are rated on a 3-point scale (1 = never, 2 = sometimes, 3 = often). Raw scores for scales are converted to standard scores by summing their items, then obtaining the corresponding *T* value for the appropriate gender and age range. Index scores are calculated by summing their respective raw scale scores, which has a related *T* value. For the total score (GEC), raw scores are summed for the BRI and MI. The total is matched with a *T* value. A standardized score equal to or greater than 65 (i.e., scores 1.5 SDs above the mean) indicate significant elevation in executive dysfunction.

The BRIEF has established convergent reliability with related measures and samples have demonstrated internal consistency reliabilities in the range of .88–.98 across all scales and indices (Gioa et al., 2000). Internal consistency alphas for the analogous indices used in the current sample ranged from .69 to .88. Attesting to the ecological validity of the BRIEF, Donders (2002) commented that the instrument assessed molar aspects of everyday behavior, and it "appears to measure something that is not routinely captured by other existing instruments, and that it may offer incremental knowledge about the daily functioning of children and adolescents" (p. 230). In relation to the instrument's capacity to discern between EF subdomains, findings from Gioia, Isquith, Retzlaff, and Espy's (2002) factor analysis supported the discriminating

value of the BRI and MI. They also provided evidence for a three factor solution structure for the BRIEF, which further supports a fractionated view of EF and was explored in post-hoc analyses of the present study.

Measures Completed by the Child/Adolescent Participant

Quality of life. The self-report version of the PedsQL (Varni, Seid, & Kurtin, 2001) was administered to participants age 12 and older in order to assess QOL from the child/adolescents' perspective. In their 2003 study, Varni and colleagues reported an internal consistency alpha of .89. Similarly, the alpha for the current sample was .90. Consistent with the findings for the parent-proxy form in the 2003 study and prior research (Varni et al., 2001), construct validity for the child self-report was supported by the relationship between poorer self-report scores and protracted school absences/general sickness. Self-report forms are scored in the same fashion as the parent forms indicated above.

Anxiety. The Self-Report of Personality (SRP) of the BASC (Reynolds & Kamphaus, 2004) was completed by children and adolescents ages 12 and older. The SRP includes items in True/False format and the four-point Likert-type scale used with the PRS. The SRP forms require 20–30 minutes to complete. Tan (2007) noted that the instrument has solid psychometric properties, with factor analytic studies yielding moderate to high loadings in support of construct validity. Test-retest reliabilities were reported in the range of upper .70s to low .80s for SRP scales for respondents below the college level (Reynolds & Kamphaus, 2004). Cronbach's alpha for the Anxiety scale in the current sample was .84. Scoring parallels that of the parent forms already discussed.

Executive functioning. The Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) was administered to child/adolescent participants ages 8 and older. This instrument assesses critical EFs (e.g., including flexibility in thinking, category switching, and the ability to inhibit automatic or dominant responses) by measuring participants' performance in a game-like format in a controlled clinic setting.

The present study utilized three specific D-KEFS tests: (a) Trail-Making, (b) Verbal Fluency, and (c) Color-Word Interference. The Trail-Making Test measures flexibility of thinking on visual motor tasks in five different conditions: Visual Scanning, Number Sequencing, Letter sequencing, Number-Letter Switching, and Motor Speed. The Verbal Fluency Test measures ability to generate verbal responses according to set rules within a 60 second time period. The following three conditions were administered: Letter Fluency, Category Fluency, and Category Switching. Lastly, a version of the Stroop Test (Stroop, 1935), the Color-Word Interference Test was given to measure inhibition of verbal responses through naming incongruent ink colors. All four increasingly more complex conditions were administered. Raw scores are converted to standardized scores (M = 10; SD = 3) for each test.

Influenced by prior factor analytic research (Latzman & Markon, 2010), composites for Inhibit and Monitoring EF subdomains were constructed using available archival data. Sufficient data were not available to assess the Shifting subdomain. It is noteworthy that whereas the Inhibition factor continues to denote the capacity to deliberately inhibit prepotent or automatic responses, the Monitoring factor "is similar to Miyake's updating dimension, [which] reflects the abilities of actively monitoring and evaluating information" (Latzman et al., p. 456).

Although internal consistency alphas in the current sample ranged from .75 to .87, the D-KEFS' psychometric properties have generated considerable debate in the literature regarding the instrument's limitations and appropriate use. According to a review by Shunk, Davis, and Dean (2006), internal consistency coefficients ranged from low to high across the tests and age groups, which extend to an upper limit of 89 years. They added that "this has been a popular criticism of the D-KEFS system but does not pose serious concern because of the difficulties associated with measuring executive functioning" (p. 277). Whereas Schmidt (2003) pointed out the scant evidence supporting the measure's validity, Delis, Kramer, Kaplan, and Holdnack (2004) retorted with a list of more than 25 studies demonstrating the D-KEFS' sensitivity to assess EF capacity in various clinical groups. Homack, Lee, and Riccio's (2005) test review further attested to the discriminant and convergent validity by citing several supporting studies. However, they also noted that it is unclear to what extent the instrument assesses EF in everyday functioning due to a lack of evidence demonstrating its ecological validity. Taken collectively, proper caution is required when interpreting D-KEFS scores, and it appears judicious to heed prior researchers' suggestions that this measure might best be characterized as a research tool that can expand our knowledge of EF within more controlled (i.e., research lab or clinic) environments (Crawford, Sutherland, & Garthwaite, 2008; Homack et al.).

Analyses

After reviewing various analytical approaches to test the hypothesis that EF mediates the effects of anxiety on QOL in youth, mediation analyses were conducted using Preacher and Hayes' (2008) *indirect.sps* macro (SPSS version 18.0.3). The first

hypothesis positioned EF (BRIEF) as a potential mediator for the effect of anxiety (BASC) on QOL (PedsQL). The second hypothesis tested a deconstructed EF model using the BRIEF's two primary indices (i.e., BRI and MI) as simultaneous mediators. Significant differences in this model inspired an exploratory post-hoc analysis of alternative empirically supported BRIEF factors (Gioa et al., 2002) as simultaneous mediators to more fully assess the mediation capacity of EF subdomains. Finally, a subsequent model investigated EF's multidimensional nature using two factors from the D-KEFS child-performance measure. All models tested the effects of age, sex, ethnicity, and ADHD as covariates as suggested by prior research (Bastiaansen et al., 2005; Barkley, 1997; Diamond et al., 2007; Utsey et al., 2002). A description of how missing data were handled is followed by the rationale for conducting the mediation analysis. Finally, a closer look at the analyses for each hypothesis concludes the chapter.

Multiple Imputation for Missing Values

Each variable or measure examined in this study demonstrated less than ten percent missing values. Step two of Table 2-1 summarizes the process followed for addressing missing values. As discussed and recommended by Schlomer, Bauman, and Card (2010), missing values were handled by utilizing the multiple imputation capabilities of the Amelia package (Honaker, King, & Blackwell, 2011) of R software (R Development Core Team, 2010). Given that Amelia relies on the assumption of multivariate normality (i.e., imputed values are drawn from a normal distribution), the imputation was conducted only after variable values were linearly transformed to achieve univariate normality in effort to enhance multivariate normality. For example, a transformed PedsQL variable was calculated as follows: Transformed PedsQL =

[max(PedsQL) + 1 - PedsQL]^.8. Different transformations were utilized for each variable to best accommodate their distributions. Subsequently, multivariate normality was tested and supported with the energy test of multivariate normality (E = 1.988, p = .593; mvnorm.etest function of ENERGY package for R; Rizzo & Szekely, 2011), even though multiple imputation with non-normal data has proven effective/successful (Graham & Schafer, 1999).

The Amelia function also requires the assumption that data are missing at random (MAR) or missing completely at random (MCAR). As suggested by Schlomer and colleagues (2010), dummy variables were calculated to indicate "missing" or "'nonmissing" for each variable and were shown to be correlated with other variables used in the analyses and imputations, thereby suggesting a MAR pattern of missingness. For example, all dummy variables were significantly correlated with age. The only exception was the PedsQL dummy variable, which lacked two values. Follow-up inspection of the omitted values yielded no discernible pattern, and thus missingness for PedsQL was assumed to be MCAR. Overall, although it appeared the data were MAR, it may not have been problematic if missingness were not missing at random (NMAR), given that multiple imputation has performed reasonably well with NMAR data (Buhi, Goodson, & Neilands, 2008).

Following the 20 multiple imputations of missing values, variable values were reverse-transformed to their original state. Samples were then created according to the measure-appropriate age-ranges indicated in step 3 of Table 2-1. Sample A consisted of all participants. Sample B consisted of child participants ages 12 years and older as appropriate for the self-report form of the BASC. Sample C consisted of child

participants ages 8 years and older as appropriate for the D-KEFS. To facilitate interpretation and reduce possible effects of multicollinearity, continuous predictor variables were then centered on their within-sample means. Dummy variables for female, minority, and ADHD were left as originally coded (i.e., female, minority, ADHD diagnosis = 1; male, White, non-ADHD diagnosis = 0).

As reported in steps four and five of Table 2-1, all analyses were performed on each multiply imputed data set. Results from these analyses were aggregated using Rubin's (1987) guidelines for combining results from multiply imputed data (*mi.inference* function of NORM package for R; Novo, Schafer, & Fox, 2011). Given that Rubin's methods rely on estimate values, their corresponding standard errors, and normal-theory, this study's bootstrap-based estimates (not having standard error estimates) and confidence intervals (CIs) were aggregated simply by calculating the mean estimate and CI across results from multiply imputed data.

Rationale for Mediation Analysis

Zanna and Fazio (1982) described the generations through which a line of research evolves. A first-generation research question for the line of research at hand would be whether anxiety and EF are related to QOL. A simple correlational analysis could examine whether these relations exist, and a multiple regression analysis could examine the size of the effect either anxiety or EF have on QOL, while controlling for the effects of each other. As reviewed herein, various studies have already demonstrated such correlations between anxiety and QOL (see reviews by Mendlowicz & Stein, 2000; Olatunji et al., 2007); anxiety and EF (e.g., Airaksinen et al., 2004; Micco et al., 2009); and EF and QOL (e.g., Klassen et al., 2004; Sherman et al., 2006).

A second-generation research question would be when, or under what conditions these relations exist or vary. For example, studies have demonstrated that variables such as age and sex have more than demographic value in pediatric QOL research. Bastiaansen and colleagues (2005) examined different factors that impact QOL in a sample of children with psychiatric disorders. Results indicated that the deleterious impact of mental disorders on children's QOL increased with age. Mental illness also affected girls' QOL more than it did boy's QOL. These findings help support the inclusion of age and sex as covariates in the present study.

The present study proposed to address what Zanna and Fazio (1982) called a third-generation research question. It examined the process or mechanism underlying the relationship between anxiety and QOL. Castaneda and colleagues (2008) underscored the value in better elucidating this relationship specifically for these variables. In accordance with ACT (Eysenck et al., 2007), it was hypothesized that EF would be a mechanism though which anxiety affects QOL. Specifically, the hypothesis was that higher anxiety would be associated with (or lead to) reduced EF, which in turn would be associated with (or lead to) reduced QOL. This is a basic mediation model, with EF mediating the effects of anxiety on QOL.

Using standard path label nomenclature (cf. Fritz & MacKinnon, 2007), Figure 2-1 presents a path diagram for a model in which the independent variable *X* (i.e., anxiety) has an effect on the dependent variable *Y* (i.e., QOL). The path coefficient *c* represents the total effect of *X* on *Y*. Compare with Figure 2-2, which presents a path diagram for a model in which the effect of *X* on *Y* is mediated by a variable *M* (e.g., EF). The path coefficient *a* represents the effect of *X* on *M* and coefficient *b* represents the subsequent

effect of *M* on *Y*. The path coefficient *c'* represents the direct effect of *X* on *Y*. The indirect effect of *X* on *Y* could be represented by the product of coefficients *a* and *b* (i.e., *ab*). Thus, Figure 2-1's total effect *c* corresponds to the sum of Figure 2-2's direct effect *c'* and indirect effect *ab*. In notation, $c = c' + ab$.

To test the hypothesis that EF mediates the effect of anxiety on QOL (Figure 2-3), the analysis must test the process' sequence of effects. The most substantive question is whether the combined effect of *X* on *M* and *M* on *Y* —of anxiety on EF, and EF on QOL—is significantly different from zero. More succinctly, the analysis must test the significance of the *ab* product, the indirect effect of anxiety on QOL.

Evaluating approaches to mediation. Over the past few decades, methodologists for mediation analysis have streamlined approaches to testing the significance of combined effects such as this. Baron and Kenny (1986) helped researchers better understand and utilize meditational analyses. Although the basic guidelines they offered appeared responsible for the subsequent proliferation of mediation analyses, many researchers have continued using them despite mediation specialists' general consensus regarding their limited accuracy and essential obsolescence (Fritz & MacKinnon, 2007; Preacher & Hayes, 2004). Some methodologists have proposed using only the second and third steps of the four steps Baron and Kenny suggested for assessing mediation (i.e., *a* is significant and *b* is significant). Although this joint significance outperforms the four steps together and is more powerful than the Sobel test (Fritz & MacKinnon; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002), other approaches described below provide a more accurate estimate of the CI for the *ab* product.

The Sobel test (1982, 1986) performs the exact statistical test the proposed study aims to conduct—the significance of the *ab* product—but ultimately under-delivers. Its tests and estimates of statistical significance assume the *ab* sampling distribution is normal, whereas it is often skewed. MacKinnon and colleagues (2002) demonstrated the resulting relatively high rate of Type II errors. As a non-parametric alternative to the Sobel test, resampling or "bootstrapping" draws a large number of samples (e.g., 1000 samples or more, with replacement) from the data, each time estimating *ab* (MacKinnon, Lockwood, & Williams, 2004; Shrout & Bolger, 2002). Preacher and Hayes (2004) described a percentile approach in which they calculated a point estimate as the mean of these *ab* estimates. They suggested calculating a 95% CI by identifying estimates corresponding to the 2.5th and 97.6th percentiles when sorting the estimates by size. If this interval does not include the value zero, the implication is that *ab* is statistically significantly different from zero, at a 95% CI. However, given that the distribution of *ab* estimates is often skewed, the percentile-based interval often does not center on the point estimate. Preacher and Hayes' (2008) *indirect.sps* macro uses bias-correction (Efron & Tibshirani, 1993) to overcome the effects of this skew. This approach has demonstrated better accuracy and power (Fritz & MacKinnon, 2007).

