Childhood Trauma and ADHD Diagnosis as Predictors of Consistently Elevated Internalizing
Symptoms

by

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Abstract

Many children adapt well to life after trauma, but children with ADHD may be at risk for post-traumatic maladaptation. ADHD is often comorbid with mental health disorders and deficits in cognitive functioning. Using data from the Multimodal Treatment of Children with ADHD study, a randomized clinical trial that tested treatments for ADHD in 579 children and later recruited 289 comparison children, the present study tests whether trauma predicts more severe levels and faster increases in depression and anxiety for children with ADHD versus developmentally typical peers. Interpersonal trauma predicted elevated depression and anxiety symptom severity, but not trajectories. Natural trauma predicted elevated anxiety symptoms, but not trajectories. Last, ADHD diagnosis predicted elevated depression symptoms, but not trajectories. There were no trauma x ADHD interactions on symptom levels or trajectories. This study informs practice and policy by guiding practitioners to use trauma history to identify children who demonstrate persistent internalizing symptoms.

KEYWORDS: Trauma, ADHD, depression, anxiety

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Table of Contents

Title page...	i
Abstract...	ii
Acknowledgement...	iii
Table of Contents...	iv
List of Tables...	v
List of Figures...	vi
Introduction...	1
Developmental Systems Theory...	2
Childhood Trauma...	6
Internalizing Psychopathology...	10
Attention Deficit/Hyperactivity Disorder...	12
The Current Study...	16
Method...	17
<i>Participants and Procedure</i> ...	17
<i>Measures</i> ...	18
<i>Data Preparation and Analysis Strategy</i> ...	23
Results...	32
<i>Depression</i> ...	32
<i>Anxiety</i> ...	38
Discussion...	45
<i>Limitations</i> ...	62
<i>Concluding Remarks</i> ...	63
References...	67

List of Tables

Table 1: <i>Symptom Criteria for ADHD Diagnosis</i> ...	13
Table 2: <i>Effects coding for ethnicity</i> ...	23
Table 3: <i>Effects coding for household advantage</i> ...	23
Table 4: <i>Demographic characteristics of sample by ADHD diagnosis</i> ...	30
Table 5: <i>Descriptive statistics for depression and anxiety symptoms</i> ...	33
Table 6: <i>Descriptive statistic and correlation matrix of select variables</i> ...	33
Table 7: <i>Growth of and effects of trauma and ADHD on depression symptoms</i> ...	35
Table 8: <i>Growth of and effects of trauma and ADHD on anxiety symptoms</i> ...	40

List of Figures

Figure 1: <i>Hypotheses between trauma, ADHD, and internalizing symptoms...</i>	17
Figure 2: <i>Depression trajectories for children with interpersonal trauma histories...</i>	36
Figure 3: <i>Depression trajectories for children with and without an ADHD diagnosis...</i>	37
Figure 4: <i>Anxiety trajectories for children with interpersonal trauma histories...</i>	41
Figure 5: <i>Anxiety trajectories for children with natural trauma histories...</i>	42

Childhood Trauma and ADHD Diagnosis as Predictors of Consistently Elevated Internalizing Symptoms

Early traumatic experiences are a common occurrence in children (Little, Akin-Little, & Somerville, 2011), among which include the sudden death of a caregiver or loved one, surviving a natural disaster, and experiencing early childhood maltreatment. Although many children show resiliency and eventually function normally after trauma, there are well documented negative consequences to experiencing trauma during childhood (Agorastos et al., 2014; Ballard et al., 2015). Early trauma has been linked to the development of psychopathology; for example, victims of childhood sexual abuse are more likely to later receive a diagnosis of major depressive disorder and to attempt suicide (Ballard et al., 2015). In addition, the type, timing, and frequency of trauma may be crucial to predicting later onset and severity of maladaptive outcomes. For example, individuals who report a high frequency of childhood abuse or neglect are more likely to have chronic depression, in addition to having more severe symptoms, onset of depression before age 21, and comorbid anxiety (Wiersma et al., 2009). However, it is unclear why some children develop without major maladaptation while others experience difficulties, which vary in degree of severity.

Cognitive functioning can greatly influence childhood outcomes post-trauma. Children with ADHD in particular show impaired executive function in domains such as impulsivity (Raiker, Rapport, Kofler, & Sarver, 2012). Impulsivity may manifest as a limited ability to apply adaptive problem-solving skills, such as comparing various solutions, when faced with adversity. In addition, children with ADHD possess smaller coping repertoires and have difficulty interpreting the context of a problem and, as a

result, applying a proper coping response (Babb, Levine, & Arseneault, 2010). Thus, children with ADHD may face increased adversity post-trauma when compared to peers without an ADHD diagnosis.

This study will examine the relations between childhood trauma and the severity and progression of internalizing symptoms (depression and anxiety) for children with and without ADHD. These relations are viewed from a developmental systems perspective, considering both inter- and intraindividual variability in symptom development. Results may inform policy and practice with a general goal of promoting positive development and facilitating early identification of children with a risk of developing severe psychopathology.

Developmental Systems Theory

Developmental systems theory takes a multilevel approach to understanding developmental processes (Lerner, 2006), and offers a helpful framework for understanding the emergence and progression of psychopathology following trauma. From this perspective, an individual is viewed as developing within his or her unique system, comprising levels that range from genes to cultural norms. Time is a fundamental component of the system and intraindividual variability and change are evidence of development unfolding. Using this theoretical perspective, researchers can triangulate what aspects of a developmental system, for which individuals, in what contexts, and during which times promote adaptive development (Lerner, 2006). The present study is guided by a developmental systems perspective to investigate why some youth struggle and others thrive post-trauma.

Historical approaches to the development of the individual cyclically shifted from theories in which either “nature”, such as biogenetics, or “nurture”, such as environmental experiences, was the primary force guiding development (Sameroff, 2010). Two such early models of human development are the *organismic* and *mechanistic* models. Organismic models of human development purport that the individual develops in universal stages (Dixon & Lerner, 1988). Individuals become more complex as they achieve each stage in a unidirectional, irreversible trajectory. There are clear concerns when considering organismic models. For example, how can the diversity of development be explained if all individuals are driven to successfully progress through universal stages? Why do people who start life with similar resources and risks often end up at very different places years later? Consideration must also be given to the role of the environment in human development. Mechanistic models, in contrast, purport that the individual is a passive machine made up of various components (Dixon & Lerner, 1988). Development occurs as the individual reacts to environmental forces, and the individual is a powerless entity that follows the path laid out by external contexts. Of concern for mechanistic models of development is the lack of purposefulness attributed to the individual, which is a weakness of this theoretical perspective.

Contemporary developmental systems theories take a *contextualist* approach, recognizing that individual, contextual, and interactional levels may all simultaneously influence development (Dixon & Lerner, 1988). The diversity of development is understood as a strength, and individual development is not unidirectional (Dixon & Lerner, 1988). Behaviours, abilities, and characteristics may regress to an earlier state

over time, and development may occur on different timelines for different people.

Current developmental science has placed much focus on contextualism and how person-context interaction can create opportunities for or place constraints on development (Dixon & Lerner, 1988; Lerner & Callina, 2014).

Developmental science has emerged into a field of study that focuses on the diversity of development across the lifespan, which is largely attributed to the interplay between multiple levels of organization that affect and are affected by the individual (Lerner, 2006; Marshall, 2013). Fundamental components of contemporary developmental systems theory include: (1) relations as developmental units, (2) reciprocal relations, and (3) plasticity.

First, developmental systems theory emphasizes that the relationship between context and organism is a primary focus of research. Lerner (2006) describes this shifting approach to developmental science: “the relations among levels and not on main effects of any level itself, as constituting the fundamental units of analysis of development” (p. 4). For example, developmental systems theory will attribute development to the interactions of multiple levels of organization that make up the complete human ecology, which includes both nature- and nurture-based factors (Gottlieb & Halpern, 2002; Lerner, 2006; Spencer et al., 2009). These range from genetic influences, to individual factors, to broader social contexts or cultural influences. For example, the development of depression in a child depends on genes (e.g., parent history of depression), exposure to adverse events, social interactions—or lack thereof—with peers, and the economic climate, all of which may influence the early detection and treatment of the disorder.

Second, levels of organization are intertwined and function in a reciprocal, or *coactional*, relationship (Gottlieb & Halpern, 2002; Lerner, 2006; Spencer et al., 2009). The levels of organization act on the individual (e.g., parenting style may influence child temperament) and the individual exerts an influence on the levels (e.g., child temperament may influence parenting style). The reciprocal relationship has been represented as individual ↔ context (Lerner, 2004).

Third, plasticity, or the ability for change, exists across the lifespan (Lerner, 2006). At all times, individuals and contexts are capable of changing developmental trajectories for better or worse. However, Lerner notes that “change in individual ↔ context relations is not limitless, and the magnitude of plasticity... may vary across the life span and history” (p. 3). Thus, intraindividual variation (i.e., within-person), along with interindividual (i.e., between-person) differences in intraindividual variation becomes a focus of research.

Given the uniqueness of any given individual’s developmental system and its influence on developmental trajectories, two important features stand out: *equifinality* and *multifinality* of development within a system (Cicchetti & Rogosch, 1996). Equifinality states that a variety of diverse conditions within any developmental system may result in the same outcome. Multifinality states that similar conditions may function differently within any developmental system and result in disparate outcomes. Thus, understanding the processes within a system *and* outcome—in the case of this study, internalizing psychopathological symptoms—becomes a primary goal of research. Cicchetti and Rogosch (1996) clarify by stating that “what might be considered error variance at the group level must be critically examined for understanding diversity in

process and outcome” (p. 598). For example, two children who grow up in physically abusive households may differentially develop, with one child but not the other exhibiting psychopathological symptoms. A typical cross-sectional study might place both children into one classification (i.e., abused) and determine that any differences in the outcome variable (e.g., psychopathology) not attributable to abuse classification or to other tested variables constitute error variance. In reality, many unexplained differences are likely due to meaningful but unmeasured intraindividual variability within each child’s unique developmental system over time. In other words, naturally occurring fluctuations in psychopathological symptoms may distort research findings when sources of intraindividual variability are not considered or accounted for. A study design that attempts to explain both inter- and intraindividual sources of variation in psychopathological outcomes following trauma is consistent with the principles of developmental systems theory and leverages its strengths.

Childhood Trauma

Childhood trauma is common (Little, Akin-Little, & Somerville, 2011). In a nationally representative sample, over 26% of Canadians retrospectively reported experiencing at least one traumatic event as a child (Colman et al., 2013). In a community sample, over 10% of individuals reported being abused as a child and the majority reported repeated or prolonged abuse (Collishaw et al., 2007). Another study suggested that one in five adolescents experienced the death of a close friend within the last year (Rheingold et al., 2004). What constitutes trauma is a topic of debate (Kennerley, 2012), but the *Diagnostic and Statistical Manual of Mental Disorders, Fifth*

Edition (DSM-V) defines criteria for posttraumatic stress disorder in children over age 6 as follows:

Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways: 1) Directly experiencing the traumatic event(s). 2) Witnessing, in person, the event(s) as it occurred to others. 3) Learning that the event(s) occurred to a close family member or close friends. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental. 4) Experiencing repeated or extreme exposure to aversive details of the traumatic event(s). (APA, 2013, p. 271)

Other studies have used the broader term, *adverse childhood experiences* (ACE), to describe trauma-like events. These can include psychological, physical, or contact sexual abuse and household dysfunctions, which are defined as exposure to substance abuse, mental illness, step/mother physical abuse, or criminal behavior (Felitti et al., 1998).

Others use the term *complex trauma* to define a similar construct, “the experience of multiple or chronic and prolonged, developmentally adverse traumatic events, most often of an interpersonal nature (e.g., sexual or physical abuse, war, community violence) and early-life onset” (p. 433; Spinazzola et al., 2005).

While the various definitions appear face valid, some aspects should be excluded. Experiencing household dysfunction would not likely be a traumatic event itself, but it is closely associated with other events that may be deemed traumatic. For example, having a parent with a mental illness is not a traumatic event, but any resulting psychological neglect may be deemed traumatic. For the purposes of the present study, *trauma* is

defined as any one-time or repeated exposure to physical, psychological, or sexual abuse or a death or near-death experience of violent or sudden nature that happened to the self, was witnessed directly, or happened to a close family member or friend.

