Somatic Symptoms and Adverse Childhood Experiences (ACEs) in Children and Adolescents

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Abstract

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Background: Somatic symptoms are commonly understood as subjective reports of physical discomfort without an identified cause. Common somatic symptoms such as headaches, stomachaches, and fatigue are particularly prevalent in children and adolescents, and are associated with impaired functioning, negative health outcomes, and unnecessary medical expenses. Previous research has suggested that adverse childhood experiences (ACEs), such as child maltreatment and household dysfunctions, are associated with somatic symptoms in childhood and adolescence. However, the longitudinal relationship between ACEs and somatic symptoms, the mediating mechanisms between ACEs and somatic symptoms, and the trajectory patterns of somatic symptoms across various stages of childhood and adolescence remain unknown.

Purpose: The overarching purpose of this dissertation is to advance the understanding of the effects of ACEs on the etiology and development of somatic symptoms in childhood and adolescence. This dissertation has three specific aims: 1) to examine the concurrent and longitudinal relationships between ACEs and somatic symptoms across adolescence (ages 12-16 years), comparing the effects between maltreatment and household dysfunctions, 2) to examine if anxiety and depression symptoms in adolescence mediate the effect of ACEs on concurrent and future somatic symptoms in adolescence (ages 12-16 years), and 3) to identify and describe the heterogeneity of trajectory patterns of somatic symptoms across childhood and adolescence (ages 6-16 years) and their potential predictors and outcomes.

Methods: This dissertation is comprised of three quantitative studies that were secondary analyses of the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN), a consortium of five longitudinal prospective studies conducted at different geographical locations across the United States using standard procedures. Participants included 1354 child and primary caregiver dyads. Data were collected when children were ages 4, 6, 8, 10, 12, 14, 16, and 18 years. The Ecobiodevelopmental framework is the guiding framework for this dissertation. Paper 1 used a longitudinal cross-lagged path analysis with the data collected when children were ages 12, 14, and 16 years. Paper 2 also used data collected when children were ages 12, 14, and 16 years to conduct a longitudinal cross-lagged path analysis and mediation analysis. Finally, paper 3 used the data from all time points (child ages 6, 8, 10, 12, 14, 16, and 18 years) to conduct a latent class growth analysis (LCGA).

Results: The findings of paper 1 indicated that a greater number of ACEs experienced at age 12 were associated with increased somatic symptoms at age 12, and concurrent exposure to household dysfunctions had stronger effects on somatic symptoms at age 12 as compared with

concurrent exposure to maltreatment. The findings suggest that the accumulation, timing, and types of ACEs have unique effects on somatic symptoms in adolescence. Paper 2's results demonstrated significant concurrent associations between ACEs and increased anxiety/depression symptoms and between anxiety/depression symptoms and increased somatic symptoms at ages 12, 14, and 16. Moreover, anxiety/depression symptoms mediated the pathway between ACEs and concurrent somatic symptoms at ages 12, 14, and 16. However, there were no significant longitudinal mediation effects. Paper 3's results identified four distinct patterns of somatic symptom trajectories across ages 6 to 16: low-stable (43.6%), increasing (24.5%), decreasing (22%), and high-stable (9.9%). Being a female and exposure to maltreatment before age 6 predicted increased chances of membership in the increasing and high-stable group compared to the low-stable group. Compared to the low-stable group, those in the increasing and the high-stable group were more likely to have a diagnosis of major depressive disorder at age 18.

Conclusion: This dissertation's findings contribute to our understanding of the relationship between somatic symptoms and ACEs across childhood and adolescence. The findings indicate that children with persistent somatic symptoms across childhood and adolescence are more likely to have experienced early childhood maltreatment, may be experiencing increased anxiety and depressive symptoms, may be concurrently exposed to ACEs, and are at increased risk for developing major depressive disorder in young adulthood. These findings call for addressing ACEs in adolescence, for example, by preventing maltreatment and family dysfunctions, and providing effective interventions for mitigating the effects of ACEs. Further research is needed to develop methods that effectively detect, address, and advocate for children experiencing somatic symptoms and ACEs in community settings.

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Chapter 1. Introduction

Background

Somatic symptoms are commonly understood as subjective reports of physical discomfort without an identified cause. Prevalent somatic symptoms include headaches, stomachaches, and fatigue. In studying somatic symptoms, it is imperative to distinguish the concepts of symptoms and diseases. Symptoms are subjective feelings and experiences of physical phenomena, whereas diseases are objectively observable evidence of abnormalities in the body. Since symptoms and diseases are two distinct constructs, symptoms can occur in the absence of diseases (Beck, 2008; Campo, 2012; Williams & Zahka, 2017). Somatic symptoms, understood as symptoms without objective diseases, will be the focus of this dissertation. Similar terms that are often interchangeably used with somatic symptoms include *functional somatic symptoms*, *somatization, medically unexplained symptoms*, and *somatic complaints*. Persistent and severe somatic symptoms are clinically diagnosed as somatic symptom disorder (APA, 2013). Somatic symptoms have historically been highly prevalent among children, with a gradual increase in adolescence (Campo, 2012; Swain et al., 2014). Somatic symptoms are often viewed as less dangerous since they are not signs of identifiable disease. However, the damaging effects of somatic symptoms in childhood and adolescence are well documented in the literature. Somatic symptoms in childhood are associated with decreased emotional well-being, decreased attention, lower academic achievement, impaired cognitive functioning, increased healthcare utilization rate, and unnecessary expenses (Fremont, 2003; Groenewald et al., 2014; Kelly et al., 2010; Stone et al., 2019; Voerman et al., 2017; Williams & Zahka, 2017). Moreover, persistent somatic symptoms during childhood and adolescence also predict future adverse health outcomes such as depression, anxiety, and suicidality (Andersen et al., 2009; Breidablik et al., 2009; Hoftun et al., 2011; Luntamo et al., 2014; Shelby et al., 2013).

A brief history of somatic symptoms research

Reviews of past research on pediatric somatic symptoms report that somatic symptoms have been common in community-based as well as clinical samples of children and adolescents (Campo, 2012). The biomedical model, which is the conceptual model of symptoms and disease upon which Western medicine was founded, viewed physical and psychological processes separately. Using this model, the etiology of somatic symptoms could not be identified (Engel, 1977). However, the paradigm shift in the medical sciences to the biopsychosocial model allowed for a more integrative view of somatic symptoms. Using the biopsychosocial perspective, somatic symptoms are perceived as presentations of the dynamic interactions among biological (e.g., sex and onset of puberty), psychological (e.g., insecure attachment, anxiety sensitivity, and depression), and socio-environmental factors (e.g., childhood trauma, social capital, and peer victimization (Engel, 1981; Hart et al., 2013; Ibeziako & Bujoreanu, 2011; Kugler et al., 2012; Nelson et al., 2017; Susman et al., 2003; Waldinger et al., 2006). With this new perception, somatic symptoms' etiological mechanisms have received increased attention across various disciplines in the past few decades (Ask et al., 2016; Brown, 2004; Dell & Campo, 2011; Kugler et al., 2012).

A brief review of Adverse Childhood Experiences

The research on adverse childhood experiences (ACEs) originated from the seminal Adverse Childhood Experiences study conducted by Kaiser Permanente and the Center for Disease Control and Prevention (CDC) between 1995 and 1997 (Felitti et al., 1998). This landmark study uncovered the strong graded dose-response relationship between childhood adversities and future health. Felitti et al. (1998) defined ACEs as negative experiences such as child maltreatment or household dysfunction occurring between ages 0 to 18, encompassing the

burdens of childhood adversity. The original ACEs measure consisted of child abuse (e.g., physical, psychological, and sexual abuse) and household dysfunction (e.g., household mental illness, household substance abuse, mother treated violently, parental separation or divorce, and criminal behavior in the household). Following over 17,000 adults, this study found strong relationships between ACEs and multiple health outcomes such as alcoholism, drug abuse, depression, suicide attempts, severe obesity, ischemic heart disease, cancer, chronic lung disease, and liver disease (Felitti et al., 1998). Since the original ACEs study, research on ACEs has proliferated, and studies have established that ACEs are important factors of psychosocial, behavioral, and physical health outcomes across the lifespan (Petruccelli et al., 2019).

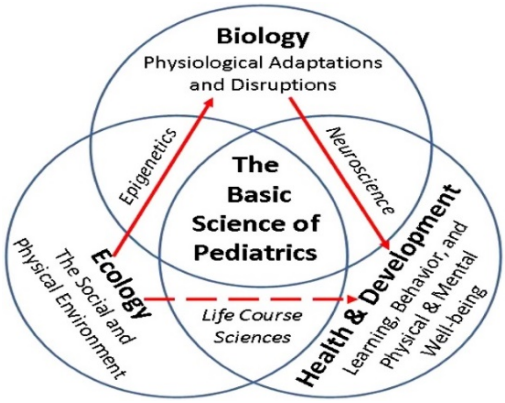
Theoretical framework

The Ecobiodevelopmental (EBD) framework (Shonkoff et al., 2012) is the guiding framework for this dissertation studying somatic symptoms and ACEs. The EBD framework incorporates the dynamic interaction between children's environments, the biology of toxic stress, and life course development to explain children's health outcomes. The EBD framework was developed by Shonkoff et al. (2012) to illustrate the relationship between early life experiences, ecological factors, and developmental and health outcomes via mechanisms of epigenetics and neuroscience. This framework conceptualizes human development as a function of an ongoing and complex interaction between biology and ecology (Shonkoff et al., 2012). In the heuristic model (Figure 1.1), the fundamental concepts of biology, ecology, and health/development are depicted as three separate circles that are integrated and meet in the middle. The concepts are connected by arrows that describe ecology influencing biology via epigenetics, biology affecting health and development by neuroscience, and ecology influencing health and development by life course sciences. This framework combines Bronfenbrenner's

Ecological model (1979) that describes multiple interacting systems of influence on a person's development and the Bio-developmental framework that proposes a science-based approach to understanding early childhood. With this framework, exposure to childhood adversity is viewed as an impetus for physiologic responses that manifest in health outcomes.

Figure 1.1

Ecobiodevelopmental Model (Shonkoff et al.,2012)



There are three major theoretical assumptions of this framework.

1) *Epigenetics*. Epigenetics explains how a person's ecology results in physiological adaptations and disruptions. The epigenetics theory posits that environmental influences and early adversity affect when, how, and to what degree different genes are expressed without altering the DNA sequence. Moreover, the epigenetic modification of genes modulates the expression of one's genotype, allowing specific personal characteristics or behaviors to be activated or prohibited. For example, exposures to early child adversities are hypothesized to affect later abdominal pain through epigenetic mechanisms leading to visceral hypersensitivity (Liu et al., 2017).

2) *Neuroscience*. A central neuroscientific mechanism between ACEs and somatic symptoms is through the concept of toxic stress. Significant stress in the lives of children

is viewed as a risk factor for the origin of unhealthy behaviors as well as an impetus for impaired physiologic responses that can manifest in the short term as well as long term health outcomes. Such results take effect through the mechanism of toxic stress. When a child is stressed, the child's body goes through a series of physiological changes in response to the stress. The child's brain activates the hypothalamic-pituitary-adrenocortical axis and the sympathetic-adrenomedullary system, releasing increased levels of stress hormones such as corticotropin-releasing hormone (CRH), cortisol, norepinephrine, and adrenaline. Simultaneously, elevated inflammatory cytokines are released in response to the parasympathetic nervous system. Although brief and mild to moderate activations of these stress hormones are necessary and beneficial for survival, excessive and prolonged exposures without protective factors can be toxic. Repeated exposure to stress is linked to prolonged activation of the allostatic systems strongly connected to the nervous, endocrine, and immune systems that mediate the allostatic processes. The allostatic process is a biological process where the body activates adaptive responses to adjust to external changes. Adversities negatively affect one's body by cumulative and prolonged exposure to toxic stress levels that burdens body functioning both physiologically and psychologically. This is also known as the "allostatic load" (McEwen, 2012). Toxic stress from ACEs and allostatic load disorders brain circuitry and other organs and metabolic systems in children. Dysregulation caused by the allostatic load is known to affect somatic symptoms through impairments in neurological functions leading to hyperarousal of the autonomic nervous system and disordered emotional processing. The allostatic load theory also hypothesizes that the frequency, type, and severity of stress have varying effects on the body. According to this theory, the effects

of maltreatment by type, frequency, and severity will be hypothesized to have varying effects on somatic symptoms (McEwen, 2012).

3) *Life-course theory*. The life course theory postulates that allostatic load and stress-induced changes in the body can have different effects throughout one's life span depending on the timing and contexts (Hussey et al., 2005; Juster et al., 2010). The life-course theory assumes that the timing of events- such as exposure to ACEs- has a differential impact on health outcomes. Mainly, life course theory highlights the importance of sensitive periods of development, such as early childhood and adolescence, on health outcomes. In line with this hypothesis, the timing of exposure to ACEs in childhood and adolescence are hypothesized to have different impacts on somatic symptoms.

In summary, using the EBD framework, ACEs are hypothesized to trigger activation of physiological and psychological processes that increase somatic symptoms. The EBD framework posits that children develop with reciprocity and mutual accommodation between the child and the dynamically nested social and physical systems. With this understanding, somatic symptoms will be viewed to be affected by childhood adversities from multiple levels of sources.

Literature review

Existing literature on somatic symptoms has mainly focused on the adult population. Although scarce, several longitudinal and cross-sectional studies document the significant association between ACEs and somatic symptoms in childhood and adolescence (Bonvanie et al., 2015, 2017; Flaherty et al., 2009; Hart et al., 2013; Paras et al., 2009; Rueness et al., 2020; Tang et al., 2020; Villalonga-Olives et al., 2011; Winding & Andersen, 2019; Zouini et al., 2019). Cross-sectional studies have found that somatic symptoms were positively associated with

family conflict among urban elementary school children in the U.S. (Hart et al., 2013). Also, Rueness et al. (2020) found a graded relationship between exposure to child abuse and somatic symptoms in adolescence and young adulthood, with differences between the types of childhood abuse. Longitudinal studies have reported a significant association between past ACEs and future somatic symptoms. For example, In a large population-based cohort study in the Netherlands, Bonvanie et al. (2017) found that ACEs in adolescence predicted higher levels of somatic symptoms during adolescence with a past-year diagnosis of anxiety and/or depression mediating the relationship between ACEs and somatic symptoms (Bonvanie et al., 2017). In a U.S. sample of children, Flaherty et al. (2009) found that experiencing five or more ACEs during childhood (Ages 0-12) was associated with increased somatic symptoms at age 12. Moreover, Winding and Andersen (2019) reported that poor family functioning at age 15 was associated with higher somatic symptoms at age 15 and 18 in Dutch adolescents.

As evidenced above, existing cross-sectional and longitudinal studies have supported that ACEs and somatic symptoms are associated with childhood and adolescence. However, few prospective studies have collected repeated measures of somatic symptoms and ACEs across different childhood and adolescent stages. Studies using prospective and repeated measures of somatic symptoms and ACEs could provide insight into the longitudinal relationships and trajectories across childhood and adolescence. Also, despite the substantial knowledge gained, several gaps remain. Among them are questions about the longitudinal relationship between ACEs and somatic symptoms in adolescence, the mediating role of anxiety and depression symptoms between ACEs and somatic symptoms, and the trajectory patterns of somatic symptoms across stages of childhood.

Purpose

The overarching goal of this dissertation is to advance the understanding of the effects of adverse childhood experiences on the etiology and development of somatic symptoms in childhood and adolescence.

Rationale

Given the high prevalence and the known adverse effects of somatic symptoms on children, families, and society, it is critical to develop and implement effective policies and practical interventions to support children experiencing somatic symptoms. To do so, a better understanding of the etiologies and development of somatic symptoms in childhood and adolescence is critical.

Specific Aims

Aim 1: Examine the longitudinal relationship between ACEs and somatic symptoms across ages 12-16 years, exploring the differential effects of accumulation, timing, and types of ACEs.

Aim 2: Examine if anxiety and depression symptoms in adolescence mediate the concurrent and longitudinal relationships between ACEs and somatic symptoms in adolescence across ages 12 to 16.

Aim 3: Identify and describe the heterogeneity of trajectory patterns of somatic symptoms across childhood ages 6-16 and potential predictors and outcomes.

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Chapter 2: The Longitudinal Relationship between Adverse Childhood Experiences (ACEs) and Somatic Symptoms in Adolescence: Exploring Accumulation, Timing, and Types of ACEs

Abstract

Objectives: This study examines the concurrent and longitudinal relationships between adverse childhood experiences (ACEs) and somatic symptoms across adolescence. This study specifically compares the effects of timing and type of ACEs on somatic symptoms in adolescents (ages 12-16 years).

Methods: This study uses prospective data of 1354 child and caregiver dyads from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN). Data collected from three time points during which child participants were ages 12, 14, and 16 are used to conduct multiple longitudinal path analyses.

Results: The results indicated a significant association between ACEs and increased concurrent somatic symptoms at age 12. Moreover, concurrent exposure to household dysfunctions had stronger effects on somatic symptoms at age 12 as compared with concurrent exposure to maltreatment.

Conclusion: These findings highlight the potential usefulness of using somatic symptoms as markers for concurrent exposure to ACEs. The findings suggest that the accumulation, timing and types of ACEs have unique effects on somatic symptoms in adolescence.

