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LOMA LINDA UNIVERSITY School of Behavioral Health in conjunction with the Faculty of Graduate Studies

Adverse Childhood Experiences, Cognitive Ability, and Psychosocial Functioning

by

Maleia Mathis

A Dissertation submitted in partial satisfaction of the requirements for the degree Doctor of Philosophy in Psychology

September 2019

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ABBREVIATIONS

ACE	Adverse Childhood Experience
ADHD	Attention Deficit Hyperactivity Disorder
CD	Conduct Disorder
GAD	Generalized Anxiety Disorder
KBIT	Kaufman Brief Intelligence Test
ODD	Oppositional Defiant Disorder
PHQ	Patient Health Questionnaire
PSC	Pediatric Symptom Checklist
SES	Socioeconomic Status

ABSTRACT OF THE DISSERTATION

Adverse Childhood Experiences, Cognitive Ability, and Psychosocial Functioning

by

Maleia Mathis

Doctor of Philosophy, Graduate Program in Clinical Psychology Loma Linda University, September 2019 Cameron Neece, Chairperson

Nearly half of all children in the U.S. have experienced at least one Adverse Childhood Experience (ACE) and 23 percent have experienced at least two ACEs. Cumulative exposure to ACEs places children at increased risk for poor psychosocial functioning in childhood and cognitive ability may mediate this relationship. The study aims were to assess the predictive ability of cumulative exposure to ACEs on poor psychosocial functioning and to determine if verbal and non-verbal cognitive ability mediated this association. Parent-child dyads from a low-income pediatric clinic were assessed. Parents were given a parent report measure to assess the child's psychosocial functioning and prospective and known exposure to ACEs. Children were given the Kaufman Brief Intelligence Test, Second Edition (KBIT-II; Kaufman & Kaufman, 2004) to assess verbal and non-verbal cognitive ability. We hypothesized that ACEs would predict elevated scores on a parent-report measure of poor psychosocial functioning and that verbal and non-verbal cognitive ability would mediate the association between child ACE score and poor psychosocial functioning. We found that ACE exposure was predictive of poor psychosocial functioning overall, as well as internalizing and externalizing behavior problems specifically. We found no significant mediating effect

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of verbal or nonverbal cognitive ability on the relationship between ACE exposure and psychosocial functioning. Future research should examine the effect of targeted interventions to decrease internalizing and externalizing symptoms in early childhood, in order to improve psychosocial functioning later in adolescence and to reduce psychopathology in adulthood.

CHAPTER ONE

INTRODUCTION

Adverse Childhood Experiences (ACEs) – including child maltreatment, substance use of a household member, mental illness or incarceration of a household member, domestic violence, and divorced or separated parents - have a well-established connection to health outcomes (Dong et al., 2004; Dube, Felitti, Dong, Giles, & Anda, 2003; Felitti et al., 1998). According to the most recent national survey of child health, nearly half of all children in the U.S. have experienced at least one ACE (Child and Adolescent Health Measurement Initiative, 2012). Furthermore, approximately 23 percent of children under age 17 have experienced two or more ACEs. Children with multiple exposures to ACEs disproportionately exhibit a range of poor mental health outcomes when compared with children who have not experienced multiple ACEs (Bethell, Newacheck, Hawes, & Halfon, 2014; Lucenko, Sharkova, Huber, Jemelka, & Mancuso, 2015). For example, children who have experienced multiple exposures to childhood adversity are at increased risk for the development of internalizing and externalizing behavior problems (Jaffee, 2017). Internalizing disorders after childhood adversity include both mood and anxiety disorders (Brown, Cohen, Johnson, & Smailes, 1999; Cohen, Brown, & Smailes, 2001). Externalizing disorders after childhood adversity include disorders of attention or impulsivity (Hunt, Slack, Berger, 2017), as well as disruptive behavior and conduct disorders (Cohen, Brown, & Smailes, 2001; Famularo, Kinscherff, & Fenton, 1992; Murray & Farrington, 2010). However, despite numerous research studies linking specific kinds of childhood maltreatment and household dysfunction with various poor psychosocial outcomes in childhood, there

remains a gap in the literature assessing the effect of cumulative exposure to multiple ACEs on overall psychosocial functioning, used here to encompass both internalizing and externalizing behavior problems, as well as problems with attention.

Evidence suggests that there are likely multiple developmental processes that are adversely impacted by ACE exposure and which may result in poor psychosocial functioning including physical, cognitive, emotional, and social development (Mulvihill, 2005). However, cognitive development stands out as one developmental process that may explain some of the relationship between ACEs and poor psychosocial functioning in childhood, because of role that cognitive ability plays in social relationships and social processes, which in turn impacts prosocial behavior and adaptive coping (Manninen et al., 2013; Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). Additionally, externalizing behavior problems are a particular area of focus within poor psychosocial outcomes, because they are associated with increased likelihood of engagement in health risk behaviors, a common outcome associated with ACE exposure in the literature (Fanti & Henrich, 2010; Jokela, Ferrie, & Kivimäki, 2009).

Researchers have consistently found that exposure to childhood maltreatment has an adverse impact on cognitive functioning (Crozier, & Barth, 2005; Pandey, 2011), and that cognitive ability is implicated in the development of poor psychosocial functioning in childhood (Dietz, Lavigne, Arend, & Rosenbaum, 1997; Kusche, Cook, & Greenberg, 1993). Thus, it may be that some children are at increased risk for the development of internalizing and externalizing behavior problems in childhood because of the impact of ACEs on their cognitive functioning. However, researchers have yet to test cognitive

ability as an explanatory mechanism for the development of poor psychosocial functioning in childhood after ACE exposure.

The present investigation addressed this gap in the literature by testing whether cumulative ACE exposure predicted poor psychosocial functioning in childhood. Additionally, this investigation tested cognitive ability as an explanatory mechanism for the relationship between ACE exposure and poor psychosocial functioning in childhood. This investigation examined 1) if increased risk of ACE exposure predicted poor psychosocial functioning; and (2) whether or not verbal and non-verbal cognitive ability mediated the relationship between ACEs and poor psychosocial functioning.

Adverse Childhood Experiences

Childhood maltreatment and household dysfunction have a well-established connection to adult health outcomes (Benjet, Borges, & Medina-Mora, 2010; Mulvihill, 2005; Shonkoff et al., 2012). In one of the largest U.S. studies of cumulative risk, Felitti, and colleagues (1998) assessed for a connection between multiple exposures to ACEs and adult health outcomes. Their line of research found that ACEs were common, often cooccurring, and collectively predicted numerous poor mental and physical health outcomes in adulthood (Dong et al., 2004; Dube, Felitti, Dong, Giles, & Anda, 2003; Felitti et al., 1998). ACE research spurred a national conversation about the impact of ACEs on adult health outcomes; and, as a result, addressing ACEs has moved to the forefront of many local and national health organization agendas (Bethell, Newacheck, Hawes, & Halfon, 2014). However, despite advances in ACE research, numerous gaps remain in understanding the full implications of ACEs on health outcomes. For example, given the

established role that ACEs play in adult mental and physical health outcomes, and the fact that 47% of US children have experienced at least one ACE, further investigation is needed to understand the impact of ACEs on childhood outcomes (Bethell et al., 2014).