Childhood trauma has frequently been linked to depression. Adults who experienced one or more adverse childhood events were more likely to be in a depressed mood for more than two weeks in the past year and to attempt suicide when compared to individuals who experienced no adverse childhood events (Anda et al., 2002; Felitti et al., 1998). The likelihood of these negative outcomes increased when the frequency of traumas increased (i.e., a *dose-dependent* relationship). Those who report at least two instances of childhood trauma are at increased risk for depression later in life (Colman et al., 2013). There is increased risk for a more severe onset of depressive disorder in adults who experienced childhood trauma and have a history of elevated depressive symptoms (Hovens, Giltay, Spinhoven, van Hemert, & Pennix, 2015). Moreover, adults who report severe childhood trauma, such as frequent and multiple types of abuse, are more likely to be chronically depressed (Wiersma et al., 2009).

Childhood trauma is also linked to anxiety disorders. Those who experienced a childhood traumatic event had a higher likelihood of current and lifetime posttraumatic stress disorder diagnoses (Wu, Schairer, Dellor, & Grella, 2010). These likelihoods increased with every additional traumatic experience. A history of sexual and physical abuse increases the risk of developing social anxiety disorder, panic disorder, and generalized anxiety disorder (Cougle, Timpano, Sachs-Ericsson, Keough, & Riccardi, 2010). A dose-dependent trend between trauma and anxiety symptoms has also been suggested (Hovens et al., 2010). Adults experiencing extreme childhood stress have

increased anxiety symptoms compared to those with no experiences (Chu et al., 2013). Many of the relationships between childhood trauma and later anxiety are stronger for those with comorbid depression or multiple anxiety diagnoses.

Children who experience trauma appear to have a poorer prognosis (i.e., they are more likely to develop psychopathology) than those who experience trauma at a later point in development (Chu et al., 2013; Zlotnick et al., 2008). In addition, adverse childhood events are associated with higher depressive symptoms in early adolescence, particularly during periods of high stress (Rudolph & Flynn, 2007). Internalizing symptoms in one study were higher for adolescents who reported potentially traumatic events as children, and these symptoms followed a similar pattern over time when compared to adolescents not reporting a trauma history (Whitson & Connell, 2016). However, these children had serious emotional dysregulation problems and were measured over a relatively short time (i.e., 36-months). Another study focused exclusively on the onset of separation anxiety disorder and did not investigate variability in symptoms *within* childhood and adolescence (i.e., the focus was a categorical diagnosis; Silove et al., 2015). No long-term studies have yet examined patterns of progression over time of depression or anxiety symptoms in children with and without histories of trauma. The development of children who experience trauma deserves particular attention due to the potentially enduring negative consequences.

Gender Differences

Gender is linked to experiencing specific forms of trauma. Girls are more likely to experience sexual abuse than boys (Dykman et al., 1997; Statistics Canada, 2013), and trauma experienced by women is more likely to be part of a class marked by sexual

assault. Men's trauma is more likely to be part of a class marked by non-sexual violence (Ballard et al., 2015).

Gender has also been linked to differences in the severity of post-trauma consequences; however, the results are equivocal. Sexually abused boys experienced worse psychopathological symptoms—internalizing and externalizing— than did girls (Dykman et al., 1997). Additionally, research has shown that being male increases the risk of externalizing behavior, after controlling for trauma history (Danielson, et al., 2009). Other research suggests that outcomes are worse for girls; trauma decreases girls' ability to deal with high levels of stress, but not boys' (Rudolph & Flynn, 2007). Still other studies report no differences in the onset of psychopathology in male or female participants (Collishaw et al., 2007), which add to the mixed findings. Thus, the association between gender and specific types of trauma must be considered as differential effects may exist and need clarification.

Internalizing Psychopathology

The abundance of research investigating developmental sequelae after experiencing trauma suggests childhood trauma increases the risk of impaired psychological functioning (e.g., Ballard et al., 2015; Brent, Melhem, Donohoe, & Walker, 2009; Collishaw et al., 2007). Two common forms of internalizing psychopathology associated with traumatic experiences are major depressive and anxiety disorders. These disorders present impairments in functioning in individuals that may require professional intervention and treatment.

There has been criticism regarding traditional diagnostic models of internalizing psychopathology, specifically with regards to anxiety and depressive (i.e., emotional)

disorders (Newby et al., 2015). For example, it is argued that anxiety and depression have the same underlying biological, cognitive, and social etiological factors and, thus, can be treated similarly. Discrete diagnostic systems can obfuscate assessment, diagnosis, and treatment; this may be reflected by the high comorbidity between internalizing disorders. More recently, *transdiagnostic* treatment has an interest of researchers and clinicians; this approach assumes that mental disorders have common etiological underpinnings, which are comprising of biological, psychological, and social factors, despite their various manifestations (McEvoy et al., 2009). In addition, treatment protocols could be unified and follow similar procedures. Transdiagnostic treatment has been shown to be effective for depressive and anxiety symptoms, and quality of life (Newby et al., 2015). Despite the noted benefits, a transdiagnostic approach has potential disadvantages and needs further evaluation (McEvoy, Nathan, & Norton, 2009). Although the transdiagnostic approach is growing and may prove to be a viable replacement to discrete-diagnosis, this study will focus on depression and anxiety disorders separately based the availability of traditional instruments that measure internalizing symptoms as separate domains.

Major depressive disorder (MDD) is marked by feelings of hopelessness and despair, a loss of interest in regular activities, problems with sleep and appetite, fatigue, and impairments in cognitive functioning (e.g., problems making decisions or focusing) (APA, 2013). Major depression is sometimes accompanied by suicidal ideation or attempts. The prevalence of MDD in a sample representative of the United States is 6.6% within 12-months (Kessler et al., 2003), while a sample representative of Canada had a slightly lower rate of 4.8% (Rhodes, Bethell, & Bondy, 2006; Statistics Canada,

2013). The prevalence in children is suggested to be less than 3%, but this number increases during adolescence (Fleming & Offord, 1990). The typical age of onset is 15 years old, with girls having a slightly earlier onset than boys (Lewinsohn, Clarke, Seeley, & Rohde, 1994). In addition, a relapse into a depressive episode is common, with 5% of adolescents relapsing within six months and 33% within four years. MDD is likely to have an earlier onset if it is comorbid with an anxiety disorder (Lewinsohn et al., 1994).

Anxiety disorders are a cluster of maladaptive cognitions, thoughts, or behaviors marked by excessive fear and anxiety (APA, 2013). While fear is the emotional response to an imminent danger or threat and usually accompanied by autonomic arousal, anxiety is the expectation of a future threat, which is usually accompanied by tension and behavior adjustments to avoid the threats. The experienced anxiety is maladaptive in that afflicted individuals overestimate the dangers of a situation and underestimate their ability to deal with it (Clark & Beck, 2010). Some anxiety disorders typically develop in childhood (e.g., Separation anxiety disorder; APA, 2013). In adolescence, specific and social phobias are the most prevalent at 20% and 8.6%, respectively, and girls are more likely than boys to develop these phobias (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). The lifetime prevalence of *any* anxiety disorder in adolescents aged 13- to 17-years old is 32.4% (Kessler et al., 2012). Children with attention deficit/hyperactivity disorder are at increased risk for developing MDD or an anxiety disorder (Kessler et al., 2006).

Attention Deficit/ Hyperactivity Disorder

Children with a diagnosis of Attention Deficit/ Hyperactivity Disorder (*ADHD*; Kessler et al., 2006) are at increased risk for comorbid mental illness. ADHD is a

Table 1
Symptom Criteria for ADHD diagnosis.

Inattention	<ul style="list-style-type: none"> - Careless errors or low attention to detail - Difficulty maintaining attention to tasks/activities - Often fails to listen when spoken to - Fails to follow instructions or finish tasks (e.g., school, chores) - Difficulty organizing tasks - Avoids, dislikes, or is hesitant to perform tasks that require maintained concentration - Often loses items - Easily distracted - Forgetful (e.g., performing chores)
Hyperactivity/ Impulsivity	<ul style="list-style-type: none"> - Fidgety - Failure to maintain in seat when required - Inappropriate actions (e.g., running/climbing) - Failure to be quiet during leisure activities - Uncomfortable with being still for extended periods - Excessive talking - Difficulty waiting turn during conversations - Difficulty waiting turn (e.g., lines) - Interruptive/ intrusive towards others

Note: adapted from DSM-5 (APA, 2013).

neurodevelopmental disorder marked by a pattern of inattention and/or hyperactivity that significantly impairs the functioning of the individual (APA, 2013). Classification for people under age 17 requires at least six inattention symptoms or six hyperactive or impulsive symptoms. When both criteria are met for at least six months, the diagnosis is *combined presentation*. If only one set of the six symptoms are met, the diagnosis is labeled as *inattentive* or *hyperactive/impulsive* presentation, respectively. See Table 1 for a listing of the relevant symptoms. In addition to the noted criteria, DSM-5 requires that

the individual's symptoms i) manifest prior to age 12, ii) persist across settings (e.g., at home and at school), iii) interfere with quality of life or functioning (e.g., inability to perform in school/work), and iv) are not attributable to other disorders. Studies have estimated that ADHD occurs in 6.6% of children and 2.6% of adults (Kessler et al., 2006). The Canadian prevalence in young children is estimated to be 2.6% and increasing (Brault & Lacourse, 2012). The estimated worldwide prevalence rate is over 5% (Polanczyk, de Lima, Horta, Biederman, & Rohdes, 2007). Recent estimates based on the sample of interest in the present study suggest that approximately 50% of those with a childhood diagnosis of ADHD continue to meet criteria in adulthood (Roy et al., 2016), while others estimate that number to be 70% (Sibley et al., 2012a). However, there is evidence of a substantial struggle with ADHD-related symptoms in adolescence and adulthood for those diagnosed in childhood but who no longer meet ADHD diagnostic criteria (Biederman, Mick, & Faraone, 2000; Sibley et al., 2012a). For example, 55-60% of adolescents with a childhood ADHD diagnosis have clinically elevated levels of impairment relevant to ADHD, but only 10-20% of these adolescents meet a formal diagnosis of ADHD (Sibley et al., 2012b). While these numbers vary depending on the type of informant, there is a consistent discrepancy between diagnosis and impairment.

ADHD is associated with impaired functioning in a variety of domains. Barkley's (1997) unified theory of ADHD describes the purported deficits in working memory, self-regulation, reflection, and reconstitution (e.g., verbal skills), and research supports his position. Children have been shown to have poorer receptive language skills—a form of language intelligence—and working memory compared to typically developing peers (Hutchinson, Bavin, Efron, & Sciberras, 2012). Other research findings suggest that

young girls with ADHD score significantly worse, although still within the normal range, on measures of cognitive ability (i.e., intelligence and achievement test) when compared to non-diagnosed peers (Hinshaw, 2002). Adolescents with elevated ADHD symptoms have also been found to have lower scores on a measure of overall functioning, compared to non-ADHD controls (Hurtig et al., 2007). Similar deficits are suggested in the domain of social cognition (Bora & Pantelis, 2016).

An ADHD diagnosis carries comorbidity risks. Although children with ADHD most commonly have comorbid Oppositional Defiant Disorder or Conduct Disorder at a younger age (Cuffe et al., 2015), an ADHD diagnosis in young adulthood is associated with an increased risk of comorbidity with depression (Turgay & Ansari, 2006). In young girls aged 6- to 11-years old, there is a strong risk of comorbid depressive symptoms compared to non-diagnosed peers (Hinshaw, 2002). A similar but less severe trend is associated with anxiety disorders (Kessler et al., 2006). An ADHD diagnosis plus anxiety or depression comorbidity presents immediate clinical concerns; treatment must be adjusted to account for symptoms and treatment resistance for both disorders. In addition, treatment becomes increasingly vital as suicide rates are higher among individuals with a comorbid diagnosis when compared to those with major depression alone (Turgay & Ansari, 2006). Understanding the etiology of comorbid psychopathology in individuals with ADHD is of paramount concern.