In sum, having reviewed different approaches to mediation analysis, the present study utilized Preacher and Hayes' (2008) *indirect.sps* macro to generate bias-corrected and accelerated bootstrap CIs for indirect effects (5,000 resamples). This approach appears to be among the most well-established and powerful (Fritz & MacKinnon, 2007; Woody, 2011), and it has been recommended for psychological research (Mallinckrodt, Abraham, Wei, & Russell, 2006). Following Preacher and Kelley's (2011) guidelines,

kappa-squared effect sizes (κ^2) were estimated using the MBESS package (Kelley & Lai, 2010) in R Software (R Development Core Team, 2010).

The number of analyses outlined in the next sections inflates experimentwise error rate (i.e., Type I error). Rather than manage this increase by adopting a lower alpha, the significance criterion was kept at $p = .05$ due to the potential to contribute to an understudied and critical topic (Bastiaansen et al., 2004; Castaneda et al., 2008). Noting the debate over what level of significance to use, Howell (2002) recommended that the decision is most appropriately based on researchers' thoughtful inspection of the implications of Type I and Type II errors in their studies. Given the exploratory nature of this research, the decision was to accept a possibly higher Type I error rate in order to optimize the sensitivity of detecting theory-predicted effects to inform future research.

Hypothesis 1

Because of the theoretical reasons cited (Eysenck & Calvo, 1994; Eysenck et al., 2007), it was hypothesized that EF would mediate the relationship between anxiety and QOL, as illustrated in Figure 2-3. This hypothesis was tested in two iterations with (a) parent and (b) child raters for the anxiety and QOL variables. In Hypothesis 1A, it was expected that increased scores on the parent report of the BASC Anxiety scale (indicating more distressing anxiety levels) would be associated with increased scores on the BRIEF EF measure (indicating greater executive dysfunction), which in turn would be associated with reduced scores on the parent report PedsQL (indicating poorer QOL). In Hypothesis 1B, the same mediation via the BRIEF was tested for pediatric self-reported anxiety scores and pediatric self-reported QOL scores. Table 2-5 presents a tabulated conceptualization of how this study's various analyses related and

differed. The table demonstrates that Hypothesis 1A and 1B each use the BRIEF total score as a mediator, but differ in using the parent versus child forms of the PedsQL and BASC Anxiety scale. Models also covaried age, sex, ethnicity, and ADHD, as indicated by prior research (Barkley, 1997; Bastiaansen et al., 2005; Diamond, 2007; Utsey et al., 2002; Zelazo et al., 2003). Preacher and Kelley (2011) described the challenges and underuse of effect size measures with mediation models, and their strategies were used to calculate effect sizes for indirect effects (i.e., κ^2).

Hypothesis 2

Moving from a more global to granular level of analysis, Hypothesis 2 examined which specific aspects of EF mediated anxiety's effects on QOL (Figure 2-4). According to ACT (Eysenck et al., 2007), anxiety compromises the inhibit and shift domains of EF. In order to examine the specific EF domains, the Behavioral Regulation Index (BRI) of the BRIEF was hypothesized to be the primary mediator because it chiefly assesses inhibition and shifting, as compared to the primary updating EF subdomain measured by the Metacognition Index (MI). Analyses were completed in two iterations, both using the BRI and MI as simultaneous mediators of the effect of anxiety on QOL. Hypothesis 2A tested the simultaneous mediation for the parent forms of the BASC Anxiety scale and PedsQL. The second iteration tested the mediation using the child self-reports of anxiety and QOL. Again, it was hypothesized that the BRI (containing the inhibit and shift EF subdomains) would exhibit a stronger mediation effect than the MI (containing updating). The variables were analyzed using the *indirect.sps* macro. Age, sex, ethnicity, and ADHD were covaried. Effect sizes were determined as earlier.

Because results provided evidence for the differential mediation power of the BRIEF's BRI and MI indices, exploratory post-hoc analyses were conducted to further

investigate the multidimensional nature of EF. Specifically, the Behavioral Regulation factor and the Emotional Regulation factor of Gioia and colleagues' (2002) BRIEF confirmatory factor analysis were compared as simultaneous mediators (Figure 2-5). A note of caution is in order due to the semantic resemblance (e.g., BRI versus BR factor) in the designation of these EF measures. Emerging from an initial exploratory factor analysis vital to the construction of the BRIEF as an "everyday world" (Gioia et al., p. 251) measure of EF, the Behavioral Regulation Index (BRI) is one of the two primary indices of the instrument, and it consists of the Inhibit, Shift, and Emotional Control scales. Subsequent research by Gioia and Isquith (2002) examined the Monitor scale, which forms a part of the other principle BRIEF index, the Metacognition Index (MI). In keeping with their hypotheses, the Monitor scale contained items reflecting two distinct dimensions: monitoring of task-related activities and monitoring of personal behavior. Not only did these dimensions demonstrate stability over time, but they also split their allegiance among the BRIEF's primary indices. Self-monitoring loads strongly on the BRI, and task-monitoring on the MI. Thus, follow-up confirmatory factor analytic work was conducted to arrive at a more nuanced conceptualization of EF via BRIEF measurement. Gioia and colleagues concluded that the initial two factor model may be supplemented by considering the better fitting three factor solution, including the Metacognition factor, the Emotional Regulation factor (ER), and the Behavior Regulation factor (BR). Due to the current study's theoretical framework positing that the inhibit and shift subdomains of EF are impacted by anxiety (Eysenck et al., 2007), exploratory analyses were performed to examine the Behavior Regulation factor (containing the Inhibit scale) and the Emotional Regulation factor (containing the Shift

scale) in order to assess if there were differences in mediation strength. No hypotheses were formed a priori. For clarification, Table 2-5 contextualizes Hypothesis 2 analyses and exploratory analyses in relation to other aspects of the study.

Whereas prior models relied on parent-assessed EF, a final model investigated EF's fractionated nature using two factors from the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001). Guided by a factor analytic study (Latzman & Markon, 2010) and subsequent research (Latzman, Elkovitch, Young, & Clark, 2010), D-KEFS data were used to form composites for inhibition and monitoring EF subdomains in order to further explore their potential mediating role in the relationship between anxiety and pediatric QOL (Figure 2-6). Potentially confusing terminology is again noteworthy. In the D-KEFS context, the Inhibition factor continues to denote the ability to deliberately inhibit prepotent or automatic responses. The Monitoring factor, however, "is similar to Miyake's updating dimension, [which] reflects the abilities of actively monitoring and evaluating information" (Latzman et al., p. 456).

Post-hoc Analyses

The lack of analyses on pharmacological interventions is noteworthy in the reviewed QOL literature (e.g., Bastiaansen et al., 2004, 2005; Varni et al., 2001, 2003) and likely stems from the divergent emphases of the cited studies. The absence of attention to medication effects does not imply the topic is unimportant. In fact, medication use could reasonably impact the predictor, mediator, and criterion variables in the current study. Effects may be both direct and indirect, or relevant to the relationships among the variables. Due to the proportion of participants reporting use of prescription medication (60% of total sample), follow-up analyses were conducted to assess effects related to participants' medication usage. With the BRIEF total score as

the mediator variable, scores for 108 individuals on the parent versions of the BASC Anxiety scale and the PedsQL were analyzed. Covariates from the prior best fitting models were included. Results were aggregated across 20 multiply imputed data sets. Finally, medication status was also assessed as a potential moderator of anxiety's effects on EF, and then again as a moderator of EF's effects on QOL (Figure 2-7).

Table 2-1. Steps in data handling and analyses

Step	Description
1	Archive
	a) Select cases from assessment period for PedsQL (dependent variable)
	b) Perform standard preparation of analysis variables for correct format (e.g., dummy coding, convert age from months to years, etc.)
	c) Calculate internal consistency reliabilities
2	Missing values
	a) Transform variable values for univariate normality in order to improve multivariate normality
	b) Perform multiple imputation for missing values, using all analysis variables (including covariates; Amelia package, R software, 20 imputations)
	c) Reverse-transform variable values
3	Sampling
	a) Determine samples by lower age limits for measures per analysis (upper limit: 18 years old)
	• Sample A: all ages (*N* = 108)
	• Sample B: ages 12 and up (*n* =42; lower age limit for BASC child self-report form used)
	• Sample C: ages 8 and up (*n* = 81; lower age limit for D-KEFS)
	b) Mean-center continuous predictor variables within each sample
4	Mediation analyses
	a) Check significance of various combinations of mediators and covariates using SPSS *indirect* macro (Preacher & Hayes, 2008)
	b) Process models for each of 20 multiply imputed data sets
	c) Aggregate each model's results across imputations using *mi.inference* function (NORM package, R software), based on Rubin's (1987) guidelines to aggregate multiply imputed data
5	Effect sizes
	a) Calculate effect size for each individual mediator variable using mediation function (MBESS package, R software), based on Preacher and Kelley's (2011) guidelines for kappa-squared
	b) Process effect size mediation models for each of 20 multiply imputed data sets
	c) Calculate mean *ab* product and effect size across multiply imputed data sets

Table 2-2. Age, sex, and ethnicity for samples

				Age			
	N	% Female	% Minority	*M*	*Mdn*	*SD*	Range
Sample A	108	28.7	29.6	10.8	10.5	3.4	4.3–18.8
Sample B	42	33.3	35.7	14.4	14.3	1.4	12.3–18.8
Sample C	81	29.6	29.6	12.2	12.6	2.6	8.0–18.8

Note. Sample A contained two individuals (ages 4.3 and 4.6 years) that fell below the standard BASC age normative guideline of 5 years old.

Table 2-3. Primary diagnoses for each sample

Diagnosis	Sample A (*n* = 108)	Sample B (*n* = 42)	Sample C (*n* = 81)
Attention-Deficit/Hyperactivity Disorder (ADHD)			
ADHD Predominantly Inattentive Type	15	9	14
ADHD Combined Type	42	14	25
ADHD Not Otherwise Specified (NOS)	3	0	2
Pervasive Developmental Disorders (PDD)			
Asperger's Disorder	5	1	2
Autistic Disorder	1	0	1
PDD NOS	7	1	6
Disruptive Behavior Disorders			
Conduct Disorder	2	1	2
Oppositional Defiant Disorder	3	2	2
Learning Disorders			
Mathematics Disorder	3	3	3
Reading Disorder	2	0	1
Disorder of Written Expression	1	1	1
Learning Disorder NOS	1	1	1
Mood Disorders			
Major Depressive Disorder, Depressive Disorder NOS, Dysthymic Disorder	3	1	3
Mood Disorder NOS	3	2	3
Anxiety Disorders			
Generalized Anxiety Disorder, Anxiety Disorder NOS	3	2	3
Obsessive Compulsive Disorder	2	1	2
Panic Disorder	1	0	1
Language Disorders			
Expressive Language Disorder	1	0	1
Mixed Receptive-Expressive Language Disorder	2	0	1
Other Diagnoses			
Cognitive Disorder NOS	1	0	1
Psychotic Disorder NOS, Schizophrenia	2	1	2
Tic Disorder NOS	1	1	1
Adjustment Disorder	1	1	1
Separation Anxiety Disorder	1	0	1
No Diagnosis	2	0	1

Note. Most participants had multiple diagnoses: 76% in Sample A, 74% in Sample B, 77% in Sample C.

Table 2-4. Primary medications reported in each sample

	Sample A (*n* = 108)	Sample B (*n* = 42)	Sample C (*n* = 81)
No medications	43	10	32
Stimulant	33	15	25
Selective serotonin reuptake inhibitor (SSRIs)	12	8	10
Non-stimulant/norepinephrine reuptake inhibitor (NRIs)	3	1	2
Atypical antipsychotic	6	3	4
Anti-hypertensive	5	1	2
Anti-convulsive	3	1	3
Norepinephrine dopamine reuptake inhibitor (NDRIs)	2	2	2
Noradrenergic and selective serotonergic antidepressant (NaSSAs)	1	1	1

Note. Percentages of participants reporting multiple medications follow: 28% in Sample A, 43% in Sample B, 33% in Sample C.

Table 2-5. Specification of models analyzed

Analysis index	Mediator 1	Mediator 2	Sample
Mediators based on BRIEF			
Parent form of PedsQL and BASC			
1A	Total	—	A
2A.1	BRI	MI	A
2A.2	ER	BR	A
Child form of PedsQL and BASC			
1B	Total	—	B
2B.1	BRI	MI	B
2B.2	ER	BR	B
Mediators based on D-KEFS			
Parent form of PedsQL and BASC			
2C	Inhibit	Monitor	C
Child form of PedsQL and BASC			
2D	Inhibit	Monitor	B

Note. All analyses tested age, sex, minority, and ADHD status covariates. BRI = Behavioral Regulation Index. MI = Metacognition Index. ER = Emotional Regulation factor. BR = Behavioral Regulation factor.

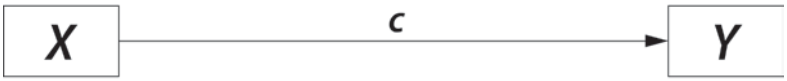

Figure 2-1. Path diagram depicting a model in which the independent variable X has an effect on the dependent variable Y. The path coefficient c represents the total effect of X on Y.