Childhood adversity widely impacts individual development and is predictive of changes in physical, cognitive, emotional, and social development in childhood (Mulvihill, 2005). Since infants and toddlers are most vulnerable to maltreatment and household dysfunction, these changes often begin at the earliest stages of development and extend throughout childhood and adolescence (Lansford, Dodge, Pettit, Bates, Crozier, & Kaplow, 2002; U.S. Department of Health & Human Services, 2017). Researchers argue that children exposed to maltreatment and household dysfunction experience a combination of hyperarousal and dissociation (Mulvihil, 2005). These states function as adaptive physiological responses that protect the individual by releasing endogenous opioids into the body, resulting in decreased arousal, decreased awareness of pain, and decreased heart rate and blood pressure (Mulvihil, 2005). However, chronic or cumulative stress exposure overwhelms the body's adaptive stress-response and the body is forced to create a new baseline in order to compensate; this internal restructuring can lead to changes in the structural organization of the brain as well as the specific cognitive process that contribute to overall brain functioning (Cicchetti, 2002; Mulvihil, 2005). Childhood maltreatment and household stress are related to electrophysiological abnormalities in the frontal lobe, reduced volume of the amygdala and hippocampus, abnormalities of the size of the corpus callosum, and highly lateralized hemispheric responses to memory recall (Navalta, Polcari, Webster, Boghossian, & Teicher, 2006; Cicchetti, 2002). These findings, and others, support that childhood adversity alters the

structure of the brain and may explain some of the adverse impact of childhood adversity on child development.

The impact of maltreatment and household dysfunction on development is also likely a result of the interaction between biological processes and environmental processes. For example, a child's propensity for language development is partially due to the child's genetic material but also partially due to exposure to a language-rich environment and to the quality of interaction between the child and caregivers (Stacks, Beeghly, Partridge, & Dexter, 2011; Eigsti & Cicchetti, 2004). In cases of childhood exposure to maltreatment and household dysfunction, children are less likely to be exposed to the types of environments that have a positive effect on language development (Eigsti & Cicchetti, 2004). Moreover, children who are exposed to chronic adversity also learn to respond to the social cues from their environment differently than children who are not exposed to chronic adversity (Eisenberg et al., 2001; Kim & Cicchetti, 2010). As children develop cognitively and socially, they learn prosocial ways of responding to problems in their environment. However, children who are exposed to adversity are more likely to rely on aggressive problem-solving strategies because their environment did not expose them to prosocial strategies, and because the changes in their brain cause difficulties with identifying appropriate ways of responding (Eisenberg, Cumberland, Spinrad, 1998; Eisenberg, et al., 2001; Hann, 2001). Taken together, the evidence of interactions between biology and environment following ACE exposures supports the farreaching effect of ACEs on various aspects of child development and may also explain the eventual development of poor psychosocial functioning in childhood.

Childhood Adversity and Childhood Psychosocial Functioning

Cumulative exposure to childhood maltreatment increases the risk of children developing internalizing disorders like depression and anxiety (Brown, Cohen, Johnson, & Smailes, 1999; Cohen, Brown, & Smailes, 2001), and externalizing disorders like attention deficit hyperactivity disorder (ADHD; Hunt et al., 2017), oppositional defiant disorder (ODD), and conduct disorder (CD; Cohen, Brown, & Smailes, 2001; Famularo, Kinscherff, & Fenton, 1992; Murray & Farrington, 2010); this holds true even after controlling for the adverse impact of socioeconomic status (SES; Bolger & Patterson, 2001; Weitzman, & Wegner, 2015). Household dysfunction has also been associated with poor psychosocial functioning; Dean and colleagues (2010) found that parental mental illness was strongly associated with externalizing and internalizing behavior problems in adolescence. Murray and Murray (2010) also found a similar association between parental incarceration and externalizing and internalizing behavior problems in children. Furthermore, in a longitudinal study Keiley and colleagues (2001) found that early maltreatment exposure was a stronger predictor of externalizing and internalizing behavior problems than later maltreatment exposure, even after controlling for SES and gender. This is significant because young children, prior to age four, are at greatest risk for maltreatment exposure (U.S. Department of Health & Human Services, 2017). These findings suggest that the developmental processes interrupted when children are exposed early in life may have a greater influence on psychosocial functioning than abuse exposure alone.

Some researchers have argued that child characteristics, like externalizing or internalizing behavior problems, place a child at increased risk for child abuse, thus

calling into question the direction of the relationship between maltreatment and psychosocial functioning (Bousha & Twentyman, 1984; Wolfe, 1985). However, evidence supports that maltreatment, and particularly cumulative exposure, leads to poor psychosocial functioning in children, rather than the other way around (Moffitt, 2005; Burke, Pardini, & Loeber, 2008; Jaffe, 2017). For example, Jaffe and colleagues (2004), in a prospective twin study, found that child maltreatment was associated with changes over time in children's antisocial behaviors, that there was a dose-response relationship between chronicity of maltreatment and severity of behavior, and that maltreatment was still predictive of abuse even after controlling for parental antisocial behavior. These findings support that maltreatment plays a significant role in behavior outcomes for children. Moreover, even when preschoolers were matched on behavioral characteristics and socioeconomic status in a longitudinal study, additional life stressors, such as relational issues between parents or parental mental health concerns, led to future child maltreatment, rather than child behavior or early parenting practices (Keiley, Howe, Dodge, Bates, & Pettit, 2001). These findings suggest that caregiver exposure to multiple stressors may have temporal precedence within the etiology of child maltreatment.

Poorer psychosocial functioning following ACE exposure is likely caused by impaired brain mechanisms; specifically, impairments within the prefrontal cortex are thought to be linked with the development of internalizing and externalizing behavior problems because of the role of the prefrontal cortex in emotion regulation and the inhibition of aggressive impulses (Burghy et al., 2012; Herringa, Birn, Ruttle, Burghy, Stodola, Davidson, & Essex, 2013; Liu, Raine, Venables, & Mednick, 2004). Externalizing behavior problems are a particular point of focus because researchers have

found that ACEs have a stronger predictive effect on externalizing behavior problems than they do on internalizing behavior problems in early childhood (Appleyard et al., 2005; Hunt et al., 2017). Additionally, externalizing behavior problems are of particular focus because they are associated with increased likelihood of engagement in health risk behaviors, whereas internalizing behaviors are not as strongly implicated in the development of health risk behaviors (Fanti & Henrich, 2010; Jokela et al., 2009; Timmermans et al., 2009); engagement in health risk behaviors were posited to explain the connection between ACEs and poor health outcomes in the original ACE study by Felitti and colleagues (1998). High levels of externalizing problems in early childhood have also been found to predict rapid increases in internalizing problems later in development; and, internalizing problems tend to increase in middle childhood and adolescence, while externalizing behavior problems tend to decrease after middle childhood (Gillion & Shaw, 2004), but externalizing behavior problems remain a focus of the early and middle childhood literature because of the predictive role of externalizing behavior problems in future problem behavior like juvenile delinquency and adult crime and violence (Cohen et al., 2001; Farrington, 2001; Hann, 2001; Liu, 2004; Retz 1995). Overall, there remains strong evidence linking exposure to childhood adversity with the development of both internalizing and externalizing behavior problems and it is unclear which element of psychosocial functioning is most strongly impacted when examining cumulative exposure in early childhood, as this literature is still emerging.