Early childhood traumatic experiences have been linked to ADHD symptomatology. Children with ADHD and a history of parental abuse had higher impulsivity and inattention scores compared to children with ADHD without the abuse history (Becker-Blease & Freyd, 2008). Other research showed that adopted children

with ADHD symptoms are more likely to have a history of preadoption abuse or neglect compared to adopted children with no symptoms (Simmel, Brooks, Barth, & Hinshaw, 2001). Similarly, girls diagnosed with combined type ADHD were more likely to have a history of childhood abuse compared to those with no diagnosis (Hinshaw, 2002). Additionally, children with ADHD had higher rates of physical and sexual maltreatment compared to children with an adjustment disorder (Ford et al., 2000).

Understanding how children with ADHD respond to adversity is of concern. Deficits in cognitive functioning may be linked to a poorer ability to cope, which is an important response to adversity. It is important to track the longitudinal responses to trauma in children with ADHD, as these children may present important differences in the development of internalizing psychopathology. No study to date has investigated these associations.

The Current Study

This study will investigate the association between childhood trauma and depression and anxiety as it unfolds into adolescence, and how these associations differ for children with and without ADHD (See Figure 1). Hypotheses for the present study are as follows:

H1) Childhood trauma will be associated with higher levels and faster increases in depression and anxiety (internalizing symptoms) through mid-adolescence,

H2) ADHD diagnosis in childhood will be associated with higher levels and faster increases in internalizing symptoms through mid-adolescence, and

H3) Longitudinal associations between trauma and internalizing symptoms will be stronger for those with a childhood ADHD diagnosis.

Figure 1

Hypotheses between trauma, ADHD, and internalizing symptoms.

Method

Participants and Procedure

Data for the present study were drawn from the Multimodal Treatment Study of Children with Attention-Deficit/Hyperactivity Disorder (MTA), a multisite clinical trial study that assessed long-term treatment efficacy in children diagnosed with ADHD. Starting in 1992, 579 children diagnosed with ADHD combined type and aged 7 to 9.9 years old were randomly assigned to one of four conditions (Medication Treatment, Behavioral Treatment, Combined Medication and Behavioral Treatment, or Community Comparison) across six sites in the U.S. and Canada. The active treatment phase was 14-months in duration. A longitudinal follow-up study was initiated 10 months after treatment concluded, and at that time 289 comparison children (the Local Normative

Comparison Group; LNCG), were recruited from the same schools as the original children and were matched on age and gender. Both samples were followed approximately biennially for 16 years after original study randomization.

The ADHD diagnosed children were 19.7% females, while the comparison group were 19.4% females. The ethnic composition of the sample of children with ADHD was 75.6% Caucasian, 12.5% Black, 4.9% Hispanic, and 7.0% mixed/ other. The corresponding ethnic composition of the LNCG sample was 65.6%, 10.7%, 13.9%, and 9.8%, respectively. Estimated annual income, before taxes, was distributed as follows for the children with ADHD and the LNCG: 12.2% and 4.2% below \$20,000, 77.7% and 84.7% between \$20,000 and \$75,000, 9.1% and 10.1% above \$75,000, with 1% in both groups missing income data.

Some children involved in the study presented with comorbid diagnoses at baseline. Although 31.8% of children had only an ADHD diagnosis, 13.6% presented a comorbid anxiety disorder, 29.5% presented a comorbid oppositional defiant disorder/conduct disorder, 0.4% presented a comorbid mood disorder, and 24.7% presented some combination of ADHD and two or more other disorders (Jensen et al., 2001).

Measures

The MTA study includes longitudinal measures of trauma, depression, and anxiety.

Depression. Depression was measured using the Children's Depression Inventory (CDI; Kovacs, 1992) at the 2-, 3-, 6-, 8-, and 10-year assessments. The CDI is a 27-item instrument that measures depressive symptoms. Each item asks the child to select a

sentence that best describes him or her in the past four weeks. Sentences differ by describing the severity of depressive symptoms on a 3-point scale (e.g., how often a child feels sad, has trouble sleeping, or feels like crying). Potential responses are 0 (e.g., *I am sad once in a while*), 1 (e.g., *I am sad many times*), and 2 (e.g., *I am sad all the time*). Scores across items are summed, with higher scores indicating more depressive symptoms. The CDI has good reliability (Sun & Wang, 2015) and criterion validity (Knight, Hensley, & Waters, 1988). In addition, the CDI can differentiate children with major depressive or dysthymic disorder from children with a different or no psychiatric diagnosis (Kovacs, 1992). Generally, a total score of 19 is used as a cut-off for depression screening (Woodley & Curtis, 2007), although the subscales *negative mood*, *interpersonal problems*, *ineffectiveness*, *anhedonia*, and *negative self-esteem* are available. The CDI total score was used for this study as an overall indicator of depressive symptoms. The internal consistency of the CDI for this study was measured using Cronbach's alpha for each MTA wave, and ranged from $\alpha = .84 - .91$.

Anxiety. Anxiety was measured using the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997) at the 2-, 3-, 6-, 8-, and 10-year assessments. This self-report measure contains 45 items that measure the severity of anxiety symptoms. Individuals respond on a 4-point scale indicating how well the item describes themselves over the past month. For example, "I worry about other people laughing at me", with responses ranging from 1 (*Never true*) to 4 (*Often true*). Scores are summed to identify anxiety symptoms, where higher scores indicate more anxiety symptoms. MASC total scores have been used as an indicator of overall anxiety symptoms in previous research (Hinshaw, 2002). Others suggested that the MASC total

score may not be beneficial to clinicians hoping to screen for specific anxiety disorders, although it is useful for screening between the general population and those referred for anxiety problems (van Gastel & Ferdinand, 2008). The MASC total score was used in this study as an overall indicator of anxiety symptoms. The MASC demonstrates good convergent validity with Spence's Anxiety Scale for Children and a moderate test-retest reliability of $r = .70$ (Baldwin & Dadds, 2007). The internal consistency of the MASC for this study was measured using Cronbach's alpha at each MTA wave and ranged from $\alpha = .90 - .92$. This is comparable to the level of 0.89 found in a psychometric evaluation of the MASC (Baldwin & Dadds, 2007).

Traumatic Events. Traumatic events were measured using the Kiddie Post Traumatic Stress Scale (March, n.d.) at the 2- and 3-year assessments. Children were asked about their lifetime occurrence of traumatic events, and responded to 15 prompts of events that may have happened "to me" or "someone I know". Examples of events are badly beaten; bad car, boat, bike, train, or plane accident; and suicide attempt or died from suicide. When multiple events were identified, children indicated which, if any, was the *worst thing* that ever happened. Some studies have used the total number of traumatic events that have happened (e.g., Wu et al., 2010), others have differentiated between interpersonal traumas (e.g., abuse, accidental death of a loved one) versus other types (e.g., natural disaster; Wamser-Nanney & Vandenberg, 2013), and sexual or physical abuse versus other types (e.g., death of a loved one; Ballard et al., 2015).

In the present study, two trauma variables were created to understand traumatic experiences: (1) *natural traumas* describe traumatic experiences that are the result of some natural disaster or other naturally occurring event: experiencing a terrible fire or

explosion; chemical or other deadly poisoning; bad storm, flood, tornado, hurricane, or earthquake; bad car boat, bike, train, or plane accident; got sick and almost died or died; and other very bad accident; (2) *interpersonal traumas* were: being badly scared or hurt by a gang or criminal; badly beaten; shot or stabbed; taken away from one's family; suicide attempt or died from suicide; kidnapped or held captive; and saw something terrible happen to a stranger. These versions of trauma variables were chosen based on studies suggesting that interpersonal trauma has distinct psychopathological implications when compared to other trauma types (e.g., Chu et al., 2013; Ford et al., 2011; Zlotnick et al., 2008). Possible total scores ranged from 0 to 4 for natural trauma and from 0 to 3 for interpersonal trauma. Given extreme sparseness at higher numbers of traumas, each variable was restricted to three categories: 0 (*no traumatic events*), 1 (*one traumatic event*), and 2 (*two or more traumatic events*). This coding strategy permitted inspection of a dose-dependent relationship between trauma occurrences and psychopathology, as has been found elsewhere (e.g., Colman et al., 2013). Lastly, the KPTS gives the option to describe whether an event happened to "someone I know". It was determined that including this option may underestimate the effects of traumatic experiences on psychopathological outcomes because: 1) the KPTS does not indicate if the event that happened to "someone I know" was witnessed in person and 2) the KPTS does not indicate the relationship of "someone I know", which is a required component of the definition of trauma (i.e., a close family member or friend). Thus, the traumas included in this study are those that were directly experienced by the child.

Demographic Information. Demographic information was collected at intake for the larger MTA study. For all categorical demographic variables, weighted effects coding

was used to permit regression coefficients to represent comparisons between each target category and the marginal mean depression and anxiety scores. There are $C-1$ weighted codes for each demographic category, where C is the number of categories within a variable. Weighted effect codes were constructed by selecting a base or reference group for each categorical variable; all non-base categories are assigned a code of “1” for their respective portion of the $C-1$ codes, while the base category is assigned a code of “ $-n_c/n_b$ ”, where n_c is the sample size for the non-base category and n_b is the sample size of the base category. Weighted effects codes are centered and less correlated than non-weighted coding schemes (see Cohen, Cohen, West, & Aiken, 2003 for a comprehensive review of weighted effects coding). Codes for *biological sex* were 1=Female, -0.248=Male. Child *race/ethnicity* was coded with three variables to represent four categories. Black, Hispanic, and Mixed/other ethnicity were contrasted with the sample average (codes summarized in Table 2). *Household advantage* was a composite variable based on parents’ education and household composition. An advantaged household is defined as a two-parent household (parents married or living common law) in which one or both parents have a college or university degree. A disadvantaged household is defined as a single-parent household (e.g., divorced or never married) in which no parent is college- or university-educated. Any other combination was referred to as neither advantaged nor disadvantaged. These three groups were coded across two variables using the latter as the reference category (codes summarized in Table 3). Family history of depression or anxiety disorders were also used as control variables in the analysis. Lastly, child history of depression or anxiety disorders was also obtained as a control variable. Child diagnosis was assessed at the baseline measurement ($n_{lncg} = 244$, $n_{adhd} =$

Table 2
Effects coding for ethnicity.

Category	Coefficient		
	γ_{ib}	γ_{ih}	γ_{io}
Black	1	0	0
Hispanic	0	1	0
Other	0	0	1
White	-0.252	-0.170	-0.161

Table 3
Effects coding for household advantage.

Category	Coefficient	
	γ_{ia}	γ_{id}
Advantaged	1	0
Disadvantaged	0	1
Neither	-1.059	-0.437

461) or, in cases where there was no baseline measurement, the 1-year follow-up ($n_{lncg} = 0$, $n_{adhd} = 9$). These mental illness history variables were coded dichotomously, with 0 = no history and 1 = a history.

Data Preparation and Analysis Strategy

All MTA data are organized in “long” format (Singer, 1998), with multiple records per participant representing each wave of assessment. Time-invariant covariates, such as ADHD diagnosis and biological sex, appeared in the dataset as the same score or variable code across waves for a single participant. Individuals’ ages were calculated using the measurement of *days from baseline*, which is included in each MTA data file, added to their age at baseline. Participants’ ages were used as a continuous longitudinal measurement of time rather than wave of assessment (e.g., 36-month follow-up). Age was centered at 13 years old based on the abundance of data points close to this age and its expected developmental significance as a time at which multiple life events may be

experienced (school transitions; going through puberty) that may act as catalysts for change in internalizing psychopathology.