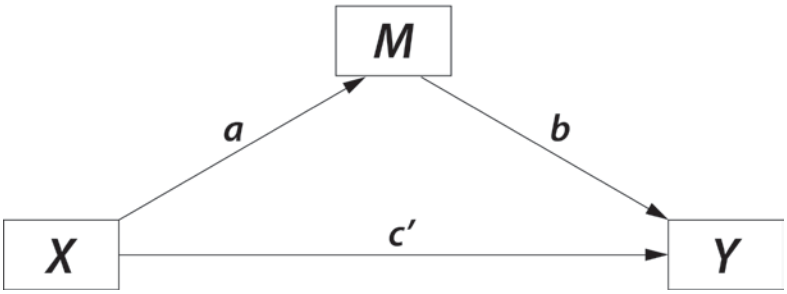

Figure 2-2. Path diagram depicting a model in which the variable M mediates the effect that variable X has on variable Y. The path coefficient a represents the effect of X on M and coefficient b represents the effect of M on Y. The path coefficient c' represents the direct effect of X on Y, whereas the ab product represents the indirect effect of X on Y.

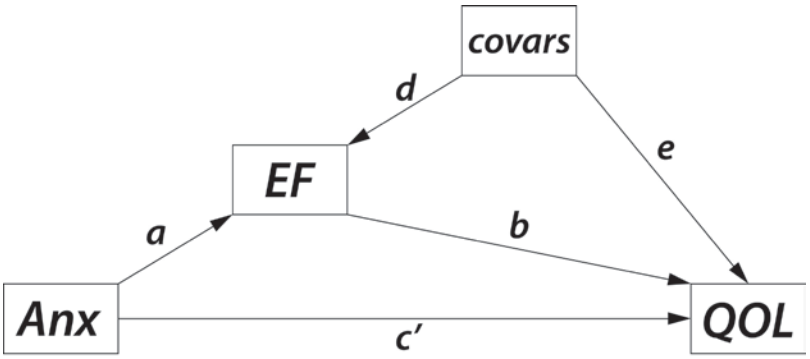

Figure 2-3. Path diagram representing the EF (BRIEF total score) mediation and the effects of covariates on the mediator and outcome.

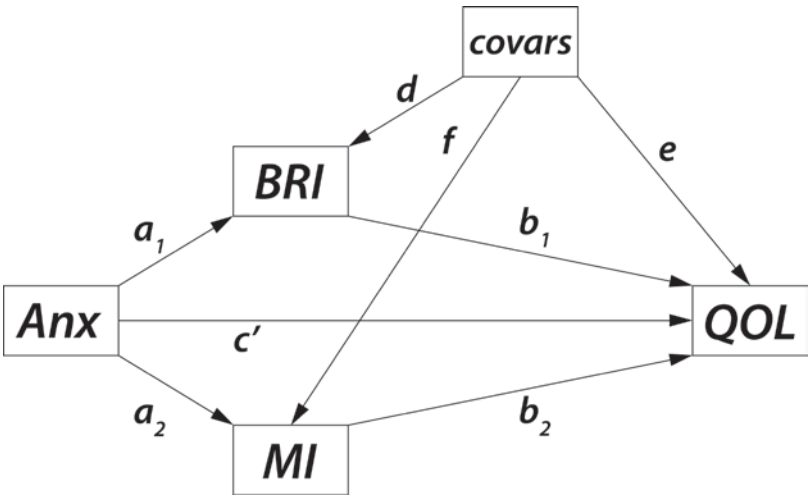

Figure 2-4. Path diagram representing simultaneous mediators and the effects of covariates on mediators and outcome. BRI refers to Behavioral Regulation Index of BRIEF. MI refers to Metacognition Index of BRIEF.

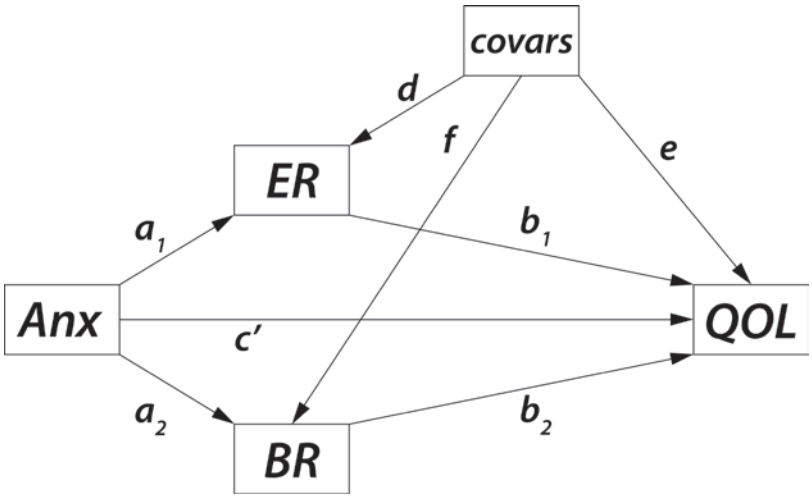

Figure 2-5. Path diagram representing simultaneous mediators and the effects of covariates on mediators and outcome. ER refers to Emotional Regulation factor of BRIEF. BR refers to Behavioral Regulation factor of BRIEF.

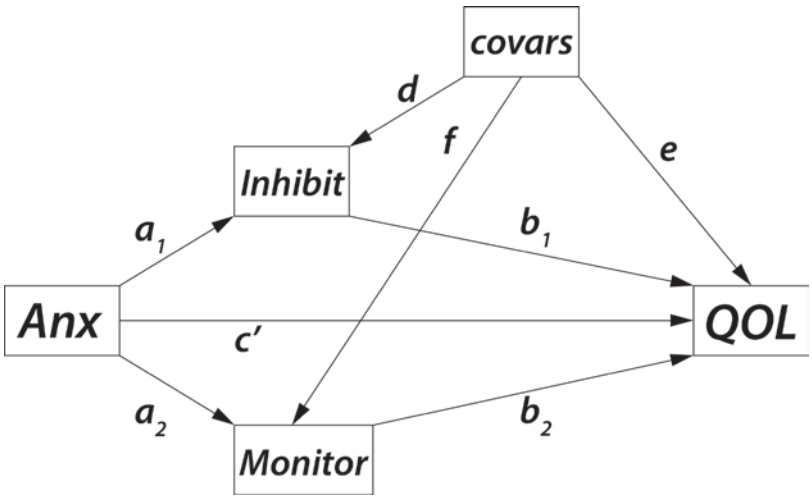

Figure 2-6. Path diagram representing simultaneous mediators and the effects of covariates on mediators and outcome. Inhibit and Monitor refer to D-KEFS factors.

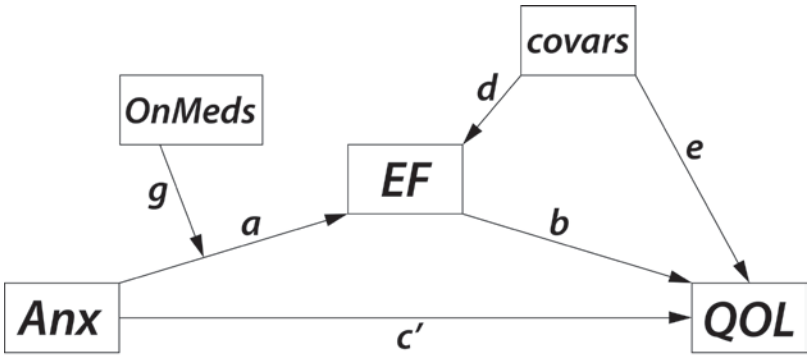

Figure 2-7. Path diagram representing the effects of anxiety on EF, as moderated by medication status. The g path is an estimate of the OnMeds variable in interaction with the anxiety variable. The OnMeds variable is also included among the covariates depicted in the model as contributing to predictions of EF and QOL.

CHAPTER 3
RESULTS

Table 3-1 presents means, standard deviations, ranges, internal consistency alphas, and (initial) percentages of missing data for the measures used in Samples A, B, and C. Internal consistency reliabilities were generally above .70 and as high as .91. The slightly lower alpha of .69 for the Behavior Rating Inventory of Executive Function (BRIEF; Gioia et al., 2000) total score was derived from combining its two primary indices (Behavioral Regulation Index [BRI] and Metacognition Index [MI]), and thus was somewhat expected in light of this study's second hypothesis assessing the multidimensional nature of executive functioning (EF). The low alpha of .56 for the BRIEF Behavioral Regulation factor (BR; Gioia et al., 2002) corresponded to an empirically derived aspect of the BRIEF examined as a post-hoc analysis. Archival constraints did not permit the omission of 4 specific items of the BR factor, which likely compromised the internal consistency. These items load on a third factor that was not of theoretical interest in this analysis. Given the exploratory nature of the post-hoc analysis model using the BR factor, the lower alpha and slightly modified factor place constraints on interpretation of the analyses, yet do not jeopardize the validity of the study's main findings that were unaffected by this issue. Similarly, the rate of missing values—less than ten percent for any variable within any sample—likely posed little threat to the validity of the results, especially given the robust handling of missing values via the multiple imputation process.

Regression assumptions were checked and supported the viability of the analyses. Checks were made for unusual or influential data (e.g., cases who were outliers for size of residuals and whose data had unusual leverage to influence model estimates, as per

Cook's D), normality of residuals, heteroscedasticity, collinearity or multicollinearity, and nonlinearity of relationships between predictors and outcomes (UCLA: Academic Technology Services, 2011). There appeared to be no trends of multicollinearity (tolerance generally above .5 and VIF generally below 2.0) or nonlinear relationships (based on examination of partial regression plots). Slight heteroscedasticity was noted in the models, which indicates that the variance of residuals was not always homogeneous across levels of the predicted values (i.e., commonly less variability for the highest and lowest predicted values). This potentially affected the accuracy of inferences regarding *p* values or statistical significance of model parameters. In a very few models, the distribution of residuals barely met significance for non-normality, which could also affect the accuracy of inferences regarding statistical significance. However, given that heteroscedasticity and non-normally distributed residuals do not affect parameter estimates themselves, the parameters of primary interest—the *ab* products representing indirect effects and the associated effect sizes—remained unaffected. Given that statistical significance of indirect effects was determined by nonparametric bootstrapping, it was not susceptible to heteroscedasticity or non-normally distributed residuals.

Table 3-2 presents the correlations among the various measures and covariates analyzed within each sample. Correlations between measures not used together within an analysis may of course be less relevant. Parent-assessed anxiety (M = 62.5, SD = 14.7) and child self-assessed anxiety (M = 52.31, SD = 10.90) were correlated (r = .45, p = .004) in the current subsample (Sample B, n = 42) where measures were obtained

from both raters.[1] Mean scores on the Behavior Assessment System for Children, Second (BASC; Reynolds & Kamphaus, 2004) Anxiety scale differed significantly between parent-report and child-report, $t(37) = 4.93$, $p < .001$, indicating higher levels of parent-observed children's anxiety, which appears consistent with available literature on internalizing disorders (Ravens-Sieberer et al., 2006).

For EF, the mean score of the BRIEF total composite was elevated ($M = 70.1$, $SD = 10.5$), in keeping with other pediatric mixed clinical samples (Gioia et al., 2000, 2002). Prior research suggests that parental of EF may not correlate with child-performance measures (Vriezen & Pigott, 2002), and a similar pattern was detected in the current study. Apart from the Delis-Kaplan Executive Function System (D-KEFS; Delis et al., 2001) factor's inverse correlation with BRIEF Metacognition Index (MI; $r = -.34$, $p < .05$), no other significant relationships emerged.

Regarding quality of life (QOL), parent and child scores were significantly correlated ($r = .52$, $p < .001$). Parent assessment ($M = 60.55$, $SD = 19.64$) was significantly lower (t (38) $= 4.93$, $p < .001$) than child self-reported QOL ($M = 69.33$, $SD = 15.75$). Mean QOL in the current study was lower than that of other studies that used the Pediatric Quality of Life Inventory Version 4.0 (PedsQL; Varni et al., 2001). For example, Bastiaansen and colleagues' (2004) clinical pediatric sample reported higher parent-assessed QOL ($M = 71.98$, $SD = 13.03$). The correlation between parent and child report resembled that of the current sample ($r = .51$, $p < .01$). Varni and colleagues' (2003) study described scores for healthy ($M = 82.29$, $SD = 15.55$) and chronically ill (i.e., diagnoses of asthma, diabetes, depression, ADHD; $M = 73.14$, $SD =$

[1] Correlations and *t*-tests for the BASC and PedsQL described in this paragraph were performed on the data prior to multiple imputation.

16.46) participants between the ages of 2 and 16 (N = 10,241). The Pearson product-moment correlation for the entire sample was .61 (p < .01). The authors calculated a minimal clinically important difference (MCID) score of 4.50 for the parent-assessed PedsQL score. An MCID refers to the "smallest difference in a score of a domain of interest that patients perceive to be beneficial and that would mandate, in the absence of troublesome side effect and excessive costs, a change in the patients' management" (Varni et al., p. 332). Notably, the current sample's mean parent-assessed QOL falls more than two MCIDs below Varni and colleagues' sample mean.

Hypothesis 1

This study's primary aim was to examine whether EF mediated the deleterious effect of anxiety on QOL in a pediatric clinical sample. It was hypothesized that the total score for EF (measured with the BRIEF) would demonstrate statistical significance as a mediator of the effect of anxiety (measured with the Anxiety scale from the BASC on the total score of the PedsQL). This hypothesis was tested with (a) parent forms for the PedsQL and the BASC Anxiety scale and (b) child self-report forms of the same measures.

Hypothesis 1A: Parent Forms for BASC Anxiety and PedsQL, BRIEF as Mediator

The first iteration of Hypothesis 1 examined scores for 108 individuals on the parent versions of the BASC Anxiety scale and the PedsQL, with the BRIEF total score as the mediator variable. Table 3-3 provides parameter estimates for the model, including age, female, minority, and ADHD covariates (i.e., female, minority, ADHD diagnosis = 1; male, White, non-ADHD diagnosis = 0). The first row of estimates in the table shows QOL regressed on anxiety (parameter c from Figure 2-1), before accounting for EF as a mediator, but controlling for the covariates (p = .002).