Childhood Adversity and Cognitive Ability

In addition to effects on psychosocial functioning, ACEs also adversely impact a child's cognitive ability. Young children, whose brains are experiencing marked neuronal activity, are most vulnerable to stress exposure and are most likely to experience long term effects (Lansford, Dodge, Pettit, Bates, Crozier, & Kaplow, 2002; Moylan, Herrenkohl, Sousa, Tajima, Herrenkohl, & Russo, 2010; U.S. Department of Health & Human Services, 2017; Watts-English, Fortson, Hooper, & De Bellis, 2006). Exposure to childhood adversity alters the way the brain is structured, its volume, and its neuronal connectivity; all of which may negatively impact overall cognitive ability (De Bellis, 2005; De Bellis, Hooper, Spratt, & Woolley, 2009; De Bellis, et al., 1999; Teicher, Ito, Glod, Andersen, Dumont, & Ackerman, 1997; Tottenham et al., 2010; Watts-English et al., 2006). Children exposed to maltreatment and household dysfunction were found to perform more poorly on cognitive tasks than those who were not exposed to these adversities, even when participants were matched on SES and IQ (Beers & De Bellis, 2002). They also had IQ scores that were 10 points below average for their age norms (Zimmerman et al., 1997), and demonstrated particular deficits in complex, higher-order cognitive tasks (Pechtel & Pizzagalli, 2011; Tottenham et al., 2010). It is likely that the extensive structural and functional changes that occur following ACE exposure are a result of the body's attempt to protect the child from his or her environment and to maintain homeostasis following the body's stress response, which then result in cognitive deficits (Mulvihil, 2005).

Sylvestre and colleagues (2016) found that children who were exposed to abuse or neglect demonstrated delays in expressive, receptive, and pragmatic language when

compared to children who were not exposed to abuse or neglect. Additionally, Sylvestre and Mérette (2010) found that cumulative exposure to multiple adversities, including neglect and household dysfunction, was more predictive of language deficits than neglect on its own. Lum and colleagues (2015) conducted a meta-analysis of language deficits in children exposed to maltreatment and found that they consistently demonstrated poorer verbal ability than non-maltreated children across various measures of vocabulary development, expressive language ability, and receptive language ability. It is likely that the pathway from maltreatment to verbal deficits involves a complex interaction between biological and environmental processes. One explanation is that maltreatment affects the development of the frontotemporal region, which affects multiple processes, including language development (Stacks et al., 2011). It is also likely that parents who are exposed to these household stressors are less likely to interact with a child in a way that enriches their language development (Stacks et al., 2011; Eigsti, & Cicchetti, 2004).

Exposure to ACEs also impacts visuo-spatial cognitive ability, which is a person's ability to identify visual and spatial relationships among objects; visuo-spatial, or non-verbal cognitive ability, is important because it mediates the brain's ability to perform higher order cognitive tasks (Davis, Moss, Nogin, & Webb, 2014; Navalta, Polcari, Webster, Boghossian, & Teicher, 2006; Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984). Tarter and colleagues (1984) found that children who were exposed to parental substance abuse performed more poorly on measures of visuo-spatial cognitive ability than a non-exposed control group. They also found that physically abused adolescents had lower Performance IQ scores (another name for visuo-spatial intellect) on average than non-abused adolescents. Navalta and colleagues (2006) found that young adult

women who had been sexually abused performed more poorly on cognitive tasks that assessed reasoning and problem solving than did control participants, even after controlling for variables that may confound cognitive ability like depression and anxiety. Davis and colleagues (2014) argue that poor integration between the left and right hemispheres of the brain, caused by the reduced size of the corpus callosum, may explain the resulting deficits in visuo-spatial cognitive ability that follow maltreatment. These findings support that childhood adversity not only negatively impacts verbal cognitive ability, but also has a negative impact on the development of non-verbal cognitive ability.

Cognitive Ability and Childhood Externalizing Behavior Problems

The impact of childhood adversity on cognitive development is concerning because the resulting cognitive deficits are implicated in externalizing behavior problems. The development of externalizing behavior problems after ACE exposure is multifactorial, often involving the interaction of various developmental processes for each individual child, including parent-child interactions and child specific characteristics (van Os, Jones, Lewis, Wadsworth, & Murray, 1997). While evidence has implicated multiple developmental processes, cognitive development has been consistently examined as a contributor to the etiology of externalizing behavior problems (Kusche, Cook, & Greenberg, 1993; van Os et al., 1997). Broadly speaking, evidence supports that lower overall cognitive ability is associated with greater externalizing behavior problems in child populations (Dietz, Lavigne, Arend, & Rosenbaum, 1997; Pihet, Combremont, Suter, & Stephan, 2012). Researchers have found that overall cognitive ability is inversely related to child behavior problems on parent report measures (Cook, Greenberg,

& Kusche, 1994); and among children with developmental disabilities, researchers have found that a cognitive, rather than a diagnosis-centered, approach to understanding behavior problems is most supported by the literature (Visser, Berger, Prins, Lantman-De Valk, & Teunisse, 2014).

Cognitive ability likely contributes to externalizing behavior problems because of its effect on social relationships (Tarter, Hedegus, Winsten & Alterman, 1984). Deficits in verbal cognitive ability are known correlates of antisocial behaviors because of their role in socialization (Manninen et al., 2013). It has even been argued that higher verbal cognitive ability may protect against antisocial behaviors by promoting prosocial behavior and self-control (Hann, 2001; Manninen et al., 2013). Children with higher verbal IQ are better able to problem solve relational difficulties with peers and illicit regulatory support from caregivers, thus strengthening the parent-child relationship (Tarter et al., 1984). Furthermore, researchers have also found that cognitive flexibility, which allows an individual to adjust their thoughts and responses appropriately across social situations, and which is considered necessary for socially appropriate conduct, has been directly associated with eternalizing behavior problems (Morgan & Lilienfeld, 2000; Visser et al., 2014).

Visuo-spatial cognitive deficits are implicated in social processes, specifically in poor attachment and difficulty with emotion recognition and emotion processing (Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999; Raine et al., 2002). This is because visuo-spatial deficits impact a child's ability to correctly identify the caregiver's emotional cues and to respond appropriately, thus interfering with parent-child attachment (Raine et al., 2002). Additionally, visuo-spatial deficits are associated with a

reduced ability to accurately identify negative and positive emotions, which may elicit a fear-response, together with a self-protective, though incorrect, aggressive response (Speltz et al., 1999; Raine et al., 2002). These cognitive deficits directly impact the child's ability to function appropriately in social situations and predispose the child to aggressive responding.

Regarding specific cognitive deficits and their role in externalizing behavior problems, there have been mixed results regarding whether verbal or non-verbal cognitive ability is a stronger predictor of the development of externalizing behavior problems in childhood (Im-Bolter & Cohen, 2007; Plomin, Price, Eley, Dale, & Stevenson, 2002; Raine et al., 2002). Im-Bolter and Cohen (2007) found that deficits in verbal abilities are implicated in many of the most common externalizing disorders from pre-school through adolescence, including: ODD, and CD. Beitchman and colleagues (1996) found that verbal cognitive abilities at age five were predictive of externalizing behavior problems on parent report measures at age 12.5. Additionally, in a metaanalysis of current literature, Hollo and colleagues (2014) found that 81 percent of children with clinical behavioral disorder diagnoses exhibited verbal abilities that were significantly below average.