The MTA data files are organized separately by study measure (e.g., CDI and MASC are separate data files). Files were compiled into one composite data file by matching observations by participant ID and assessment wave, and relevant scale scores were computed. Data were screened for outlying observations. Outcome variables (i.e., depression and anxiety) were screened for outlying observations using the guideline that semistudentized residual scores greater than 4 are considered outliers and continuous predictor variables were screened for outlying observations using the guideline that leverage values greater than 0.5 are considered outliers (Kutner, Nachtsheim, Neter, & Li, 2005). If cases for depression or anxiety measures met criteria as an outlier on both, as identified by conducting regression analyses on the purported full model for each assessment wave, DFFITS were observed to determine their influence on the model. For depression, eight observations across seven participants were identified as outliers and influential for depression across all assessment waves. For anxiety, six observations across four participants were identified as outliers and influential for anxiety across all assessment waves. However, for both depression and anxiety, results of models including and excluding potentially influential observations showed virtually identical model parameters and patterns of significance test results; thus, these observations were retained in subsequent analyses.

Depression and anxiety symptoms were plotted over age to determine the best functional forms of change over time for each construct. While depression appeared to change in a relatively linear fashion, anxiety appeared to decline over age nonlinearly in a

gradually decelerating trajectory, suggesting that a quadratic rate of change might fit the data well. Visual inspection of depression and anxiety scores separately for ADHD versus LNCG groups and separately by level of trauma supported the planned hypothesis tests.

Multilevel modelling (MLM) was used for data analysis. MLM—also known as hierarchical linear modeling, random coefficients regression, or mixed-effects regression—explicitly accommodates the nested structure of a data set such as multiple observations obtained from a single participant. Observations within a cluster, or nest, violate the assumption of independence of error terms from an ordinary least squares regression model (OLS), making OLS regression inappropriate (Nezlek, 2008). MLM is analogous to OLS, such that a set of independent variables are used to predict an outcome, or dependent variable. However, to account for dependent variation within clusters, MLM allows for cluster-level equations to be predicted. Both fixed and random effects are estimated through MLM. Fixed effects are regression coefficients that estimate population parameters (i.e., are the associations significantly different from zero?) and random effects estimate within- and between-person deviations (Nezlek, 2003). These deviation estimates are represented as variance parameters. Importantly, MLM allows for an interpretation of the variance that occurs within- *and* between-clusters. Within-cluster effects can be model as a level-1 equation and between-cluster effects as level-2 equations. In MLM, independent variables at level-1 are modeled as dependent variables at level-2 (Nezlek, 2008). Both equations can be simplified (i.e., reduced), which separates deviations into those within- and between-clusters. Variables can be added at either level of the model, which depends on if they vary within or

between a cluster. For example, in a longitudinal study in which daily measurements of mood are clustered within individuals, sex could be a level-2 predictor variable (i.e., does not change for each person's daily observation) and daily caloric intake could be a level-1 predictor variable (i.e., changes for each person's daily observation). In 2-level longitudinal designs, these are also known as time-invariant and time-variant predictors, respectively. This technique is appropriate for addressing many research questions informed by a developmental systems perspective (Marshall, 2013). Multilevel models accommodate repeated measures and easily permit tests of interactive relations among variables over time. Importantly, MLM accommodates different kinds of outcome distributions (e.g., positively skewed) in the same way as OLS regression analysis and employs full information maximum likelihood estimation (FIML), which accommodates cases with missing waves of assessment, eliminating the need for listwise deletion. Typically, MLM involves model building to test the suitability of a set of predictors and to determine the proportion of random effects explained by predictors.

Developing the Model

This study will test a two-level model (i.e., level-1 as within person differences, level-2 between person differences) which allows both the intercept and slope to vary across individuals. The model with no predictors, the *unconditional model*, represents an individual's depression or anxiety score (i.e., outcome variable) and is expressed as:

Level-1:

$$y_{ti} = \beta_{0i} + r_{ti}$$

where y_{ti} is the outcome for person i at time t . Thus, a depression or anxiety score at time t for person i is comprised of person i 's mean and a deviation from that person mean at time t , expressed as r_{ti} . The unconditional model allows the intercept (β_{0i}) to randomly

vary across individuals. The variance in the random intercept is expressed in the level-2 equations as:

Level-2:

$$\beta_{0i} = \gamma_{00} + \mu_{0i}$$

where β_{0i} is composed of the grand mean (γ_{00}) and a deviation from the grand mean for individual i , (μ_{0i}). Thus, the combination of level-1 and -2 equations allow a unique understanding of both within- and between-person effects. The full model is expressed as:

Full model:

$$y_{ti} = \gamma_{00} + \mu_{0i} + r_{ti}$$

Thus, an individual's depression (or anxiety) score at any time-point is a composite of the grand mean depression score (γ_{00}) plus an average deviation for person i from the grand mean (μ_{0i}), plus a time-specific deviation for person i at time t (r_{ti}). The following example expands these equations to include both time-varying (i.e., level-1; age) and time-invariant (i.e., level-2; ADHD diagnosis) predictors. The level-1 equation can be expressed as:

Level-1:

$$y_{ti} = \beta_{0i} + \beta_{1i}(AGE) + r_{ti}$$

Thus, a depression or anxiety score comprises a person's mean depression score across all times (β_{0i}), plus the effect of age (β_{1i}), plus any further deviation for person i at time t (r_{ti}). However, depending on the functional form of internalizing symptoms over time, as determined by visual inspection of the symptoms over age plots, an additional predictor variable may be added to account for nonlinearity (e.g., $y_{ti} = \beta_{0i} + \beta_{1i}(AGE) + \beta_{2i}(AGE)^2 + r_{ti}$). It is important to note that both the intercept, β_{0i} , and slope, β_{1i} , can

randomly vary across individuals. These will be represented by two level-2 equations, expressed as follows:

Level-2 (Intercept):

$$\beta_{0i} = \gamma_{00} + \gamma_{01}(ADHD) + \mu_{0i}$$

Level-2 (Slope):

$$\beta_{1i} = \gamma_{10} + \gamma_{11}(ADHD) + \mu_{1i}$$

where an person's slope and intercept are comprised of the mean intercept (γ_{00}) and slope (γ_{10}) for the complete sample, plus the effects of ADHD diagnosis on the intercept (γ_{01}) and slope (γ_{11}), plus any further deviation for person i , from the mean intercept (μ_{0i}) and slope (μ_{1i}). Thus, the full model would be expressed as:

Full Model:

$$y_{ti} = \gamma_{00} + \gamma_{01}(ADHD) + \gamma_{10}(AGE) + \gamma_{11}(ADHD)(AGE) + \mu_{1i}(AGE) + \mu_{0i} + r_{ti}$$

where a depression or anxiety score for person i at time t is comprised of the grand mean (γ_{00}), plus the fixed effects of age (γ_{10}), ADHD diagnosis (γ_{01}), and ADHD interaction with age (γ_{11}). In addition to the fixed effects, added are the deviation for person i at time t (r_{ti}), of person i from the average intercept (μ_{0i}), and the deviation of person i from the average rate of change over age (μ_{1i}).

In this study, model building started with an unconditional model (i.e., a simple random intercept model for depression/anxiety with no predictor variables) to determine the proportion of total variance that is accounted for by between-person differences, or the *intraclass correlation*, ρ , which can be expressed as:

$$\rho = \frac{\tau_{00}}{(\sigma^2 + \tau_{00})}$$

where τ_{00} is the between-person variance (i.e., variance of μ_{0i}) and σ^2 is the intraindividual or within-person variance (i.e., variance of r_{ti}). Second, age was included

in the model as a level-1 predictor to determine the unconditional growth model. Third, a conditional model was investigated where history of trauma is added at level-2 to investigate its effects on the associations between internalizing symptoms and age. Fourth, ADHD is added to the model to determine its associations with internalizing symptoms and age. Lastly, interactions between ADHD and trauma are added to identify any potential moderating effects of trauma and ADHD on the progression of internalizing symptoms over time.

Missing Data

Participants with missing data on the dependent, but not the predictor, variable(s), can be included under FIML (Nezlek, 2003). Thus, of the original 868 participants that were considered for inclusion in this study, 95 participants had no measure of depression and anxiety and 59 had missing data on a level-2 (i.e., time-invariant) variable. The final sample size is 714. Table 4 presents the demographic characteristic of the final sample. Of these 714 children, 88, or 12.3%, (13.8% of ADHD children and 9.4% of LNCG children) were missing at least one wave measurement of depression and anxiety, respectively, from among the full set of assessments for which they were eligible. Comparisons were made between participants included in the analyses (i.e., possessing complete predictor data and at least one outcome variable) versus excluded (i.e., missing at least one predictor or had no outcome data), and between those with complete depression/anxiety data versus those missing depression/anxiety scores at one or more waves (Note: these results were identical for depression and anxiety and, thus, are reported as single results).

Included versus excluded children

Table 4
Demographic characteristics of sample by ADHD diagnosis.

	ADHD (<i>n</i> = 470)	LNCG (<i>n</i> = 244)
	<i>N</i> (% of group)	<i>N</i> (% of group)
Female	92 (19.57)	50 (20.49)
Ethnicity		
White	296 (62.98)	160 (65.57)
Black	89 (18.94)	26 (10.66)
Hispanic	35 (7.45)	34 (13.93)
Other	50 (10.64)	24 (9.84)
Advantaged household	189 (40.21)	114 (46.72)
Disadvantaged household	91 (19.36)	34 (13.93)
Neither advantaged or disadvantaged household	190 (40.43)	96 (39.34)
Child previously diagnosed with depression	12 (2.55)	1 (0.41)
Child previously diagnosed with anxiety disorder	67 (14.26)	18 (7.38)
History of family depression	181 (38.51)	71 (29.10)
History of family anxiety disorder	117 (24.89)	32 (13.11)

Note: Percent is within-group (e.g., ~29% of LNCG children had a family history of depression).

Comparisons between included and excluded children were performed on 22 baseline variables to evaluate the assumption that data were *missing at random* (Baraldi & Enders, 2010). These comparisons were performed separately by ADHD status. Only 2 of 22 baseline variables differed for ADHD-diagnosed children who were included versus excluded. Excluded children were more likely to be part of a family with lower income or public assistance. LNCG children who were included versus excluded differed on one baseline variable: excluded children were more likely to have experienced divorce. Excluded participants had missing predictor variable data; children from families with low income or using public assistance may have additional barriers regarding research participation or attrition (e.g., lack of transportation, more likely to change residences), which may manifest as more missing variables. For LNCG children, divorce may present similar circumstances such as increased financial strain and stress, or an increased likelihood of residential relocation. However, there is no reason to assume that the likelihood of missing data points are *related* to the missing values themselves.

Thus, the data appears to fulfill the missing at random assumption. This assumption posits that “missingness is related to other measured variables in the analysis model, but not to the underlying values of the incomplete variable” (Baraldi & Enders, 2010, p.7).

Complete versus incomplete repeated measures

A multilevel model was analysed to determine if individuals with and without missing waves of assessment differed on depression or anxiety scores after controlling for ADHD diagnosis. Depression ($t(711) = 0.87, p = .3836$) and anxiety scores ($t(711) = 1.05, p = .2960$) did not differ for participants with complete versus incomplete data. In addition, comparisons between individuals with complete versus incomplete data were performed on 22 baseline variables to ensure that data fit the assumption to be missing at random. These analyses were performed separately by ADHD status. Seven of 22 baseline variables differed for ADHD-diagnosed children with complete versus incomplete depression or anxiety data. Those with missing data were more likely to be part of a family with lower income; have public assistance, welfare, or social security income; have younger biological mothers; be a younger school-level grade on study recruitment; to be male; and be expelled. In addition, ADHD children with missing wave data were less likely to have a father with mental health problems, but 20% of respondents did not have data available on this variable. LNCG children with complete versus incomplete data differed on 5 of 22 baseline variables. Those with missing data were more likely to be part of a family with lower income; have public assistance, welfare, or social security income; have younger biological mothers; be expelled; and have repeated a grade. Most socio-demographic variables tested were the same for children with complete and incomplete data. The differences that were observed between

children with and without missing waves are social adversity variables, which are partially accounted for in the “household advantage” variable used for in this study. In addition to the points listed in the previous section, the fact that most baseline differences were disparate for ADHD versus LNCG children suggests that these differences were random and, likely, has no systematic relation to the missing values themselves. Again, the data appears to fulfill the missing at random assumption (Baraldi & Enders, 2010).