The second portion of the table provides estimates for a model including the BRIEF total score as a mediator of the effect of anxiety on QOL. The first estimate is for the *a* path, or the BRIEF total score regressed on the BASC anxiety score. The estimate indicates that a one-point increase on the anxiety scale corresponded to a .204 increase on the BRIEF, indicating increased executive dysfunction ($p = .011$). The estimate for the *b* path corresponds to PedsQL scores regressed on the BRIEF. The estimate indicates that a one-point increase on the BRIEF (i.e., increased executive dysfunction) corresponded to a 1.131 decrease on the QOL measure ($p < .001$). The *c'* parameter indicates the direct effect of anxiety on QOL after removing the indirect effect via the mediator variable. The *c'* estimate suggests a similar negative relationship between anxiety and QOL—a one-point increase in the former corresponded to a .168 decrease in the latter—but this relationship was not statistically significant ($p = .093$). However, the main estimate of interest in Table 3-3 is the indirect effect of anxiety through the BRIEF total mediator: the *ab* product. This estimate indicates that the indirect effect of a one-point increase on the BASC anxiety measure through the EF mediator corresponded to a .229 point decrease in QOL (95% confidence interval [CI], -.417 to -.070).

Statistical significance does not address effect size. Preacher and Kelley (2011) developed the mediation function (MBESS package, R software) to calculate a number of effect size measures for indirect effects, ultimately recommending kappa-squared (κ^2) as the preferred statistic. Its values range from zero to one, indicating the ratio of the obtained indirect effect estimate in relation to how large it could have possibly been, given the data and model specification. However, Preacher and Kelley's mediation

function only estimates simple mediation models (i.e., a single mediator and no covariates).[2]

Due to these software constraints, best practice was followed and effect sizes were calculated on the simple mediation model with only the BRIEF total score as mediator, without inclusion of covariates. The indirect effect estimate was significant as shown in the lower portion of Table 3-3 (95% CI, -.329 to -.043). Preacher and Kelley (2011) recommended that κ^2 values can be evaluated with Cohen's (1988) guidelines of small (.01), medium (.09), and large (.25). This effect was medium to large (κ^2 = .150; 95% CI, .036 to .264).

Covariates in the model are listed in Table 3-3 as parameters d_1 to d_4 (in relation to the mediator) and e_1 to e_4 (in relation to the dependent variable). Refer to Figure 2-3. The only significant covariates were minority and ADHD in relation to the BRIEF, and ADHD in relation to QOL. Individuals of minority (non-White) status had BRIEF scores that were 4.5 points lower on average (i.e., less executive dysfunction), whereas individuals with a primary diagnosis of ADHD had BRIEF scores that were 4.388 points higher on average (i.e., more executive dysfunction). Individuals with an ADHD diagnosis had QOL scores that were 10.303 points higher, on average, than individuals with other diagnoses (e.g., internalizing disorders, learning disorders, Pervasive Developmental Disorders) in the mixed clinical sample.

Given that the model reported in Table 3-3 included a mix of parameters—some significant and others not—including covariates with no significant effects at all, a pared

[2] Preacher confirmed that there is no existing prepared software function or macro to calculate κ^2 for more complex models, and that to create one would be a formidable challenge requiring matrix algebra and mathematical software programming far exceeding that used in the *indirect* macro or the MBESS mediation function (K. Preacher, personal communication, October 23, 2011).

down model was estimated. Table 3-4 presents estimates for a model omitting the age and female covariates, retaining the minority and ADHD covariates. The general trend of the estimates and their interpretation remains the same, and the adjusted R^2 for the pared down model demonstrates superior model fit with the nonsignificant parameters removed.

Hypothesis 1B: Child Forms for BASC Anxiety and PedsQL, BRIEF as Mediator

The second iteration of Hypothesis 1 differed from the first only in that it examined child self-report forms of the BASC Anxiety scale and the PedsQL (Table 3-5). The archive included fewer individuals in the proper age range (12 years and older) for the self-report forms (n = 42; Sample B). The indirect effect for this model is nonsignificant, along with most other parameters in the model. The effect size estimate is low (κ^2 = .053; 95% CI, .003 to .191) but the CI allows for the possibility that the effect may be medium.

Hypothesis 2

As a follow-up to the first hypothesis' examination of whether general EF appears to mediate the relationship between anxiety and QOL, Hypothesis 2 examined specific EF subcomponents for their relative strength in the mediation role. It was expected that in accordance with Attentional Control Theory (ACT; Eysenck et al., 2007), the Behavioral Regulation Index (BRI) of the BRIEF would demonstrate a stronger mediation effect than the Metacognition Index (MI) of the BRIEF, if the latter were to demonstrate any mediation effect at all. The BRI primarily assesses inhibition and shifting, whereas the MI evaluates updating, task initiation, and monitoring of performance. Collectively, these two indices comprise the BRIEF total score.

A first set of Hypothesis 2 analyses used the BRI and MI indices as simultaneous mediators of the effect of anxiety on QOL. The first iteration examined the parent forms of the BASC Anxiety scale and PedsQL, and the second iteration assessed the child self-report forms of each. The significant differences found between these mediators supported the hypothesis that particular subdomains of EF would demonstrate different mediation strength and spurred additional exploratory analyses comparing empirically derived and theoretically relevant alternative BRIEF *factors* (i.e., Behavior Regulation factor and Emotional Regulation factor) as simultaneous mediators (Gioia et al., 2002). Finally, a set of analyses compared the mediation strength of two EF subdomains as assessed by the D-KEFS child performance measure. Subdomain composition was informed by factor analysis (Latzman & Markon, 2010) and subsequent research (Latzman et al., 2010).

Hypothesis 2A.1: Parent Forms for BASC Anxiety and PedsQL, BRI and MI as Mediators

The first iteration for Hypothesis 2 examined scores for 108 individuals on the parent versions of the BASC Anxiety scale and the PedsQL, with the BRIEF BRI and MI as simultaneous mediator variables. Table 3-6 presents the estimates for this simultaneous mediator model, including all covariates. The BRI indirect effect ($a_1 \times b_1$) was statistically significant (95% CI, -.388 to -.095) and the MI indirect effect ($a_2 \times b_2$) was not (95% CI, -.159 to .008).

Before interpreting these results, the nonsignificant covariates of age and sex were eliminated from the model (Table 3-7) and the adjusted R^2 demonstrated better model fit. The model retained the minority and ADHD covariates due to significant relationships with EF and QOL, respectively. After shedding the nonsignificant

parameters, the MI indirect effect demonstrated statistically significance (95% CI, -.171 to -.014). The BRI indirect effect estimate had a greater absolute value than that of the MI, and significance testing for the contrast of indirect effects indicated that they differed significantly (95% CI, -.295 to -.026). Tested in single mediator models as per software limitations, the BRI effect size appeared medium-large (κ^2 = .193; 95% CI, .073 to .310) and the MI effect size (κ^2 = .072; 95% CI, .007 to .165) appeared small-medium. Compared to the effect size of .150 for the BRIEF total examined in Hypothesis 1A, these results may suggest that the BRI dimension of EF (i.e., the inhibit and shift subdomains) functions as a purer mediator of anxiety's negative effect on QOL.

Estimates for the covariates in Table 3-7 indicate two statistically significant relationships. Again, covariates were dummy coded (i.e., female, minority, ADHD diagnosis = 1; male, White, non-ADHD diagnosis = 0). Minority was associated with lower BRIEF MI scores by 5.165 points, on average, suggesting less executive dysfunction in this domain (p = .01). The presence of an ADHD diagnosis was associated with higher PedsQL scores (p < .001), indicating that those with the externalizing disorder reported a 9.88 higher QOL score, on average, indicating better QOL than those with internalizing (e.g., anxiety and mood disorders) and other disorders in this mixed clinical sample.

Hypothesis 2B.1: Child Forms for BASC Anxiety and PedsQL, BRI and MI as Mediators

The above analysis comparing simultaneous mediators of the BRIEF BRI and MI was repeated in a second iteration using child self-report forms of the BASC Anxiety scale and the PedsQL (Table 3-8). The archive included fewer individuals in the proper age range (12 years and older) for these forms (n = 42; Sample B). Again, the smaller

sample size carried with it diminished power to detect effects. The indirect effect and covariates were not statistically significant. The effect size of .015 for the BRI is rather small and indistinguishable from zero. The effect size for the MI (κ^2 = .079; 95% CI, .005 to .265) is closer to medium and its wide CI ranges from small to large.

Analysis 2A.2: Parent Forms for BASC Anxiety and PedsQL, ER and BR as Mediators

The significant differences found between the BRIEF BRI and MI as simultaneous mediators in Hypothesis 2A.1 supported the idea that specific dimensions of EF would demonstrate different strengths of mediation and spurred this additional exploratory analysis driven by a prior factor analytic study. Gioia and colleagues (2002) outlined a three-factor model of the BRIEF subscales. Two factors were of particular interest for the present study because resultant analyses could shed light on potential differences in mediation strengths between the inhibit and shift EF subdomains. The Emotional Regulation (ER) factor was comprised of the Shift and Emotional Control subscales. The Behavioral Regulation (BR) factor was comprised of the Monitor and Inhibit subscales. (Note, as explained earlier, the Behavioral Regulation *factor* is linguistically similar though compositionally different than the Behavioral Regulation *Index*.) Archival limitations did not allow for the selection of specific items of the Monitor scale, thereby resulting in a BR factor that contained 4 additional task-monitoring items. These 4 items load on the Metacognition factor, which was not of theoretical interest in this exploratory analysis. Exploratory Analysis 2A.2 used the parent forms of the BASC Anxiety scale and the PedsQL, with the BRIEF ER and BR factors as simultaneous mediators (Figure 2-5). These analyses examined data for the 108 individuals of Sample A.

Table 3-9 presents the estimates for this simultaneous mediator model, including all covariates. The ER indirect effect ($a_1 \times b_1$) was statistically significant (95% CI, -.510 to -.177), whereas the BR indirect effect (a2 × b2) was not. Nonsignificant parameters were eliminated to test a more parsimonious model (Table 3-10). Model fit was not compromised (i.e., adjusted R^2 of .539 decreased to .532). The model retained the ADHD covariate, which again demonstrated a positive association with QOL (p = .008). The effect size for the ER indirect effect in a simple mediator model appeared large (κ^2 = .273; 95% CI, .160 to.383), larger than both the effect sizes for BRIEF total in Hypothesis 1A and BRI in Hypothesis 2A.1, which suggests that the shifting subdomain of EF functioned as the most potent mediator of anxiety's effect on QOL observed in this study, cross-sectional design implications notwithstanding.

Analysis 2B.2: Child Forms for BASC Anxiety and PedsQL, ER and BR as Mediators

The above analysis comparing simultaneous mediators of the BRIEF ER and BR was repeated in a second iteration using child self-report forms of the BASC Anxiety scale and the PedsQL (Table 3-11). The archive included fewer individuals in the proper age range (12 years and older) for these forms (n = 42; Sample B). Again, the smaller sample size compromises statistical power and influences interpretation of findings. Although the mediation indirect effect was not statistically significant, the effect size of .091 for ER was medium to possibly large (95% CI, .009 to .261).

Hypothesis 2C: Parent Forms for BASC Anxiety and PedsQL, D-KEFS Inhibit and Monitor as Mediators

A final comparison of simultaneous child-performance mediators using the D-KEFS was influenced by prior factor analytic research (Latzman & Markon, 2010) and subsequent research employing the factors (Latzman et al., 2010). Composites for

Inhibit and Monitoring EF subdomains were based on Latzman and colleagues' design (Figure 2-6). As referenced earlier, although the Inhibition factor retains its usual meaning of the capacity to inhibit dominant responses, the Monitoring factor resembles Miyake and colleagues' (2000) updating subdomain, which refers to the capacity to evaluate and monitor information. The Inhibit factor is the mean of two other composites: the mean of the five Trail-Making conditions and the mean of the Color-Word Inhibit and Inhibit-Switch conditions. The Monitor factor is the mean of the three Verbal-Fluency conditions. Archival limitations did not allow for strict adherence to Latzman and colleagues' suggested incorporation of the Design Fluency mean.[3] Hypothesis C tested a mediation model with these simultaneous mediators using the parent forms of the PedsQL and BASC anxiety scale. Given that the D-KEFS is appropriate for ages 8 and above, the resulting Sample C included 81 individuals.

Table 3-12 presents the results of this model, demonstrating nonsignificance for essentially all parameters. Effect sizes for the D-KEFS Inhibit and Monitor indirect effects are rather small at .002 and .010 respectively, which seems to suggest that the lack of significant findings was not simply due to low statistical power. The only significant covariates were minority (i.e., higher Inhibit scores) and age (i.e., older individuals having higher Monitor scores).

Hypothesis 2D: Child Forms for BASC Anxiety and PedsQL, D-KEFS Inhibit and Monitor as Mediators

The above analysis comparing simultaneous mediators of the D-KEFS Inhibit and Monitor factors was repeated in a second iteration using child self-report forms of the

[3] Attempted correspondence with the D-KEFS lead author regarding this measurement concern was unsuccessful.

BASC Anxiety scale and the PedsQL (Table 3-13). The archive included fewer individuals in the proper age range (12 years and older) with the self-report forms (*n* = 42; Sample B). Size constraints and power concerns remained and may represent plausible explanations as to why the indirect effects for this model were not statistically significant. However, because the Monitor indirect effect had a κ^2 of .049, significant results may have been obtained in a larger sample.

Post-hoc Analyses: Possible Effects Related to Medication Status

The absence of analyses on pharmacological interventions is somewhat surprising in the reviewed QOL literature (e.g., Varni et al., 2001, 2003), especially when considering how medications may influence the effects of this study's variables in clinical samples (e.g., Bastiaansen et al., 2004, 2005). Although few studies have examined QOL in relation to anxiety (Sherbourne et al., 1996), Mogotsi, Kaminer, and Stein (2000) reported that medications have been associated with enhanced QOL in anxiety disorder patients. They added that further empirical evidence is needed, especially in relation to the links between treatment and QOL change. Though neither a treatment nor an outcome study, the current research may partly illuminate the anxiety/medication relationship, at least insofar as it pertains to a mixed clinical sample.