Introduction

Adolescence is a sensitive developmental period with remarkable plasticity for growth and risk for the onset of various health problems (Patton et al., 2016; Viner et al., 2017). Somatic symptoms, commonly understood as self-reported complaints of physical discomfort without an identified cause, have historically been highly prevalent among adolescents (Campo, 2012; Swain et al., 2014). The existing body of somatic symptoms suggest the damaging impacts of experiencing somatic symptoms during childhood and adolescence, including decreased emotional well-being, decreased attention, lower academic achievement, impaired cognitive functioning, increased healthcare utilization rate, and unnecessary expenses (Fremont, 2003; Groenewald et al., 2014; Kelly et al., 2010; Stone et al., 2019; Voerman et al., 2017; Williams & Zahka, 2017). Also, persistent somatic symptoms during childhood and adolescence predict future adverse health outcomes such as depression, anxiety, and suicidality (Andersen et al., 2009; Breidablik et al., 2009; Hoftun et al., 2011; Luntamo et al., 2014; Shelby et al., 2013). Despite the significant burdens that somatic symptoms inflict on the child, family, and society, the longitudinal dynamics and etiological mechanisms of somatic symptoms are not fully understood. This study addresses this gap in the literature by assessing ACEs as a significant contributing factor of somatic symptoms in adolescence using prospective longitudinal data.

Adverse childhood experiences and somatic symptoms

Common somatic symptoms in adolescents include headaches, stomachaches, fatigue, and muscle tension/pain (Campo, 2012; Williams & Zahka, 2017). Previous studies have argued that somatic symptoms tend to peak between the ages of 12 and 18 years (Rhee et al., 2005) and show a gradual increase from early to late adolescence (Campo, 2012; Swain et al., 2014). Due to the lack of identifiable organic causes, somatic symptoms cause a great deal of confusion for

children, families, and professionals. Historically, the traditional biomedical model of medicine that viewed the body and the mind separately could not explain nor provide adequate treatment for somatic symptoms (Engel, 1977). The paradigm shift to the biopsychosocial model within the past few decades has allowed for a more integrative view of somatic symptoms. Within the biopsychosocial perspective, somatic symptoms are understood as presentations of the dynamic reciprocity among biological, psychological, and socio-environmental factors (Engel, 1981; Nelson et al., 2017). With this advanced perception, the etiological mechanisms of somatic symptoms have received increased attention across various disciplines in the past few decades (Ask et al., 2016; Brown, 2004; Dell & Campo, 2011; Kugler et al., 2012). Notably, recent progress in neuroscience and epigenetics has provided a framework to describe the mechanisms and effects of stressors on physical health through the concept of toxic stress (Juster et al., 2010; McEwen & Gregerson, 2019; Shonkoff et al., 2012). Consequently, researchers have argued that significant stressors in childhood such as ACEs are strongly associated with both concurrent and future somatic symptoms (Bonvanie et al., 2015, 2017; Flaherty et al., 2009; Hart et al., 2013; Paras et al., 2009; Rueness et al., 2020; Tang et al., 2020; Villalonga-Olives et al., 2011; Winding & Andersen, 2019; Zouini et al., 2019). Exposure to ACEs causes somatic symptoms through interactions of biological (e.g., accelerated cellular aging, disruptions in neural development), emotional (e.g., increased emotional sensitivity, impaired emotion regulation), and social (e.g., increased hostile attribution bias, lack of social support) processes (Drury et al., 2014; Glaser et al., 2006; McLaughlin et al., 2020; Price & Glad, 2003). A growing body of literature has emphasized the significance of incorporating different aspects of ACEs, such as the accumulation, timing, and type, to more thoroughly examine the complex relationship between ACEs and health outcomes (Atzl et al., 2019; Negriff, 2020a; Schroeder et al., 2020).

Accumulation of adversity

The dose-response relationship between the accumulation of ACEs and somatic symptoms has been well-established by previous studies (Balistreri & Alvira-Hammond, 2016; Flaherty et al., 2009; Kerker et al., 2015; McCrae et al., 2019; Rueness et al., 2020; Tang et al., 2020; Thompson et al., 2015). To date, the majority of studies on ACEs have relied on index scores that sum the number of retrospective self-reports of ACEs to measure the accumulation of ACEs and their relationship with future health outcomes (Turner et al., 2020). Potential recall bias limits the validity of such an instrument. The prospective design in measuring ACEs in childhood may address this limitation (Reuben et al., 2016). However, very few studies have used the prospective longitudinal measures of ACEs to study their association with concurrent somatic symptoms.

Timing of adversity

The timing of when ACEs occur is a significant factor in the etiology of somatic symptoms—evidence for the importance of timing of adversity links to the sensitive periods of neural development. For example, Anderson et al. (2008) reported in a sample of young women with histories of childhood sexual abuse that hippocampal volume was most significantly associated with sexual abuse between the ages of 3 to 5 and 11 to 13. In contrast, the mid-portion of the corpus callosum and the prefrontal cortex gray matter volume were most susceptible between the ages of 9 to 10 and 14 to 16. Furthermore, Tomoda (2012) showed that portions of the visual cortex appeared to be most susceptible to witnessing domestic violence between ages 11 and 13. Studies focused on maltreatment generally agree that earlier ACEs are more strongly related to later anxiety, depression, and suicidal ideation in adolescence and adulthood (Dunn et al., 2013; Kaplow & Widom, 2007). However, recent findings suggest that these results may

differ by the type of adversity. For example, Niederkrotenthaler et al. (2012) noted that parental deaths and suicide had stronger effects when experienced at earlier ages. In Khan et al. (2015), suicidal ideation was predicted by parental verbal abuse at age 5 in males and sexual abuse at age 18 in females (Khan et al., 2015). Moreover, Harpur et al. (2015) found that late and cumulative maltreatment predicted depressive symptoms while early maltreatment predicted anxiety symptoms (Harpur et al., 2015). The relatively smaller amount of evidence regarding the timing of ACEs and mental health is due to the methodological limitations of previous studies, mainly using cumulative measures of ACEs (Fisher et al., 2011).

Studies that have assessed the importance of maltreatment timing and risk for psychopathology have traditionally dichotomized childhood exposure into early and later childhood. The results from these studies have been mixed. However, dichotomizing timing into early and late, or childhood and adolescence may be too simplistic. Previous literature supports that exposure during brief periods may have different impacts on mental health. For example, Kaplow and Widom (2007) have found that maltreatment exposure at ages 3 to 5 years was associated with higher post-traumatic stress disorder (PTSD) and depression rates than exposure at ages 0 to 2 or 6 to 8. Similarly, Dunn et al. (2013) found that exposure to maltreatment at ages 3 to 5 was more strongly associated with depression and suicidality than exposure at ages 0 to 2 or 6 to 8. However, no study to date has focused on shorter periods (such as two years) in adolescence.

Type of Adversity

Another significant aspect of ACEs is the type of adversity. ACEs are commonly categorized by two types: maltreatment and household dysfunctions. Among the subtypes, the most widely assessed types of adversities include the original CDC-Kaiser Permanent ACE study

items: physical abuse, emotional abuse, sexual abuse, neglect, the mother being treated violently, substance abuse in the household, mental illness in the household, parental separation or divorce, and an incarcerated household member (Felitti et al., 1998). The impact of different types of adversities on children's health outcomes has gained growing attention in the literature. When conventional ACEs index scores are used, all adversities are weighted equally, suggesting a linear effect (Schilling et al., 2008). Although this approach is useful in finding the cumulative effects of ACES, it does not convey information regarding the varying impacts of individual adversity on outcomes. Increasing evidence shows the different effects of the types of adversities on mental health outcomes (Atzl et al., 2019; Higgins, 2004; Narayan et al., 2017; Negriff, 2020a; Ryan et al., 2000). There are mixed results from these studies. For example, some studies point to maltreatment as a risk for internalizing symptoms, whereas household dysfunction variables are more predictive of externalizing problems (Higgins, 2004; Ryan et al., 2000). In contrast, other studies show that maltreatment experiences have more substantial effects on mental health outcomes (Atzl et al., 2019; Narayan et al., 2017; Negriff, 2020a). Atzl et al. (2019) compared the effects of childhood maltreatment and family dysfunction experiences on depression and PTSD symptoms among low-income pregnant women. This study's results indicated that higher total ACEs score and higher maltreatment scores predicted higher PTSD and depression symptoms during pregnancy, while family dysfunction did not. In a cross-sectional study, Narayan et al. (2017) compared the effects of child maltreatment and family dysfunction on the socioemotional problems of children aged four to six. This study's findings showed that childhood exposure to maltreatment, but not family dysfunction, significantly predicted elevations in children's emotional problems. Finally, Negriff (2020)'s cross-sectional study among youth (mean age=18) using self-reports of ACEs showed that different types of

adversities had different effects. For example, those who witnessed intimate partner violence reported symptoms of depression, anxiety, and trauma. At the same time, all of the maltreatment items were associated with significantly higher scores of depression, trauma, externalizing, and anxiety symptoms. In other words, when household dysfunction and maltreatment sum scores were entered into the model together, maltreatment primarily accounted for the majority of mental health symptoms. However, limited studies have compared the effects of maltreatment and household dysfunctions on somatic symptoms in adolescents.

In summary, ACEs have a lasting and harmful impact on one's health across the life course. Early identification and effective intervention are critical to prevent and mitigate ACEs' damaging effects (McEwen & Gregerson, 2019). However, the identification of individuals who are experiencing ACEs remains a significant barrier to sufficient intervention. Previous studies have suggested the usefulness of somatic symptoms in identifying children experiencing ACEs in community settings such as schools, primary care, and social services (Tietjen et al., 2015). Therefore, it is imperative to study the longitudinal relationship between ACEs and somatic symptoms in adolescence to better understand somatic symptoms' etiology related to ACEs.

Taken together, the literature on ACEs and somatic symptoms suggests that the accumulation, timing, and types of ACEs may have unique effects on somatic symptoms in adolescents. However, previous research on the longitudinal impact of ACEs on somatic symptoms has mainly focused on adult populations (Kisely et al., 2018; McCall-Hosenfeld et al., 2014; Smith & Flannery-Schroeder, 2013). Although a few studies on adolescents exist, most of them are limited to child maltreatment, such as abuse and neglect (Rueness et al., 2020; Tietjen et al., 2015). Furthermore, the majority of these studies depend on retrospective self-reports of ACEs (Petruccelli et al., 2019).

Current Study

The present study used a longitudinal path analysis method to investigate the longitudinal relationship between ACEs and somatic symptoms in adolescence by testing three hypothesized models (Figure 2.1) with the following specific aims and hypotheses based on the previous literature.

Aim1: To examine the effects of ACEs on concurrent and future somatic symptoms. It was hypothesized that experiencing a higher number of ACEs will predict higher concurrent and future somatic symptoms.

Aim2: To examine the autoregressive effects of somatic symptoms and ACEs. A higher number of ACEs was hypothesized to predict a higher number of ACEs in the following time points. Also, higher somatic symptoms in the past were hypothesized to predict higher somatic symptoms in the future.

Aim 3: To examine if the relationship between ACEs and somatic symptoms differ by the timing of ACEs. It was hypothesized that ACEs exposure during earlier periods of adolescence (i.e., measured at age 12, encompassing ACEs at 10-12 years) would have more significant effects on concurrent and longitudinal somatic symptoms compared to later periods of adolescence (i.e., measured at age 16, encompassing ACEs during 14-16 years).

Aim4: To compare the difference in effects mentioned above between the two ACEs types: maltreatment and household dysfunction. Maltreatment was hypothesized to have more potent effects on concurrent and future somatic symptoms compared with household dysfunction.

Methods

Design & Procedure

This study was a secondary data analysis from a longitudinal prospective design study, the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN; (Runyan et al., 2014). LONGSCAN was a consortium of five longitudinal studies that used standard procedures conducted at five different sites: East (EA), Midwest (MW), Northwest (NW), Southwest (SW), and South (SO). The sites' settings were urban for EA, MW, and NW, suburban for SW, and a combination of urban, suburban, and rural for the SO site. LONGSCAN's purpose was to study the etiology and effects of child maltreatment on various ecological level factors. LONGSCAN followed 1,354 child and primary caregiver dyads until the children were 18 years old. Data were collected when children were ages 4, 6, 8, 12, 14, 16, and 18 years using comprehensive face-to-face interviews and Audio Computer-Assisted Self Interviews (ACASI) method. Data collection started in 1991 and ended in 2012. This current study will use data from waves 4, 5, and 6, collected when children were ages 12, 14, and 16 years.

Participants

Each site had a unique sampling methodology that allowed for systemic variation in the level of risk for maltreatment. NW site's sample included children reported to Child Protective Services (CPS) with two groups based on maltreatment substantiation status. MW site included a sample of children reported to CPS and neighborhood controls. EA sample consisted of children at risk for failure to thrive in the first year, parent HIV infection or drug use, and neighborhood control. SO site's sample included children who were at high risk of maltreatment at birth and consisted of two groups based on the indication of CPS reports at age 4. The SW site's sample

included children who were removed from their homes and placed into foster care due to substantiated maltreatment. The baseline sample of LONGSCAN included 1354 participants. Of the 1354 participants who were recruited at baseline, 977 (72.2%) completed either child or caregiver interview at age 12, 962 (71.0%) completed either child or caregiver interview at age 14, and 901 (66.5%) completed either child or caregiver interview at age 16. Across the three-time points of 12, 14, and 16 years, 1137 (84.0%) had at least one interview completed. 999 (73.8%) completed at least two interviews out of three-time points. 704 (52.1%) completed interviews at all three-time points. Chi-square tests and t-tests were conducted to test the difference in demographic variables between the various levels of study completion. Results indicated that participants from the MW site, compared to those from other sites, were more likely to have completed all three interviews and at least two interviews. Also, dyads with a child identified as Black/African American at baseline were more likely to have complete at least one interview. Therefore, the models were controlled for site and race in addition to the child's sex. Using the Full-Information Maximum Likelihood (FIML) estimation, which estimates parameters using any available information in the dataset, all 1354 samples were included in the analysis (Enders, 2001). Table 2.1 describes the demographic characteristics of the sample population.

Table 2.1

Baseline demographic characteristics of sample population (N=1354)

	n	%
Child's sex		
Female	697	51.48
Male	657	48.52
Child's race		
Black	721	53.3
White	354	26.1
Hispanic	97	7.2
Other[2]	181	13.4
Site		
EA	282	20.83
MW	245	18.09
NW	254	18.76
SO	243	17.95
SW	330	24.37
Caregiver Marital Status		
Not married[3]	893	65.95
Married	444	32.79
Caregiver Education		
≤12 years	458	33.83
≥12 years	882	65.14
Household Annual Income		
<$15,000	703	51.92
≥$15,000	619	45.72

Note.[1] Measured at age 4 or 6. [2] [1]Other included Native American, Asian, Mixed Race, and Other races. [3]Includes never married, separated, divorced, or widowed. EA=East, MW=Midwest, NW=Northwest, SO=South, SW=Southwest

Measures

Somatic symptoms

Somatic symptoms were assessed using the Child Behavior Checklist (CBCL) 4-18 (Achenbach, 1991). The CBCL is a widely used tool for assessing children's internalizing and externalizing behaviors. This study used the somatic complaints subset of the CBCL measure. Primary caregivers were asked to evaluate the child's behavior over the past six months on a 3-point Likert scale (0=not true (as far as you know), 1= somewhat true or sometimes true, 2= very true or often true). The somatic complaints subset asked about nine physical problems without known medical causes- including feels dizzy, overtired, aches or pains, headaches, nausea, eye problems, rashes or skin problems, stomachaches or cramps, and vomiting or throwing up. Raw scores of somatic complaints items were calculated by summing the item scores with a possible range from 0 to 18. Cronbach's alpha at age 12,14, and 16 were 0.723, 0.68, 0.72 respectively. Raw scores were used in the structural equation models. Clinically significant and borderline levels of somatic symptoms were calculated using the age-standardized T-scores and were used for descriptive purposes.

Adverse Childhood Experiences (ACEs)

Assessment of ACEs in the past year was based on those used in the original ACEs study (Felitti et al., 1998). Nine total variables were used to assess ACEs. Dichotomous scores (0=No, 1=Yes) on each variable were summed to create an overall ACEs index score, with scores ranging from 0 to 9 and higher score indicating more exposure to ACEs. Table 2.2 summarizes the instruments and time points at which each ACE item was measured. ACEs assessed in this

study included two broad categories of childhood experiences: child maltreatment and household dysfunction.

Table 2.2

Measurement Instruments Used to Construct ACEs Scores

	ACE	Instrument	Respondent	Time Frame
Child maltreatment	Physical abuse	MMCS	Study staff	Past two years
	Sexual abuse	MMCS	Study staff	Past two year
	Emotional abuse	MMCS	Study staff	Past two year
	Neglect	MMCS	Study staff	Past two year
Household dysfunction	Caregiver Divorce/Separation	Child Life Events[b]	Caregiver	Past year
	Caregiver Depressive Symptoms	CES-D	Caregiver	Past week
	Caregiver Treated Violently	Partner to Partner Conflict Tactics Scale[d]	Caregiver	Past year
	Household Criminal Activity	Child Life Events[b]	Caregiver	Past year
	Household Substance Abuse	Risk Behaviors of Family & Friends[e]	Child	Currently

Note.

MMCS= Modified Maltreatment Coding System, Barnett,Manly,& Cicchetti (1993).

[b] LONGSCAN (1992).

CES-D=Center for Epidemiological Studies Depression Scale, Radloff (1977).

[d] Straus et al (1998); [e]LONGSCAN (1998).