Results

Descriptive statistics of mean depression and anxiety scores are presented in Table 5. Symptom scores are also broken down by wave for convenience. A correlation matrix of variables of interest are presented in Table 6. Approximately 16% of children experienced one interpersonal trauma ($n_{\text{lncg}} = 34$, $n_{\text{adhd}} = 78$) and 2% experienced two or more interpersonal traumas ($n_{\text{lncg}} = 0$, $n_{\text{adhd}} = 16$). Approximately 24.4% of children experienced one natural trauma ($n_{\text{lncg}} = 61$, $n_{\text{adhd}} = 112$) and 10.9% experienced two or more natural traumas ($n_{\text{lncg}} = 21$, $n_{\text{adhd}} = 57$). Children in the ADHD group were more likely to have a past diagnosis of depression ($\chi^2(1) = 4.13$, $p = .044$) or anxiety ($\chi^2(1) = 7.25$, $p = .005$) and a family history of depression ($\chi^2(1) = 6.23$, $p = .013$) or anxiety ($\chi^2(1) = 13.49$, $p = .0002$). Over 50% of children with a history of depression also had a history of anxiety. Lastly, children with ADHD were more likely to have experienced interpersonal trauma $\chi^2(1) = 4.15$, $p = .041$, but not natural trauma $\chi^2(1) = 0.39$, $p = .533$.

Depression

Covariates and Skewness

The CDI score distribution was, not surprisingly, positively skewed. All analyses were repeated with a log transformation such that the new CDI score was equal to the

Table 5
Descriptive statistics for depression and anxiety symptoms.

	Wave					
	2-year	3-year	6-year	8-year	10-year	Total
Anxiety	2.27 (0.44)	2.56 (0.38)	1.92 (0.40)	1.84 (0.38)	1.88 (0.39)	2.03 (0.43)
<i>n</i>	708	675	618	521	194	2716
Depression	0.22 (0.22)	0.18 (0.19)	0.21 (0.22)	0.25 (0.27)	0.24 (0.28)	0.21 (0.23)
<i>n</i>	708	675	617	518	194	2712

Table 6
Descriptive statistic and correlation matrix of select variables.

	Int. Symp.	Interpersonal	Natural	ADHD	Sex
Int. Symp	-	.078***	.068***	-.010	.131***
Interpersonal	.156***	-	.217***	.086***	-.099***
Natural	.071***	.219***	-	.056**	-.074***
ADHD	.105***	.088***	.055**	-	-.003
Sex	.048*	-.098***	-.073***	-.002	-

Note: Int. Symp. = Internalizing symptoms. Below the diagonal are for depression and above are for anxiety. * denotes $p < .05$. ** denotes $p < .01$. *** denotes $p < .001$.

natural log of the old CDI score plus one (i.e., $\log[\text{CDI}+1]$; this method retains a “0” point). No differences in significant fixed or random effects emerged; thus, the skewed data do not appear to disguise any relevant effects and allows for results to be interpreted in the CDI’s original metric.

An *unconditional* random intercept model was analysed to determine the amount of variability in depression scores between and within persons, $\chi^2(1) = 284.11$, $p < .0001$, $-2LL = -581.5$. The grand mean depression score at age 13 across all children was an average CDI item score of 0.216. Thus, the average depression score was relatively low, as the CDI scale ranges from 0-2. The intraclass correlation in the unconditional model for depression was:

$$\rho = \frac{\tau_{00}}{(\sigma^2 + \tau_{00})} = \frac{0.016}{(0.037 + 0.016)} = 0.302$$

This indicates that approximately 30.2% of the variability in depression scores can be attributed to between-persons differences, while 69.8% of the variability in depression is within-person.

Model 2 incorporates *age* as a predictor variable (centered at 13 years old) to test the rate of linear growth (slope) in depression scores. The unconditional growth model was an improvement from the unconditional model, $\Delta\chi^2(2) = 138.37, p < .0001, -2LL = -726.4$. The grand mean depression score at age 13 was an average CDI score of 0.215. There was a small, but positive, association with age and depression scores, $\gamma_{10} = 0.005$ ($SE = 0.002$), $p = .0058$. Each one year increase in age predicted an increase of 0.005 in depressive symptoms reported. Unexpectedly, there was a 12.0% increase of intercept variance (i.e., τ_{00}). There was approximately a 24% reduction in individual error when age was included as a predictor of depression scores, which is expressed as:

$$\begin{aligned} \text{Reduction in error} &= \frac{(\text{Model 1 } r_{ij} - \text{Model 2 } r_{ij})}{\text{Model 1 } r_{ij}} = \frac{(0.03669 - 0.02784)}{0.03669} \\ &= 0.2412 \end{aligned}$$

A quadratic growth model was considered; however, visual inspection of the data previously indicated that the data were linear.

Next, a model was built that included all demographic variables (“controlled model”)—sex, ethnicity, past family and child diagnoses, and household status (e.g., advantaged). The association between age and depression scores remained significant, $\gamma_{10} = 0.005$ ($SE = 0.002$), $p = .0048$. There was a 6.98% reduction in intercept variance between the unconditional growth model and the controlled model.

Hypothesis 1

To test hypothesis 1, trauma was incorporated into the model—controlling for covariates—to determine its associations with level (intercept) and linear growth (slope) of depression. Results are shown in Table 7 (under the column heading *Trauma*).

Interpersonal trauma was significantly associated with the depression intercept, but not

Table 7
Growth of and effects of trauma and ADHD on depression symptoms.

	Trauma		ADHD		Trauma x ADHD	
	γ (SE)		γ (SE)		γ (SE)	
Intercept	0.178	(0.009)***	0.176	(0.011)***	0.170	(0.011)***
IT	0.081	(0.013)***	-		0.044	(0.028)
NT	0.015	(0.009)	-		-	
ADHD	-		0.044	(0.013)**	0.026	(0.013)
IT x ADHD	-		-		0.049	(0.031)
NT x ADHD	-		-		-	
Slope	0.007	(0.002)**	0.002	(0.003)	0.003	(0.003)
IT	<0.000	(0.004)	-		-0.004	(0.008)
NT	-0.005	(0.003)	-		-	
ADHD	-		0.004	(0.004)	0.004	(0.004)
IT x ADHD	-		-		0.002	(0.009)
NT x ADHD	-		-		-	
Random Effects	Estimate (SE)		Estimate (SE)		Estimate (SE)	
σ^2	0.028	(0.001)***	0.028	(0.001)***	0.028	(0.001)***
τ_{00}	0.015	(0.001)***	0.016	(0.001)***	0.015	(0.001)***
τ_{01}	0.0006	(0.0003)*	0.001	(0.0003)*	0.0006	(0.0003)*
τ_{11}	0.0009	(0.0001)***	0.001	(0.0001)***	0.0009	(0.0001)***

Note: * $p < .05$, ** $p < .01$, *** $p < .0001$. IT = interpersonal trauma. NT = natural trauma.

growth. Natural trauma was not associated with the slope or intercepts. Individuals with histories of interpersonal trauma had higher overall depression scores, but did not have different growth trajectories than those with no history of trauma. This relationship can be described as “dose dependent” and is depicted in *Figure 2*. Those who experienced two or more traumas had an average of 0.081 higher mean depression scores than those with only one experience, which is the same average increase for those with one interpersonal traumatic experience when compared to those without any instances. In addition, depression mean scores increased at a rate of 0.007 per year. Incorporating trauma in the model reduced the intercept variance by approximately 7.6%.

Hypothesis 2

To test hypothesis 2, ADHD group was incorporated into the model—controlling for covariates but excluding traumas—to determine its unique effect on depressive levels

Figure 2

Depression trajectories for children with interpersonal trauma histories.

and growth (Table 7, column heading *ADHD*). Having an ADHD diagnosis was significantly associated with higher depression intercepts, but not growth. Individuals with an ADHD diagnosis had higher overall depression scores, an average of 0.044 higher mean score, but did not have different depression growth trajectories than those without an ADHD diagnosis (see *Figure 3*). After adding ADHD as a predictor in the

Figure 3

Depression trajectories for children with and without an ADHD diagnosis.

model, in fact, the previously significant rate of change in depression become non-significant, suggesting stability in depression scores over time. The ADHD model reduced intercept variance by 2.4% when compared to the controlled model.

Hypothesis 3

To test hypothesis 3 a model including ADHD, interpersonal trauma, and potential interactions was analysed (Table 7, column heading *Trauma x ADHD*). When both trauma and ADHD were included in the model the previously significant main effects were no longer significant. Children with trauma histories and/or an ADHD diagnosis did not have different depression intercepts or slopes. There were no trauma x ADHD interactions, suggesting that depression scores for children with ADHD is the same regardless of the frequency of trauma experienced. Like the ADHD-only model, the slope was non-significant, which indicates stable depression scores over time. This model reduced error variance by 10.99% when compared to the controlled model.

Several covariates were consistently significant in the depression models. While their significance was not featured in any hypothesis, their results may provide insight for this study's results and future research. Not surprisingly, a past diagnosis of depression (versus no past diagnosis) was significantly associated with higher depression scores for each model (γ s ranging from 0.088 - 0.098, all $ps < .05$). Sex was also significantly associated with higher depression scores compared to the sample average, with girls having higher depression scores across all models (γ s ranging from 0.0242 - 0.031, all $ps < .05$). Lastly, identifying as Black (γ s ranging from 0.027 - 0.040, all $ps < .05$) or Hispanic (γ s ranging from 0.0466 - 0.0541, all $ps < .005$), was associated with higher overall depression scores compared to the sample average.

Anxiety

An unconditional model was analysed, which determines the amount of variability in anxiety scores without accounting for any control or predictor variables, and growth, $\chi^2(1) = 311.80, p < .0001, -2LL = 2295.42$. The grand mean anxiety score at age

13 across all children was an average MASC score of 2.032. The intraclass correlation in the unconditional model for anxiety was:

$$\rho = \frac{\tau_{00}}{(\sigma^2 + \tau_{00})} = \frac{0.0595}{(0.1307 + 0.0595)} = 0.3218$$

This indicates that approximately 32.2% of the variability in anxiety scores can be attributed to between person differences; over 67.8% of the variability in anxiety is within-person variance.

Model 2 incorporates age as a predictor variables (centered at 13 years old) to determine the linear growth, or slope, of anxiety scores over time. Both linear growth and quadratic acceleration/deceleration terms were tested based on the initial visual inspection of the functional form of the anxiety scores. The unconditional growth model was an improvement when compared to the unconditional model, $\Delta\chi^2(5) = 350.41, p < .0001$, $-2LL = 2203.91$. The grand mean anxiety score at age 13 was an average MASC score of 1.99. There was a small, but negative, association with age and anxiety scores, $\gamma_{10} = -0.070, p < .0001$. In addition, there was a significant positive deceleration, $\gamma_{20} = 0.009, p < .0001$. There was approximately a 46% reduction in individual error when linear and quadratic growth were included as predictors of anxiety scores.

Next, a model was built that included all control variables (“controlled model”)—sex, ethnicity, past family and child diagnoses, and household status (e.g., advantaged). This model was a good fit for the data. The new conditional mean anxiety score at age 13 was an average MASC score of 1.961. The association between age and anxiety scores remained significant, $\gamma_{10} = -0.070, p < .0001$; also, acceleration remained significant, $\gamma_{20} = 0.0009, p < .0001$. There was a 6.75% reduction in intercept variance between the unconditional growth model and the controlled model.

Table 8
Growth of and effects of trauma and ADHD on anxiety symptoms.