Child and adolescent participants' medication status was gathered during the clinical interview as part of the assessment. Because 60% of the total sample reported current use of prescription medication, follow-up analyses were conducted to assess effects related to participants' medication usage. No hypotheses were formed a priori. Due to limited numbers of each particular medication type (Table 2-4), pediatric participants formed two medication-status groups: (a) a medication-naïve group, including participants not taking prescription medications for psychiatric conditions, and

(b) a medication group, composed of children currently prescribed stimulants, selective serotonin reuptake inhibitors, non-stimulant/norepinephrine reuptake inhibitors, norepinephrine dopamine reuptake inhibitor, noradrenergic and selective serotonergic antidepressant, anti-convulsives, anti-hypertensives, and atypical antipsychotics. Worthy of note is the vast range of medication-types contained within the medicated subgroup. Accordingly, interpretation of the following post-hoc analyses may be constrained by the dichotomizing of “medicated” versus “non-medicated” participants as well as the wide variety of psychiatric diagnoses for which the medications were prescribed (Table 2-3).

Hypothesis 1A was revisited adding medication status as a covariate. With the BRIEF total score as the mediator variable, scores for 108 individuals on the parent versions of the BASC Anxiety scale and the PedsQL were analyzed. Covariates from the prior best fitting model were included (i.e., minority, ADHD diagnosis, on medication = 1; White, non-ADHD diagnosis, medication-naïve = 0). Results were aggregated across 20 multiply imputed data sets. All trends in the results remained identical. Most notably, the mediation remained significant, suggesting that EF as a unitary construct mediated the anxiety and QOL relationship even in the presence of controls for the effects of medications on EF and QOL. Similarly, trends were also identical for Hypothesis 2A.1 and Exploratory Analysis 2A.2 when the medication status covariate was added to the models. These findings suggest that the particular EF subdomains demonstrated similar mediation properties despite controls for effects of medication status on EF and QOL.

Whereas the post-hoc analyses above covaried medication status in models to examine and control for its effect on EF and QOL, medication status was also examined as a potential moderator of anxiety's effects on EF, and then again as a moderator of EF's effects on QOL. A series of models tested the significance of the dummy-coded medication status variable (i.e., on medication = 1, medication-naïve = 0) in interaction with anxiety as a predictor of EF, and in interaction with the various EF measures (e.g., BRIEF total, BRI, MI, ER, and BR) as predictors of QOL. Anxiety and EF were mean-center to minimize multicollinearity problems and facilitate interpretation. Interaction terms were nonsignificant and thus were not retained in any models.

However, two potential interactions may be of note (Figure 3-1). Estimates for the negative effect of anxiety on EF were larger for individuals taking prescription medications, yet the differences were not statistically significant. Specifically, anxiety's association with executive dysfunction measured by the BRIEF total score (as in Hypothesis 1A) appeared to occur almost exclusively among the group of individuals taking medications. Their anxiety parameter estimate was .260 higher (p = .089) than the medication-naïve group estimate of .035 (p = .784). Similarly, the medication group estimate for anxiety in relation to the BRIEF MI index (as in Hypothesis 2A.1) was .260 higher (p = .064), than the medication-naïve group estimate of .141 (p = .390). Whereas the p values did not meet the pre-established criterion for significance, some may be close enough to warrant attention in future studies of how medication usage may moderate the effect of anxiety on EF. Extant studies on clinical levels of pediatric anxiety and EF tend to not examine medication status of participants (e.g., Emerson et al., 2005; Kendall & Chansky, 1991; Micco et al., 2009; Toren et al., 2000), perhaps in

part due to the nascence of this line of investigation and the consequent prioritization of other research questions within mixed clinical samples. Nevertheless, questions pertinent to medication use and its associated effects merit consideration. As noted, cautious interpretation of the current findings is warranted given the heterogeneity of the medications assessed in addition to the fact that they were prescribed for a range of psychiatric disorders rather than anxiety disorders or anxious symptomatology per se.

Results Summary

Table 3-14 provides a conceptual structure for how the various iterations of models and analyses fit together and build upon one another. In sum, Hypothesis 1 tested EF as a unitary construct, whereas hypothesis 2 used three pairs of EF variables as simultaneous mediators: the first two pairs from the parent-assessed BRIEF, and the third pair from the child-performed D-KEFS. For the independent and dependent variables, all analyses were performed using (a) the parent forms and (b) the child self-report forms of the BASC Anxiety scale and PedsQL. There were two main findings. First, results from tests of Hypothesis 1 provided supporting evidence that EF may in fact function as a mediator of the relationship between anxiety and QOL. Secondly, results from tests of Hypothesis 2 supported EF's multidimensionality and the differing strength of specific subcomponents as mediators of the relationship between anxiety and QOL. Table 3-14 also indicates which mediators demonstrated significant indirect effects and provides their effect sizes.

Table 3-1. Means, SDs, ranges, alphas, and percent missing for measures

Variables	*M*	*SD*	Range	Alpha	% missing
Sample A (*n* = 108)					
PedsQL Parent	63.9	17.7	13–99	.91	1.9
BASC-Anx Parent	56.8	13.7	28–90	.84–.90	.9
BRIEF Total	70.1	10.5	40–96	.71	6.5
BRIEF BRI	66.9	13.3	35–96	.82	6.5
BRIEF MI	69.7	9.8	41–95	.88	6.5
BRIEF ER	64.0	12.8	37–93	.83	6.5
BRIEF BR	66.6	10.2	39–91	.70	6.5
Sample B (*n* = 42)					
PedsQL Child	71.3	15.6	12–99	.90	2.4
BASC-Anx Child	50.9	11.2	33–82	.84	9.5
BRIEF Total	72.0	10.3	40–88	.69	.0
BRIEF BRI	68.5	14.0	41–96	.80	.0
BRIEF MI	71.2	8.9	41–85	.87	.0
BRIEF ER	65.5	13.1	42–93	.85	.0
BRIEF BR	67.7	10.1	43–91	.56	.0
D-KEFS Inhibit	8.6	2.4	1.8–12.5	.87	.0
D-KEFS Monitor	9.4	3.3	3.0–18.3	.79	2.4
Sample C (*n* = 81)					
PedsQL Parent	62.2	17.5	13–99	.91	2.5
BASC-Anx Parent	58.9	13.3	35–90	.84–.90	.0
D-KEFS Inhibit	8.3	2.6	1.8–13.5	.87	6.2
D-KEFS Monitor	9.1	2.9	3.0–18.3	.75	7.4

Note. Range of BASC alphas corresponds to age-appropriate forms.

Table 3-2. Correlations for measures and covariates

	Variables	1	2	3	4	5	6	7	8	9	10	11	12
Sample A (*n* = 108)													
1	PedsQL Par	—											
2	BASC-Anx Parent	-.35**	—										
3	BRIEF Total	-.66**	.22*	—									
4	BRIEF BRI	-.66**	.29**	.86**	—								
5	BRIEF MI	-.53**	.13	.91**	.57**	—							
6	BRIEF ER	-.71**	.37**	.77**	.93**	.49**	—						
7	BRIEF BR	-.49**	.11	.87**	.81**	.75**	.61**	—					
8	Age	-.07	.28**	.06	.02	.07	.02	.01	—				
9	Female	-.12	.21*	.09	.04	.16	.05	.12	.03	—			
10	Minority	.09	.05	-.18	-.08	-.24*	-.08	-.10	.14	.04	—		
11	ADHD	.24*	-.28**	.13	.06	.14	-.05	.20*	-.09	-.13	-.03	—	
Sample B (*n* = 42)													
1	PedsQL Child	—											
2	BASC-Anx Child	-.57**	—										
3	BRIEF total	-.26	-.09	—									
4	BRIEF BRI	-.27	.05	.87**	—								
5	BRIEF MI	-.21	-.15	.90**	.58**	—							
6	BRIEF ER	-.46**	.21	.79**	.91**	.52**	—						
7	BRIEF BR	-.07	-.15	.84**	.84**	.68**	.60**	—					
8	D-KEFS Inhibit	-.08	.09	-.27	-.11	-.34*	-.11	-.18	—				
9	D-KEFS Monitor	-.04	.27	-.25	-.14	-.29	-.20	-.17	.60**	—			
10	Age	.12	.12	-.23	-.18	-.22	-.24	-.04	.19	.23	—		
11	Female	-.28	.31	.05	.08	.11	.12	.06	.21	.09	-.20	—	
12	Minority	.03	.17	-.22	-.12	-.25	-.10	-.11	.14	.19	.34*	-.11	—
13	ADHD	.22	-.25	.17	.04	.22	-.08	.16	-.17	-.06	-.01	-.27	-.02

Note. * $p < .05$. ** $p < .01$

Table 3-2 (cont.). Correlations for measures and covariates

	Variables	1	2	3	4	5	6	7	8	9	10	11	12
Sample C (*n* = 81)													
1	PedsQL Parent	—											
2	BASC-Anx Parent	-.27*	—										
3	D-KEFS Inhibit	.03	.05	—									
4	D-KEFS Monitor	.04	.12	.62**	—								
5	Age	.06	.15	.14	.18	—							
6	Female	-.16	.27*	.23*	.14	.01	—						
7	Minority	.05	.08	.06	.16	.23*	.05	—					
8	ADHD	.26*	-.19	-.05	-.04	.08	-.17	-.06	—				

Note. * $p < .05$. ** $p < .01$

Table 3-3. Estimates for hypothesis 1A mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Par → PedsQL Par	*c*	-.398	.129	.002	-.652	-.145
Model with mediator						
Predictors for mediator (BRIEF total)						
BASC-Anx Par	*a*	.204	.080	.011	.047	.361
Age	d_1	.112	.333	.737	-.542	.765
Female	d_2	1.487	2.277	.514	-2.978	5.951
Minority	d_3	-4.500	2.166	.038	-8.747	-.254
ADHD	d_4	4.388	2.056	.033	.358	8.418
Predictors for outcome (PedsQL Par)						
BRIEF total	*b*	-1.131	.122	< .001	-1.370	-.893
BASC-Anx Par	*c'*	-.168	.100	.093	-.363	.028
Age	e_1	.191	.396	.630	-.587	.968
Female	e_2	.143	2.749	.958	-5.246	5.533
Minority	e_3	-.830	2.698	.759	-6.119	4.460
ADHD	e_4	10.303	2.582	< .001	5.242	15.364
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.527	—	< .001	—	—
Indirect effect (BRIEF total)	$a \times b$	-.229	—	—	-.417	-.070
Model with mediator and no covariates						
Indirect effect (BRIEF total)	$a \times b$	-.175	—	—	-.329	-.043
Effect size for indirect effect	κ^2	.150	—	—	.036	.264

Table 3-4. Estimates for hypothesis 1A mediator model (selected covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Par → PedsQL Par	*c*	-.404	.122	.001	-.643	-.165
Model with mediator						
Predictors for mediator (BRIEF total)						
BASC-Anx Par	*a*	.221	.074	.003	.075	.366
Minority	d_1	-4.362	2.141	.042	-8.559	-.166
ADHD	d_2	4.270	2.037	.036	.278	8.263
Predictors for outcome (PedsQL Par)						
BRIEF total	*b*	-1.126	.121	< .001	-1.363	-.889
BASC-Anx Par	*c'*	-.155	.093	.096	-.339	.028
Minority	e_1	-.631	2.664	.813	-5.854	4.591
ADHD	e_2	10.253	2.548	< .001	5.258	15.249
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.534	—	< .001	—	—
Indirect effect (BRIEF total)	*a* × *b*	-.248	—	—	-.421	-.105
Model with mediator and no covariates						
Indirect effect (BRIEF total)	*a* × *b*	-.175	—	—	-.329	-.043
Effect size for indirect effect	κ^2	.150	—	—	.036	.264

Table 3-5. Estimates for hypothesis 1B mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Child → PedsQL Child	*c*	-.800	.218	< .001	-1.228	-.372
Model with mediator						
Predictors for mediator (BRIEF total)						
BASC-Anx Child	*a*	-.016	.171	.927	-.352	.320
Age	d_1	-1.277	1.291	.323	-3.807	1.254
Female	d_2	1.289	3.827	.736	-6.212	8.790
Minority	d_3	-3.234	3.618	.371	-10.325	3.857
ADHD	d_4	3.630	3.412	.287	-3.057	10.317
Predictors for outcome (PedsQL Child)						
BRIEF total	*b*	-.453	.206	.028	-.856	-.050
BASC-Anx Child	*c'*	-.809	.206	< .001	-1.213	-.405
Age	e_1	1.172	1.576	.457	-1.918	4.261
Female	e_2	-.754	4.636	.871	-9.841	8.334
Minority	e_3	1.118	4.445	.801	-7.595	9.831
ADHD	e_4	3.835	4.216	.363	-4.430	12.099
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.365	—	.002	—	—
Indirect effect (BRIEF total)	$a \times b$	.007	—	—	-.145	.192
Model with mediator and no covariates						
Indirect effect (BRIEF total)	$a \times b$	.039	—	—	-.087	.201
Effect size for indirect effect	κ^2	.053	—	—	.003	.191

Table 3-6. Estimates for hypothesis 2A.1 mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Par → PedsQL Par	c	-.398	.129	.002	-.652	-.145
Model with mediator						
Predictors for mediator 1 (BRIEF BRI)						
BASC-Anx Par	a_1	.338	.102	.001	.139	.537
Age	d_1	-.198	.428	.643	-1.038	.642
Female	d_2	-.338	2.872	.906	-5.968	5.292
Minority	d_3	-2.392	2.750	.384	-7.782	2.998
ADHD	d_4	3.980	2.620	.129	-1.156	9.116
Predictors for mediator 2 (BRIEF MI)						
BASC-Anx Par	a_2	.104	.074	.160	-.041	.249
Age	f_1	.233	.306	.447	-.367	.833
Female	f_2	3.424	2.097	.103	-.687	7.536
Minority	f_3	-5.463	2.000	.006	-9.382	-1.543
ADHD	f_4	3.848	1.901	.043	.122	7.574
Predictors for outcome (PedsQL Par)						
BRIEF BRI	b_1	-.671	.113	< .001	-.893	-.449
BRIEF MI	b_2	-.486	.158	.002	-.795	-.177
BASC-Anx Par	c'	-.121	.100	.227	-.317	.075
Age	e_1	.043	.395	.913	-.732	.818
Female	e_2	-.104	2.731	.970	-5.458	5.250
Minority	e_3	.005	2.687	.998	-5.261	5.271
ADHD	e_4	9.887	2.532	< .001	4.924	14.850
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.547	—	< .001	—	—
Indirect effect 1 (BRIEF BRI)	$a_1 \times b_1$	-.225	—	—	-.388	-.095
Indirect effect 2 (BRIEF MI)	$a_2 \times b_2$	-.050	—	—	-.159	.008
Contrast of indirect effects 1 and 2		-.175	—	—	-.338	-.045
Models with mediators and no covariates						
Indirect effect 1 (BRIEF BRI)	$a_1 \times b_1$	-.227	—	—	-.389	-.086
Effect size for indirect effect 1	κ^2_1	.193	—	—	.073	.310
Indirect effect 2 (BRIEF MI)	$a_2 \times b_2$	-.086	—	—	-.213	.014
Effect size for indirect effect 2	κ^2_2	.072	—	—	.007	.165