However, despite this emphasis on the role of verbal cognitive ability, Plomin and colleagues (2002) found that in a young child population, externalizing behavior problems on parent report measures were associated with both non-verbal and verbal cognitive ability, and were more strongly associated with non-verbal cognitive ability. Raine and colleagues' (2002) longitudinal study found that non-verbal, and not verbal, cognitive ability was implicated in externalizing disorders at eight and at 17 years of age.

Flouri and colleagues (2011) found that non-verbal cognitive ability might actually play a protective role for children after exposure to adversity because of the role of problem solving in social adjustment. These mixed findings suggest that further research is needed to determine the role of verbal and non-verbal cognitive ability in the development of externalizing behavior problems in childhood.

The Present Investigation

The cognitive abilities affected by maltreatment impact a child's ability to express him- or herself, to integrate his or her experiences, and to respond appropriately to social situations (Speltz et al., 1999; Raine et al., 2002). After experiences of childhood adversity, these skills would be particularly necessary to allow children to understand and process what they have experienced. Additionally, despite a wealth of information linking specific kinds of childhood maltreatment and household dysfunction with various poor psychosocial outcomes in childhood, there remains a gap in the literature assessing the effect of cumulative exposure on psychosocial functioning. Researchers have also not yet examined cognitive ability as a potential mediator in the relationship between exposure to childhood adversity and externalizing behavior problems, despite that evidence supports these associations. The present investigation aims to address these gaps in the literature by examining cumulative ACE exposure and its effect on childhood psychosocial functioning, and by clarifying the role of verbal and non-verbal cognitive ability in the relationship between ACEs and externalizing behavior problems.

Aims and Hypotheses

The present investigation addressed the following aims and hypotheses:

 <u>Aim one:</u> Determine if ACEs are associated with poor psychosocial functioning.

> Hypothesis 1a: Higher ACE score will be positively associated with scores on a parent report measure of poor psychosocial functioning. Hypothesis 1b: Higher ACE score will be positively associated with scores on a parent report measure of a child's externalizing behavior problems.

2. <u>Aim two:</u> Determine if verbal and non-verbal cognitive ability mediate the relationship between ACEs and psychosocial symptoms.

Hypothesis 2a: Verbal and non-verbal cognitive ability will both mediate the relationship between ACEs and scores on a parent report measure of externalizing behavior problems.

Hypothesis 2b: Verbal cognitive ability will be a stronger mediator for externalizing behavior problems than non-verbal cognitive ability.

Hypothesis 2c: Verbal and non-verbal cognitive ability will both mediate the relationship between ACEs and scores on a parent report measure of total behavior problems.

Hypothesis 2d: Verbal cognitive ability will be a stronger mediator on total behavior problems than non-verbal ability.

3. While the literature has established a connection between ACEs and internalizing emotional problems, the literature has demonstrated smaller effect sizes for the pathway between ACEs and internalizing emotional problems when compared with the pathway between ACEs and externalizing behavior problems. We expected to be under power for these analyses; nevertheless, we chose to run these analyses in order to look at the direction of effect and observed effect sizes in the current sample. The following aim was deemed "exploratory" given our expectations about power.

Exploratory aim one: Determine if ACEs are associated with internalizing emotional problems.

Exploratory Hypothesis 1a: Higher ACE score will be positively associated with scores on a parent report measure of internalizing emotional problems.

Exploratory Hypothesis 1b: Verbal and non-verbal cognitive ability will both mediate the relationship between ACEs and internalizing emotional problems.

METHOD

Participants and Procedures

Participants were primarily recruited from the Loma Linda University Pediatric Resident Clinic during Well-Child visits for children ages five to 11 years old; the remaining participants were recruited from the Faculty Pediatric Clinic. The Loma Linda University Pediatric Resident Clinic serves low-income patients, most of whom receive Medicaid. The Loma Linda University Faculty Pediatric Clinic serves patients with private insurance. After the Well-Child visit, families received an informational letter about the study in the mail. Participants then received a telephone call recruiting them to participate in the study and, if they consented, were scheduled to come in for a research visit, mailed research visit supplies, and received further information about participation in the study. Within eight weeks of the Well-Child visit, parent-child dyads participated in a one-time research visit; during the visit, parents completed self-report measures with demographic information, their child's ACE score, and their child's psychosocial functioning. In addition, children were assessed using the Kaufman-Brief Intelligence Test, Second Edition (KBIT-II) by a trained research assistant. There were two participants excluded from these analyses due to not providing data on a relevant measure.

Of the children who were included in the study (N=99), 51% of the children were female, 49% of the children were Hispanic / Latino, 6.3% were African American / Black, 17.7% are Caucasian / White, and 22.9% belonged to more than one ethnic group (Table 2). 92% of the parents were female, 78% were recruited from the Pediatric Resident Clinic, 10.2% of parents reportedly completed less than a high school education,

25% of parents reportedly completed their high school education, 25.5% reportedly completed a bachelor's degree, and 6% of parents reportedly completed additional education beyond a bachelor's degree.

Measures

Demographic Control Variables

Participants reported demographic information including age, gender, ethnicity, and highest level of education obtained (Appendix A). Parental education was used as a proxy variable to assess the family's socioeconomic status (SES).

Child ACE Score

Child ACE score was assessed as a zero-eleven count of total ACEs, reported at the research visit (Appendix B). Parents reported their child's ACE exposure, and risk of ACE exposure, and each ACE that was reported received a score of one. An overall ACE score was summed and a total ACE score was assigned based on the total number of ACEs endorsed.

Physical abuse

Parents reported if their child "ever lived with a parent or other adult who pushed, kicked, physically hurt, or threw something at the child?" Parents also reported if they needed "to hit/spank" their child? One point toward the total Child-ACE score was counted if parents responded affirmatively to either question.

Sexual abuse.

Parents reported if they "know or are concerned that [their] child was ever touched, or asked to touch, an adult or someone at least 5 years older sexually?" Parents also reported if their child "ever lived away from home for more than a month." One point toward the total Child-ACE score was counted if parents responded affirmatively to either question.

Emotional abuse

Parents reported if they "ever swear at or insult their child?" And, parents reported if they felt their child was "difficult to take care of." One point toward the total Child-ACE score was counted if parents responded affirmatively to either question.

Physical Neglect

Parents reported "how difficult was it for [the participant's] family to meet expenses for basic needs like food, clothing, and housing in the last year." Parents responded on a 5-point scale from "not at all" to "very" difficult (Pudrovska, Schieman, Pearlin, & Nguyen, 2005). One point toward the total Child-ACE score was counted if parents responded "somewhat," "fairly," or "very" difficult.

Emotional Neglect

Parents reported if they "look out for each other, feel close to each other and support each other?" One point toward the total Child-ACE score was counted if parents did not endorse this question.

Parental Substance Abuse

Parents reported if their child has "ever lived with anyone who had a problem with drugs or alcohol?" Additionally, parents reported if they "have had more than 4 drinks containing alcohol in one day?" One point toward the total Child-ACE score was counted if parents responded affirmatively to any of these questions.