	Trauma		ADHD		Trauma x ADHD	
	γ	(SE)	γ	(SE)	γ	(SE)
Intercept	1.923	(0.019)***	1.950	(0.024)	1.950	(0.029)***
IT	0.066	(0.031)*	-		-0.028	(0.067)
NT	0.056	(0.020)**	-		<0.001	(0.036)
ADHD	-		-0.012	(0.029)	-0.047	(0.035)
IT x ADHD	-		-		0.084	(0.075)
NT x ADHD	-		-		0.080	(0.044)
Slope	-0.065	(0.004)***	-0.072	(0.005)***	-0.070	(0.007)***
IT	-0.006	(0.008)	-		-0.005	(0.016)
NT	-0.008	(0.005)	-		-0.005	(0.009)
ADHD	-		0.003	(0.007)	0.008	(0.008)
IT x ADHD	-		-		-0.001	(0.018)
NT x ADHD	-		-		-0.006	(0.011)
Acceleration	0.009	(0.001)***	0.011	(0.002)***	0.010	(0.002)***
IT	0.008	(0.001)	-		0.003	(0.005)
NT	-0.001	(0.002)	-		0.001	(0.003)
ADHD	-		-0.003	(0.002)	-0.002	(0.003)
IT x ADHD	-		-		-0.003	(0.006)
NT x ADHD	-		-		-0.003	(0.003)
Random effects	Estimate	(SE)	Estimate	(SE)	Estimate	(SE)
σ^2	0.068	(0.003)***	0.068	(0.003)***	0.068	(0.003)***
τ_{00}	0.079	(0.007)***	0.082	(0.007)***	0.078	(0.007)***
τ_{11}	0.003	(0.0004)***	0.003	(0.0004)***	0.003	(0.0004)***
τ_{22}	0.00009	(0.00004)*	0.00008	(0.00004)*	0.00008	(0.00004)*
τ_{01}	0.0005	(0.0012)	0.0005	(0.0012)	0.006	(0.0012)
τ_{02}	-0.0014	(0.0004)**	-0.0013	(0.0004)**	-0.0013	(0.0004)**
τ_{12}	-0.0003	(0.00009)**	-0.0003	(0.00009)**	-0.0003	(0.00009)**

Note: * $p < .05$, ** $p < .01$, *** $p < .0001$. IT = interpersonal trauma. NT = natural trauma.

Hypothesis 1

To test hypothesis 1, trauma was incorporated into the model—controlling for covariates—to determine its associations with the intercept and linear growth slope of anxiety (Table 8, column heading *Trauma*). Both trauma types were significantly associated with anxiety intercepts, but not growth. Individuals experiencing either interpersonal or natural trauma had higher overall anxiety scores, but did not have different growth trajectories than those who did not experience trauma. Specifically, for every interpersonal trauma and natural trauma experienced, mean anxiety scores

Figure 4

Anxiety trajectories for children with interpersonal trauma histories.

increased by 0.066 and 0.056, respectively (see *Figures 4* and *5*, respectively). In addition, mean anxiety scores decreased at a rate of 0.065 per year and had a positive quadratic change that decreased the negative linear growth by 0.009 per year. Incorporating trauma in the model reduced the residual variance by 2.7%, intercept variance by 7.03%, and acceleration variance by 38.81% when compared to the

Figure 5

Anxiety trajectories for children with various natural trauma histories.

controlled model.

Hypothesis 2

To test hypothesis 2, ADHD was built into a model to determine its unique effect on anxiety levels and growth (Table 8, column heading *ADHD*). The results are listed in

Table A. Having an ADHD diagnosis was not associated with higher anxiety intercepts, growth, or acceleration. The ADHD model reduced intercept variance by 0.2% when compared to the controlled model.

Hypothesis 3

To test hypothesis 3, a model including, ADHD, trauma, and potential interactions was analysed (Table 8, column heading *Trauma x ADHD*). The typical anxiety trajectory was a decrease in symptoms over time that slowly leveled off (i.e., negative linear growth and positive quadratic growth). For example, at ages 9, 13, and 17 the rates of change in anxiety symptoms were -1.10, -0.70, and -0.030, respectively. The associations between interpersonal trauma, natural trauma, ADHD diagnosis, and their interactions with anxiety levels, growth, or acceleration were not significant. Thus, children with a history of trauma or ADHD diagnosis did not differ in symptom scores or trajectories. This model reduced error variance by 2.45%, intercept variance by 8.09%, slope variance by 1.38%, and acceleration variance by 44.78% when compared to the controlled model.

Sensitivity Analyses

Main effects for depression and anxiety disappeared in the models that included trauma, ADHD, and their interaction. A sensitivity analysis was considered to determine if main effects remained when the combined model dropped its interaction term. Based on the trauma- and ADHD-only models for depressive symptoms, a sensitivity analysis contained linear age, covariates, interpersonal trauma, and ADHD diagnosis as predictors. The results of this model suggest an intercept of $\gamma_{00} = 0.1645$, $p < .0001$ and a non-significant linear growth term of $\gamma_{10} = 0.002$, $p .4011$. However, both interpersonal

trauma ($\gamma_{0,INT} = 0.083, p < .0001$) and ADHD ($\gamma_{0,ADHD} = 0.033, p = .0072$) main effects retained significance. Additionally, both main effects did not affect depression symptom trajectories.

The same analysis was repeated for anxiety. Like the trauma and ADHD-only models of anxiety, both forms of trauma had significant associations, while ADHD did not. Additionally, the effects were almost identical with interpersonal and natural trauma having associations of $\gamma_{0,INT} = 0.066, p = .034$ and $\gamma_{0,NAT} = 0.056, p = .0063$, respectively (standard errors were also the same as the trauma-only model; see Table 8). Anxiety symptom trajectories were not associated with trauma or ADHD.

Covariates

Several covariates were consistently significant in the anxiety models. While their significance was not featured in any hypothesis, their results may provide insight for this study's results and future research. A child's past diagnosis of anxiety was significantly associated with higher overall anxiety scores for each model (γ s ranging from 0.081- 0.086, all $ps < .05$). A family history of anxiety disorders was also significantly associated with higher child anxiety scores for the interaction model, ($\gamma = 0.058, p = .046$). Sex was also significantly associated with higher anxiety scores, with girls having higher anxiety scores across all models (γ s ranging from 0.127 – 0.136, all $ps < .0001$). Coming from an advantaged household was associated with higher anxiety scores across all models (γ s ranging from 0.032-0.035, all $ps < .02$). Identifying as Hispanic (γ s ranging from 0.066-0.070, all $ps < .05$) was associated with higher overall anxiety scores in all models. Lastly, Black children had higher anxiety scores for the controlled ($\gamma = 0.051, p = .0466$) and ADHD models ($\gamma = 0.053, p = .0370$).

Discussion

This study is the first to examine longitudinal associations between trauma and internalizing psychopathology in children with/without ADHD. Depression and anxiety were tracked, using multi-level modelling, to determine if symptom levels and trajectories varied as a function of trauma, ADHD, and their interaction. It was hypothesized that histories of trauma and ADHD would be independently associated with higher symptom levels that increase over time more quickly for people with a history of trauma or ADHD, compared to non-trauma and non-ADHD counterparts. Additionally, it was hypothesized that ADHD would moderate the association between trauma and internalizing symptom level and trajectory. Specifically, ADHD was expected to exacerbate (i.e., worsen) symptom levels and trajectories for children with a trauma history compared to children without ADHD.

Key findings were, first, that interpersonal trauma was positively associated with depressive and anxiety symptoms, but not trajectories. Thus, children who experienced interpersonal trauma had, on average, higher symptom levels than children without such experiences, but followed a similar pattern of symptom development. Children who experienced natural trauma had higher anxiety, but not depression, symptom levels than children without such experiences, but followed a similar pattern of symptom development. Second, children with ADHD had higher depression, but not anxiety, symptom levels than children without a diagnosis. The two groups did not differ in trajectories. Last, all main effects for depression and anxiety symptom levels dissipated in a model that included both ADHD, trauma, and their interaction.

Unconditional Symptom Growth

An unconditional model investigating the symptom level and growth trajectory of depression showed that depressive symptoms followed a modest, positive increase from age 8- to 18-years old. However, depressive symptom increases were not statistically significant in the conditional models that incorporated ADHD diagnosis or a combination of childhood trauma and ADHD diagnoses; these models suggest a consistent depressive pattern from childhood to adolescence, irrespective of trauma or ADHD history. This pattern is similar to that found by Dekker and colleagues (2007), who tracked depression symptoms in youth aged 4 to 18 years old. Their results suggested that most boys and girls fit trajectories that could be described as slightly decreasing, stable and low, or slightly increasing. A previous study making use of the present sample of children from the MTA noted significant changes in depressive symptoms when comparing the MTA children with ADHD and LNCG group (ADHD children were compared by treatment randomization and a series of latent classes) (Molina et al., 2009). However, Molina et al. (2009) used measurement wave, as opposed to age, as a time metric, which did not consider variability in age. The present study clarifies that there are no differences in LNCG and ADHD children when modeling depression as a function of age. In addition, evidence from the present study suggests that depressive symptoms remain relatively stable from ages 8- to 18-years old. This finding differs from other studies showing pronounced increases in depression from late childhood through adolescence (Badlursdottir et al., 2017; Maughan, Collishaw, & Stringaris, 2013), but these differences may reflect unique features of the MTA study. First, trajectories were not modeled as a function of sex. Females may have very distinct depression trajectories with levels that are typically above male counterparts (Dekker et al., 2007). Eighty

percent of the MTA sample, both ADHD and LNCG groups, were male. This puts the study at a disadvantage when investigating effects that are predominantly female-driven, which may be the case for depression trajectories.

Second, the CDI may be a poor measure for the tracking sensitive changes in the developmental course of childhood *and* adolescent depression. For example, some CDI questions appear to be tailed more for children than adolescents such as asking children how often they ‘do what they’re told’ or how much they ‘have to push myself to do my homework’.

Last, depressive symptoms, at least as measured by the CDI, may be more prone to short-term fluctuations in response to life stressors or events, which are not captured by this multi-year longitudinal analysis with assessments spaced every two years. While the patterns in this study may reflect a child’s typical depressive trajectory, which was consistent or slightly increasing, more frequent assessments of depressive symptoms in response to specific life events or transitions may suggest a different interpretation. Regardless, depressive symptoms measured with large intervals suggest that children seem to settle into a depressive symptom pattern by age 8-years old, which lasts up until approximately age 18.

Global anxiety symptoms unexpectedly decreased on average from age 8 to 18. However, the rate of decrease slowed each year. This suggests that children in this sample are more anxious in childhood compared to adolescence. Furthermore, this pattern was evident in unconditional as well as conditional models of anxiety, which buttresses the finding. This aligns with a study that investigated specific subscale measures of anxiety disorders in youth aged approximately 12 to 20 years old (Nelemans

et al., 2014). The majority of anxiety symptoms decrease from age 12.5 years old onward with a positive quadratic or cubic acceleration, but the prognosis was better for boys and those with lower initial symptoms. The general decrease in symptoms may be attributed to how children develop stronger cognitive and social resources, which help them manage and cope with anxiety. Coping has been found to act as a mediator between stressful life events and depressive symptoms (Evans et al., 2015), but it is unclear if similar mechanisms exist for anxiety symptoms. Despite the similarities between this study and that of Nelemans et al. (2014), the decrease in anxiety symptoms over time in the present study was unexpected. As children develop and experience significant life transitions (e.g., puberty and entry into secondary school) the importance of self-concept as an individual and in reference to social groups becomes prominent. During these transitions, anxiety symptoms are expected to manifest, be exacerbated or, at best, remain relatively stable. Such patterns were demonstrated using latent class analysis in one study (Lauterbach & Armour, 2016). Three of four classes, representing approximately 93% of the sample, demonstrated stability or an increase in anxiety symptoms, while one group displayed elevated initial levels that decreased (7% of the sample). The marked difference between the initially high group and a class showing low and stable symptoms over time was that the former had fewer visits to a primary care provider of mental health professional regarding emotional/behavioral problems. This suggests that children in an initially high, then decreasing symptom trajectory may not have adequate support systems in place that facilitate treatment. In this study, it may be that many children enrolled in the MTA had parents who failed to identify or act on their children's increased anxiety symptoms. This may be a result of selection bias; children

enrolled in the MTA study were recruited from the community and tertiary care settings rather than clinics, raising the possibility that parents may have disproportionately lacked the means to seek out care provided by mental health professionals. An additional point of interest is that the majority of changes in symptoms noted by Lauterback and Armour (2016) occurred between ages 4- and 8-years old. Trajectories post-eight years old seemed to taper off. Thus, children may learn to cope with their anxiety symptoms through increased and more complex social interaction in a school context. An initial peak in anxiety, as reflected at age 8, could be representative of new peer interactions without the supervision of a parent or guardian. As a child learns to deal with the anxiety associated with the separation of a guardian and fending for oneself in unsupervised social contexts, anxiety symptoms may begin to lower and, eventually, taper off. This trend is plausible when considered the anxiety trajectory present in the current study.