Table 3-7. Estimates for hypothesis 2A.1 mediator model (selected covariates)

Model	Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)							
	BASC-Anx Par → PedsQL Par	c	-.404	.122	.001	-.643	-.165
Model with mediator							
	Predictors for mediator 1 (BRIEF BRI)						
		BASC-Anx Par	a_1	.323	.094	.001	.138	.508
		Minority	d_1	-2.584	2.705	.339	-7.886	2.718
		ADHD	d_2	4.020	2.595	.121	-1.066	9.106
	Predictors for mediator 2 (BRIEF MI)						
		BASC-Anx Par	a_2	.141	.070	.043	.004	.278
		Minority	f_1	-5.165	2.005	.010	-9.095	-1.235
		ADHD	f_2	3.582	1.909	.061	-.160	7.323
	Predictors for outcome (PedsQL Par)						
		BRIEF BRI	b_1	-.671	.111	< .001	-.888	-.453
		BRIEF MI	b_2	-.484	.153	.002	-.784	-.184
		BASC-Anx Par	c'	-.119	.092	.198	-.300	.062
		Minority	e_1	.051	2.635	.985	-5.114	5.215
		ADHD	e_2	9.880	2.495	< .001	4.989	14.770
		Adjusted R^2 for DV model	$R^2_{Y,MX}$	.554	—	< .001	—	—
	Indirect effect 1 (BRIEF BRI)		$a_1 \times b_1$	-.216	—	—	-.364	-.098
	Indirect effect 2 (BRIEF MI)		$a_2 \times b_2$	-.068	—	—	-.171	-.014
	Contrast of indirect effects 1 and 2			-.149	—	—	-.295	-.026
Models with mediators and no covariates							
	Indirect effect 1 (BRIEF BRI)		$a_1 \times b_1$	-.227	—	—	-.389	-.086
	Effect size for indirect effect 1		κ^2_1	.193	—	—	.073	.310
	Indirect effect 2 (BRIEF MI)		$a_2 \times b_2$	-.086	—	—	-.213	.014
	Effect size for indirect effect 2		κ^2_2	.072	—	—	.007	.165

Table 3-8. Estimates for hypothesis 2B.1 mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Child → PedsQL Child	c	-.800	.218	< .001	-1.228	-.372
Model with mediator						
Predictors for mediator 1 (BRIEF BRI)						
BASC-Anx Child	a_1	.108	.232	.641	-.346	.562
Age	d_1	-1.606	1.807	.374	-5.148	1.936
Female	d_2	.943	5.334	.860	-9.511	11.398
Minority	d_3	-2.307	5.062	.649	-12.229	7.615
ADHD	d_4	1.838	4.775	.700	-7.520	11.196
Predictors for mediator 2 (BRIEF MI)						
BASC-Anx Child	a_2	-.077	.146	.599	-.363	.210
Age	f_1	-.788	1.085	.468	-2.915	1.339
Female	f_2	3.193	3.228	.323	-3.134	9.521
Minority	f_3	-3.232	3.040	.288	-9.190	2.727
ADHD	f_4	4.260	2.867	.137	-1.360	9.879
Predictors for outcome (PedsQL Child)						
BRIEF BRI	b_1	-.081	.185	.659	-.443	.281
BRIEF MI	b_2	-.446	.309	.149	-1.052	.160
BASC-Anx Child	c'	-.829	.214	< .001	-1.249	-.409
Age	e_1	1.268	1.596	.427	-1.861	4.397
Female	e_2	.181	4.816	.970	-9.260	9.622
Minority	e_3	.958	4.545	.833	-7.951	9.868
ADHD	e_4	4.237	4.385	.334	-4.359	12.834
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.347	—	.005	—	—
Indirect effect 1 (BRIEF BRI)	$a_1 \times b_1$	.007	—	—	-.293	.060
Indirect effect 2 (BRIEF MI)	$a_2 \times b_2$	.045	—	—	-.096	.290
Contrast of indirect effects 1 and 2		-.038	—	—	-.333	.134
Models with mediators and no covariates						
Indirect effect 1 (BRIEF BRI)	$a_1 \times b_1$	-.016	—	—	-.161	.085
Effect size for indirect effect 1	κ^2_1	.015	—	—	.000	.059
Indirect effect 2 (BRIEF MI)	$a_2 \times b_2$	.063	—	—	-.050	.243
Effect size for indirect effect 2	κ^2_2	.079	—	—	.005	.265

Table 3-9. Estimates for analysis 2A.2 mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Par → PedsQL Par	c	-.398	.129	.002	-.652	-.145
Model with mediator						
Predictors for mediator 1 (BRIEF ER)						
BASC-Anx Par	a_1	.391	.096	< .001	.204	.578
Age	d_1	-.298	.396	.452	-1.075	.480
Female	d_2	-.768	2.716	.777	-6.092	4.556
Minority	d_3	-2.421	2.586	.349	-7.490	2.648
ADHD	d_4	1.300	2.457	.597	-3.516	6.115
Predictors for mediator 2 (BRIEF BR)						
BASC-Anx Par	a_2	.121	.080	.129	-.035	.277
Age	f_1	.000	.337	.999	-.662	.662
Female	f_2	2.764	2.244	.218	-1.634	7.162
Minority	f_3	-2.381	2.155	.269	-6.605	1.843
ADHD	f_4	5.309	2.041	.009	1.310	9.309
Predictors for outcome (PedsQL Par)						
BRIEF ER	b_1	-.793	.137	< .001	-1.061	-.524
BRIEF BR	b_2	-.296	.163	.071	-.616	.025
BASC-Anx Par	c'	-.052	.105	.618	-.259	.154
Age	e_1	-.173	.396	.663	-.951	.605
Female	e_2	-1.339	2.750	.626	-6.729	4.052
Minority	e_3	1.652	2.613	.527	-3.469	6.773
ADHD	e_4	7.938	2.602	.002	2.837	13.039
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.539	—	< .001	—	—
Indirect effect 1 (BRIEF ER)	$a_1 \times b_1$	-.305	—	—	-.510	-.177
Indirect effect 2 (BRIEF BR)	$a_2 \times b_2$	-.036	—	—	-.125	.006
Contrast of indirect effects 1 and 2		-.269	—	—	-.494	-.146
Models with mediators and no covariates						
Indirect effect 1 (BRIEF ER)	$a_1 \times b_1$	-.324	—	—	-.502	-.177
Effect size for indirect effect 1	κ^2_1	.273	—	—	.160	.383
Indirect effect 2 (BRIEF BR)	$a_2 \times b_2$	-.066	—	—	-.187	.037
Effect size for indirect effect 2	κ^2_2	.054	—	—	.003	.146

Table 3-10. Estimates for hypothesis 2A.2 mediator model (selected covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Par → PedsQL Par	c	-.398	0.122	.001	-.637	-.159
Model with mediator						
Predictors for mediator (BRIEF ER)						
BASC-Anx Par	a_1	.363	.089	< .001	.189	.537
ADHD	d_1	1.430	2.440	.558	-3.351	6.212
Predictors for outcome (PedsQL Par)						
BRIEF ER	b_1	-.946	.101	< .001	-1.145	-.748
BASC-Anx Par	c'	-.055	.097	.573	-.245	.135
ADHD	e_1	6.721	2.517	.008	1.787	11.655
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.532	—	< .001	—	—
Indirect effect 1 (BRIEF ER)	$a_1 \times b_1$	-.343	—	—	-.525	-.190
Models with mediators and no covariates						
Indirect effect (BRIEF ER)	$a \times b$	-.324	—	—	-.502	-.177
Effect size for indirect effect	κ^2	.273	—	—	.160	.383

Table 3-11. Estimates for analysis 2B.2 mediator model (all covariates)

Model Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)						
BASC-Anx Child → PedsQL Child	c	-.800	.218	< .001	-1.228	-.372
Model with mediator						
Predictors for mediator 1 (BRIEF ER)						
BASC-Anx Child	a_1	.295	.209	.160	-.116	.705
Age	d_1	-2.472	1.635	.130	-5.676	.732
Female	d_2	-.645	4.819	.894	-10.090	8.800
Minority	d_3	-1.560	4.584	.734	-10.545	7.425
ADHD	d_4	-.572	4.324	.895	-9.047	7.904
Predictors for mediator 2 (BRIEF BR)						
BASC-Anx Child	a_2	-.129	.171	.450	-.463	.206
Age	f_1	.205	1.294	.874	-2.331	2.741
Female	f_2	3.021	3.845	.432	-4.516	10.558
Minority	f_3	-1.649	3.625	.649	-8.754	5.455
ADHD	f_4	3.153	3.417	.356	-3.544	9.849
Predictors for outcome (PedsQL Child)						
BRIEF ER	b_1	-.475	.224	.034	-.913	-.036
BRIEF BR	b_2	.157	.279	.574	-.390	.703
BASC-Anx Child	c'	-.643	.227	.005	-1.088	-.197
Age	e_1	.543	1.657	.743	-2.705	3.791
Female	e_2	-2.114	4.673	.651	-11.272	7.044
Minority	e_3	2.108	4.363	.629	-6.444	10.660
ADHD	e_4	1.431	4.232	.735	-6.864	9.726
Adjusted R^2 for DV model	$R^2_{Y,MX}$	.373	—	.002	—	—
Indirect effect 1 (BRIEF ER)	$a_1 \times b_1$	-.144	—	—	-.529	.028
Indirect effect 2 (BRIEF BR)	$a_2 \times b_2$	-.010	—	—	-.231	.046
Contrast of indirect effects 1 and 2		-.135	—	—	-.519	.110
Models with mediators and no covariates						
Indirect effect 1 (BRIEF ER)	$a_1 \times b_1$	-.105	—	—	-.428	.015
Effect size for indirect effect 1	κ^2_1	.091	—	—	.009	.261
Indirect effect 2 (BRIEF BR)	$a_2 \times b_2$	.035	—	—	-.018	.234
Effect size for indirect effect 2	κ^2_2	.039	—	—	.002	.205

Table 3-12. Estimates for hypothesis 2C (all covariates)

Model	Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)							
	BASC-Anx Par → PedsQL Par	c	8.278	3.940	.036	.556	15.999
Model with mediator							
	Predictors for mediator 1 (D-KEFS Inhibit)						
		BASC-Anx Par	a_1	-4.156	2.922	.155	-9.882	1.571
		Age	d_1	.250	.519	.630	-.768	1.268
		Female	d_2	.761	.580	.189	-.375	1.898
		Minority	d_3	6.797	3.207	.034	.511	13.084
		ADHD	d_4	.525	3.256	.872	-5.856	6.907
	Predictors for mediator 2 (D-KEFS Monitor)						
		BASC-Anx Par	a_2	-.047	.489	.923	-1.007	.913
		Age	f_1	.538	.083	< .001	.375	.701
		Female	f_2	.046	.096	.630	-.142	.234
		Minority	f_3	.814	.531	.125	-.227	1.855
		ADHD	f_4	-.336	.548	.539	-1.411	.739
	Predictors for outcome (PedsQL Par)						
		D-KEFS Inhibit	b_1	-.301	.154	.050	-.603	.000
		D-KEFS Monitor	b_2	.147	.981	.881	-1.777	2.070
		BASC-Anx Par	c'	7.041	3.946	.074	-.693	14.774
		Age	e_1	.310	.871	.722	-1.398	2.018
		Female	e_2	.386	.780	.620	-1.142	1.915
		Minority	e_3	-2.972	4.479	.507	-11.751	5.808
		ADHD	e_4	2.340	4.352	.591	-6.191	10.870
		Adjusted R^2 for DV model	$R^2_{Y,MX}$	.053	—	.140	—	—
	Indirect effect 1 (D-KEFS Inhibit)		$a_1 \times b_1$	1.246	—	—	-.250	4.964
	Indirect effect 2 (D-KEFS Monitor)		$a_2 \times b_2$	-.051	—	—	-1.192	1.076
	Contrast of indirect effects 1 and 2			1.297	—	—	-.552	4.900
Models with mediators and no covariates							
	Indirect effect 1 (D-KEFS Inhibit)		$a_1 \times b_1$	.002	—	—	-.021	.068
	Effect size for indirect effect 1		κ^2_1	.002	—	—	.000	.013
	Indirect effect 2 (D-KEFS Monitor)		$a_2 \times b_2$	.012	—	—	-.015	.123
	Effect size for indirect effect 2		κ^2_2	.010	—	—	.000	.077

Table 3-13. Estimates for hypothesis 2D (all covariates)