Child Maltreatment

Each site systematically reviewed the CPS administrative records for child maltreatment allegations every two years. CPS records were reviewed and coded by highly trained coders using the Modification of Maltreatment Classification System (MMCS; (Barnett et al., 1993; English & LONGSCAN, 1997). Using the MMCS, four types of categories were created: physical abuse, sexual abuse, emotional abuse, and neglect. Physical abuse was defined as "caregiver or responsible adult inflicts physical injury upon a child by other than accidental means." Sexual abuse was defined as "any sexual contact or attempt at sexual contact occurs between a caregiver or other responsible adult and a child, for purposes of the caregivers' sexual gratification or financial benefit." Emotional maltreatment was defined as" persistent or extreme thwarting of children's basic emotional needs such as parental acts that are harmful because they are insensitive to the child's developmental level." Neglect was defined as "failure to provide the child's physical needs and/or failure to provide supervision that ensures the child's safety" (Barnett et al.,1993). Maltreatment allegations between ages 10-12, 12-14, and 14-16 were included in this analysis. Dichotomous codes based on the absence or presence of four maltreatment allegations (e.g., physical abuse, emotional abuse, sexual abuse, neglect) in each time point were summed to create an index maltreatment score ranging from 0 to 4 were created for each time point with a higher score indicating more exposure to maltreatment.

Household dysfunction

Household dysfunction included five items: caregiver separation or divorce, caregiver depressive symptoms, caregiver treated violently, household criminal activity, and household substance use.

Caregiver Separation or Divorce. The Child Life Events measure (LONGSCAN, 1992) was administered to caregivers at ages 12, 14, and 16 years and asked about caregiver separation or divorce in the past year. An affirmative response to caregiver separation or divorce between parents or parent-figures of the child was coded as 1.

Caregiver Depressive Symptoms. Caregiver depressive symptoms were measured using the Center for Epidemiological Studies Depression Scale (CES-D) (Radloff, 1977) administered to caregivers at ages 12,14, and 16 years. The CES-D scale assesses symptoms associated with depression in the past week. CES-D scores were dichotomized to the widely used cut off points at 16 or greater=1 and less than 16=0 (Lewinsohn et al., 1997), with higher scores indicating more depression symptoms.

Caregiver Treated Violently. Intimate partner violence within the past year was assessed using the Partner to Partner Conflict Tactics Scale (Straus et al., 1998) administered at ages 12,14, and 16 years. This measure assessed the type and severity of conflict tactics used between the child's primary caregiver and their partner during the past year. The caregiver's partner could be partners that the caregivers were dating, cohabiting, or marital relationships. Physical assault subscale within this measure -which consisted of items such as partner threw something, twisted or pulled hair, pushed or shoved, grabbed, slapped, used a knife or gun on me, punched or hit, choked, slammed against the wall, beat up, burned or scalded, kicked. The Sum score of physical assault was dichotomized to 0=none and 1=score greater than 1. The Southwest site did not collect this measure at age 14, and the South and Southwest sites did not collect this measure at the age 16 interview.

Household Criminal Activity. The household criminal activity was assessed by the Child Life Events Measure (LONGSCAN, 1992), which was administered to primary caregivers at the

age 12, 14, and 16 interviews. It asked if anyone in the child's family or household was jailed, imprisoned, or kept in a juvenile residential facility for breaking the law. Answers were dichotomized to 0=No and 1=Yes.

Household Substance Use. Household substance use was measured by the Risk Behaviors of Family and Friends measure developed by LONGSCAN, 1998. This measure was administered to the child at age 12,14, and 16 and asked if anyone the child lives with uses drugs or alcohol. An affirmative answer to using marijuana, cocaine or crack, meth, injection drugs, other drugs, and getting drunk or high was coded as 1, otherwise 0.

Demographic control variables. Control variables included the child's biological sex assigned at birth (male, female), race/ethnicity, and geographical sites of data collection. Race/ethnicity and geographical sites were entered into the models using dummy-coded variables.

Statistical Analyses

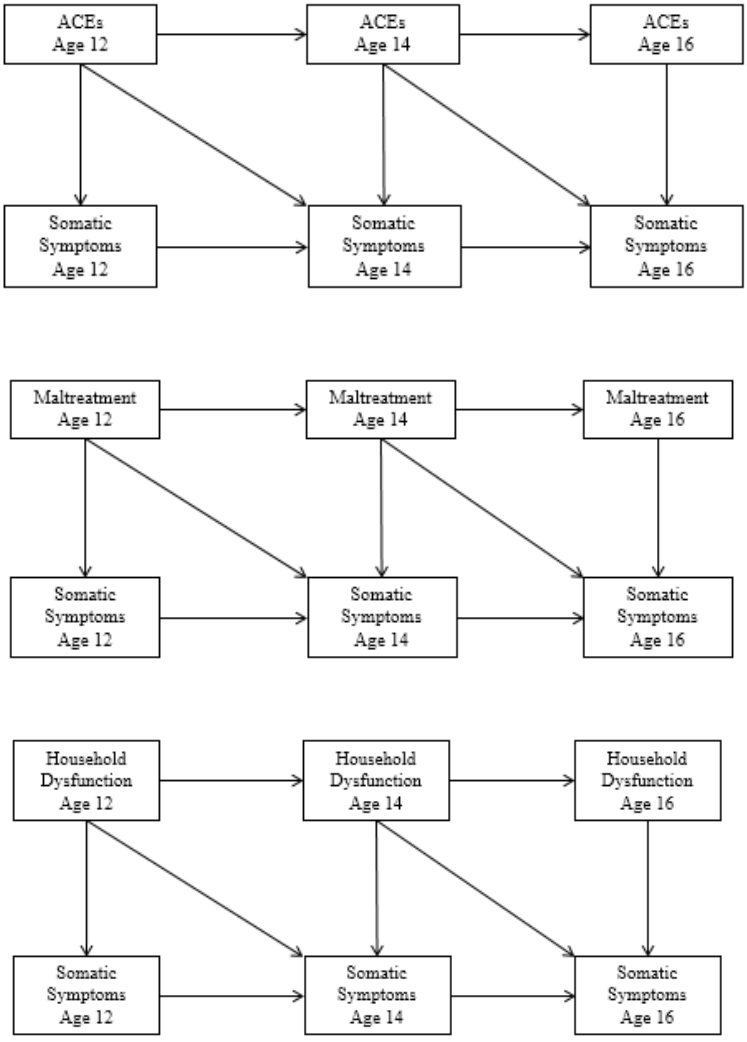
Descriptive and bivariate correlation analyses were conducted using SPSS Version 27.0. Listwise and pairwise deletion for missing data were used in descriptive and bivariate correlations, respectively. T-tests, ANOVA, and chi-square tests were used to assess whether there were significant differences in somatic symptoms and ACEs between demographic variables. Longitudinal path analyses were conducted using Mplus Version 8.0 (Muthén & Muthén, 2017). To answer aims 1 and 2, a longitudinal path analysis was conducted to simultaneously determine the autoregressive, concurrent, and cross-lagged effects between ACEs and somatic symptoms across ages 12,14, and 16. For study aim 3, two different longitudinal

path analyses were conducted with maltreatment and household dysfunction variables. Each model was controlled for the child's sex, race, and sites, which were dummy coded.

Figure 2.1 depicts the hypothesized path analysis models. To account for missing data, models were estimated using the full-information maximum likelihood (FIML), which uses all available observed data to produce unbiased parameter estimates and standard errors when data are missing at random (MAR) or missing completely at random (MCAR) (Enders, 2001). To account for the non-normal distribution of the outcome variables, robust maximum likelihood estimation (MLR) was used to estimate the parameters and standard errors. Model fit was evaluated by jointly assessing non-significant chi-square test statistics of the exact-fit hypothesis, Root Mean Squared Error of Approximation (RMSEA) below 0.06, Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) greater than 0.95, and Standardized Root Mean Residual (SRMR) below 0.08 (Hu & Bentler, 1999; Kline, 2015). Benjamini-Hochberg false discovery rate correction was conducted due to the large number of tests conducted (Benjamini & Hochberg, 1995; C. E. Smith & Cribbie, 2013). All parameters that were initially reported to be significant remained significant after the correction.

Figure 2.1

Hypothesized Models



Results

Descriptive Analyses

Table 2.3 displays the descriptive statistics and bivariate correlations among the variables in the study. No correlation coefficients were above .85, suggesting the absence of multicollinearity (Kline, 2015). Somatic symptoms at ages 14 and 16 were positively correlated with being a female. ACEs were significantly correlated with somatic symptoms, although the strength of correlations was generally weak (r=.11-.2).

Table 2.3

Descriptive Statistics and Correlations Among Study Variables

Variable	N	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Sex[a]	1354	0.51	0.50	--												
2. Age12 SOM	951	1.53	2.05	.05	--											
3. Age14 SOM	930	1.51	1.97	.09**	.44**	--										
4. Age16 SOM	867	1.48	2.07	.14**	.33**	.40**	--									
5. Age12 ACEs	918	0.94	1.16	-.03	.23**	.15**	.11**	--								
6. Age14 ACEs	907	0.90	1.16	-.01	.17**	.16**	.16**	.39**	--							
7. Age16 ACEs	831	0.86	0.98	.02	.16**	.16**	.15**	.32**	.37**	--						
8. Age12 MT	1354	0.23	0.63	.00	.08*	.12**	.10**	.62**	.22**	.16**	--					
9. Age14 MT	1354	0.20	0.61	.01	.09**	.10**	.15**	.24**	.64**	.21**	.28**	--				
10. Age 16 MT	1354	0.17	0.57	.07**	.06	.10**	.12**	.12**	.18**	.59**	.16**	.22**	--			
11. Age 12 HD	918	0.69	0.92	-.03	.23**	.10**	.08*	.83**	.34**	.29**	.07*	.11**	.01	--		
12. Age 14 HD	907	0.70	0.90	-.02	.18**	.14**	.11**	.35**	.84**	.34**	.07*	.13**	.09**	.38**	--	
13. Age 16 HD	831	0.71	0.80	-.03	.15**	.13**	.13**	.31**	.34**	.86**	.07*	.11**	.09*	.34**	.36**	--

Note. N=1354. [a]0=male, 1=female.

*p<.05,**p<.01.

SOM=Somatic symptoms, ACEs= Adverse Childhood Experiences, MT=Maltreatment, HD=Household Dysfunction.

Table 2.4 summarizes the descriptive statistics of somatic symptoms across time points. Headache was the most prevalent somatic complaint through age 12, 14, and 16. Being overtired was the second most prevalent, and stomachaches were the third most prevalently reported somatic symptoms across ages 12, 14, and 16. Between 8-10% of children scored somatic symptoms scores that are significant for borderline/clinical range.

Table 2.4

Descriptive Statistics of Somatic Symptoms

	Age 12		Age14		Age 16	
	n=951		*n*=930		*n*=867	
	M	SD	M	SD	M	SD
Somatic symptoms raw score (0-18)[1]	1.53	2.05	1.51	1.97	1.48	2.07
Somatic symptoms individual item scores						
Headaches	0.33	0.53	0.32	0.53	0.30	0.52
Stomachaches	0.24	0.48	0.23	0.48	0.21	0.45
Overtired	0.26	0.49	0.30	0.52	0.30	0.54
Rashes/skin	0.18	0.44	0.19	0.48	0.17	0.44
Aches/pain	0.19	0.46	0.15	0.41	0.16	0.41
Nausea	0.13	0.36	0.13	0.35	0.11	0.35
Dizzy	0.07	0.28	0.07	0.27	0.08	0.31
Vomiting	0.06	0.25	0.04	0.23	0.07	0.28
Eye problems	0.07	0.30	0.08	0.33	0.08	0.34
	n	%	*n*	%	*n*	%
Borderline or clinical range[2]						
Yes	92	9.7	87	9.4	71	8.2
No	859	90.3	843	90.6	796	91.8

Note.

[1] sum of individual item scores, [2]T score ≥67

Table 2.5 presents the descriptive analysis of ACEs scores. Among maltreatment variables, neglect was the most frequently reported across ages 12, 14, and 16. Sexual abuse was the least common adversity across all time points. Among household dysfunction variables, caregiver

depressive symptoms were the most prevalent adversity throughout ages 12, 14, and 16. Caregiver divorce/separation was least frequently reported across the time points. More than half of the participants reported experiencing one or more ACEs across all time points.

Table 2.5

Descriptive *Statistics of ACEs Variables*

Maltreatment	Age 12		Age 14		Age 16	
	n=1354		*n*=1354		*n*=1354	
	n	*%*	*n*	*%*	*n*	*%*
Physical abuse	90	6.6	82	6.1	60	4.4
Sexual abuse	29	2.1	31	2.3	32	2.4
Neglect	113	8.3	89	6.6	78	5.8
Emotional abuse	80	5.9	71	5.2	59	4.4
Household Dysfunction	**Age 12**		**Age 14**		**Age 16**	
	n=956		*n*=937		*n*=868	
	n	*%*	*n*	*%*	*n*	*%*
Caregiver Divorce/Separation	77	5.7	69	7.4	44	5.1
Caregiver Depressive Symptoms	238	26.9	232	27.4	254	31.5
Caregiver Treated Violently	102	14.2	63	11.4	28	7.4
Household Criminal Activity	103	10.8	120	12.8	128	14.9
Household Substance Problem	127	14.4	158	18.4	161	20.1
Number of ACEs	**Ages 10-12**		**Ages 12-14**		**Ages 14-16**	
	n=918		*n*=907		*n*=831	
	n	*%*	*n*	*%*	*n*	*%*
0	443	48.3	437	48.2	372	44.8
1	237	25.8	263	29.0	281	33.8
2	135	14.7	118	13.0	119	14.3
3+	103	11.2	89	9.8	59	7.1

Note. % indicates the percentage of participants who answered yes

Aim 1 &2: Longitudinal path analyses of ACEs and somatic symptoms

To examine aim 1 and 2, the hypothesized model with ACEs and somatic symptoms (see Figure 2.1) was fitted. The initial model yielded poor model fit with $\chi 2$ (6)=41.503, p<0.0001, CFI=0.948, TLI=0.478, SRMR=0.023, and RMSEA=0.066. Modification indices suggested further improvements could be made by freely estimating the path from somatic symptoms at age 12 to somatic symptoms at age 16 (MI=13.450, EPC=0.163, STDYX E.P.C.=0.160) and the path from ACEs at 12 to ACEs at 16 (MI=18.519, EPC=0.170, STDYX E.P.C.=0.200). Re-specified model with above paths freed achieved a good model fit, $\chi 2$ (4)=8.103, p=0.089, CFI=0.994,TLI=0.909,SRMR=0.011,and RMSEA=0.028 and modification indices did not suggest any additional improvements to the model. As hypothesized, each variable in the previous wave positively predicted the next wave(s). These results indicate the stability of ACEs and somatic symptoms over time. Concurrent paths between age 12 ACEs and age 12 somatic symptoms were significant. None of the cross-lagged paths were significant.

Aim 3: Longitudinal Path Analyses with Maltreatment and Somatic symptoms vs. Household Dysfunction and Somatic symptoms

Applying the earlier ACEs model results, the paths between age 12 and 16 of ACEs and somatic symptoms were estimated in both the maltreatment and household dysfunction model. The maltreatment model showed acceptable fit with $\chi 2$ (4)=4.124, p=0.3895, CFI=1.00,TLI=0.997,SRMR=0.007,and RMSEA=0.005. Household dysfunction model also demonstrated acceptable fit with $\chi 2$ (4)=9.223, p=0.0558, CFI=0.993,TLI=0.889,SRMR=0.011,and RMSEA=0.031. Similar to the ACEs model, both maltreatment and household dysfunction models resulted in significant autoregressive paths; in other words, past maltreatment predicted higher maltreatment in the following waves, and past household dysfunction predicted higher household

dysfunction in the following waves. All concurrent and cross-lagged paths across ages 12, 14, and 16 in the maltreatment model were non-significant. However, household dysfunction at age 12 significantly and positively predicted somatic symptoms at age 12. Table 2.6, Figure 2.2, Figure 2.3, and Figure 2.4 present the path analysis results. The effects of control variables are not displayed in the figures for clarity of the figures.

Table 2.6

Summary of Longitudinal Path Analyses Results

ACEs Model		
	β	SE
Autoregressive paths		
Age 12 SOM→Age14 SOM	0.41***	0.04
Age 14 SOM→Age16 SOM	0.28***	0.05
Age 12 SOM→Age16 SOM	0.18**	0.05
Age 12 ACEs→Age14 ACEs	0.39***	0.04
Age 14 ACEs→Age16 ACEs	0.29***	0.05
Age 12 ACEs→Age16 ACEs	0.20***	0.04
Concurrent paths		
Age 12 ACEs→Age 12 SOM	0.21***	0.03
Age 14 ACEs→Age 14 SOM	0.07	0.04
Age 16 ACEs→Age 16 SOM	0.03	0.04
Cross-lagged paths		
Age 12 ACEs→Age 14 SOM	0.03	0.04
Age 14 ACEs→Age 16 SOM	0.05	0.06
Maltreatment Model		
	β	SE
Autoregressive paths		
Age 12 SOM→Age14 SOM	0.42***	0.04
Age 14 SOM→Age16 SOM	0.28***	0.05
Age 12 SOM→Age16 SOM	0.18**	0.05
Age 12 MT→Age14 MT	0.24***	0.04
Age 14 MT→Age16 MT	0.17***	0.05
Age 12 MT→Age16 MT	0.10**	0.04
Concurrent paths		
Age 12 MT →Age 12 SOM	0.06	0.03
Age 14 MT →Age 14 SOM	0.04	0.04
Age 16 MT →Age 16 SOM	0.04	0.05
Cross-lagged paths		

Age 12 MT →Age 14 SOM	0.05	0.04
Age 14 MT →Age 16 SOM	0.06	0.05
Household Dysfunction Model		
	β	SE
Autoregressive paths		
Age 12 SOM→Age14 SOM	0.42***	0.04
Age 14 SOM→Age16 SOM	0.28***	0.05
Age 12 SOM→Age16 SOM	0.18**	0.05
Age 12 HD→Age14 HD	0.38***	0.04
Age 14 HD →Age16 HD	0.26***	0.05
Age 12 HD →Age16 HD	0.25***	0.04
Concurrent paths		
Age 12 HD →Age 12 SOM	0.22***	0.03
Age 14 HD →Age 14 SOM	0.06	0.03
Age 16 HD →Age 16 SOM	0.05	0.03
Cross-lagged paths		
Age 12 HD →Age 14 SOM	-0.002	0.04
Age 14 HD →Age 16 SOM	0.007	0.04

Note.