Trauma and Internalizing Symptom Trajectories

A trauma-only model was analysed and suggested that depression symptoms differed for children with and without experiences of interpersonal trauma. While traumatic events were coded 0, 1, and 2 for independent traumatic events, it has been noted in past research that interpersonal traumas are rarely isolated or one-time occurrences. In one study involving pregnant women, Grote et al. (2012) found that 92% of participants who reported a form of childhood abuse endorsed at least one other form of abuse (e.g., both sexual abuse and emotional neglect). Like this study, those who reported more versus fewer interpersonal traumas, specifically abuse, also endorsed more depressive symptoms. The results of this study align with others' findings (e.g., Chu et al., 2013; Grote et al., 2012; Lauterback & Armour, 2016), despite the core difference in

samples. Although the definition of interpersonal trauma in this study was more inclusive—for example a suicide loss was included—the results suggest that trauma involving another person is associated with significantly higher depression symptoms. Perhaps the transformation of primary relationships, such as the loss of trust in a caretaker or sudden and unexpected death of a loved one, is integral in engendering a sense of hopelessness or guilt. The unique role interpersonal trauma plays is further supported by the lack of significance in natural trauma's association with depressive symptoms, which was demonstrated in another study (Chu et al., 2013). Natural trauma, while potentially detrimental and important for an individual's development, presents fewer concerns for depressive symptoms through childhood and adolescence. Natural trauma may be more important for developing a sense of unpredictability or anticipation regarding dangerous events, which is a core feature of anxiety; examples of natural traumas, such as flooding, hurricanes, or fires, are typically unexpected.

Interpersonal and natural traumas were associated with increased global anxiety symptoms, but not trajectories. Thus, children who experience such traumas typically have increased symptoms, but follow a similar symptom development pattern as those without traumatic experiences. This association between trauma and anxiety symptoms (Martin et al., 2014) and disorders (Legerstee et al., 2010) has been demonstrated in other research with children. Anxiety symptoms revolve around the *anticipation* of threat (APA, 2013); naturally occurring trauma typically involve unexpected, and often unpredictable, events. Children experiencing such events may develop an excessively cautious disposition that generalizes across various life contexts, including those that do not warrant anticipation of danger. The result may manifest as higher anxiety symptoms

(i.e. increased perception that things can and will go awry). The fact that trajectories did not interact with trauma suggest that the changes in anxiety symptoms reflects a purely developmental process that is independent of trauma history.

Trauma, specifically interpersonal trauma such as physical or sexual abuse, rarely occurs as a single incident event. Despite this, 43.84% of participants in this study experienced at least one traumatic event, while only 9.25% experiencing two or more interpersonal or natural traumas, or a combination. However, the frequency of any one traumatic event remains unknown; a child who reports being “badly beaten” may have been a victim once, or one-hundred times. Accounting for the frequency of traumatic occurrences may have changed the results of this study.

One major, and unequivocally traumatic event, that was excluded from the KPTS is sexual abuse. The lack of sexual abuse as an option for a childhood traumatic event and information regarding trauma frequency suggests that the effects observed in this study may be attenuated relative to true relations in the population between trauma and internalizing symptoms. It is believed that the trajectories observed in this study reflect typical development of internalizing symptoms, but additional research can build on this hypothesis. Trauma, as defined and measured in this study, did not influence the typical progression of internalizing symptoms. Future studies may incorporate sexual abuse into a similar research question to test its potential effects on symptom levels and growth. Sexual abuse seems to have distinct effects when compared to other forms of interpersonal trauma (Ballard et al., 2015) and may have distinct effects on the trajectory of internalizing psychopathology.

Childhood ADHD and Internalizing Symptom Trajectories

Children with ADHD in this study had significantly higher depression scores when compared to children without a diagnosis in the ADHD-only model. However, once ADHD was included as a model predictor, depression growth trajectories dissipated (i.e., the overall slope became non-significant). There was also no slope by ADHD interaction, indicating that children with ADHD follow similar depression symptom trajectories when compared to their non-diagnosed peers. A significant ADHD effect for depression, but no ADHD by Time interaction, was also observed in a previous MTA study (Molina et al., 2009). These results align with another finding that suggested that childhood ADHD is linked to increased depressive symptoms at ages 18 to 25 years old (Meinzer et al., 2016). This latter research also had a non-significant slope by ADHD interaction when tracking depressive symptoms from age 18 to 25. However, Meinzer et al. (2016) reported a significant negative slope and the associations between childhood ADHD diagnosis and depressive symptoms were non-significant once controlling for current ADHD symptoms. Despite the differences between Meinzer's sample and the MTA, there is evidence to suggest that depression may be consistently higher for children with ADHD from early childhood to early adulthood. Current ADHD symptom levels and impairment, however, may be the vital component in predicting later depressive symptomology (Hechtman et al., 2016). Hechtman et al. followed up the MTA children at 16-years after baseline and found that children with ADHD and the LNCG group did not differ with rates of anxiety or mood disorders (Hechtman et al., 2016). However, significant differences were found when comparing ADHD-desisters (i.e., those who no longer meet diagnostic criteria) and persisters; ADHD-desisters and the LNCG children had similar rates of mood and anxiety disorders. This pattern suggests that children with

ADHD, especially those with persistent symptoms, may be at risk for development of comorbid depression and are candidates for extra attention from mental health care professionals. If professionals conduct check-ins with children with ADHD, especially those with persistent impairment, additional depression screening may be valuable for identifying additive levels of clinically significant and depression-related impairment in this at-risk population. Mental health care should be sensitive to multiple, simultaneous conditions (e.g., McKee, 2017) that require equal attention from a collaboration of general practitioners, mental health professionals, and primary support systems.

Children with an ADHD diagnosis did not differ in anxiety symptoms and trajectories compared to children without a diagnosis. It is unlikely that the MTA's randomized treatment (i.e., medication, behavioral management, etc.) may have influenced the null results. Treatment assignment did not affect depression or anxiety symptom levels at treatment completion, or 10-month pre-baseline of this study (i.e., children in community care did not differ from children with medication, behavior management, or a combination of the two; Jensen et al., 2001; March et al., 2000). However, children assigned to the behavioral treatment or community care group did fare better than the medication or combined group at the 6-year follow up; these children were less likely to have a formal depression or anxiety diagnosis (Molina et al., 2009). Although the association between ADHD and anxiety symptoms was expected to be similar and of a similar magnitude to depression, as based on extant literature (Kessler et al., 2006), the lack of an association is surprising. ADHD does not appear to promote new or exacerbate existing anxiety symptoms. It may be that the manifestation of anxiety is subjectively different for children with ADHD. Thus, while symptoms levels may be

like those of non-diagnosed peers, the manifestation of measurable impairment may be disparate and unexpected when compared to non-ADHD peers. For example, it may be that children with ADHD have anxiety symptoms that are revolve around negative social interactions (March et al., 2000). Approximately, 14% of ADHD children had comorbid anxiety disorders at baseline in this study, as opposed to 7% of LNCG children. The manifestation of anxiety in ADHD children may have been picked up within a comprehensive diagnostic interview, such as the DISC did in assessing anxiety and mood disorders, whereas the MASC may be restricted to the expected manifestation of anxiety in typically-developing children.

The lack of effects may largely be due to the recruitment process implemented by the MTA Co-operative Group. Children in this study were largely community-recruited, as opposed to the prototypical method of recruitment for clinical trials (e.g., clinic- or institution-based recruitment). ADHD impairment or resiliency may be lower—or higher for the latter—in a community-based sample when compared to those recruited clinically. Children recruited through hospitals, psychologists' offices, or other tertiary care centers are likely to present increased impairment in various facets of functioning, which is largely the reason why they seek those settings in the first place. A lower level of impairment in children participating in the MTA study is a plausible explanation for the lack of association between ADHD diagnosis and anxiety symptoms. As opposed to depression, perhaps anxiety symptoms are more likely to be associated with a more severe ADHD profile. Children with ADHD may be more likely to ruminate on past negative events, which can predict depressive symptoms (Broderick & Korteland, 2004), than to anticipate future danger. Indeed, the tendency for individuals with ADHD to

ruminate more than non-ADHD peers as a form of emotion regulation has been demonstrated in the literature (Matthies & Philipsen, 2014). This would explain the unique association of ADHD with depression and not anxiety symptoms.

Last, and as suggested by previous work with the MTA sample, anxiety symptoms may affect a child's response to treatment of ADHD (Jensen et al., 2001). Despite the lack of association between ADHD and anxiety symptoms in this study, clinicians would still benefit from assessing anxiety symptoms to determine a child may be better suited to a behavioral form of ADHD treatment (Jensen et al., 2001).

Joint Contributions of Childhood ADHD and Trauma History

The effects of interpersonal trauma and ADHD on increased depression and anxiety symptoms were not evident in a combined model. This suggests that multicollinearity between ADHD diagnosis and trauma history may have had an impact on the already small estimates of their effects. This is evidenced by the large increase in the standard error of the interpersonal trauma effect when ADHD is included in the model. If trustworthy, the non-significant interaction is an encouraging finding for children with ADHD and a trauma histories: the associations between trauma and depression will be similar for children with and without ADHD, which may allow children with and without ADHD to follow similar treatment guidelines —when necessary—after traumatic interpersonal experiences.

A sensitivity analysis was investigated to determine the additive, but not interactive, effects of interpersonal trauma and ADHD on depressive symptoms by removing their interaction from the analysis. The results suggest both effects significantly predict increased depression symptoms. Thus, while ADHD does not

change the association between trauma and depressive symptom, a child with both ADHD and a trauma history may have higher depressive symptoms due to their additive effects. This ‘no-interaction’ model results suggest that children with ADHD interpret or integrate traumatic experiences similar to children without ADHD—with respect to the manifestation of depressive symptoms. A sensitivity analysis for anxiety symptoms, which included trauma, ADHD, but not the interaction, revealed identical trauma main effects and standard errors as the trauma-only model. Thus, trauma has similar effect when controlling for ADHD diagnosis. Both sensitivity analyses corroborate the poignant, adverse effects of childhood traumatic experiences. Although trajectories are unaffected, children with traumatic experiences appear to have consistently higher internalizing symptoms.

The main effects of trauma and ADHD suggest that depression and anxiety are different manifestations of internalizing psychopathology. Although transdiagnostic treatment is a growing body of interest for research and clinicians, and proving to be relatively effective (Newby et al., 2015), this study suggests that depression and anxiety progress differently over time and are affected differently by trauma and ADHD. Interpersonal trauma and ADHD affect depression symptoms, while interpersonal and natural trauma, but not ADHD, affect anxiety symptoms. It may be that children with ADHD are more vulnerable to a depressogenic manifestation of the underlying emotional, or internalizing, disorders. The fact that anxiety disorders were more prevalent at baseline when compared to depressive counterpart in children in this study suggest that there are, at least, differences in the developmental expression of anxiety and depressive disorders. The results of this study mark the importance of testing the efficacy

and effectiveness of a transdiagnostic treatment approach on specific subsamples, such as those with trauma history. These children have elevated depression and anxiety symptoms that may obfuscate classic treatment approaches, making transdiagnostic treatment a favoured approach.

A continuation of this study, in which internalizing symptoms are tracked further into young adulthood, would be fruitful. Adulthood presents important developmental transitions, such as living independently from parents or starting serious romantic relationships, which may misalign an individual's needs from what is available in the context (Schulenberg, Sameroff, & Cicchetti, 2004). For example, women aged 18-23 who continue to live or must move back in with parents over a four-year span experience significant increases in stress (Bell & Lee, 2008). In addition, other research suggests an increase in depressive symptoms between the summer before and second semester of the first year of college (Alfeld-Liro & Sigelman, 1998). Thus, a peak in internalizing symptoms may have a delayed effect for some people, in response to challenging transitions such as these. Interpersonal trauma may obfuscate these transitions due to their marked interpersonal nature. This aligns with the accentuation principle that states that psychological dispositions, such as increased depression after a traumatic event, may become accentuated during life transitions (Graber & Brooks-Gunn, 1996). While it would be expected that individuals with a history of interpersonal childhood trauma would continue to have significantly higher levels of internalizing symptoms when compared to those without such experiences, differences in trajectories may propagate during important life transitions. The continued measurement of internalizing symptoms would buttress the assumption that the trajectory similarities noted in this study are truly

reflected of the normative developmental process of depression and anxiety symptoms or if different component of a developmental system can clarify differences in trajectories.