Mental Illness in the Family

Parents reported if their child has "ever lived with anyone who was depressed, mentally ill, or suicidal?" One point toward the total Child-ACE score was counted if parents responded affirmatively to this question.

Current Mental Illness

Parents were also asked to respond to the two-item Patient Health Questionnaire (PHQ-2) and the Generalized Anxiety Disorder scale (GAD-2; Löwe et al., 2010; Löwe, Kroenke, & Gräfe, 2005). One point toward the total Child-ACE score was counted if parents respond positively to the family mental health question, or score 2 or more on either the PHQ-2 or the GAD-2.

Parental Incarceration

Parents reported if their child has "ever lived with anyone who went to prison, jail, or other correctional facility?" One point toward the total Child-ACE score was counted if parents respond positively to this question.

Domestic Violence Exposure

Parents reported if their child has ever "witnessed adults in the home pushing, hitting, kicking, or physically threatening each other." One point toward the total Child-ACE score was counted if parents respond affirmatively to this question.

Parental Divorce / Separation

Parents reported if their "child's parents [are] separated, divorced, or not living together?" One point toward the total Child-ACE score was counted if parents are separated, divorced, or not living with a partner.

Cognitive Ability

Verbal and non-verbal cognitive ability were assessed using the Kaufman Brief Intelligence Test, Second Edition (KBIT-II). The KBIT-II can be administered to individuals ages four through 90 and provides a brief measure of verbal and non-verbal intelligence (Kaufman & Kaufman, 2004). The verbal cognitive score measures word knowledge, a range of general information, verbal concept formation, and verbal reasoning ability. The verbal cognitive score is assessed using the verbal knowledge (tests receptive vocabulary and general knowledge) and riddles (comprehension, reasoning, and vocabulary knowledge) subtests. The non-verbal cognitive score measures problem-solving skills by assessing an individual's ability to perceive relationships and complete visual analogies. The non-verbal cognitive score is assessed using the matrices subtest (ability to complete visual analogies and understand relationships). Per the administrative manual, item responses were scored dichotomously: correct responses received a score of one and incorrect responses received a score of zero. Raw scores were then converted to standard scores and percentile ranks using the tables in the manual.

The KBIT-II has an internal consistency coefficient of .90 for verbal IQ for children and adolescents ages four through 18 (Kaufman & Kaufman, 2004). It has an internal consistency coefficient of .86 for nonverbal IQ for children and adolescents ages four through 18. It has an internal consistency coefficient of .92 for IQ composite for children and adolescents ages four through 18. The adjusted test-retest reliability of the KBIT-II for children ages four through 12 for verbal is r = .88, for non-verbal is r = .76, and for IQ composite of r = .88. KBIT-II scores have also been correlated with other IQ assessments to determine validity. When compared with the Wechsler intelligence scale's measure for verbal IQ the KBIT-II had an adjusted correlation r = .80, for non-verbal IQ it had an adjusted correlation r = .62, and for IQ composite it had an adjusted correlation r = .81.

Psychosocial Functioning

The Pediatric Symptom Checklist (PSC) is a parent report measure with subscales that screen for attention, internalizing symptoms, and externalizing symptoms (Appendix C). PSC questions also assess for somatic complaints and various school concerns. Taken together these subscales and items give an overall score for psychosocial symptoms. The PSC cut-off score of 28 has a specificity of .68 and a sensitivity of .95. The test-retest reliability of the PSC ranges from r = .84 to r = .91 (Jellinek, Murphy, Robinson, Feins, Lamb, & Fenton, 1988; Murphy et al., 1992). Researchers found a

strong internal consistency for the PSC (Cronbach α = .91, *p* < .001; Jellinek et al., 1988). Parents reported if each of the 35 items happen "never," "sometimes," or "often" and items were scored zero, one, or two respectively (Jellinek, et al., 1988). Overall PSC scores were summed and a total score was given; scores above 28 indicate clinically significant psychosocial problems.

Within the PSC-35 are 17 questions that make up three subscales; these subscales are used to determine if children are demonstrating concerns in the area of attention, internalizing, or externalizing symptoms (Gardner et al., 1999). Responses that indicated children "Feel sad, unhappy," "Worry a lot," "Feel hopeless," "Seem to be having less fun," or "Down on yourself" indicated internalizing problems. Reponses that indicated children "Fight with other children," "Tease others," "Do not listen to rules," "Refuse to share," "Do not understand other people's feelings," "Does not listen to rules," or "Take things that do not belong to you" indicated externalizing problems. Responses that indicated children are "Fidgety, unable to sit still," "Distracted easily," or "Has trouble concentrating," "Acts as if drive by a motor," or "Daydreams too much," indicated attention-related behavior problems. The subscales performed as well as commonly used screening measures, such as the Child Depression Inventory, the Child Anxiety-Related Disorders, and the Child Behavior Checklist subscales (aggression, attention, anxiousdepressed, internalizing, externalizing, and total behavior) in predicting diagnoses of attention-deficit/hyperactivity disorder, externalizing disorders, and depression (Gardner, Lucas, Kolko, & Campo, 2007). However, the internalizing items are less successful at identifying anxiety-related disorders (Gardner et al., 2007).

Data Analytic Plan

Descriptive statistics were reported for each of the variables to provide an overview of sample characteristics (Table 1). Control variables under consideration included child age, child gender, SES, and child ethnicity. Age of onset of adverse exposure has been found to impact psychosocial outcomes in childhood, such that for certain maltreatment types like neglect and physical abuse earlier age of onset was correlated with worse outcomes (Maikovich-Fong & Jaffe, 2010; Mulvihill, 2005). Evidence supports that there is an interaction between gender and child maltreatment on externalizing behavior problems (Jung, Herrenkohl, Lee, Hemphill, Heerde, & Skinner, 2015; Maschi, Morgen, Bradley & Hatcher, 2008). Socioeconomic status has also been found to impact externalizing behavior problems in childhood (Eamon, 2000; Slopen, Fitzmaurice, Williams, & Gilman, 2010). Finally, evidence suggests that ethnic identity may play a role in psychosocial adjustment (Serrano-Villar & Calzada, 2016; Yasui, Dorham, & Dishion, 2004). If age, gender, SES, and ethnicity, were significantly correlated with the outcome variables, we controlled for their effect in the analysis. Participants were excluded from this analysis if they did not provide data on any of the relevant measures.