*p<.05, **p<.01, ***p<.001.

β = standardized beta.

SOM=Somatic symptoms, ACEs= Adverse Childhood Experiences, MT=Maltreatment, HD=Household Dysfunction.

Figure 2.2

Standardized Results of Longitudinal Path Analyses Between ACEs and Somatic symptoms

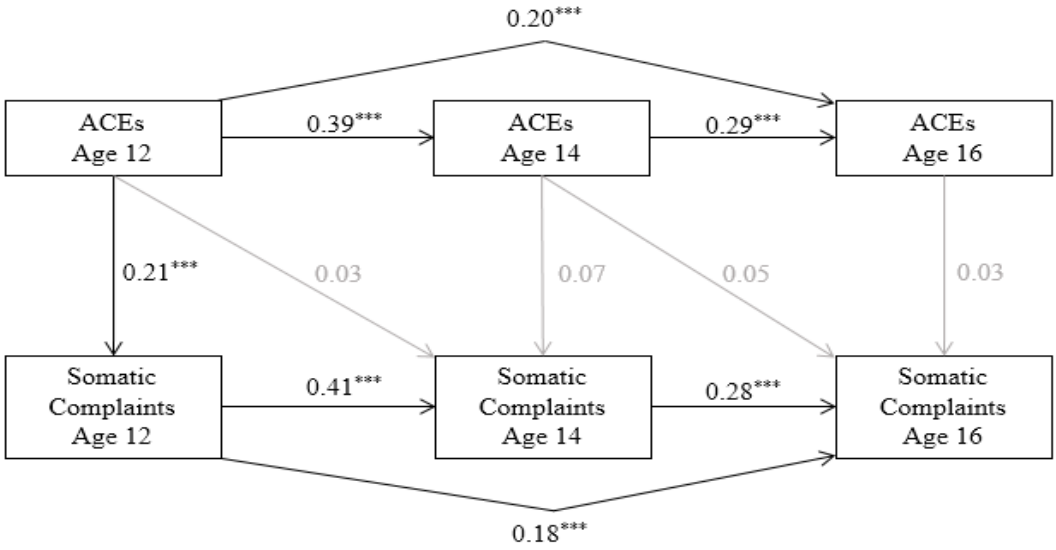


Figure 2.3

Standardized Results of Longitudinal Path Analyses Between Maltreatment and Somatic symptoms

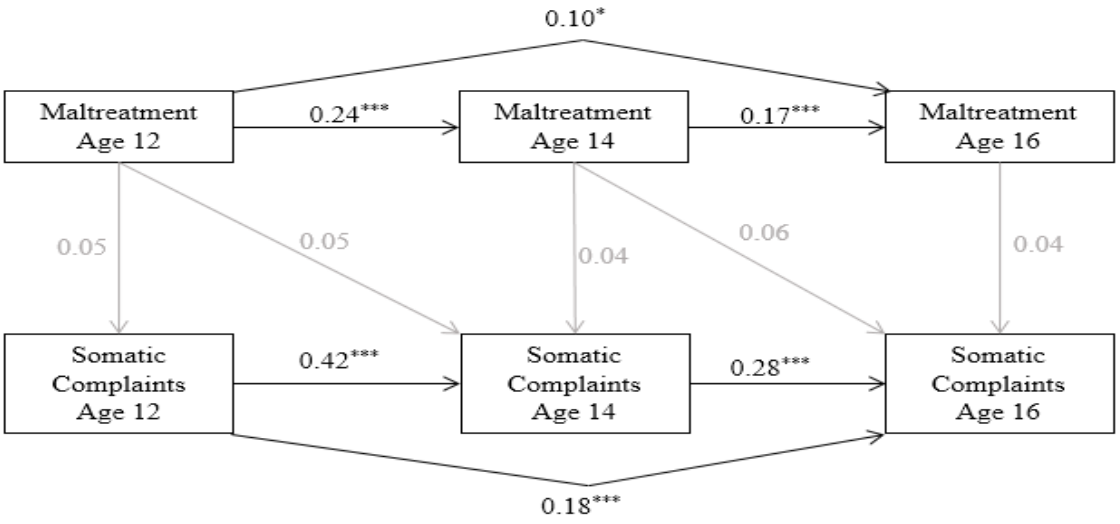
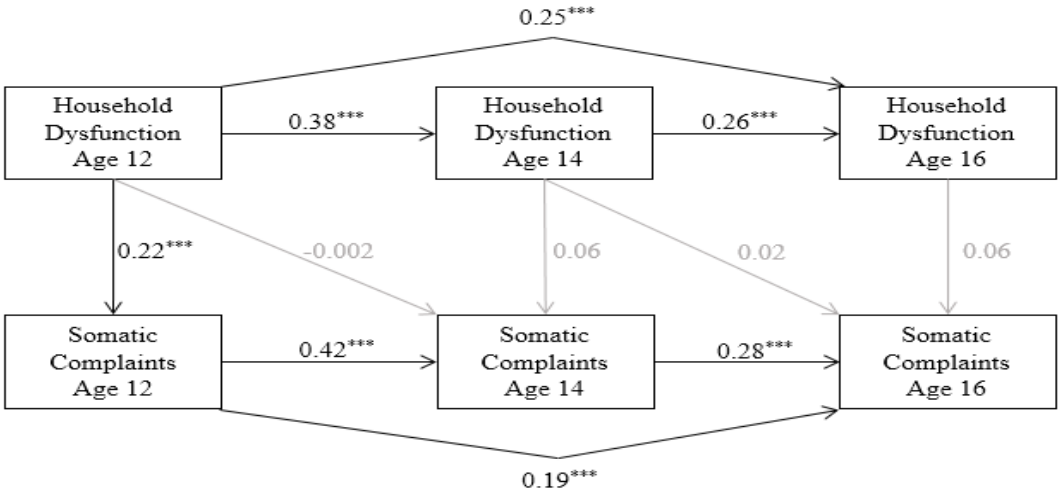


Figure 2.4

Standardized Results of Longitudinal Path Analyses Between Household Dysfunction and Somatic symptoms



Discussion

Despite the well-established link between ACEs and somatic symptoms, significant uncertainty remains regarding the longitudinal relationships between ACEs and somatic symptoms. The present study examines the relationship between ACEs and somatic symptoms across ages 12-16 years.

Concurrent and future effects of ACEs on somatic symptoms

This study's first aim was to examine the effect of ACEs on concurrent and future somatic symptoms in adolescents. The study's results do not support the hypothesis that ACEs are predictive of future somatic symptoms in adolescents. None of the models' cross-lagged paths show evidence that there was a significant relationship between ACEs and somatic symptoms after two years. This result is contrary to the findings of other prospective longitudinal studies that suggest associations between ACEs and somatic symptoms in the future (Bonvanie et al., 2017; Groenewald et al., 2020; McCall-Hosenfeld et al., 2014; Wooldridge et al., 2020). For example, Bonvanie et al. (2017) have found that the severity of adverse life events experienced in the previous two to three years significantly predicted somatic symptoms in adolescents ages 13-19 (Bonvanie et al., 2017). Moreover, Groenwald et al. (2020) found that cumulative ACEs predicted chronic somatic symptoms in children ages six to 17 years (Groenewald et al., 2020). A possible explanation for this discrepancy may be the relatively long lag of two years in the current study. Furthermore, the ACEs captured in this study refer to experiences in the past one to two years. Therefore, the ranges of the ACE counts were not as wide as those of the cumulative ACE counts often used in other studies, which limits the strength of the association. The concurrent paths reveal that the association between ACEs and somatic symptoms was significant at age 12 but not at age 14 and 16. This significant concurrent

association at age 12 aligns with the association observed in previous research (Winding & Andersen, 2019). The difference in the strength and significance of associations by age may be partly explained by developmental differences at ages 12, 14, and 16. During early adolescence, children experience higher proximity to the family environment, which may increase the effects of family dysfunctions compared with middle adolescence (Sawyer et al., 2012; Steinberg, 2005). In summary, this finding for the first aim suggests that current ACEs experienced by children have a more substantial impact on somatic symptoms than ACEs experienced in the recent past. This finding supports the use of somatic symptoms as potential markers for current ACEs in adolescents.

The longitudinal stability of ACEs and somatic symptoms

The second aim of this study was to examine the autoregressive effects of somatic symptoms and ACEs. The results indicate that higher somatic symptoms at age 12 predict higher somatic symptoms at ages 14 and 16. This finding supports the well-established literature on the stability of somatic symptom levels across childhood and adolescence (Janssens et al., 2010; Kim et al., 2019). This result is also supported by the descriptive analysis results, which reveal that approximately 8-10% of children persistently scored somatic symptom levels that were borderline or clinically significant. The path coefficients for ACEs show that ACEs at age 12 predict higher ACEs at ages 14 and 16. ACEs at age 14 also predict higher ACEs at age 16. This finding highlights the persistence of ACEs across adolescence, which aligns with the findings of other longitudinal studies (Dubowitz et al., 2019; Thompson et al., 2015).

Different effects of maltreatment and household dysfunction

This study's third aim was to compare the effects of two ACE types: maltreatment and household dysfunction. The autoregressive path coefficients for both maltreatment and household dysfunctions were significant in this study. However, the path coefficients for household dysfunctions were stronger, suggesting that household dysfunctions across adolescence are more enduring as compared with maltreatment. The concurrent path results demonstrate that household dysfunctions were stronger predictors of somatic symptoms at age 12 than maltreatment. This finding differs from the results of previous studies showing stronger effects of maltreatment as compared with household dysfunctions on depression, trauma, and anxiety symptoms in adolescence and adulthood (Atzl et al., 2019; Narayan et al., 2017). This inconsistency may be partly explained by the difference in ACE measures. In this study, the official CPS allegations of maltreatment were compared to the self-reports used in other studies (Atzl et al., 2019; Negriff, 2020b). The use of CPS reports is a strength of this current study as reliance on self-reports for longitudinal assessments of maltreatment may lead to self-report bias. However, it is possible that the official records of CPS allegations may not accurately capture the prevalence or perception of maltreatment experienced by the child (Dubowitz et al., 2019). Moreover, maltreatment in this study was measured for the past two years. It is possible that maltreatment that the children were concurrently experiencing at the time of the interview were not included. In addition, four out of five household dysfunction measures were based on caregiver reports in this study, whereas Atzl et al. (2019) and Negriff (2020a) used self-reported measures of household dysfunctions. There may be biases in caregiver reports, and official records may limit reliable assessments of adversities. Future research should consider using multi-informant methods to assess ACEs. Overall, the results support the broader hypothesis that

the types of adversities have a distinct impact on somatic symptoms (McLaughlin et al., 2014; Turner et al., 2020).

Limitations

Several limitations of this study should be discussed. First, somatic symptoms in this study were measured using the caregiver reported CBCL. Although the caregiver-reported CBCL is a widely-used instrument to assess somatic symptomatology, poor to low parent-child agreement has been reported (Salbach-Andrae et al., 2009). Combining youth self-reports and/or other informant reports may provide a more accurate and comprehensive view. Furthermore, internal consistency among somatic symptom items was moderate (0.68 - 0.72). Thus, a possible measurement error must be acknowledged. Second, although longitudinal path analysis models are useful in simultaneously estimating multiple paths, this model does not account for the variability in longitudinal changes across individuals. Future research should utilize alternative methods, such as latent growth modeling, to capture individual variabilities. Third, maltreatment in this study was measured by official CPS records, and family dysfunctions were mostly assessed using caregiver reports. Such measures may not accurately assess children's ACEs due to overreporting or underreporting (Hardt & Rutter, 2004). Future studies should consider using multi-informant methods to measure ACEs. Fourth, this study relies on the original ACE study to select the ACE items (Felitti et al., 1998). However, many researchers have criticized the predictive power of these items and note that this index fails to include broader types of adversities, such as poverty, community violence, homelessness, and racism, that have been known to be strongly associated with health outcomes (Finkelhor, 2018; McEwen & Gregerson, 2019; Turner et al., 2020). Finally, this study used a sample from a longitudinal study with significant attrition. A possible selection bias should be considered.

Conclusion

In summary, this study's findings suggest that the accumulation, timing, and types of ACEs have unique effects on somatic symptoms in adolescence. The results highlight that ACEs experienced at age 12 were significantly associated with somatic symptoms at age 12. Moreover, compared with maltreatment, household dysfunctions were more strongly related to somatic symptoms at age 12. The significant concurrent effects of ACEs on somatic symptoms at age 12 suggest that somatic symptoms are a potentially useful indicator of concurrent exposure to ACEs, particularly in early adolescence. Parents, teachers, school nurses, counselors, and primary health care providers are often the first people to recognize somatic symptoms in adolescents. Providers should consider that somatic symptoms at age 12 may indicate concurrent experiences of adversities and make evidence-based care decisions such as screening for ACEs and making appropriate referrals.

Further work is needed to delineate the mechanism in which different aspects such as the accumulation, timing, and types of ACEs impact somatic symptoms. Future work should also focus on the impact of ACEs on somatic symptoms at age 12 and investigate other predictors and protective factors. The strong effects of household dysfunction on somatic symptoms suggest that interventions on the family and individual levels should be implemented to mitigate the effects of ACEs on concurrent somatic symptoms.

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Chapter 3. The Mediating Role of Anxiety/Depression Symptoms between ACEs and Somatic Symptoms in Adolescents

Abstract

Objectives: This study seeks to examine the relationships among adverse childhood experiences (ACEs), somatic symptoms, and anxiety/depression symptoms during adolescence and investigate whether anxiety/depression symptoms mediate the relationship between ACEs and somatic symptoms.

Methods: This study uses data from the LONGSCAN (Longitudinal Studies of Child Abuse and Neglect) of 1354 children and their primary caregivers to conduct a longitudinal cross-lagged path analysis among ACEs, anxiety/depression symptoms, and somatic symptoms at three points during adolescence (ages 12, 14, and 16 years).

Results: The results indicated significant concurrent associations between ACEs and increased anxiety/depression symptoms and between anxiety/depression symptoms and increased somatic symptoms at ages 12, 14, and 16 years. Moreover, anxiety/depression symptoms significantly mediated the relationship between ACEs and concurrent somatic symptoms at ages 12, 14, and 16. However, there was no significant relationship between past ACEs and somatic symptoms. The mediation effects of anxiety/depression symptoms between ACEs and later somatic symptoms were also non-significant.

Conclusion: The findings suggest that anxiety/depression symptoms mediate the concurrent relationships between ACEs and somatic symptoms at ages 12, 14, and 16. Providers should consider assessing anxiety/depression symptoms and possible concurrent exposure to adversities when caring for adolescents who present somatic symptoms.

Introduction

Somatic symptoms are commonly known as physical complaints without identified organic causes (Campo, 2012). Somatic symptoms such as headache, stomachaches, nausea, and fatigue are particularly prevalent in adolescents (Swain et al., 2014). The pervasiveness and adverse effects of somatic symptoms on children's health, well-being, and future outcomes have been well-documented (Kelly et al., 2010; Stone et al., 2019; Voerman et al., 2017). Childhood adversities, commonly conceptualized as adverse childhood experiences (ACEs), are one of the significant contributors of childhood somatic symptoms (Bonvanie et al., 2015; Rueness et al., 2020). However, the mechanism of how ACEs affect the onset of somatic symptoms is not fully understood. Recently, increasing evidence suggests that anxiety/depression symptoms may mediate the relationship between ACEs and somatic symptoms (Bonvanie et al., 2017; Bugge et al., 2017; Hammond et al., 2019; Kugler et al., 2012; McCall-Hosenfeld et al., 2014; Wooldridge et al., 2020). Understanding the mediating mechanisms between ACEs and somatic symptoms may facilitate the development and delivery of appropriate assessments and interventions to children who present somatic symptoms. This study addresses this gap in knowledge by examining relations among ACEs, anxiety/depression symptoms, and somatic symptoms across adolescence.

ACEs, anxiety, depression, and somatic symptoms

Somatic symptoms are a part of the "internalizing cluster," representing symptoms of internalizing disorders, including anxiety, depression, and somatization. Anxiety, depression, and somatization often co-occur and are highly prevalent in adolescents (Achenbach et al., 2016; Narusyte et al., 2017). The concurrent and longitudinal association between ACEs, anxiety, depression, and somatic symptoms has been well-established in the literature (Crandall et al.,

2020; Elmore & Crouch, 2020; Grasso et al., 2016; Hunt et al., 2017; Turney, 2020). For example, in a recent cross-sectional study using data from the 2016-2017 National Survey of Children's Health, Elmore & Crouch (2020) found that a higher number of lifetime ACEs was associated with significantly higher odds of both anxiety and depression among children and adolescents ages 8 to 17 years (Elmore & Crouch, 2020). Also, Groenewald et al. (2020) found that exposure to more ACEs was associated with increased odds for frequent or chronic somatic symptoms (Groenewald et al., 2020). Furthermore, longitudinal studies have reported an association between early childhood ACEs and increased internalizing problems in middle childhood (Hunt et al., 2017; Schroeder et al., 2020) and adulthood (Tang et al., 2020). Winding and Andersen (2019) found a significant association between experiencing ACEs and concurrent somatic symptoms at age 15 and 18 (Winding & Andersen, 2019). Given the high prevalence, co-morbidity, and lasting harmful effects of internalizing symptoms, there has been a growing interest in studying the differential effects of ACEs on the three internalizing symptoms. However, parsing the relationships among anxiety, depression, and somatic symptoms is difficult due to the similarities and complex associations between the three different symptoms.