Noteworthy Covariates

Some covariates included in this study's analyses should be of concern for practitioners and researchers seeking to understand or treat depression symptoms. First, children identifying as Black had higher depressive symptom scores across the only-trauma, only-ADHD, and sensitivity models (ranging from a higher mean score of 0.029-0.038 when compared to the weighted average). Although the effect was small, it is concerning when considering the results of a study that report that African-Americans are less likely to attend multiple sessions of psychotherapy compared to non-Latino white participants, even when controlling for depressive symptom severity (Gonzalez et al., 2010). They also reported more general impairment because of depressive symptoms compared to white counterparts. Another study suggests that father education and family financial status, and not ethnicity, are variables of concern for depression (Doi et al., 2001). However, the association between black ethnicity and depressive symptoms found in this study remained after controlling for household advantage, a composite variable that included father's education, in addition to mother's education and parental marital status. Practitioners assessing or treating clients of black ethnicity should be aware of the increased risk of depressive symptoms, even if the increase is modest, as they are likely to be more impaired and more reluctant to complete treatment.

Second, a history of depressive disorders was also associated with depressive symptoms for all models except the ADHD-only model. For all other models, children with past diagnoses had significantly higher mean depression scores at age 13 (ranging

from 0.090-0.098, $ps < .05$). While not surprising, this demonstrates the prolonged nature of depressive symptoms. The diagnoses were made at the baseline measurement or, in cases where this data were unavailable, the one-year follow-up used in this study, indicating that elevated symptom levels—at least a significantly higher amount—persist up until at least age 13.

There were several covariates that were consistently associated with anxiety symptoms. First, girls had consistently higher mean anxiety scores compared to boys, ranging from 0.1169-0.1254 (all $ps < .0001$). This effect was large and almost doubled the magnitude of the main effects of trauma in the trauma-only model. This aligns with other studies that suggested that females are more likely to experience anxiety symptoms (March et al., 1997; Martin et al., 2014), especially for general and social anxiety disorder symptoms (Nelemans et al., 2014). Women are more likely to experience sexual abuse or assault (Ballard et al., 2015; Dykman et al., 1997; Statistics Canada, 2013). Perhaps interpersonal traumas of a sexual-nature, which were excluded from this study, are partially mediating the relationship between being female and having increased anxiety symptoms.

Second, a history of a childhood anxiety disorder was positively associated with anxiety symptoms for all models. Although this is not surprising, it marks the importance of long-term follow-up for clinicians. Children with a history will continue to have increased symptoms that may reach severe levels when faced with important life transitions or stress, which are also associated with increased psychopathology (Schulenberg, Sameroff, & Cicchetti, 2004).

Third, coming from an advantaged household was associated with more anxiety symptoms. This contradicts the general association of lower SES and higher anxiety symptoms (e.g., Vine et al., 2012). It should be noted that *advantaged household* was a composite variable that did not include parental income. Rather, it included maternal and paternal education—when available—and parental marital status. Perhaps this is due to increased parental expectations and pressure on a child, which leads to increased anxiety regarding academic, social, or extracurricular failure. For example, perceived parental expectations act as a mediator for test anxiety in females (Peleg, Deutch, & Dan, 2016); perhaps perceived expectations are the underlying mechanisms for increased anxiety symptoms in children with parents who achieved higher education.

Lastly, children within the *other* ethnicity group (10.4% of the sample), which included those identifying as Asian, Native American, mixed ethnicity, and other, had significantly higher anxiety scores in the trauma-only and sensitivity model. This was unexpected; a large study drawing data from the Collaborative Psychiatric Epidemiology Studies data suggest that individuals identifying as Caucasian have the highest lifetime prevalence of anxiety disorders (Asnaani et al., 2010). Furthermore, Asnaani et al. suggest that Asian Americans had the lowest prevalence rates. It may be that prevalence rates are not indicative of overall group mean symptom levels. In other words, while Caucasians may have the most cases of individuals meeting diagnostic criteria, those identifying as mixed/other may have higher overall mean scores.

The magnitude of the effects in this study needs to be addressed in the context of the findings. Despite statistically significant main effects, a majority of the magnitudes are relatively small. As an example, consider the association between interpersonal

trauma and depressive symptoms in the trauma-only model. A child who experienced two or more interpersonal traumas had an average mean score that was 0.162 points higher than a child without any experiences. These can be translated to total CDI scores at age 13 of 4.81 and 9.18 for children without versus with two or more interpersonal trauma experiences, respectively (assuming all other covariates are held constant at 0). This is a modest effect using a standard deviation of 6.21. In addition, overall depression scores were modest for the whole sample. The suggested CDI cut-off scores are 20 for the general population and 13 for a clinical sample (Matthey & Petrovski, 2002), which suggests the average child in this study had scores that do not warrant concern, even for those with trauma or depressive disorder history. It may be that children in the study are under-reporting their symptoms, which could be clarified using a more comprehensive depression measurement or method (i.e., clinical interview). Regardless, Matthey & Petrovski note that the CDI cut-offs are based on flawed data and should not be used for screening purposes. Rather, the CDI is best suited to track affect changes over time. Similarly, the magnitude of anxiety differences were low and scores fell in the normal range, such as the mean score of 2.24 on the MASC for a community-based sample of children approximately 13-years old (March et al., 1997). Children with and without a trauma history in this study fell below or around a mean score of 2 on the MASC at age 13 years old. The recruitment method, which was previously noted, may explain the sub-clinical levels of internalizing symptoms found in this study. While there were specific effects for trauma and ADHD on internalizing symptoms, this study suggests that the average child is resilient against a drastic increase in depression or anxiety symptoms regardless of trauma history or ADHD diagnosis.

Self-report methods may also be responsible for some of the smaller effects found in this study. A positive response bias in children with ADHD may be present, such that these children overestimate their competencies, compared to an external observer such as a teacher or parent, to levels that are on par with non-diagnosed peers in an effort to self-protect (Hoza et al., 2010). Children engaging in self-protection may automatically or unconsciously deny personal deficits and overestimate their abilities in efforts to maintain a positive self-concept. Hoza et al. found that children in the MTA had higher depressive scores as well as an increased tendency to positive self-bias when compared to non-diagnosed peers. In addition, children with ADHD had relatively consistent levels of self-bias over time, suggesting that children with ADHD engage in social positive bias consistently from ages 8- to 17-years old. Thus, it is likely that children with ADHD are underestimating their symptoms of depression and anxiety. Future studies may benefit from taking into consideration collateral reports in addition to self-reports; both assessments may triangulate on a truer reflection of internalizing symptoms.

Limitations

The results of this study should be interpreted in the context of its limitations. First, there is an abundance of research that demonstrates the perseverating nature of ADHD symptoms that cause significant impairment, regardless of whether the individual continues to meet a formal diagnosis (e.g., Sibley et al., 2012). Thus, there is likely to be variability in ADHD symptoms from ages 9 (i.e., closer to baseline in this study) to 18. Using ADHD diagnosis as an indicator will incorporate much of the variability in ADHD symptoms. However, future studies should track intraindividual variability in ADHD symptom severity from childhood to adolescence, as opposed to a baseline diagnosis,

which may elucidate the associations between ADHD, trauma, and internalizing symptoms. Second, some of the instruments used in this study could be improved. The Kiddie Posttraumatic Stress Scale does not provide a timeline for trauma history. As a result, important temporal differences could not be detected and, instead, may act as confounds; trauma that occurred during early childhood is likely to affect internalizing symptoms differently than a trauma that occurred one month before study recruitment. In addition, many *potentially* traumatic experiences were excluded from analyses because it could not be determined who experienced the event. It was never definitive if trauma that occurred “to someone I know” fit DSM’s definition of a traumatic experience and, thus, were excluded. In addition, the KPTS has limited psychometric properties; a published psychometric assessment paper could not be found, which calls into question its validity and reliability.

KPTS assessments were measured at the baseline of this study or, when unavailable, one-year post-baseline. It is plausible, and likely, that children experienced traumatic events after these assessments. This is undoubtedly a strong, yet unalterable, limitation of the current study. Evidence of trauma after these assessments may have elucidated some of the findings in this study, in addition to being able to test the existing hypothesis that children trauma has worse consequences when compared to trauma during later years.

Concluding Remarks

Interpersonal trauma has a unique relationship with depression and anxiety symptoms that remain after controlling sex, ethnicity, and internalizing disorder history. Experiencing interpersonal trauma appears to put children at risk of consistently elevated

levels of anxiety or depression. Natural trauma only affected anxiety symptoms. These results would inform practitioners whom work with children to assess trauma history, regardless of ADHD diagnosis, as children with trauma histories may require prolonged or disparate forms of intervention that target the elevated internalizing symptoms. It is unclear from the results of this study if intervention can bring children with trauma histories' internalizing symptoms to levels like no-experience peers.

Future studies can tease apart the differential effects of trauma and internalizing symptoms in children with and without ADHD by segregating types of interpersonal or natural trauma. A study segregating interpersonal-based childhood adversity into deprivation (e.g., neglect) and threat (e.g., sexual abuse) found important differences in psychopathological outcome and physiological mechanisms underlying stress responses (Busso, McLaughlin, & Sheridan, 2017). Others have noted the distinct outcomes for different types of interpersonal trauma (Ballard et al., 2015). The associations of trauma have been, and continue to be, an area of increased interest for development scientists.

One important avenue for investigation is the temporal sequence of traumatic experiences and ADHD symptoms. While there is a clear link between trauma and ADHD symptom severity (e.g., Becker-Blease & Freyd, 2008), the question of “Does trauma increase risk for ADHD, or vice-versa?” still remains. Longitudinal studies tracking both trauma and ADHD from birth into childhood are invaluable to answering such questions. These studies may provide insight into the relationship between trauma, ADHD, and later psychopathological symptoms.

This study elucidated the various trajectories for children with and without trauma histories and ADHD diagnoses. While trajectories remained relatively consistent

regardless of a child's trauma history, depression and anxiety symptom levels were significantly higher for some children. The most prominent effect was for interpersonal trauma, marking the negative consequences of such events. While primary prevention should be a clear focus for policy makers and community support systems, which manifest as a reduction in the likelihood that children will go through interpersonal traumas, mental health professionals and primary supports should be mindful of a child's trauma history. The more interpersonal trauma a child endures, the higher their risk for depression and anxiety compared to peers with no (or less) trauma history.

Children with ADHD may be at risk for elevated depression levels from childhood to adolescence. While ADHD symptom management is a clear focus for mental health professionals, deficits in other domains in functioning, such as potentially impaired social functioning (e.g., Bora & Pantelis, 2016), should continue to be a priority. Reports completed by parents suggest that adolescents with persistent ADHD, when compared to non-ADHD controls, are more rejected by their peers and have fewer close friends (Bagwell, Molina, Pelham, & Hoza, 2001). A child's ability to function socially may largely affect depressive symptoms based on peer acceptance—or lack thereof.

A child grows within a complex developmental system that can adaptively or maladaptive influence development. This study has highlighted important components of this system by identifying trauma history and ADHD diagnosis as specific markers for increased anxiety or depressive symptoms—or in the case of interpersonal trauma history, both. However, researchers can benefit from this perspective and add additional components of an individual's developmental system to determine how they influence the

development of internalizing symptoms, whether additively or by interacting with other components. The substantial amount of individual variability in symptoms levels and trajectories that remain in this study, after including core effects of ADHD and trauma, mark the importance of considering and including additional contextual features. Including components of the context, which may range from genetic composition to broad, social influences can only strengthen our understanding of human development.

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