Model	Parameter		Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator (total effect of IV on DV)							
	BASC-Anx Child → PedsQL Child	c	-.800	.218	< .001	-1.228	-.372
Model with mediator							
	Predictors for mediator 1 (D-KEFS Inhibit)						
		BASC-Anx Child	a_1	-.012	.038	.746	-.086	.062
		Age	d_1	.377	.301	.210	-.213	.966
		Female	d_2	1.248	.887	.159	-.489	2.986
		Minority	d_3	.526	.841	.532	-1.124	2.175
		ADHD	d_4	-.530	.794	.504	-2.086	1.025
	Predictors for mediator 2 (D-KEFS Monitor)						
		BASC-Anx Child	a_2	.062	.052	.239	-.041	.164
		Age	f_1	.453	.407	.266	-.345	1.251
		Female	f_2	.538	1.206	.655	-1.825	2.902
		Minority	f_3	.682	1.143	.550	-1.557	2.922
		ADHD	f_4	.114	1.076	.916	-1.996	2.224
	Predictors for outcome (PedsQL Child)						
		D-KEFS Inhibit	b_1	-1.113	1.156	.336	-3.377	1.152
		D-KEFS Monitor	b_2	.898	.857	.295	-.783	2.578
		BASC-Anx Child	c'	-.871	.232	< .001	-1.327	-.415
		Age	e_1	1.761	1.698	.300	-1.567	5.090
		Female	e_2	-.434	5.073	.932	-10.378	9.510
		Minority	e_3	2.556	4.712	.588	-6.680	11.792
		ADHD	e_4	1.505	4.464	.736	-7.244	10.254
		Adjusted R^2 for DV model	$R^2_{Y,MX}$	.279	—	.021	—	—
	Indirect effect 1 (D-KEFS Inhibit)		$a_1 \times b_1$	.017	—	—	-.071	.240
	Indirect effect 2 (D-KEFS Monitor)		$a_2 \times b_2$	.053	—	—	-.031	.439
	Contrast of indirect effects 1 and 2			-.036	—	—	-.446	.117
Models with mediators and no covariates							
	Indirect effect 1 (D-KEFS Inhibit)		$a_1 \times b_1$	-.004	—	—	-.098	.026
	Effect size for indirect effect 1		κ^2_1	.004	—	—	.000	.026
	Indirect effect 2 (D-KEFS Monitor)		$a_2 \times b_2$	.049	—	—	-.009	.331
	Effect size for indirect effect 2		κ^2_2	.049	—	—	.003	.261

Table 3-14. Summary of indirect effect estimates and effect sizes across models

Analysis index	Mediator 1 (κ^2)	Mediator 2 (κ^2)	Difference in mediators	Significant covariates	Sample
Mediators based on BRIEF					
Parent form of PedsQL and BASC					
1A	Total* (.15)	—	—	Minority, ADHD	A
2A.1	BRI* (.19)	MI* (.07)	(BRI > MI)*	Minority, ADHD	A
2A.2	ER* (.27)	BR (.05)	(ER > BR)*	ADHD	A
Child form of PedsQL and BASC					
1B	Total (.05)	—	—	—	B
2B.1	BRI (.02)	MI (.08)	—	—	B
2B.2	ER (.09)	BR (.04)	—	—	B
Mediators based on D-KEFS					
Parent form of PedsQL and BASC					
2C	Inhibit (.00)	Monitor (.01)	—	Age, Minority	C
Child form of PedsQL and BASC					
2D	Inhibit (.00)	Monitor (.05)	—	—	B

Note. * $p < .05$ for mediators' indirect effects or for difference between mediators' indirect effects. BRI = Behavioral Regulation Index. MI = Metacognition Index. ER = Emotional Regulation factor. BR = Behavioral Regulation factor.

CHAPTER 4
DISCUSSION

The current study assessed executive functioning's (EF) hypothesized mediation of the relation between anxiety and quality of life (QOL) in a pediatric clinical sample. With design constraints (i.e., cross-sectional data) as the interpretational backdrop for the findings, support for EF's mediating role was detected. In keeping with a Processing Efficiency Theory-based prediction that anxiety compromises EF as a unitary construct (PET; Eysenck & Calvo, 1994), current findings supported this disruption for the parent-assessed models. Anxiety-related executive dysfunction was in turn associated with decreased pediatric QOL. Further analyses assessed a prominent model of EF as a fractionated construct (Miyake et al., 2000) and concomitant theoretical advances regarding anxiety's effect on particular subdomains of EF. Attentional Control Theory (ACT; Eysenck et al., 2007) posits that anxiety impacts the inhibit and shift subdomains of EF, and the present findings generally aligned with this theoretical prediction as well. In fact, relative to a monolithic EF construct, inhibition and shifting more strongly mediated the anxiety-QOL relationship. Furthermore, exploratory analyses demonstrated the even greater mediation capacity for additional empirically derived and theoretically sound EF subdomains (Gioia at al., 2002). This chapter will contextualize current findings within the extant literature, offering limitations and future directions, followed by a brief summary and conclusions.

Findings Contextualized in Current Literature

This section discusses current findings related to EF as a global then fragmented mediator. Parent versus child rater results are examined in light of the available research, followed by a consideration of the implications of EF mode of measurement

(i.e., parent assessment versus child-performance). Finally, additional overarching findings related to pediatric anxiety and QOL are discussed.

Mediation with EF as a Unitary Construct

Design implications notwithstanding, findings provide preliminary support for Airaksinen and colleagues' (2005) postulation that EF deficits would predict eroded QOL related to anxious symptomatology. This study fits well within the extant literature on pediatric anxiety and executive dysfunction (Emerson et al., 2005; Francis, 1988; Kendall & Chansky, 1991; Micco et al., 2009; Toren et al., 2000). Just as Francis (1988) found that children with higher anxiety levels also had significantly more task-inhibiting thoughts, this study provided preliminary evidence that EF serves as a mechanism for how anxiety impacts QOL. The impairments in cognitive flexibility detected in Toren and colleagues' (2000) sample resemble the current findings supporting the plausibly adverse effects of anxiety on EF, and specifically, the shifting and inhibiting subdomains.

The current results fit well with Micco and colleagues' (2009) findings. As referenced earlier, their study provided tentative data regarding direction of effects by examining children of parents with anxiety and depressive disorders. Due to prior research indicating higher prevalence of psychopathology in the offspring of affected parents as compared to the offspring of healthy parents, Micco and colleagues expected to find EF impairments in children at risk for depression and anxiety. Their results, however, revealed no relationship between offspring status and executive dysfunction, leading them to conclude that impaired EF may not serve as a trait marker for developing anxiety or depression. Conversely, they found that children with current mood and anxiety symptoms also exhibited EF impairment, which suggested that

executive dysfunction may be symptomatic of depression and anxiety rather than causal. Micco and colleagues' conclusions aligned with Eysenck and colleagues' (1992, 2007) theories on anxiety and disrupted EF, which collectively set the stage for the current study.

Driven by Eysenck and Calvo's (1992) theory on anxiety's impairing effects on general EF, the current study's initial model using the parent-assessed Behavior Rating Inventory of Executive Function (BRIEF; Gioa et al., 2000) as the mediator—which has been noted to possess greater ecological validity than narrowly focused performance measures, as described later—demonstrated statistical significance as well as a medium to large effect size for the mediation. Due to mathematical limitations (K. Preacher, personal communication, October 23, 2011), recall that the effect sizes were only calculated in simple mediation models, and thus did not account for simultaneous mediators or the effect of covariates.

Interpretation is not as clear in the child self-reported models analyzing the hypothesized BRIEF mediation. All self-report analyses for the Pediatric Quality of Life Inventory Version 4.0 (PedsQL; Varni et al., 2001) and the Behavior Assessment System for Children, Second Edition (BASC; Reynolds & Kamphaus, 2004) Anxiety scale examined Sample B, consisting of 42 individuals ages 12 and older. Essentially all parameters in these models were nonsignificant. Sometimes the effect sizes were large enough to suggest that with larger samples significant effects would potentially be detected. Consider Hypothesis 1B. The low sample size and resulting diminished power to detect effects are obvious potential considerations for why the indirect effect of anxiety on QOL is nonsignificant, along with most other parameters in the model.

Although the effect size estimate was low, the CI's expanse allowed for the possibility of a medium effect. The likelihood of whether a larger sample would have detected a significant effect is unclear though probable. Fritz and MacKinnon (2007) suggested that analyses such as the bias-corrected, bootstrap mediation used in this study require at least 71 individuals in order to achieve a statistical power of .80 for detecting medium effects. Detection of small effects requires 148 to 462 individuals. The current sample, however, contained only 42 children and adolescents. In conjunction with the effect size and CI yielded herein, detection of significant findings in larger samples appears probable, at least sufficiently enough to justify future research.

Although there is a paucity of research on the role of EF in pediatric anxiety with regard to QOL, studies have demonstrated links between executive dysfunction and eroded QOL related to ADHD (Klassen et al., 2004), traumatic brain injuries (Horneman et al., 2005), and epilepsy (Sherman et al., 2006). The current findings add to this literature, now with an emphasis on anxious symptomatology. Castaneda and colleagues (2008) highlighted the importance of exploring third variables influencing the anxiety and QOL relationship, and the current study shed light on EF's potentially mediating role as a unitary and fragmented construct. Findings suggested that particular subdomains of EF may exert even greater mediation strength.

Mediation with EF subdomains

Eysenck and colleagues' (2007) current theory of the relationship between anxiety and EF capitalized on research advances related to what is often called "the unity and disunity of EF." Investigating latent variables with a confirmatory factor analysis, Miyake and colleagues (2000) detected a three-factor model of EF that has become prominent in the field and continues to garner attention (Latzman & Markon, 2010). Their results

indicated that three separable yet related subdomains constitute EF: inhibition, or the ability to exert control over automatic responses; shifting, or the capacity to perform multiple operations and switch from one task to another while experiencing interference; and updating, or the capacity to monitor and assess the relevance of novel information while performing the task at hand. Basing their predictions on this deconstructed model of EF, Eysenck and colleagues further specified that anxiety impacts shifting and inhibiting subcomponents of EF.

This study's findings supported EF's multidimensionality. As mediators, the BRIEF Behavioral Regulation Index (BRI) containing the inhibition and shifting subcomponents of EF outperformed the updating subdomain as represented by the BRIEF Metacognition Index (MI). Although both indices demonstrated significant mediation effects for parent-assessed anxiety and QOL, contrast estimates suggested the primacy of inhibition and shifting as mediating subdomains. Additionally, in Hypothesis 2A.1, a greater effect size was detected for the inhibition/shifting index than the updating analog. The larger effect is somewhat expected in light of the BRI's advantage derived from containing two EF subdomains in comparison to the single subdomain occupying the MI. Notably, the strength of effect for the inhibit/shift composite exceeded the effect size for the BRIEF total as examined earlier, perhaps suggesting that the inhibit/shift subdomains function as a purer mediator of anxiety's effect on QOL.

More precisely, the shifting subdomain—or the ability to move freely between tasks or to transition from one aspect of a problem to another in response to contextual demands—may exhibit greater potential as a mediator in the relationship between anxiety and QOL. Promising results for the differential mediation power inspired

exploratory post-hoc analyses based on findings from Gioia and colleagues' (2002) BRIEF confirmatory factor analysis. Whereas initial analyses showed some support for stronger mediation properties in the inhibit/shift subdomains in comparison to the updating domain, there was interest in determining how inhibition and shifting would operate independent of one another. Thus, the inhibit versus shift subdomains competed as simultaneous mediators, and the Emotional Regulation (ER) factor corresponding to the shift subdomain significantly mediated anxiety's effects on QOL. It had a large effect size, exceeding the effect sizes of unitary EF and the inhibit/shift subdomain composite. The shifting subdomain of EF appeared to function as the strongest mediator of anxiety's negative effect on QOL observed in this study.

Child self-reports, again, presented ambiguity in relation to assessing varying strengths of EF subdomains. In the model examining the differential power of the inhibit/shift subcomponents (BRI) versus the updating subcomponent (MI), the updating dimension had a greater effect size than inhibit/shift. Considered independently, this inconsistency is difficult to interpret, and it may represent an artifact of a model with limited significant parameters. The finding may also stem from differences related to self-report versus parent-report. However, interpretation of this difference should be tempered with caution given that the respective indirect effects did not differ at a level of statistical significance. Although the model was not significant, power was again compromised due to the small sample size. Recall that the sample size of 42 individuals for this analysis fell below Fritz and MacKinnon's (2007) .80 threshold to detect medium-sized effects. Clearly, additional research is needed to shed light on the disparities between parent and child assessment in relation to EF's potential mediation

of anxiety and QOL. Divergences among raters—and measurement instruments—are not novel in the pediatric QOL research, and the discussion now turns to this topic.

Rater and Measurement Divergences

In the models using the parent forms for the PedsQL and the BASC Anxiety scale, the EF mediation appeared consistently significant with the BRIEF (global EF composite and subdomains as mediators), but not with the D-KEFS factors. These findings raise at least two important methodological questions related to raters and measures. As recommended for pediatric QOL research (Bastiaansen et al., 2005), data from child/adolescent participants and parents/primary caregivers were examined. Previous research suggested that parent-assessed QOL was reliable in a sample of asthmatic children (Le Coq, Boeke, Bezemer, Bruil, & Van Eijk, 2000), yet overall findings for parent-child agreement are mixed (Ravens-Sieberer et al., 2006). Reliability and validity are challenging issues in pediatric QOL assessment (Connolly & Johnson, 1999; Matza et al., 2004), particularly with abstract psychological constructs such as anxiety. Ravens-Sieberer and colleagues (2006) noted that lower concordance rates predominate for internalizing or emotional content areas, and in the current study, parent-assessed anxiety were significantly greater than child self-reported anxiety.

Generally, parent-proxy report is thought to offer greater reliability, perhaps at the cost of decreased validity (Matza et al., 2004). It is difficult to assess the extent to which skewed parental or child reporting is operating. On the one hand, the mere presence of mental health symptoms may confound self-assessment (Saintfort et al., 1996), especially for children and complex mental health-related concepts (Rebok et al., 2001). On the other hand, the individual's unique perspective takes on even greater relevance when speaking of internalizing disorders such as anxiety, given the

experience is often one of private misery: undetected, misconstrued, or deliberately avoided by even the most well intentioned observer. The tension between the parent and child perceptions is hard to reconcile and extends beyond the scope to the current study, though it is worthy to note that it is often the parents' perspective that determines whether a child will seek treatment. Accordingly, the current mixed overall findings fit well within extant literature, given that low concordance rates between children and significant others have been noted. Future studies involving multiple "objective" raters (e.g., health care providers, teachers) may help clarify rater discrepancies in this line of anxiety, EF, and QOL research. Even though additional perspectives are also vulnerable to reporting bias, additional raters enrich prospects for cross-validation.