Table 1							
Demographic Table	ble						
	African American/	Latino/ Hispanic	Asian/ Pacific Islander	Caucasian/ White	Multiethnic Mean (SD)	Other Mean (SD)	All Subjects Mean (SD)
(96 = N)	Black Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)			
Child Age	6.5 (3.67)	7.85 (2.80)	7.67 (1.25)	8.47 (1.91)	7.55 (1.88)	6.00 (0.00)	7.81 (2.07)
Child Gender		53 100V		1 100/	CO 100/		
Female % Male %	50.0% 50.0%	53.19% 46.81%	33.33% 66.67%	41.18% 58.82%	59.10% 40.90%	0.0% 100%	51.0% 49.0%
Parental Education	14.83 (2.14)	12.48 (4.95)	16.33(0.47)	15.06 (3.27)	13.88 (3.53)	18.00 (0.0)	13.61 (2.34)
ACEs Risk	2.0 (1.53)	2.06 (2.05)	0.33 (0.47)	2.61 (2.87)	2.88 (2.46)	0.0 (0.0)	2.13 (2.13)
KBIT Total	98.17 (16.27)	96.06 (12.96)	114.0 (18.68)	107.00 (11.73)	99.96 (15.82)	114.0 (0.0)	100.03 (14.44)
Verbal	98.83 (12.75)	94.68 (11.43)	108.67 (21.03)	102.50 (12.21)	98.83 (18.67)	106.0 (0.0)	98.12 (14.21)
Non Verbal	96.83 (20.06)	97.85 (15.51)	115.33 (12.74)	109.11 (13.05)	100.83 (16.83)	118.0 (0.0)	101.38 (16.20)
Total Behavior Problems	4.50 (5.43)	8.68 (8.28)	12.67 (12.66)	15.56 (13.15)	11.58 (11.58)	1.0 (0.0)	9.71 (9.14)
Externalizing Behavior Problems	0.33 (0.52)	2.13 (2.63)	1.33 (2.31)	3.11 (3.14)	2.0 (2.78)	1.0 (0.0)	0.94 (1.52)
Internalizing Behavior Problems	0.33 (0.52)	0.66 (1.09)	2.33 (4.04)	2.17 (2.62)	1.21 (1.96)	1.0 (0.0)	1.98 (2.52)

Table 1

We tested the assumptions of a hierarchical linear regression analysis to test our aim and our exploratory aim. Fit of relationship between predictor and outcome variables were determined based on a scatter plot of the predictor variable on the outcome variable, and the predictor variable on the residuals of the outcome variable, with a superimposed fit (loess) line. Correct specification of predictor variables was determined based on the literature. Measurement error of predictor variables was addressed to the best of our ability by using reliable and valid measures. Constant variance of residuals was determined by plotting the residuals of the outcome variable on a scatter plot to see if the residual was roughly constant across all values of the predictor variable; we then examined the plots for the suggestion of heteroscedasticity. Independence of residuals was tested by evaluating index plots and looking for clustering and serial dependency. Normality of residuals was tested by examining a normal probability plot. Outliers were determined by evaluating the leverage, discrepancy, and influence of any outlier variables; when appropriate outliers were either corrected or deleted. Multicollinearity was assessed using Variance Inflation Factor tests.

Additional assumptions that were tested for the mediation model for aim two included specification of causal order and direction (reverse causal effects). These assumptions were assessed using theory and research. Misspecification caused by unmeasured variables was accounted for by using research to determine appropriate variables for the model. Measurement error in the mediator was accounted for by selecting a tool with strong psychometric properties.

Hypothesis (1-2): ACEs will predict poor psychosocial functioning overall as well as externalizing behavior problems: Assumptions for parametric statistics were tested as

listed above. Using a hierarchical regression model, ACE score predicting overall PSC symptoms and PSC externalizing symptoms was tested after controlling for gender and financial difficulty.

Hypotheses (3-5): verbal and non-verbal cognitive ability will mediate the relationship between ACEs and externalizing behavior problems: Assumptions were tested as listed above. A multiple mediation analysis using bootstrapping was conducted to determine if verbal and non-verbal cognitive ability mediated the relationship between ACEs and PSC externalizing symptoms, after controlling for gender and financial difficulty. Multiple mediation was used because it allows for multiple mediator variables to be tested simultaneously, while reducing Type 1 error and parameter bias (Preacher & Hayes, 2008a). Statisticians recommend the use of bootstrapping, because it tests indirect effects, does not assume that the sampling distributions are normal, and is a more robust test in the case of smaller sample sizes (Hayes, 2009).

Our aims were tested using SPSS, and the multiple mediation macro "Indirect," developed by Preacher and Hayes (2008a), to determine if verbal and non-verbal cognitive ability mediated the relationship between ACEs and psychosocial problems. This macro uses bootstrapping, which takes a sample (n) with replacement from the original sample, estimates the coefficients from predictor to mediator and from mediator to outcome variable, and then calculates an indirect path (mediation effect). Afterwards, the computer runs through that same procedure (k) number of times in order to create a sampling distribution. This method produces the total mediation effect, as well as specific coefficients for the effect of each mediator variable. "Indirect" also gives the standard error and a 95% confidence interval (CI) for each indirect effect. Statisticians recommend

reporting and interpreting bias-corrected and accelerated bootstrap confidence intervals because they are the most accurate (Preacher & Hayes, 2008a).

Power

Power analyses were run to determine the sample size needed to conduct the following analyses. Since power analysis techniques are still developing to accurately calculate the power associated with multiple mediation (Thoemmes, MacKinnon, & Reiser, 2010), a power analysis was conducted to calculate the sample size needed to ensure we had enough power to detect the direct effect of our predictor variable on the outcome variable. Multiple mediation with bootstrapping is a more robust test in the case of smaller sample sizes, and, therefore, calculating the power associated for a direct effect served as an adequate proxy for the sample size needed for a multiple mediation analysis (Hayes, 2009).

Appleyard and colleagues (2005) found that the direct effect for ACEs and externalizing behavior problems yielded an r^2 of .13. These results were used to calculate an f^2 effect size $[f^2 = r^2 / 1 - r^2]$. Then, G* Power 3.1, a software to calculate statistical power, was used to calculate needed sample size (Faul, Erdfelder, Buchner, & Lang, 2009). It was determined that a sample size of 89 was needed to have the power necessary to detect the direct effect of ACEs on externalizing behavior problems. Given that the measure for poor psychosocial functioning was designed to capture total symptom presentation it stands to reason a sample with sufficient power to detect a moderate direct effect ($f^2 = .15$), will be sufficient for this study. Therefore, it was

determined that a sample size of 89 was needed to have the power necessary to detect the direct effect of ACEs on total symptom presentation of psychosocial functioning.

Appleyard and colleagues (2005) also found that the direct effect for ACEs on internalizing behavior problems yielded an r^2 of .08. It was determined that a sample size of 147 would be needed to have the power necessary to detect this direct effect. Since we knew we were likely to be under power to detect an effect given our sample size (n=99), we proposed examining internalizing behavior problems as an exploratory analysis that would be explored along with our study aims, but which were not main study aims.

RESULTS

Preliminary Analyses

Preliminary analyses examined correlations between variables of interest (Table 2). When ACEs were predicting internalizing behavior problems, externalizing behavior problems, and overall psychosocial functioning, child gender was significantly correlated and was used as a control in the mediation analyses. There were two participants excluded from these analyses due to not providing data on relevant measures and an additional two participants excluded from these analyses to relevant measures that represented significant data outliers.