Anxiety and depression on somatic symptoms

To understand the complex relationships among internalizing symptoms, it is essential to describe and distinguish their features clearly. The tripartite model is useful for conceptualizing anxiety and depression. The tripartite model, initially proposed by Clark & Watson (1991), posits that anxiety and depression contain both shared and unique components. The shared component between anxiety and depression is negative affectivity, which is generally defined as feelings of emotional distress (Watson et al., 1988). The distinct components between depression and anxiety include anhedonia and hyperarousal. Depression is characterized by anhedonia, and

anxiety is distinguished by physiological tension and hyperarousal, such as psychomotor tension and autonomic hyperactivity (Clark & Watson, 1991). By using the tripartite model, several studies have demonstrated that experiencing symptoms of anxiety and depression are antecedents of the onset of somatic symptoms (Halder et al., 2002; Janssens et al., 2010; Larson et al., 2004). Anxiety and depression are known to influence somatic symptoms through heightened physical sensations and altered cognition. Anxiety, characterized by fear, avoidance, and physiological arousal, affects somatic symptoms through increased physical sensations and altered perceptions (Mallorquí-Bagué et al., 2016). Physiological hyperarousal, a distinct component of anxiety, is known to cause somatic symptoms through increased autonomic and psychomotor agitations (e.g., increased heart rate, shortness of breath, and muscle tension). Such physical changes result in somatic discomfort and pain. Moreover, anxious individuals are more focused on physical sensations, which may heighten the intensity of the symptoms (Crawley et al., 2014; Stegen et al., 2001). Depression is distinguished from anxiety by anhedonia (i.e., the absence of positive affect). Anhedonia is known to induce somatic symptoms through maladaptive cognition. Neurobiological imaging studies have shown that anhedonia is associated with heightened sensitivity to pain (Gorwood, 2008; Hermesdorf et al., 2016; Penninx et al., 2013). Furthermore, anhedonia is associated with increased bodily focus due to ruminative self-focusing (Harshaw, 2015). Finally, negative affectivity, a shared element of anxiety and depression, has also been identified as influencing somatic symptoms through the increased perception of physical sensations as unpleasant (Aronson et al., 2006; Stegen et al., 2001).

The mediating role of anxiety and depression between ACEs and somatic symptoms

Increasing evidence suggests that anxiety/depression symptoms may mediate the relationship between ACEs and somatic symptoms (Bonvanie et al., 2017; Bugge et al., 2017;

Hammond et al., 2019; Kugler et al., 2012; McCall-Hosenfeld et al., 2014; Wooldridge et al., 2020). For example, Kugler et al. (2012) conducted a cross-sectional study on children (ages 8-17 years) residing in foster homes and have found that anxiety symptoms mediated the relationship between sexual abuse and somatic symptoms (Kugler et al., 2012). Moreover, Hammond et al. (2019) found that depression and anxiety symptoms mediated the relationships between family dysfunction during the first 6 years and migraine diagnosis in adolescence (ages 14-15). In summary, there is some evidence that anxiety and depression mediate the relationship between ACEs and somatic symptoms. However, previous studies have mainly used cross-sectional designs examining retrospective reports of ACEs, limiting the reliability of measurements. Furthermore, no study has examined the mediating role of anxiety/depression symptoms between the concurrent relationship of ACEs and somatic symptoms in adolescence.

Implications

Adolescence is a critical period wherein children experience many biological, psychological, and ecological changes. Such changes often contribute to the onset of new problems, such as anxiety and depression. Additionally, exposure to adversities is highly prevalent in adolescence (Crouch et al., 2019). Despite the advances made in the literature regarding ACEs and internalizing disorders in adolescents, the difficulties in identifying individuals experiencing ACEs and internalizing symptoms remains a significant obstacle in service delivery (Barnes et al., 2020; Costello et al., 2014). Determining the mediating role of anxiety and depression between ACEs and somatic symptoms may provide critical evidence in identifying children experiencing anxiety/depression symptoms and current and past exposure to adversities.

The current study

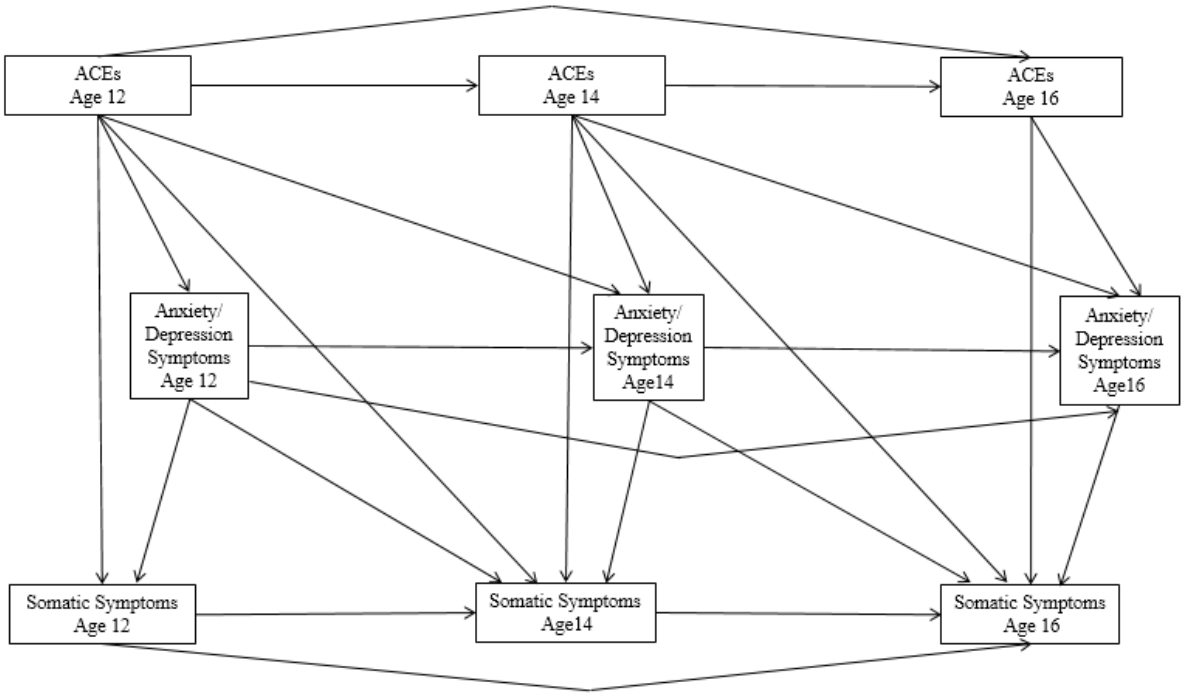
This study sought to address these gaps in knowledge by utilizing a longitudinal cross-lagged path analysis to examine the longitudinal relationships among ACEs, anxiety/depression symptoms, and somatic symptoms in adolescents. This study tested the hypothesized model (Figure 3.1) with the following specific aims and hypotheses:

Aim 1: To examine the concurrent and longitudinal relationships among ACEs, anxiety/depression symptoms, and somatic symptoms across adolescence. It was hypothesized that experiencing a higher number of ACEs predicts increased concurrent and future somatic symptoms and anxiety/depression symptoms. It was also hypothesized that higher scores of anxiety/depression symptoms predict increased concurrent and future somatic symptoms.

Aim 2: To examine if anxiety/depression symptoms mediate the concurrent and longitudinal relationships between ACEs and somatic symptoms in adolescents. It was hypothesized that anxiety/depression symptoms mediate concurrent and longitudinal relationships between ACEs and somatic symptoms in adolescents.

Figure 3.1

Hypothesized Mediation Model



Methods

Design and Participants

This study used data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN; Runyan et al., 2014), a consortium of five studies conducted to study the predictors and effects of child maltreatment. LONGSCAN followed 1,354 children and their primary caregivers from ages 4 to 18 years. Data were collected when children were ages 4, 6, 8, 10, 12, 14, 16, and 18. This study used data collected from ages 12, 14, and 16. Participants were recruited from five sites across the United States. The sites included East (EA), Midwest (MW), Northwest (NW), South (SO), and Southwest (SW). The sampling criteria varied across the sites to include various levels of risk or exposure to maltreatment. NW, MW, and SO sites included children referred to Child Protective Services (CPS). SW site recruited children who were placed into foster care before age 4 years, and EA site recruited children who were considered at risk for maltreatment based on prenatal and neonatal risk factors. Detailed descriptions of the methodology of the LONGSCAN can be found in Runyan et al. (2014). A total of 1,354 children and caregiver dyads were in the baseline sample. Among the original sample, 977 (72.2%), 962 (71.0%), 901 (66.5%) completed either child or caregiver interviews at age 12, 14, and 16 respectively. 1137 (84.0%) completed at least one interview, 999 (73.8%) completed at least two interviews, and 704 (52.1%) all three interviews at age 12, 14, and 16. There were a few statistically significant differences between samples with different completion patterns. The MW site was more likely to have completed all three interviews. Also, dyads with a child identified as Black/African American at baseline were more likely to have completed at least one interview. Table 3.1 presents the demographic characteristics of the baseline sample (N=1354).

Measures

Somatic Symptoms

The Child Behavior Checklist (CBCL) 4-18 (Achenbach, 1991) was administered at ages 12, 14, and 16. The somatic complaints subscale of the CBCL asked the primary caregivers to rate the frequency of nine somatic problems without known medical cause- within the past six months. Nine items of somatic symptoms included feeling dizzy, overtired, aches or pains, headaches, nausea, eye problems, rashes or skin problems, stomachaches or cramps, and vomiting or throwing up. Caregivers rated the child on a 3-point Likert scale: 0=Not true (as far as you know), 1=somewhat true or sometimes true, 2=very true or often true. Raw scores of somatic symptoms items calculated by adding the scores were used in the analysis with possible ranges from 0 to 18, with higher scores indicating higher severity of somatic symptoms. Cronbach's alpha at age 12, 14, and 16 were 0.72, 0.68, 0.72 respectively.

Anxiety/Depression Symptoms

The Child Behavior Checklist's anxious/depressive symptom subscale (CBCL) 4-18 (Achenbach, 1991) was used. Primary caregivers were asked to evaluate the child's anxious/depressive behaviors over the past six months on a 3-point Likert scale: 0=Not true (as far as you know), 1= Somewhat true or sometimes true, 2= Very true or often true. There were 14 total items including complaints of loneliness, cries a lot, fears he/she might think or do something bad, feels he or she has to be perfect, feels or complains that no one loves him/her, feels others are out to get him/her, feels worthless or inferior, nervous, high-strung, or tense, too fearful or anxious, feels too guilty, self-conscious or easily embarrassed, suspicious, unhappy, sad, or depressed, and worries. Raw scores were summed with possible ranges from 0 to 28 with

higher scores indicating higher severity of anxiety/depression symptoms. Cronbach's alpha at age 12, 14, and 16 were 0.84, 0.86, 0.86 respectively.

Adverse Childhood Experiences (ACEs)

An ACE index score was calculated by summing dichotomous scores (0=No, 1=Yes) on nine variables. The variables were selected using the original CDC-ACEs study as a reference (Felitti et al., 1998). The composite score ranged from 0 to 9, with higher scores indicating a greater number of ACEs experienced. The ACEs assessed in this study included two broad categories of childhood experiences: child maltreatment and household dysfunction. Four types of maltreatment and five types of household dysfunction were included in the study.

Child Maltreatment

Child maltreatment was assessed using CPS records. CPS records were reviewed and coded by highly trained coders using the Modification of Maltreatment Classification System (MMCS; (Barnett et al., 1993; English & LONGSCAN, 1997). Four types of non-mutually exclusive categories included physical abuse, sexual abuse, emotional abuse, and neglect. The CPS records were assessed by the study staff every two years. Maltreatment allegations between ages 10-12, 12-14, and 14-16 years were included in this analysis. The Interrater reliability of the MMCS was relatively high, with kappa ranging from .73 for psychological abuse to .87 for physical abuse (English, 1997). Dichotomous indicators based on the absence or presence of maltreatment allegations in each time point were summed to create an index maltreatment score, which ranged from 0 to 4. Index maltreatment scores were created for each time point. Allegations of maltreatment rather than substantiations were used based on the previous studies'

findings that allegations and substantiations of maltreatment were similarly associated with child outcomes. (Drake, 1996; Hussey et al., 2005).

Household Dysfunction

Household dysfunction included caregiver separation or divorce, caregiver depressive symptoms, caregiver treated violently, household criminal activity, and household substance use.

Caregiver Separation or Divorce. The Child Life Events measure (LONGSCAN, 1992) was administered to caregivers at ages 12, 14, and 16 and asked about caregiver separation or divorce in the past year. An affirmative response to caregiver separation or divorce between parents or parent-figures of the child was coded as 1.

Caregiver Depressive Symptoms. Caregiver depressive symptoms were measured using The Center for Epidemiological Studies Depression Scale (CES-D) (Radloff, 1977) administered to caregivers at ages 12,14, and 16. The CES-D scale assesses symptoms associated with depression in the past week. CES-D scores were dichotomized to widely used cut off points at 16 or greater=1 and less than 16=0 (Lewinsohn et al., 1997).

Caregiver Treated Violently. Intimate partner violence within the past year was assessed using the Partner to Partner Conflict Tactics Scale (Straus et al., 1998) administered at ages 12,14, and 16. This measure assessed the type and severity of conflict tactics used between the child's primary caregiver and their partner during the past year. The partners could be partners that the caregivers are dating, cohabiting, or married to. Physical assault subscale within this measure -which consisted of items such as partner threw something, twisted or pulled hair, pushed or shoved, grabbed, slapped, used a knife or gun on me, punched or hit, choked, slammed against the wall, beat up, burned or scalded, kicked. The Sum score of physical assault was

dichotomized to 0=none and 1=score greater than 1. Southwest did not collect this measure at age 14, and South and Southwest sites did not collect this measure at age 16 interviews.

Household Criminal Activity. The household criminal activity was assessed by the Child Life Events Measure (LONGSCAN, 1992) was administered to primary caregivers at age 12, 14, and 16 interviews. It asked if anyone in the child's family or household was jailed, imprisoned, or kept in a juvenile residential facility for breaking the law. Answers were dichotomized to 0=No and 1=Yes.

Household Substance Use. Household substance use was measured by the Risk Behaviors of Family and Friends measure developed by LONGSCAN, 1998. This measure was administered to the child at age 12,14, and 16 and asked if anyone the child lives with uses drugs or alcohol. An affirmative answer to using marijuana, cocaine or crack, meth, injection drugs, other drugs, and getting drunk or high was coded as 1, otherwise 0.

Demographic control variables

The child's biological sex assigned at birth, race/ethnicity, and site were collected at baseline. Binary dummy variables were used.

Statistical Analyses

Descriptive and bivariate correlation analyses were conducted using SPSS Version 27.0. Listwise and pairwise deletion for missing data were used in descriptive and bivariate correlations, respectively. The longitudinal mediation model was tested using Mplus Version 8.0 (Muthén & Muthén, 2017).

First, Little's MCAR test using all variables used in the model was tested using SPSS version 27.0. The missingness pattern was MCAR, χ^2=188.73(158), p=.048, suggesting that the pattern of missingness in the data was MCAR. Full information maximum likelihood (FIML) was used to address missingness (Arbuckle, 1996). FIML assumes the data to be missing at random or missing completely at random (MCAR) and results in unbiased parameters. FIML is known to produce more valid estimates than listwise deletion or multiple imputations, even if the assumptions regarding MAR and MCAR are not fully met (Arbuckle, 1996). Therefore, data on all 1,354 participants were used in the analysis. Because dependent variables were non-normally distributed, robust maximum likelihood estimation (MLR) was used to provide the model fit and statistical inference robust to non-normality. Fit indices such as non-significant chi-square test statistics of the exact-fit hypothesis, Root Mean Squared Error of Approximation (RMSEA) below 0.06, Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) greater than 0.95, and Standardized Root Mean Residual (SRMR) below 0.08 were used to assess the fit of the model to the data (Hu & Bentler, 1999; Kline, 2015a). Finally, significant mediation effects were calculated using the bias-correcting bootstrapping method in Mplus using the MODEL INDIRECT command.

Results

Descriptive Analyses

Table 3.1 displays the descriptive statistics of the demographic characteristics of the sample. Table 3.2 presents the descriptive statistics and bivariate correlations among the study variables. The mean scores of somatic symptoms, anxiety/depression symptoms, and ACEs scores were all highest at age 12 and gradually decreased. There were no correlation coefficients above .85, indicating the absence of multicollinearity (Kline, 2015).

Table 3.1

Descriptive Statistics of Study Variables

	N	%
Child's sex		
Female	697	51.5
Male	657	48.5
Child's race/ethnicity		
Black	721	53.2
White	354	26.1
Hispanic	97	7.2
Other[1]	181	13.4
Site		
EA	282	20.8
MW	245	18.1
NW	254	18.8
SO	243	17.9
SW	330	24.4
Annual Household Income at age 12		
≤$14,999	262	28.8
≥$15,000	649	71.2
Caregiver Education Attainment		
≤ 11 years	284	29.8
≥ 12 years	669	70.2

Note.

[1]Other included Native American, Asian, Mixed Race, and Other

Table 3.2

Descriptive Statistics of Zero-Order Correlations Among Study Variables

Variable	n	M	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Sex[1]	1354	0.51	0.50	--									
2. Age12 SOM	951	1.53	2.05	.05	--								
3. Age14 SOM	930	1.51	1.97	.09**	.44**	--							
4. Age16 SOM	867	1.48	2.07	.14**	.33**	.40**	--						
5. Age 12 AD	951	3.52	3.85	-.01	.47**	.32**	.27**	--					
6. Age 14 AD	930	3.46	4.07	.07*	.33**	.44**	.32**	.65**	--				
7. Age 16 AD	867	3.08	3.95	.11**	.28**	.35**	.53**	.54**	.61**	--			
8. Age12 ACEs	918	0.94	1.16	-.03	.23**	.15**	.11**	.27**	.25**	.17**	--		
9. Age14 ACEs	907	0.90	1.16	-.01	.17**	.16**	.16**	.26**	.31**	.24**	.39**	--	
10. Age16 ACEs	831	0.86	0.98	.02	.16**	.16**	.15**	.29**	.19**	.23**	.32**	.37**	--

Note. [1]0=female, 1=male. *p<.05,**p<.01.