Measurement concerns also pertain to EF assessment. Evidence suggests that executive dysfunction may be detected better by family members who can assess children's performance in everyday life situations (Sherman et al., 2006). Yet, as noted, the possibility of parental misreporting has been documented in the literature, especially in relation to children's internalizing mental disorders and neuropsychological dysfunction (Matza et al., 2004). The present study detected statistically significant findings in the EF mediation models employing the parent forms for the PedsQL and the BASC Anxiety scale. Child performance on the D-KEFS, however, yielded nonsignificant findings. In conjunction with disparate rater-related findings, another rival—and perhaps more likely—explanation for the discrepancy in EF measurement stems from the nature of the measures themselves. For instance, Vriezen, Pigott, and Pelletier (2001) found that behavioral ratings and performance-based assessment were not correlated, which may imply that children can excel on restricted testing in controlled

clinical environments and still experience significant impairment in everyday living. The current findings were consonant with this lack of correlation between behavioral ratings and performance-based assessment. Of course, there are proponents of clinical EF assessment (e.g., Kalinian, 2003; Latzman et al., 2010), as it appears to discriminate between different groups in relation to executive dysfunction. Others have argued that clinical instruments such as the D-KEFS may not adequately detect compromised EF due to the highly controlled, one-on-one testing environment (e.g., Schmidt, 2003; Strauss, Sherman, & Spreen, 2006). Participants are given explicit rules and time-frames. Although a "white coat" effect may threaten optimal performance, the structured, quiet ambience sets the stage for effective problem solving and task performance.

Accordingly, a general theme in the literature is that clinical evaluation may not be as ecologically valid as an instrument like the BRIEF, which appears more attuned to EF deficits in daily activities and thus better suited to capture "real-world" behavior and difficulties (Denckla, 2002; Donders, 2002; Sherman et al., 2006). Banich summarized the challenges of clinical EF measurement well, noting that "the very nature of executive functioning makes it difficult to measure in the clinic or laboratory; it involves an *individual* guiding his or her behavior, especially in novel, unstructured, and non-routine situations that require some degree of judgment" (p. 89, 2009). It is perhaps for this reason that current findings with only the BRIEF provided preliminary evidence for EF's mediation of anxiety and QOL.

That is not to say that EF assessment by third-party observers is immune from bias and subsequent critical interpretation. Denckla (2002) noted two important

interpretational caveats. Not unlike other modes of assessment, a level of linguistic competence is required in the parent, and such an understanding is largely influenced by divergences in colloquial versus professional denotations of words. Whereas Denckla highlighted the common tendency to erroneously pathologize organized and meticulous—though otherwise functionally "normal"—behavior as "Obsessive-Compulsive Disorder," she also noted that semantic misunderstandings are possible though less likely to occur with the BRIEF. A greater threat to validity comes in the form of emotional biases of the third-party observer, who may knowingly or unknowingly alter responses in effort to exaggerate or minimize children's dysfunctional behavior. Such questions of perception and the many influences that inform it are not unique to parent-assessed EF, though responsible interpretation of results requires awareness of these factors.

Additional Contributions to Pediatric Anxiety, EF, and QOL Research

Implicit within the mediation analyses, findings were consistent with prior research demonstrating the association between anxiety and impaired EF (Emerson et al., 2005; Francis, 1988; Kendall & Chansky, 1991; Micco et al., 2009; Toren et al., 2000). Of note, however, were the relatively low parent-assessed and child self-reported anxiety scores in the current sample. Anxious symptomatology, when evaluated by either rater, did not meet the level of clinical significance according to the measure's guidelines. Given the study's aim of broadly assessing anxious symptoms in a naturally occurring clinical setting, this sample characteristic was somewhat unexpected in light of the high prevalence of pediatric anxiety (Saddock et al., 2009). The diagnostic composition of the sample also made the low level of anxiety surprising, given that anxiety is one of the most frequent comorbid disorders in children with ADHD (Tannock, 2000).

The preponderance of ADHD diagnoses in the sample was not anticipated, nor was it uncommon due to the generally high frequency of children referred to outpatient clinics for ADHD assessment. Sixty of the 108 participants had ADHD diagnoses, which influenced the decision to control for ADHD status in order to minimize interpretational confounds. Greater executive dysfunction appeared associated with ADHD in keeping with prior research. Somewhat counterintuitive, perhaps, was that individuals with an ADHD diagnosis had QOL scores that were 10.3 points higher on average than those without an ADHD diagnosis in the overall sample when examined in Hypothesis 1. Although this finding was incongruent with at least one other study that revealed no statistically significant QOL difference across diagnostic categories (Bastiaansen et al., 2004), this finding may be reasonable within the context of a mixed clinical sample, considering that individuals with ADHD were compared to individuals diagnosed with a mental disorder. In fact, as illustrated in Table 2-3, only 2 of the 108 individuals in Sample A had no diagnosis, and perhaps the non-ADHD diagnoses (e.g., Pervasive Developmental, Disruptive Behavior, Learning, Mood, and Anxiety Disorders) were simply associated with lower QOL.

The current sample reported relatively poorer QOL than others in the literature. Bastiaansen and colleagues' (2004) clinical sample exhibited a mean score approximately half a standard deviation higher than what was observed in the current study. Speculations could abound as to the many unexamined third variables or other between-sample differences, though an obvious potential confound is cultural relativism. That is, their study was conducted in the Netherlands, and cross-cultural comparison of QOL in the absence of culture-specific norms is challenging at best, and the interested

reader can pursue this topic further. Analyses closer to home help contextualize the impoverished QOL within the current sample. Evaluating a U.S. based pediatric sample, Varni and colleagues' (2003) also detected a mean QOL score that exceeded the current sample by half a standard deviation. Their sample consisted of chronically ill youth (i.e., diagnoses of asthma, diabetes, depression, and ADHD) between the ages of 2 and 16. In relation to their minimal clinically important difference (MCID)[1], the current sample average fell two units below Varni and colleagues' sample mean, suggesting an overall poor report for children's psychosocial, physical, and emotional well-being. Prior research has documented the impaired QOL of children with psychiatric disorders in comparison to healthy and physically ill children (Sawyer et al., 2002), and the current study further attests to the compromised QOL in a child psychiatric clinical sample.

Limitations and Future Directions

This study has a number of strengths and limitations. In terms of diversity, some analyses demonstrated significantly better EF in racial-ethnic participants as compared to White counterparts. Standardization of the utilized measures and a search of the available literature revealed no documented effects for racial-ethnic status (Delis et al., 2001; Gioia et al., 2000; Tan, 2007). Future studies might examine this effect more deeply, perhaps with increased multiculturally heterogeneous samples. Regarding sample composition, the mixed clinical sample examined here is a considerable strength in that it may provide practicing psychologists with preliminary data relevant to clinical settings, though future replication and further elaboration of this work is needed.

[1] As noted earlier, an MCID refers to the "smallest difference in a score of a domain of interest that patients perceive to be beneficial and that would mandate, in the absence of troublesome side effect and excessive costs, a change in the patients' management" (Varni et al., 2003, p. 332).

This study adhered to the aspirational yet pragmatically challenging practice of incorporating disparate points of view (Matza et al., 2004; Ravens-Sieberer et al., 2006). As referenced, future studies might also benefit from additional third-party raters (e.g., teacher or health care professionals) in order to cross-validate parent reports. Additionally, this study gathered self-report data for children ages 12 years and older, an age range that seemed appropriate for self-report on a complex psychological variable such as anxiety. Debate on age appropriateness for child self-report of internalizing psychiatric symptomatology is unresolved (Bastiaansen et al., 2004, 2005; Matza et al.), and future work may consider deeper investigation of this topic or incorporate larger samples than the self-report subsample examined herein. In keeping with third party involvement, some variables that may have been relevant and ideal to control for statistically (Kessler, 1987) were not measured. Perhaps parents' or caregivers' overcompensation for children's lower EF resulted in children with very low EF experiencing somewhat higher QOL, in which case it would have been ideal to attempt to measure this effect and include it in the analyses. Even though measurement of this phenomenon may be challenging, future research may benefit from integrating parental level of accommodation.

The choice to keep the significance criterion at $p = .05$ is relevant for interpretation of current results. Inflation of experimentwise error rate (i.e., Type I error) was deemed acceptable and responsible given the limited research in pediatric QOL (Bastiaansen et al., 2004) and the anxiety/EF relationship (Castaneda et al., 2008). Howell (2002) underscored the debate as to what significance criterion is appropriate, concluding that researchers' informed views on the trade-off between Type I and Type II errors may be

the wisest guide to set an alpha level. The exploratory nature of this research provided a rationale for the decision, and results may best be interpreted in light of this analytic approach.

Well-respected and psychometrically solid instruments were utilized, apart from some reservations about the D-KEFs as noted (Crawford et al., 2008; Homack et al., 2005). The BRIEF is widely used due to its ecological validity and clinical utility. Future studies may benefit from utilizing item level data for this instrument, which would allow for assessment of the BR factor (tested here in a post-hoc exploratory analysis) with the 4 task-monitoring items omitted as prescribed by a prior confirmatory factor analysis (Gioia et al., 2002). In light of the stronger ER results, this follow-up work appears less critical, though this line of research is in its incipient phases and replication is warranted. Similarly, the D-KEFS factors could also be enriched by a non-restrictive archive to better replicate Latzman and colleagues' (2010) composite structure. Nonetheless, the earlier caveats related to child-performance assessment may render this a moot point. Additionally, future studies may be enriched by utilizing a dedicated anxiety measure (e.g., Multidimensional Anxiety Scale for Children; March, 1997). Now that preliminary evidence supporting the EF mediation hypothesis has been detected, additional contributions can be made regarding which specific domains of QOL (i.e., physical, emotional, social, or academic) are most involved in the relationship between anxiety and EF.

Driven by Eysenck and Calvo's (1992) and Eysenck and colleagues' (2007) theories, results provided evidence for the hypothesis that EF is a mechanism through which anxiety conveys its effect on QOL. However, any study of mediation is up against

serious challenges. Psychologists are appropriately concerned about causality in mediation models (Spencer, Zanna, & Fong, 2005). According to Woody (2011), one challenge is the relative ease of obtaining apparently significant tests of mediation even when the models are nonsensical. He cautioned well, reminding that "establishing mediation is difficult, requiring multiple converging approaches in a creative program of research. It is not, and never will be, reducible to any formulaic statistical test, no matter how sophisticated" (p. 243). One limitation is the cross-sectional nature of these data (i.e., for each individual, data were collected for all measures on a single occasion). Thus, the data are especially susceptible to uncertainties related to causality and temporal ordering of the effects. Maxwell and Cole (2007) pointed out how cross-sectional data insufficiently address the causal effects hypothesized by mediation. However, the present study's use of an empirically supported theoretical framework (see Derakshan & Eysenck, 2009) clearly bolsters the findings, which in turn invite replication. The current study sets the stage for related future work in an obviously important area of research.

Careful interpretation is imperative so as to not extend too far beyond the design implications. Although the present study's theory-driven approach yielded supporting evidence for EF's role as a mechanism through which anxiety affects QOL, the dynamic interplay between this set of variables appeals to reason. The likely reciprocity of influence between anxiety and QOL has been noted (Bastiaansen et al., 2004; Mogotsi et al., 2000). Some evidence points toward executive dysfunction as symptomatic of anxiety (Micco et al., 2009) yet bi-directional effects cannot be ruled out by any means (Emerson et al., 2005; Francis, 1988; Kendall & Chansky, 1991; Toren et al., 2000).

Again, this area of inquiry is clearly in its nascence, and the current findings may catalyze future investigation and substantiate the investment associated with more advanced designs. Ideal mediation studies overcome the shortcomings of cross-sectional data by using longitudinal designs, which appear well founded in light of the current findings. Dwyer (1983), for instance, described the benefits of longitudinal data for evaluating reciprocal causation between variables. As noted by MacKinnon and Luecken (2008), a diversity of approaches is of substantial value in research that examines third variables (i.e., mediators and moderators) such as EF in relation to anxiety and QOL.

Summary and Conclusion

In light of the scarcity of research on the role of EF in pediatric anxiety and QOL, the present study sought to expand the empirical knowledge base by addressing this critical gap in the literature. Using theory-based predictions, the study tested the hypothesis that EF would mediate the relationship between anxiety and QOL. For parent-assessment of EF, results supported the predictions for (a) EF as a unitary mediator and (b) EF's multidimensional nature, including the differing mediation strengths of the inhibit and shift subdomains. These preliminary findings offer empirical support for conducting more strategically designed studies to corroborate, contradict, or expand on the current findings. Evidence supporting the EF mediation may provoke further investigation into the potential for anxiety-focused clinical interventions that emphasize bolstering EF. Because "Daily EF 'exercise' appears to enhance EF development much as physical exercise builds bodies" (Diamond et al., 2007, p. 1388), possible therapeutic approaches to anxiety—the nation's most prevalent mental disorder—may be enriched by future investigation of the EF/anxiety/treatment nexus.

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BIOGRAPHICAL SKETCH

Born and raised in New Jersey, Robert Merrell spent his sophomore year in Andalucía, Spain, at the Universidad de Sevilla. A scholarship later allowed him to study in South America at the Universidad de Concepción in southern Chile. A Phi Beta Kappa member, he earned his Bachelor of Arts (*summa cum laude*) and Master of Arts in Spanish Literature from Villanova University outside of Philadelphia, Pennsylvania.

Influenced by his minor-related studies in philosophy and passion for helping others move toward optimizing personal growth, he began studying psychology and received his Master of Science degree from the University of Florida. After completing his doctoral studies in December 2011, he plans to remain in his current position within the University of Florida's Department of Psychiatry. His interests range from clinical supervision of psychiatry residents to the provision of exposure and response prevention treatment for Obsessive-Compulsive Disorder for children and adults. Future career plans may involve further specialization in the treatment of anxiety and mood disorders.