Table 2

Correlations for Measured	Variables (N=99)								
	1	2	3	4	5	6	7	8	9	10
1. Child Age	1.00									
2. Child Gender	-0.22	1.00								
3. Parental Education	-0.04	0.27**	1.00							
4. ACEs Risk	-0.01	-0.15	0.01	1.00						
5. KBIT Total	0.18	-0.09	0.27**	-0.05	1.00					
6. Verbal	0.17	-0.15	0.20*	-0.09	0.80**	1.00				
7. Non Verbal	0.13	-0.02	0.23*	0.00	0.83**	.34**	1.00			
8. Total Behavior Problems	-0.05	0.24*	0.06	0.42**	0.12	-0.08	0.24*	1.00		
9. Externalizing Behavior Problems	-0.11	0.22*	0.01	0.34**	0.05	-0.10	0.16	0.82**	1.00	
10. Internalizing Behavior Problems	0.07	0.24*	0.14	0.35**	0.21*	0.04	0.28**	0.78*	0.47**	1.0

Main Aims

Results indicated that when controlling for child gender, risk for ACE exposure accounted for a significant amount of the variance in total behavior problems ($r^2 = 0.35$, p < .001); such that, as risk for ACE exposure increased by one additional exposure, total behavior problems increased by 2.30 points (Figure 1). Additionally, when controlling for child gender, risk for ACE exposure accounted for a significant amount of the variance in externalizing behavior problems ($r^2 = 0.23$, p < .001). As risk for ACE exposure increased by one additional exposure, externalizing behaviors increased by .49 points.

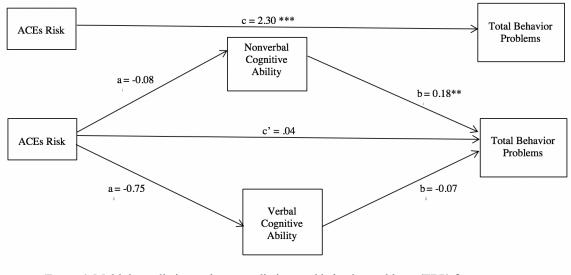


Figure 1. Multiple mediation pathway predicting total behavior problems (EBP) from ACE score through the effect of verbal and non-verbal cognitive ability. * Denotes statistical significance, P < .05; **Denotes statistical significance, p < .01; ***Denotes statistical significance, p < .01.

A mediation analysis using bootstrapping was conducted to test whether nonverbal and verbal cognitive ability were mediators of the relationship between risk for ACE exposure and externalizing behavior problems (Table 3). Non-verbal cognitive ability did not mediate the relationship between risk for ACE exposure and externalizing behavior problems ($a_{1}^{b} = -0.00, 90\%$ CI = [-.04, .04], SE = 0.02). Verbal cognitive ability also did not mediate the relationship between risk for ACE exposure and externalizing behavior problems $a_{2}^{b} = 0.01, 90\%$ CI = [-.01, .05], SE = 0.02.

	Indirect Effect	95% CI	SE
Total	0.01	[04, .07]	0.03
Non-verbal	-0.00	[04, .04]	0.02
Verbal	0.01	[02, .05]	0.02
Contrast	0.01	[03, .07]	0.03

Table 2

Table 4

Multiple Mediat	tion predicting Total B	ehavior Problems		
	Indirect Effect	95% CI	SE	
Total	0.04	[18, .40]	0.16	
Non-verbal	-0.01	[22, .29]	0.14	
Verbal	0.05	[05, .25]	0.08	
Contrast	0.06	[20, .40]	0.16	

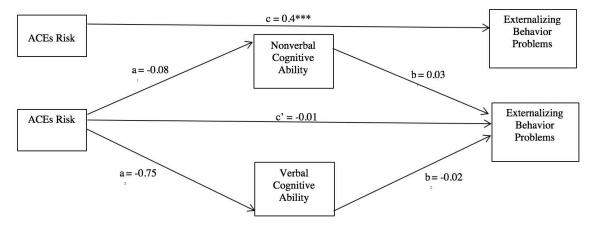


Figure 2. Multiple mediation pathway predicting externalizing behavior problems (EBP) from ACE score through the effect of verbal and non-verbal cognitive ability. ****Denotes** statistical significance, p < .01; *****Denotes** statistical significance, p < .001.

Exploratory Aims

Results indicated that when controlling for child gender, risk for ACE exposure accounted for a significant amount of the variance in internalizing emotional problems ($r^2 = 0.26$, p < .001); such that, as risk for ACE exposure increased by one additional exposure, internalizing emotional problems increased by .34 points (Table 5). Additionally, a mediation analysis using bootstrapping was also conducted to test whether non-verbal and verbal cognitive ability were mediators of the relationship between risk for ACE exposure and internalizing emotional problems (Figure 3). Nonverbal cognitive ability did not mediate the relationship between risk for ACE exposure and internalizing emotional problems ($a_{1}b_{1} = -0.00, 90\%$ CI = [-.04, .04], SE = 0.03; Figure 3). Verbal cognitive ability also did not mediate the relationship between risk for ACE exposure and internalizing emotional problems ($a_{2}b_{2} = 0.00, 90\%$ CI = [-.02, .02], SE = 0.01; Figure 3). Of note, within the model examining the indirect effect of ACEs on internalizing emotional problems, the pathway from non-verbal cognitive ability to internalizing emotional problems was significant (Figure 3).

Table 5				
Multiple Mediat	ion predicting Internal	lizing Emotional Pi	oblems	
	Indirect Effect	95% CI	SE	
Total	-0.00	[04, .05]	0.03	
Non-verbal	-0.00	[04, .04]	0.03	
Verbal	0.00	[02, .02]	0.01	
Contrast	0.00	[04, .05]	0.03	

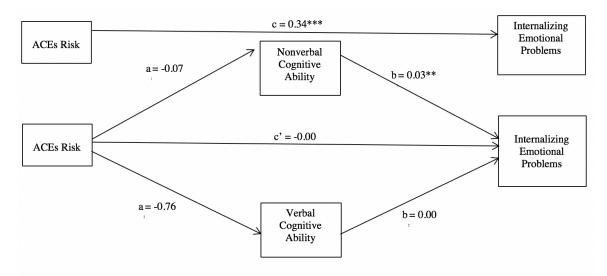


Figure 3. Multiple mediation pathway predicting internalizing behavior problems (EBP) from ACE score through the effect of verbal and non-verbal cognitive ability. ****Denotes** statistical significance, p < .01; *****Denotes** statistical significance, p < .001.

Post-hoc Analyses

Given that there is evidence suggesting that maltreatment type may impact a child's cognitive development, a post-hoc analysis was completed examining whether the maltreatment-specific ACE variables (physical, sexual, and emotional abuse, together with emotional and physical neglect) would significantly predict psychosocial functioning and to test whether non-verbal and verbal cognitive ability were mediators of the relationship between risk for exposure to maltreatment and total behavior problems. Results indicated that after controlling for child gender, risk for exposure to maltreatment accounted for a significant amount of the variance in total behavior problems ($r^2 = 0.38$, p < .001); such that, as risk for ACE exposure increased by one additional exposure, total behavior problems increased by 4.39 points. A mediation analysis using bootstrapping was then conducted to test if non-verbal and verbal cognitive ability were mediators of the relationship between risk for exposure to maltreatment and total behavior problems.

Non-verbal cognitive ability did not mediate the relationship between risk for exposure to maltreatment and total behavior problems ($a_{11}^{b} = 0.16, 90\%$ CI = [-.16, .51], SE = 0.20). Verbal cognitive ability also did not mediate the relationship between risk for exposure to maltreatment and total behavior problems ($a_{22}^{b} = 0.09, 90\%$ CI = [-.11, .44], SE = 0.17).