SOM=Somatic symptoms, AD=Anxiety/Depression symptoms, ACEs= Adverse childhood experiences.

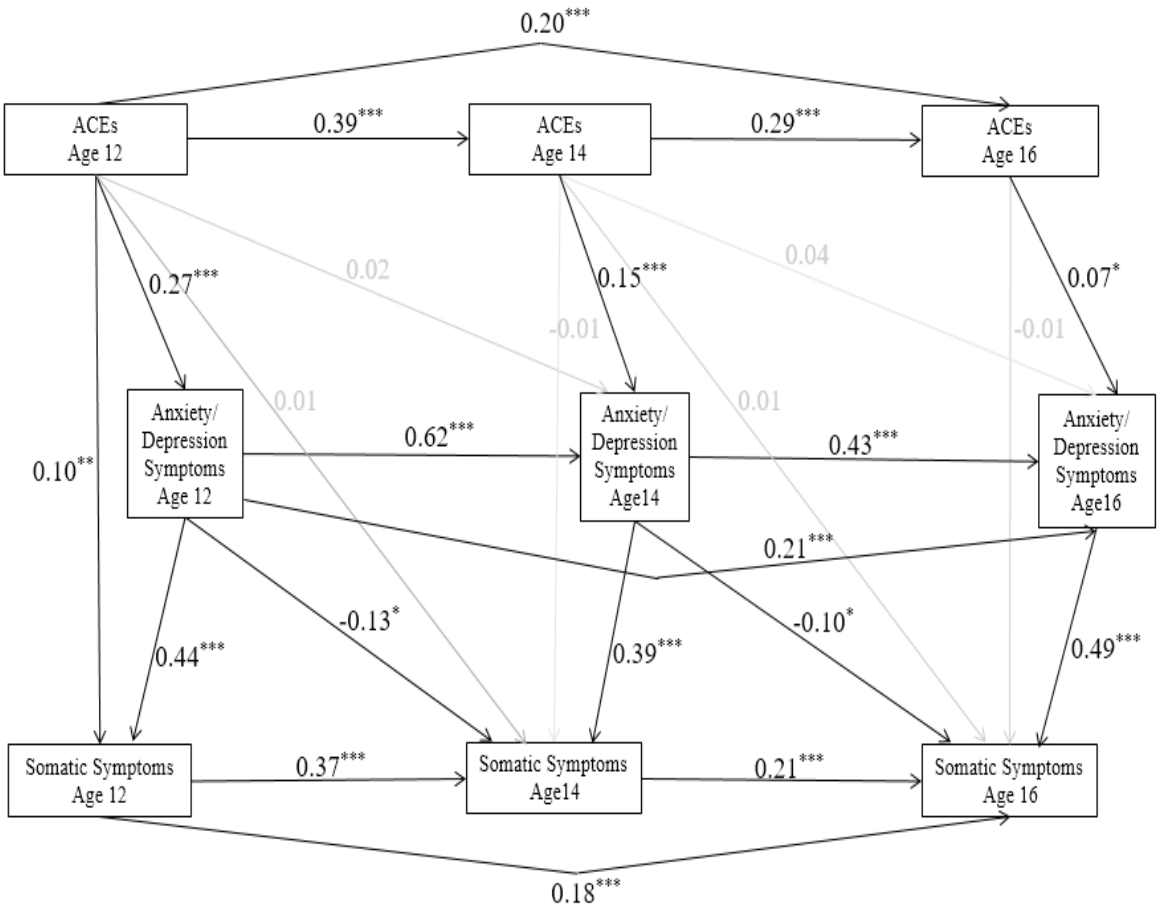
Path Models

Main effects. Overall, the model showed an acceptable fit with $\chi^2(10)=39.07$, p=<.001, RMSEA=0.046, CFI=0.99, TLI=0.85, and SRMR=0.023. As shown in Figure 3.2 and Table 3.3, ACEs at age 12 had significant positive effects on anxiety/depression symptoms at age 12 and somatic symptoms at age 12. ACEs at age 14 had significant positive effects on anxiety/depression symptoms at age 14, but not on somatic symptoms at age 14. Similarly, ACEs at age 16 had significant positive effects on anxiety/depression symptoms at age 16, but not on somatic symptoms at age 16. Longitudinally, ACEs did not predict anxiety/depression symptoms or somatic symptoms in the next time point. Anxiety/depression symptoms at age 12 had significant positive effects on concurrent somatic symptoms at age 12, 14 and 16 years. All three variables showed stable and positive autoregressive effects longitudinally.

Indirect effects. The bootstrapped estimates of the indirect effect and 95% confidence intervals are presented in Table 3.3. There was a significant indirect effect from ACEs to concurrent somatic symptoms via concurrent anxiety/depression symptoms at ages 12, 14, and 16. However, anxiety/depression symptoms did not mediate the longitudinal relationship between ACEs and somatic symptoms.

Figure 3.2

Path Model with Standardized Coefficients



*p<.05, **p<.01, ***p<.0001.

Table 3.3

Summary of Mediation Analysis Results

	β	se	CI (95%)
Age 12 ACEs→Age 12 SOM via Age 12 AD	0.12***	0.02	[0.08-0.15]
Age 14 ACEs→Age 14 SOM via Age 14 AD	0.06***	0.02	[0.03-0.09]
Age 16 ACEs→Age 16 SOM via Age 16 AD	0.04*	0.02	[0.001-0.07]
Age 12 ACEs→Age 14 SOM via Age 14 AD	0.01	0.01	[-0.02-0.04]
Age 14 ACEs→Age 16 SOM via Age 14 AD	-0.01	0.01	[-0.02-0.01]

Note.

*p<.05, **p<.01, ***p<.001. β = standardized beta.

SOM=Somatic symptoms, AD=anxiety/depression symptoms, ACEs= Adverse Childhood Experiences.

Discussion

This study's objectives were as follows:1) to examine concurrent and longitudinal relationships among ACEs, anxiety/depression symptoms, and somatic symptoms; and 2) to examine if anxiety/depression symptoms mediate the concurrent and longitudinal relationship between ACEs and somatic symptoms in adolescence. It was hypothesized that: 1) higher scores of ACEs will predict increased concurrent and future anxiety, depression, and somatic symptoms; 2) higher scores of anxiety/depression symptoms will predict increased concurrent and future somatic symptoms, and 3) anxiety/depression symptoms will mediate the effect of ACEs on concurrent and longitudinal somatic symptoms.

The path analysis results present that ACEs were positively associated with concurrent anxiety/depression symptoms at ages 12, 14, and 16. Broadly, this result confirms the effect of ACEs on concurrent anxiety and depression (Balistreri & Alvira-Hammond, 2016; Elmore & Crouch, 2020; Hunt et al., 2017; Negriff, 2020a). However, ACEs did not predict future anxiety/depression symptoms measured in the subsequent time point after two years. An explanation for this may be that concurrent ACEs, as compared with past ACEs, have a more substantial impact on anxiety/depression symptoms.

Furthermore, anxiety/depression symptoms were positively associated with concurrent somatic symptoms at ages 12, 14, and 16. In other words, higher anxiety/depression symptoms predicted increased concurrent somatic symptoms at ages 12, 14, and 16. This result aligns with the existing literature that consistently demonstrates the strong concurrent association between anxiety/depression symptoms and somatic symptoms in adolescents (Agnafors et al., 2019; Crawley et al., 2014; Janssens et al., 2010; Kim et al., 2019; Lallukka et al., 2019; Lee & Vaillancourt, 2019; Shanahan et al., 2015; Soltani et al., 2019). In contrast to our hypothesis,

there were negative relationships between anxiety/depression symptoms and future somatic symptoms. This perplexing result can be explained by the suppression effect, which has been widely documented in longitudinal mediation studies. When the path coefficient between predictor and outcome variables presents the opposite sign to the Pearson correlation, a third variable's suppression effect is suspected. The suppressor variable is significantly associated with the predictor and the outcome variables and changes the relationship between them (Kline, 2015). Since the suppression effect of the mediating variable is often observed in mediation analyses, most researchers agree that mediation analyses can be conducted when the relationship between the predictor and the outcome is negative or non-significant (Kline, 2015; MacKinnon et al., 2007).

ACEs had positive effects on somatic symptoms at age 12, which partly supported the hypothesis. This result is partially aligned with the previous literature that demonstrates a significant association between ACEs and somatic symptoms in adolescents (Groenewald et al., 2020; Lee & Vaillancourt, 2019; Petruccelli et al., 2019). However, the paths between ACEs and somatic symptoms at both ages 14 and 16 were negative. The suppression effect can also explain this perplexing result. In the suppression effect, the total effect between the predictor and outcome is typically small since the direct and indirect effects tend to cancel each other out (Kenny, 2008; Kline, 2015; MacKinnon et al., 2007).

Finally, the mediation analysis shows the mediating effect of anxiety/depression symptoms for the relationship between ACEs and concurrent somatic symptoms at age 12, 14, and 16. In other words, a higher number of ACEs predicted higher levels of anxiety/depression symptoms, which in turn predicted higher levels of somatic symptoms. These results align with previous studies' findings that highlight the mediating role of anxiety and depression for the

relationship between traumatic life events and somatic symptoms (Bugge et al., 2017; Hammond et al., 2019; Kugler et al., 2012).

In this study, both the total and indirect effects of ACEs on concurrent somatic symptoms were the strongest at age 12 compared to age 14 and 16. The effects of ACEs on anxiety/depression symptoms were the strongest at age 12 and subsequently decreased at age 14 and 16. This difference in the strength and significance of the association between ages may be partly explained by the developmental differences at ages 12, 14, and 16. During early adolescence, children experience higher proximity to the family environment, which may increase the effects of family dysfunctions compared with middle adolescence (Sawyer et al., 2012; Steinberg, 2005). Moreover, many neurobiological studies have noted that the period of early adolescence is more sensitive to ACEs than middle and late adolescence. Neurobiological plasticity and the physiological changes caused by the onset of puberty may make early adolescence more sensitive to ACEs (Andersen & Teicher, 2008; Bingham et al., 2011; Crews et al., 2007; Harpur et al., 2015). In contrast, the effect of anxiety/depression symptoms on somatic symptoms was the weakest at age 12 and strongest at age 16, although the differences were marginal. Several factors might explain this result. For instance, older adolescents with anxiety/depression symptoms might have heightened sensitivity to physical arousal and maladaptive perceptions, known as causal mechanisms of somatic symptoms. Further research is needed to delineate the differential effects of anxiety and depression on somatic symptoms in distinct adolescence phases. In summary, the mediation analysis results indicate that anxiety/depression symptoms mediate ACEs' effect on concurrent somatic symptoms at ages 12, 14, and 16.

Strengths and limitations

The findings of this study should be considered in the context of several potential limitations. One of the limitations is that this study used CBCL reported by primary caregivers to assess both anxiety/depression symptoms and somatic symptoms. This method may have resulted in a shared measure variance. The caregiver report of somatic symptoms may have been unreliable as many internalizing symptoms (e.g., anxiety, depression, and somatization) are often self-limiting, especially in adolescents (Sourander et al., 1999). Future studies should consider including symptom reports from other informants, such as the adolescent themselves and teachers, to examine how the relationship differs. Another limitation of this study relates to the measurement of anxiety/depression symptoms. The CBCL measured a combined construct of anxiety/depression symptoms. However, the literature demonstrates that anxiety and depression are unique and have differential effects on somatic symptoms (Anderson & Hope, 2008; Ask et al., 2016; Bekhuis et al., 2015; Charak et al., 2019). Future research should use different measures of anxiety and depression symptoms to assess their potentially distinct mediating effects. Furthermore, this study used CPS reports of allegations for maltreatment indicators and caregivers' reported measures to construct the index scores of ACEs. There may be biases stemming from the use of official records and caregivers' reports of a child's adversities (A. Shaffer et al., 2008). Future research is needed to replicate the study findings using reports from other informants and examine differences in findings based on various sources of ACEs. Finally, the findings from this sample may not be generalizable to the overall adolescent population**.** The majority of participants in the LONGSCAN sample were considered at high-risk for child maltreatment at recruitment. This study's sample characteristic may have resulted in higher rates of anxiety, depression, and somatic symptoms due to unaccounted confounding factors.

Moreover, this study's sample comprised a high proportion of African American children and families living in poverty compared with the representative sample of U.S. children. Although demographic factors have been controlled for in the analysis, there may be potential unknown confounders. Despite these limitations, this study also presents several important strengths. The prospective longitudinal design allowed for optimal precision in examining the relationships among the variables across three different periods of adolescence. Moreover, the high risk of exposure to child maltreatment of this sample may have resulted in higher ACEs levels in adolescents than in the general population. The high rates of reported ACEs in this sample may have strengthened the power to test the effects of ACEs.

Conclusion

In summary, this study has found significant concurrent effects between ACEs and anxiety/depression symptoms and between anxiety/depression symptoms and somatic symptoms across ages 12, 14, and 16 years. Furthermore, anxiety/depression symptoms significantly mediated the relationship between ACEs and concurrent somatic symptoms at ages 12 and 14 years. The findings suggest that anxiety/depression symptoms may provide an intermediary mechanism by which ACEs impact somatic symptoms. Taken together, the findings of this study provide several clinical implications. First, the findings support the usefulness of somatic symptoms as potential markers of current anxiety, depression, and ACE exposure in adolescents. Caregivers, school staff, and primary care providers are often the first to recognize somatic symptoms in adolescents. In community settings, it is crucial for caregivers and school staff, such as school nurses, counselors, and teachers, to recognize that adolescents' somatic symptoms may imply other emotional problems such as anxiety and depression. Particular attention must be paid to adolescents with high somatic symptoms and make appropriate referrals for care and

service. Clinically, it is essential to assess family-level factors, such as family functioning, and other internalizing problems, such as anxiety and depression, as a part of the assessment for adolescents who present with somatic symptoms. Moreover, identifying families experiencing disruptions and providing appropriate interventions to mitigate the effects of exposure to concurrent ACEs may reduce symptoms of anxiety, depression, somatization, and the subsequent burdens on children and families. Further work is needed to examine the mechanisms of how anxiety and depression may differently mediate the relationship between ACEs and somatic symptoms. Furthermore, investigations on if and how this mediating relationship may differ by other factors such as type of ACEs and child's characteristics are needed.

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Chapter 4. Longitudinal Trajectories of Somatic Symptoms in Children and Adolescents: Heterogeneity, Predictors, and Outcomes

Abstract

Objectives: The primary objectives of this paper were to 1) examine the heterogeneity of trajectories of somatic complaints across children ages 6-16 years, 2) to assess the extent to which child sex and child maltreatment allegation before age 6 predict membership of certain trajectory classes, and 3) to examine if membership in a trajectory class predicts a diagnosis of depression symptoms at age 18.

Methods: Latent Class Growth Analysis (LCGA) was used to examine different trajectories of somatic symptoms across ages 6 to 16 in a sample of 1354 children using data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN). Predictors and outcomes of trajectories were analyzed using multiple logistic regression.

Results: Four distinct patterns of trajectories of somatic symptoms across ages 6-16 were identified: low-stable (43.6%), increasing (24.5%), decreasing (22%), and high-stable (9.9%). Relative to the low-stable group, membership in the increasing and the high-persistent group was predicted by being female and having exposure to maltreatment before age 6. Compared to the low-stable group, individuals in the increasing and the high-persistent group were more likely to have a diagnosis of major depressive disorder at age 18.

Conclusion: This study's results indicate that heterogeneity in trajectories exists in somatic symptoms in children. Health and service providers should consider heterogeneity in longitudinal patterns of somatic symptoms and risk factors when developing or delivering interventions for children presenting with somatic symptoms. This result suggests that persistent or increasing somatic symptoms in childhood may be used as an early marker of risk for major depressive disorder in young adulthood.

Introduction

Somatic symptoms, commonly known as bodily discomfort without an identifiable organic cause, are prevalent during childhood and adolescence (King et al., 2011; Swain et al., 2014). The profound impact of childhood somatic symptoms on long-term health highlights the need to understand the development of somatic symptoms. Modeling and characterizing the trajectory patterns of somatic symptoms and their predictors and outcomes are useful in identifying long-term risk profiles that facilitate tailored interventions (Bi et al., 2015; Mulvaeny et al., 2006). However, the investigations of heterogeneity, predictors, and outcomes of the somatic symptom trajectory in childhood have been limited.

Somatic symptom trajectory subgroups during childhood

From a developmental psychopathology perspective, the patterns of change in somatic symptoms vary across life stages based on individuals' biological, psychological, and ecological contexts (Lijster et al., 2019; Nummi et al., 2017). Previous prospective and longitudinal studies among both clinical and community samples of children have continually demonstrated that earlier somatic symptoms during childhood predict later somatic symptoms (Engel et al., 2018; Horst et al., 2014). However, most of these studies have used variable-oriented statistical analysis methods that assume generalizable and homogenous growth patterns and do not account for individual variabilities. Due to the limitations of such traditional research methods, person-centered methods, such as growth mixture modeling (GMM), have increased in popularity. As compared with variable-centered approaches, which describe the relationships among variables, person-centered approaches address individual differences in patterns of change over time (Jung & Wickrama, 2008). Recent studies have used mixture modeling to identify trajectory subgroups in depressive symptoms, anxiety symptoms, and internalizing symptoms in childhood and

adolescence. These studies' findings reveal that heterogeneity in symptom trajectories exists and suggests multiple predictive factors and outcomes related to the trajectory subgroups (Broeren et al., 2013; Lauterbach & Armour, 2016; Yoon, 2017). Modeling trajectories and identifying predictors of trajectory patterns may help characterize individuals associated with trajectory group membership. While transient and mild somatic symptoms are often do not have long-lasting impact, persistent somatic symptoms are linked with impaired functioning and well-being and future health problems (Groenewald et al., 2014; Kelly et al., 2010; Voerman et al., 2017). Therefore, understanding individuals who are more likely to experience persistent somatic symptoms across childhood is imperative in developing and implementing appropriate interventions to reduce the risk. However, only a handful of studies have examined trajectory patterns of somatic symptoms (Bi et al., 2015; Janssens et al., 2014; Lallukka et al., 2019; Mulvaeny et al., 2006; Nummi et al., 2017). Among those studies, only a couple of studies have focused on childhood and adolescence (Janssens et al., 2014; Mulvaeny et al., 2006). The findings of these studies demonstrate that long-term trajectories of somatic symptoms across childhood are heterogeneous. Despite slight variations in the number and shape of trajectories among the studies, the findings all highlight distinct subgroups. The two subgroups that are most common across these studies are those who experience low or no symptoms and those who experience persistently high levels of symptoms (Janssens et al., 2014; Mulvaeny et al., 2006). For example, in a prospective study of a community sample of Dutch adolescents, Janssens et al. (2014) identified four trajectory patterns across ages 10 to 17 (n = 2230). In this study, 4.1% of participants had persistently high somatic symptoms, 10.5% had a decreasing trajectory, 22% had a slightly increasing trajectory, and 63.3% had consistently low symptoms. In a five-year longitudinal study of a clinical sample of children ages 6-18 who were patients at a pediatric

gastroenterology clinic, Mulvaeny et al. (2006) identified three trajectory classes of somatic symptoms. The trajectory classes were described as persistently high/high-risk (14%), decreasing/short-term risk (16%), and persistently low/low risk (70%). Although scarce, the existing literature on the heterogeneity of somatic symptom trajectories supports that there are distinct subgroups of somatic symptom trajectories across childhood and adolescence. However, no known studies have explored the heterogeneity in somatic symptom trajectories across childhood and adolescence among a non-clinical sample of U.S. children.

Predictors of somatic symptom trajectory classes

Describing and characterizing individuals in subgroups help identify risk factors of persistent somatic symptoms. Although several factors such as sex, insecure attachment, anxiety sensitivity, childhood trauma, and social capital (Engel, 1981; Hart et al., 2013; Ibeziako & Bujoreanu, 2011; Kugler et al., 2012; Nelson et al., 2017; Susman et al., 2003; Waldinger et al., 2006) are known to be associated with higher occurrences of somatic symptoms, less is known about the predictors of somatic symptom trajectories. Janssens et al. (2014) examined somatic symptom trajectories of Dutch adolescents ages 10-17 years and identified being a female, poor self-rated health at age 10, and depressive symptoms at age 10 to predict membership in the persistently high somatic symptom trajectory. Mulvaeny et al.(2006)'s result from a 5- year longitudinal study looking at somatic symptom trajectories in children ages 6-18 in abdominal pain clinic identified higher anxiety and depression scores, lower perceived self-worth, and more negative life events reported at baseline to predict the persistently high trajectory (Mulvaeny et al., 2006). In the present study, early childhood maltreatment and sex will be explored as predictors of trajectory subgroups.

Early childhood maltreatment

Early childhood maltreatment is commonly defined as any form of physical, sexual, or emotional abuse and neglect experienced in early childhood, which is the period from birth to age five. Early childhood maltreatment has unique effects on long-term health, as ages zero to five represent a particularly sensitive period with significant developmental plasticity (National Scientific Council on the Developing Child, 2020; Turecki et al., 2014). Previous studies using variable-centered methods show substantial evidence that early child maltreatment increases the occurrence of somatic symptoms across in later life stages. In a seminal study, Rogosch et al found that a history of child maltreatment predicted increased somatic symptoms among children ages 8-10. Moreover, this study compared the association between allostatic load-measured by salivary cortisol and dehydroepiandrosterone (DHEA), body-mass index, waist-hip ratio, and blood pressure, and somatic symptoms based on maltreatment history. The results indicated that allostatic load was associated with higher somatic symptoms only among children with maltreatment history (Rogosch et al., 2011). Moreover, Flaherty et al. (2009) found that experiencing five or more maltreatment or household dysfunction exposure during the first six years of life was associated with greater somatic symptoms at age 12. In a recent large retrospective population-based cohort study in the United Kingdom, Chandan et al. found that childhood maltreatment history was associated with higher levels of somatic and visceral pain syndromes in adults (Chandan et al., 2020). However, no known study has examined whether exposure to early childhood maltreatment predicts persistent somatic symptom trajectories across childhood and adolescence. Using the biopsychosocial perspective, the literature suggests that exposure to early childhood maltreatment inflicts later somatic symptoms through the interactions among multiple biological, psychological, and ecological factors (Nelson et al.,

2017). Major biological processes known to affect later somatic symptoms are impairments in neurological and neuroendocrine functions. In response to stressors, such as experiencing maltreatment in early childhood, the child's brain activates the hypothalamic-pituitary-adrenocortical axis and the sympathetic-adrenomedullary system, which releases increased levels of stress hormones, such as corticotropin-releasing hormone (CRH), cortisol, norepinephrine, and adrenaline. At the same time, elevated inflammatory cytokines are released in response to the parasympathetic nervous system. Repeated activations of such a stress response system result in biological processes that impair healthy brain development and interrupt physiological responses (Gatchel et al., 2007; B. S. McEwen & Kalia, 2010). Such impairments in neurological and neuroendocrine functions are known to increase one's perception of pain (Juster et al., 2010). Moreover, early child maltreatment affects somatic symptoms through perceptions of threat and pain. Several studies have reported that children exposed to family violence show increased neural reactivity in response to situations they perceive as dangerous (McCrory et al., 2011). Such distortions in cognitions may result in maltreated children perceiving somatic symptoms as more threatening (Nelson et al., 2017). The biological and psychological processes of ACEs can be further complicated by the child's ecological environment, such as a lack of social support. There is also an increased risk of compounded victimization, which is known to be experienced by children with a history of early child maltreatment (Charak et al., 2019; Kendall-tackett, 2002). In summary, there is substantial evidence that early child maltreatment increases the occurrence of somatic symptoms across different life stages. However, whether exposure to early childhood maltreatment predicts persistent somatic symptom trajectory across childhood and adolescence is unknown.

Sex

Previous studies have noted that differences in the etiology and development of somatic symptoms between males and females result from variations in biological and social processes that lead to distinct perceptions and presentations (Gijsbers Van Wijk & Kolk, 1997; Martel, 2013; Padgaonkar et al., 2020). Several studies have demonstrated differences across biological sex (e.g., male and female) in the prevalence and development of somatic symptoms in both children and adults (Creed & Barsky, 2004; Glise et al., 2014; Mayes et al., 2020; Swain et al., 2014). During childhood, somatic symptoms are generally more prevalent among females (Campo, 2012; Dhossche et al., 2001; Swain et al., 2014), although this may differ based on age. Studies suggest that the prevalence of somatic symptoms between males and females remains relatively similar in childhood until early adolescence. This is when females begin to report more somatic symptoms as compared to males (Campo, 2012; Shelby et al., 2013). In studies examining somatic symptom trajectory subgroups, being a female was a risk factor for persistent somatic symptoms trajectory. For instance, in Janssens et al. (2014), found that females were 4.69 times more likely to be in persistently high somatic symptom trajectories across ages 10 to 17. However, whether sex is a risk factor of persistent somatic symptoms across a broader childhood age range has not been examined in previous studies.

Outcomes of somatic symptom trajectory subgroups

Depression at age 18. Depression is highly prevalent in young adulthood and impacts approximately 13.1% of young adults aged 18-25 in the U.S. (Substance Use and Mental Health Administration, 2015). Depression in young adulthood is often comorbid with other problems, such as substance abuse and anxiety disorder (Mahmoud et al., 2012). It is also linked with decreased life satisfaction, maladaptive coping strategies, and increased suicidality (Castaneda et al., 2008). Such high

prevalence and adverse implications highlight the need to address depression during young adulthood. Examining the relationships between somatic symptom trajectories and depression in young adulthood can help develop assessment and intervention strategies for children who present somatic symptoms with potential risk for depression. Several longitudinal studies have provided evidence regarding an association between somatic symptoms and an increased risk for depressive disorders. In a prospective study that followed a community sample (n = 1420) of U.S. children into adulthood, Shanahan et al. (2015) reported that frequent and persistent somatic symptoms during childhood (ages nine to 16) predicted an increased risk of depressive disorders during young adulthood. Similarly, Engel et al (2018) found that somatic symptoms in preschool age predicted increased anxious and depressive symptoms at school among a community sample of U.S. children. However, these studies have used variable-centered methods that do not account for variabilities in continuities of somatic symptoms within individuals. To date, no study has examined whether somatic symptom trajectory patterns predict increased risk of later depressive disorders.

The present study

This study's primary purpose is to apply growth mixture modeling analyses to investigate the heterogeneity, predictors, and outcomes of somatic symptom trajectory subgroups across childhood and adolescence. The specific aims and hypotheses of this study include the following:

Aim 1: To examine and describe the heterogeneity of classes of somatic complaint trajectories across ages 6 to 16 years. It was hypothesized that there are at least three patterns of trajectories: persistently low, persistently high, and moderate. Moreover, the majority of individuals are expected to be in the persistently low group, with the smallest number in the persistently high group.

Aim 2: To examine whether sex and child maltreatment status at age six predicts the membership of certain trajectory classes. It was hypothesized that being a female and having exposure to early life maltreatment predicts membership in the persistently high group.

Aim 3: To examine whether membership in a trajectory class predicts depression or anxiety symptoms at age 18. Being in the persistently high trajectory group was hypothesized to predict an increased risk of major depressive disorder diagnosis at age 18.

Methods

Design and Sample

This study used data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN; Runyan et al., 2014). LONGSCAN was a consortium of five longitudinal studies conducted in five different geographical settings across the United States. The sites included East (EA), Midwest (MW), Northwest (NW), Southwest (SW), and South (SO). LONGSCAN followed 1,354 child-primary caregiver dyads from ages 4 to 18 to study the effects of maltreatment on child health and development. The child and the caregiver completed separate interviews beginning at age 12. Data collection started in 1991 and ended in 2012. Each site employed different sampling methodologies to represent varying levels of risk or exposure to child maltreatment. The baseline sample of LONGSCAN included 1354 participants. Table 4.1 summarizes the demographic characteristics of the sample population. The sample experienced attrition throughout the study period. Table 4.2 presents the baseline characteristics of the sample population by study assessment periods. The attrition rates for age 8, 10, 12, 14, and 16 were 84.2% (n=1140) at age 8, 72.1% (n=976) at age 12, 71%(n=961) at age 14, 66.1% (895) at age 16 and 69% (n=932) at age 18. Those who had caregiver interviews at age 6, 8, 10, 12, 14, and

16 included 83.5%(n=1135), 75.0% (n=1015), 70.6% (n=956), 69.3% (n=938), 63.6% (n=861), and 69%(n=932) respectively.

Table 4.1

Baseline Demographic Characteristics of the Sample Population (N=1354)

	Age 4 or 6	
	n	%
Child's sex		
Female	697	51.48
Male	657	48.52
Child's race		
Black	721	53.25
White	354	26.14
Mixed Race	161	11.89
Hispanic	97	7.16
Other	20	1.48
Site		
EA	282	20.83
MW	245	18.09
NW	254	18.76
SO	243	17.95
SW	330	24.37
Caregiver Marital Status		
Not married[1]	893	65.95
Married	444	32.79
Caregiver Education		
≤12 years	458	33.83
≥12 years	882	65.14
Household Annual Income		
<$15,000	703	51.92
≥$15,000	619	45.72

Note. [1]includes never married, separated, divorced, or widowed

Measures

Somatic Symptoms

Somatic symptoms were assessed using the Child Behavior Checklist (CBCL) 4-18 years (Achenbach, 1991). The CBCL is a widely used tool for assessing children's internalizing and externalizing behaviors. This study used the somatic complaints subset of the CBCL measure. Primary caregivers were asked to evaluate the child's behavior over the past six months on a 3-point Likert scale (0=not true (as far as you know), 1= somewhat true or sometimes true, 2= very true or often true). The somatic complaints subset asked about nine physical problems without known medical causes- including feels dizzy, overtired, aches or pains, headaches, nausea, eye problems, rashes or skin problems, stomachaches or cramps, and vomiting or throwing up. Raw scores of somatic complaints items calculated by adding the scores were used in the analysis with possible ranges from 0 to 18 with higher scores indicating greater severity of somatic symptoms.

Child's sex. The child's sex assigned at birth was collected at baseline—sex as dichotomized to 0=Male, and 1= Female.

Maltreatment status at age 6. Maltreatment status was measured by the Modified Maltreatment Classification System [MMCS: English & LONGSCAN Investigators, 1997; a LONGSCAN-modified version of the Barnett, Manly, and Cicchetti (1993) classification system]. Investigators at the five sites reviewed CPS records to determine the presence and nature of maltreatment allegations among children in their samples. This review was done using the Children were assigned a dichotomous code based on the presence of maltreatment allegations.

Diagnosis of Major Depressive Disorder (MDD) at age 18. Diagnosis for major depressive disorder was assessed using the National Institute of Mental Health Diagnostic Interview Schedule for Children (DISC-IV) (D. Shaffer et al., 2000). The interview was administered to the child participants using the A-CASI (Audio-Computer Assisted Self-Interview) method at 18-year-old. A Major Depressive Disorder (MDD) classification was made if the youth reported five or more symptoms in the past 2-week period, and it was not due to a bereavement. Symptoms of MDD included depressed mood, diminished interest or pleasure, weight loss or weight gain or appetite change, insomnia or hypersomnia, psychomotor agitation, fatigue or loss of energy, worthlessness or guilt, concentration problem or indecisiveness, and thoughts of death, suicidal ideation, suicide attempt or plan.

Statistical analyses

Descriptive analyses were conducted using SPSS, version 27. Latent Class Growth Analysis (LCGA) was performed using Mplus version 8.0 (Muthén & Muthén, 2017)**.** Growth mixture modeling (GMM) is a method that allows for the identification of latent classes that are characterized by distinct trajectories of change in an outcome over time given patterns in the data (Jung & Wickrama, 2008). LCGA is a type of GMM in which all intercept and slope variances are fixed to zero and do not allow within-class variation, whereas GMM allows within-class variations. Although GMM with random intercept and growth factors would allow for more flexible modeling of classes, LCGA was chosen over GMM since LCGA models with within-class variations in this sample resulted in inadmissible results. Since the distribution of somatic symptoms was non-normal across observations, Poisson distribution was applied (Infurna & Grimm, 2018). LCGA analysis involved steps as recommended by Wickrama (2016) (Wickrama, 2016).

First, a traditional latent growth curve model was estimated. This step allows the researcher to examine the data's overall fit to a trajectory with a single shape or form before considering trajectory heterogeneity. The latent curve models were estimated with linear, quadratic, and cubic growth factors. A model with a quadratic curve had the best model fit, so the quadratic model was chosen. The estimated variance of intercept, slope, and quadratic factors were all significant, indicating that there is heterogeneity in the trajectory (Wickrama, 2016). Then, a latent class growth analysis (LCGA) models with an increasing number of classes up to five classes were estimated. Maximum likelihood (ML) was used for model estimation. Multiple sets of the model were run with 600 initial random start values, optimizing the best 120 final stage values, and confirmed that the maximum log-likelihood value was replicated multiple times to protect against locally optimal solutions. The optimal number of classes were selected based on fit indices, successful convergence, class size (>5%of the total sample), high posterior probabilities, entropy, and theoretical interpretability (Jung & Wickrama, 2008; Ram & Grimm, 2009). Fit indices used included the Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), Vuong–Lo–Mendell–Rubin Likelihood Ratio test (LMR–LRT), and Bootstrapped Likelihood Ratio Test (BLRT). As recommended by Nylund et al. (2007), the BIC was given more significant consideration when the results were inconsistent compared to the LMR test and the information fit statistics (Nylund et al., 2007). Finally, the latent trajectory class memberships were saved as a variable and multinomial logistic regression was conducted to examine if sex and maltreatment status before age 6 were significant predictors of membership in the identified classes and assess if certain trajectory classes predicted increased risk for depression diagnosis at age 18.

Results

Descriptive Analyses

The total sample size included 1307 participants. This sample excluded those who did not have any observations of somatic complaints between ages 6 and 16. Table 4.2 summarizes the descriptive statistics for study variables.

Table 4.2

Descriptive Statistics for Study Variables

	N	Mean or n	SD or %	Range
Maltreatment before age 6	1354			
Yes		875	64.6%	
No		479	35.4%	
Child's sex	1354			
Male		657	48.5	
Female		697	51.5	
Somatic Symptoms Raw score				
Age 6	1218	0.99	1.50	0-12
Age 8	1124	1.15	1.73	0-13
Age 10	1015	1.23	1.93	0-14
Age 12	951	1.53	2.05	0-13
Age 14	930	1.51	1.97	0-12
Age 16	867	1.48	2.07	0-14
Borderline or Clinical SOM[1]				
Age 6	1218	72	5.9%	
Age 8	1124	84	7.5%	
Age 10	1015	93	9.2%	
Age 12	951	92	9.7%	
Age 14	930	87	9.4%	
Age 16	867	71	8.2%	
Diagnosis for MDD[2] at age 18	827	22	2.7%	
Diagnosis for GAD[3] at age 18	831	6	0.7%	
Diagnosis for alcohol abuse at age 18	809	44	5.4%	

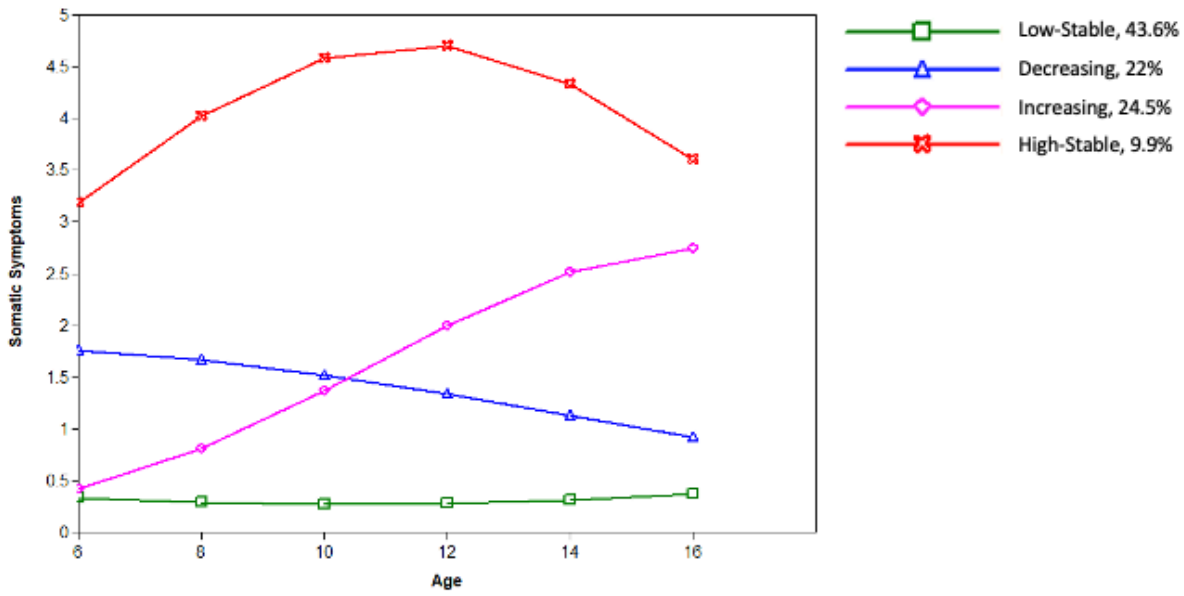
Note. [1]SOM=somatic symptoms, [2]MDD=major depressive disorder, [3]GAD=generalized anxiety disorder

Latent Class Growth Analyses

Although the BLRT p-value was non-significant for class 4, BIC was smaller for the 4-class model than the 3-class model. Per the recommendation by Nylund et al. (2017), BIC was given more significant consideration over LMR *p* values (Nylund et al., 2007). The 5-class model had the lowest BIC score, but the plot's visual inspection suggested poorly separated classes. Thus, the 4-class model was selected. Table 4.3 presents the model fit indices for the LCGA somatic symptom trajectory models. The 4-class somatic complaints LCGA model (Fig. 4.1) consisted of the following trajectory subgroups: low-stable, increasing, decreasing, and high-stable.

Figure 4.1

Estimated Latent Trajectories of Somatic Symptoms



Approximately 43.6% of the sample was in the low-stable group. Children in this group showed persistently low levels of somatic symptoms with low mean intercept (i), negative slope (s), and small quadratic term (q). (i=-1.10, s=-0.18, q=0.04). The increasing group consisted of

about 24.5% of the sample. This group started with low levels at age six and had a consistently increasing trajectory. This group was characterized by positive slope and negative quadrative terms i=-0.88,s=0.74, q=-0.07. There were 22% of the sample in the decreasing group. This group showed a moderate level of somatic symptoms at age six, which slowly decreased over time. This group had a negative slope and negative quadratic terms (i=0.56, s=-0.03, q=-0.02). Finally, 9.9% were in the high-stable group. Children in this group exhibited high levels of somatic symptoms at age six and persistently reported similarly high levels across ages 6-16 with a peak at age 12 and slowly decreased from age 12 to 16. This group had high intercept, positive slope and negative quadratic term (i=1.16, s=0.29, q= -0.05).

Table 4.3

Fit Statistics for LCGA on Somatic Symptoms

Model	AIC	BIC	BLRT *p* value	VLMR *p* value	ALMR-LRT(p)	% in the smallest class	Entropy
1 class	22,419	22,435	<0.00001	-	-		-
2 classes	19,515	19,551	<0.00001	<0.001	<0.001	33.4%	0.789
3 classes	18,952	19,009	<0.00001	0.001	0.0012	10.3%	0.742
4 classes	**18,730**	**18,807**	**1.00**	**0.2018**	**0.2018**	**10.4%**	**0.665**
5 classes	18,593	18,691	1.00	1.00	1.00	5.4%	0.658

Note. AIC= Akaike Information Criterion; BIC=Bayesian information criterion; BLRT=Bootstrapped likelihood ratio test; VLMR=Vuong-Lo-Mendell-Rubin likelihood ratio test; ALMR-LRT=Adjusted Vuong-Lo-Mendell-Rubin likelihood ratio test.

Predictors and outcomes of class membership

Table 4.4 displays the multinomial logistic regression analysis results examining the predictors and outcomes of class membership. The low-Stable group was fixed as a reference group.

Table 4.4

Logistic Regression Results (reference group: Low-stable)

	Decreasing		Increasing		High-stable	
	OR [95% CI]	p	OR [95% CI]	p	OR [95% CI]	p
Predictors						
Female	0.81 [0.61-1.08]	.15	1.33* [1.01-1.76]	.054	1.70* [1.14-2.55]	.01
Early maltreatment	1.17 [0.86-1.58]	.32	1.08* [1.08-1.97]	.01	1.66* [1.07-2.58]	.02
Diagnosis at age 18						
Depression	2.11 [0.42-10.58]	.36	5.88** [1.62-21.32]	.007	5.33* [1.17-24.28]	.03

Increasing class

Female children were 1.33 times more likely to be in the increasing class compared to males. Children who experienced early childhood maltreatment were 1.08 times more likely to be in the increasing class than the low-stable class. Children in the increasing class were 5.88 times more likely to have a diagnosis of major depressive disorder at age 18.

High-stable class

Females were 1.70 times more likely to be in the high-stable group compared to the low-stable group. Children who experienced early maltreatment were 1.66 times more likely to be in the high-stable group than the low-stable group. Those in the high-stable group were 5.33 times more likely to have a diagnosis for MDD at age 18.

Discussion

The primary objectives of this paper were as follows: 1) examine the heterogeneity of trajectories of somatic complaints across ages 6 to 16; 2) assess the extent to which child sex and child maltreatment allegations at age six predict the membership of certain trajectory classes; and 3) examine whether membership in a trajectory class predicts a diagnosis of depression or anxiety symptoms at age 18. The results from the LCGA demonstrate four distinct patterns across ages six to 16 (low-stable, increasing, decreasing, and high-stable). This result aligns with the previous literature and suggests heterogeneity in childhood somatic symptoms (Janssens et al., 2014; Mulvaeny et al., 2006). This study shows similar results to those of Janssens et al. (2014), who suggest four trajectory classes of somatic symptoms among Dutch children ages 10-16. The four trajectory classes identified in Janssens et al. (2014) are categorized into persistent low, increasing, decreasing, and persistent-high, which is similar to findings in the present study (Janssens et al., 2014). Approximately 10% of the children were in the high-stable group, meaning that they were consistently experiencing somatic symptoms across ages six to 16. This result is similar to Dunn et al.'s (2011)’s result that 12% of adolescents (ages 11-14) had persistent pain (Dunn et al., 2011). The percentage of this group is slightly higher than that of the Dutch adolescents in Janssens et al. (2014), which was 4.1%. This discrepancy might be due to the difference in measurement time points or potential differences of other characteristics between the U.S. and Dutch children.

First, experiencing early childhood maltreatment was a significant risk factor for membership in the increasing and high-stable classes. Exposure to early childhood maltreatment is associated with higher somatic symptoms through the interactions among multiple biological, psychological, and ecological factors (Nelson et al., 2017). However, specific mechanisms of

how exposure to early childhood maltreatment may affect somatic symptom trajectories across childhood are unknown. Future studies should investigate the processes of how early maltreatment affects persistent or increasing somatic symptoms across childhood.

Furthermore, being a female and experiencing early childhood maltreatment were significant risk factors for the increasing and high-stable classes. The sex differences as predictors of the increasing trajectory of somatic symptoms may be explained by the physiological changes associated with puberty (Susman et al., 2003). Puberty is a significant precursor for mood and behavior changes. Studies have found that early-onset puberty, which is more frequently found in females, is associated with increased somatic symptoms (Beck, 2008; Williams & Zahka, 2017). Previous studies have noted that differences in the etiology and development of somatic symptoms between males and females result from variations in biological and social processes that lead to distinct perceptions and presentations (Gijsbers Van Wijk & Kolk, 1997; Martel, 2013; Padgaonkar et al., 2020). Future studies should further examine how being a female may be associated with persistent somatic symptoms across childhood.

Finally, being in the increasing group and high-stable groups was significantly associated with MDD diagnosis at age 18. This finding aligns with previous studies' results (Campo, 2012; Shanahan et al., 2015). A potential explanation of this finding is that children who experience persistent somatic symptoms might have increased attentional bias/perception of pain and irritability, resulting in increased irritability and social withdrawal (Beck et al., 2011). Somatic symptoms can also cause sensitization of the stress reactivity system, resulting in inflammatory pathways to psychopathology such as MDD (Dantzer et al., 2008; Raison et al., 2006). Young adulthood is a developmental phase that critically shapes interpersonal, educational, and

economic growth across different life stages. Depression in young adulthood is a costly burden for families, workplaces, and society (Bonnie et al., 2015). Therefore, early identification of depression during this period is essential in reducing the long-term burdens of mental illnesses. However, identifying individuals at risk for later mental illnesses remains a significant barrier to prevention and appropriate service delivery (Costello et al., 2014). This study's findings suggest that intervention efforts should pay closer attention to children who experience persistence and increasing patterns of somatic symptoms in childhood and adolescence. The findings also suggest that close follow-up of children with a history of early maltreatment, especially females, is crucial given the high-risk nature of this population. Moreover, the results suggest that closer attention and appropriate interventions should be provided to children who are persistently complaining of somatic symptoms across the childhood period, as they are at greater risk for depression as young adults.

Strengths and limitations

This study's strengths include using longitudinal person-centered analyses to capture the latent heterogeneity of somatic symptoms across the childhood period. Additionally, this study utilizes a large sample size across the U.S., capturing diverse races/ethnicities with different exposures to maltreatment. However, several limitations of this study must be acknowledged. First, the study includes many children who were considered at-risk for maltreatment at baseline. This may limit the generalizability of the findings to other populations. Second, this study utilizes the caregiver reported CBCL to measure somatic complaints across ages six to 16. Although CBCL is widely used to measure somatic complaints and other behavior problems in children, CBCL may not be a reliable measure of somatic symptoms. Future studies should use more reliable measures of somatic symptoms and incorporate and compare reports by other

informants, such as the children themselves, schoolteachers, and secondary caregivers. Third, there may be other factors, such as anxiety/depression symptoms, comorbidities, ACEs, and child temperaments, that may confound the relationship between the persistence of somatic symptoms and the diagnosis of MDD at age 18. Since this study did not control for such factors, it is difficult to determine the extent to which somatic symptom classes predict MDD at age 18. Finally, there are many other outcomes at young adulthood that are known to be associated with trajectories of somatic symptoms in childhood, including generalized anxiety disorder, increased suicidality, and oppositional disorders (Copeland et al., 2009; M. L. Engel et al., 2018; Shanahan et al., 2015; Shelby et al., 2013). Future studies should examine the association between trajectory patterns and more diverse outcomes.

Implications

This study's findings offer several important clinical implications. Persistent somatic symptoms among children are often observed by caregivers, school staff, and primary care providers. Health and service providers should close attention to children reporting frequent somatic symptoms, as they are at a higher risk for developing MDD. Moreover, providers should deliver interventions to help children manage somatic symptoms before they become persistent. Lastly, providers should consider assessing past exposure to trauma and applying appropriate interventions to mitigate the effects of childhood trauma.

Conclusion

In summary, this study identifies four distinct trajectory classes of somatic symptoms. Being a female and exposure to early childhood maltreatment were risk factors of persistent and increasing trajectory groups. Health and service providers should consider heterogeneity in

longitudinal patterns of somatic symptoms and risk factors and develop or deliver interventions for children who present somatic symptoms. Additionally, this study has found that persistent or increasing somatic symptoms in childhood were a risk factor for the diagnosis of MDD at age 18. This result suggests that persistent or increasing somatic symptoms in childhood may be used as an early marker of risk for MDD in young adulthood. Somatic symptoms are often reported to caregivers, teachers, school nurses, and primary care providers. Identifying children with frequent and recurrent somatic complaints and appropriate interventions may help prevent late emotional disorders. Future research should examine more potential predictors of somatic symptom trajectories such as parenting styles, personality traits, and early childhood attachment strategies.

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Chapter 5. Conclusion

Findings

The overall purpose of this dissertation was to advance the understanding of the effects of ACEs in the etiology and development of somatic symptoms in childhood and adolescence.

Chapter 2 examined the concurrent and longitudinal relationships between ACEs and somatic symptoms across adolescence (ages 12-16 years). This study specifically compared the effects of timing and type of ACEs on somatic symptoms in adolescents. The results of this study indicated that ACEs at age 12 were associated with increased somatic symptoms at age 12, and household dysfunction had stronger effects on concurrent somatic symptoms at age 12 as compared with maltreatment. This study found no longitudinal associations between ACEs and somatic symptoms in adolescence. This study's findings highlight the association between ACEs and concurrent somatic symptoms at age 12. In addition, household dysfunction had stronger effects on concurrent somatic symptoms compared to maltreatment. The findings suggest that the accumulation, timing, and types of ACEs have unique effects on somatic symptoms in adolescence.

Chapter 3 used longitudinal mediation analysis to examine the relationships among ACEs, somatic symptoms, and anxiety/depression symptoms during adolescence and investigated whether anxiety/depression symptoms mediate the relationship between ACEs and somatic symptoms. This study's findings indicated significant concurrent associations between ACEs and increased anxiety/depression symptoms and between anxiety/depression symptoms and increased somatic symptoms at ages 12, 14, and 16. Moreover, anxiety/depression symptoms significantly mediated the pathway between ACEs and concurrent somatic symptoms at ages 12 and 14. These findings encourage addressing ACEs in adolescence, such as by preventing maltreatment and family dysfunction and providing effective interventions for mitigating the

effects of ACEs. This may reduce unnecessary stress-related symptoms and subsequent burdens on children and families

In chapter 4, growth mixture modeling was used to examine the heterogeneity, predictors, and outcomes of somatic symptom trajectories across ages 6-16. This study's result identified four distinct patterns of trajectories: low-stable (43.6%), increasing (24.5%), decreasing (22%), and high-stable (9.9%). Being a female and exposure to maltreatment before age six predicted membership in the increasing and high-stable group. Compared to the low-stable group, membership in the increasing and the high-persistent group were more likely to be diagnosed with the major depressive disorder at age 18. This study's findings suggest heterogeneity in longitudinal patterns of somatic symptoms and risk factors should be considered when developing or delivering interventions for children presenting with somatic symptoms. Moreover, this study's findings suggest that persistent or increasing somatic symptoms in childhood may be used as an early marker of risk for MDD in young adulthood.

Relevance and implications to the field of nursing

Children's health is defined as "The extent to which individual children or group of children are able or enabled to develop and realize their potential, satisfy their needs, and develop the capacities that allow them to interact successfully with their biological, physical, and social environments" (Stein, 2005). The goal of the discipline of nursing is to promote and improve the health of individuals, families, and communities (Grady, 2017). Accordingly, addressing somatic symptoms in children is an imperative part of pediatric nursing. The findings of this dissertation provide several important implications for future nursing research and practice. This dissertation's findings indicate that children with persistent somatic symptoms across childhood are more likely to have experienced early childhood maltreatment, may be

experiencing increased anxiety and depression symptoms, concurrent exposure to ACEs, and are at increased risk for major depressive disorder at age 18. Nurses in community settings such as schools, primary care clinics, and public health often encounter children with somatic symptoms. Nurses in these fields are well-positioned to detect, address, and advocate for children with somatic symptoms. In particular, school nurses regularly care for children with persistent somatic symptoms with frequent visits to the nurse's office (Shannon & Bergren, 2010). Nurses must consider underlying factors associated with somatic symptoms such as ACEs and anxiety/depression symptoms and implement appropriate assessments, interventions, and referrals. For example, when caring for children with persistent somatic symptoms, nurses may consider screening for past and concurrent and past ACEs and anxiety and depression symptoms. Furthermore, the implementation of appropriate interventions, as well as referrals, need to be made. Moreover, this dissertation's findings of the significant impact of ACEs in early childhood and adolescence calls for improvement of policy and practice efforts to prevent ACEs and/or mitigate the harmful effects of ACES. For example, when caring for children with persistent somatic symptoms, family-based interventions addressing family dynamics should be considered. Future research efforts should be made to develop effective strategies to detect, address, and advocate for children experiencing somatic symptoms and ACEs in community settings. Such methods may include standardized screening protocols for concurrent ACEs, utilizing school nursing data for screening those with persistent somatic symptoms, or nurse-led interventions such as mindfulness and self-management support for children experiencing somatic symptoms.

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