DISCUSSION

The aims of this study were to determine if risk for childhood ACE exposure was associated with poor psychosocial functioning in childhood and to determine if verbal and non-verbal cognitive ability mediated that relationship. There are numerous retrospective adult studies that have supported a connection between ACEs and poor psychosocial functioning (Dong et al., 2004; Dube, et al., 2003; Felitti et al., 1998); there is also evidence to support that specific maltreatment exposures in childhood are associated with poor psychosocial functioning later in development (Cicchetti & Toth, 2000; Hunt et al., 2017 Murray & Farrington, 2010). Finally, there is a strong body of evidence supporting structural and functional changes to the brain after exposure to childhood maltreatment (DeBellis et al., 2009; Tottenham et al, 2010). Given this, and due to the vulnerability of the young child's developing brain (Moylan et al., 2010), we posited that within a pediatric population, prospective and parent report of ACE exposure would predict poor psychosocial functioning and that cognitive ability would serve as an explanatory mechanism for the relationship between exposure to multiple ACEs and poor psychosocial outcomes. Our study found that in a sample of children ages five to 11, prospective and parent report of cumulative ACE exposure was predictive of poor psychosocial functioning overall, as well as internalizing emotional problems and externalizing behavior problems specifically. We found no significant mediating effect of verbal or nonverbal cognitive ability on the relationship between ACE risk exposure and psychosocial functioning.

While there is a sizable body of evidence supporting the association between exposure to individual types of maltreatment and household dysfunction and poor

psychosocial outcomes in children and adolescents (Cohen, Brown, & Smailes, 2001; Dean et al., 2010; Weitzman, & Wegner, 2015), and strong evidence supporting the predictive effect of ACE exposure on adult psychosocial outcomes (Dong et al., 2004; Felitti et al., 1998; Schilling, Aseltine, & Gore, 2007), this study represents a significant contribution to the literature by examining prospective and parent reports of exposure to ACEs in a pediatric population. Researchers have found that the combination of prospective report (risk of exposure) and self-report leads to the most comprehensive identification of participants who have suffered abuse (Shaffer, Huston & Egeland, 2008). Shaffer and colleagues also found that when using this method, the identified participants accurately reflected those with the greatest number of maltreatment exposures and those displaying the most internalizing and externalizing problems in adolescence. Given this, our study represents a unique contribution to the literature in that it identifies ACE exposure from prospective and parent report and uses these scores to predict psychosocial functioning. While the adult retrospective studies offer an important perspective by looking at the impact of ACEs over the life course, it is difficult to parse out at which stage in development we begin to see ACE exposure adversely impact developmental outcomes and thus to identify when to intervene. Researchers have found that internalizing and externalizing problems in middle childhood predict greater internalizing and externalizing disorders in adolescence and adulthood, and are associated with increased health risk behaviors and poor psychosocial outcomes in adulthood (Cohen et al., 2001; Gillion & Shaw, 2004; Lewis et al., 2011; Liu et al., 2004); given this, it is important to determine the earliest point in development that ACE exposure begins to impact psychosocial outcomes.

Our study supports that with data taken from a cross-sectional sample of children in middle childhood, for those with multiple ACE exposures, these exposures are already predicting greater internalizing and externalizing symptoms specifically, and poorer psychosocial functioning overall. These findings support the need for early identification and intervention support for children who are at increased risk for ACE exposure, as our study found that risk and exposure to ACEs in middle childhood predicted poor psychosocial functioning overall and both internalizing and externalizing symptoms specifically. Given the connection between poor psychosocial functioning early in development, with poor psychosocial functioning and increased health risk behaviors later in development (Cohen et al., 2001; Gillion & Shaw, 2004; Lewis et al., 2011; Liu et al., 2004); and, given that we can reliably assess risk for ACE exposure in childhood in a pediatric primary care setting (Marie-Mitchell et al., In Progress), these findings suggest that early identification and targeted intervention may play an important role in decreasing internalizing and externalizing disorders in adulthood by reducing the adverse impact of ACE exposure on childhood internalizing and externalizing symptoms.

The results of this study also raise the question of the role of cognitive ability in the relationship between ACE exposure and poor psychosocial functioning. While much evidence supports the pathway linking maltreatment and structural and functional changes in the brain with psychosocial outcomes (De Bellis, 2005; De Bellis, Hooper, Spratt, & Woolley, 2009; Herringa et al., 2013), our study did not find that cognitive ability was a mediating variable between ACE risk exposure and poor psychosocial functioning broadly, or internalizing and externalizing behavior problems specifically. Of note, we did find that the pathway between nonverbal cognitive ability and

psychosocial functioning was significant across models. Researchers have found that nonverbal cognitive ability is implicated in social processes because of its role in the identification and interpretation of emotions (Speltz et al., 1999; Raine et al., 2002). Our study supports this earlier research, and given that the path between nonverbal cognitive ability and psychosocial functioning was significant, while the path between verbal cognitive ability and psychosocial functioning was not significant, our findings may support that nonverbal cognitive ability may in fact play a stronger role in psychosocial development than verbal cognitive ability after childhood adversity. This may be because when a child is unable to appropriately recognize and respond to the emotional cues from their environment, they are more likely to exhibit emotional and behavioral dysregulation; however, further research is needed to examine this relationship.

Prior research has found that cognitive ability mediates the relationship between childhood malnutrition and psychosocial outcomes (Liu et al., 2004). Given this, posthoc analyses were conducted to test if the path between maltreatment exposure specifically and poor psychosocial functioning was mediated by cognitive ability; our study did not find that cognitive ability was a significant mediator in this relationship. It may be that cognitive ability is more strongly implicated in the relationship between exposure to particular forms of maltreatment and poor psychosocial functioning, rather than in the relationship between cumulative exposure to maltreatment and poor psychosocial functioning. This may be because exposure to certain types of maltreatment may impact cognitive development more directly; specifically, neglect and physical abuse – with their impact on nutritional status, and therein on brain growth and development, as well as potential exposure to traumatic brain injury – may have a

stronger impact on the development of cognitive ability at this stage of childhood and may thus explain why cognitive ability serves as a mediating mechanism in some studies, though not in ours. Glewwe and King (2001), in a longitudinal study of the impact of malnutrition on cognitive performance, found that malnutrition adversely impacts cognitive performance; they also found that malnutrition later in life had a stronger negative impact on cognitive performance, rather than malnutrition earlier in life. Malnourishment is thought to decrease brain cell growth, to change the brain's neurochemistry, and to increase neurotoxicity within the brain (Liu, Raine, Venables, & Mednick, 2006); it is also thought that these mechanisms predispose the brain to dysfunction and result in an increase in externalizing behavior problems in childhood (Liu & Raine, 2006). Additionally, De Bellis and colleagues (2009) argue that the structural changes that may occur in the brain as a result of physical abuse may impact psychosocial outcomes in children. It may be that cognitive ability mediates the pathway between exposure to neglect or physical abuse, rather than between risk of cumulative ACE exposure or risk of cumulative maltreatment exposure and psychosocial functioning, because of the direct effect of neglect or physical abuse on cognitive development.

Furthermore, some previous research has also noted that severity of abuse may factor into the effect of exposure to childhood adversity on psychosocial functioning (Zanarini, Yong, Frankenburg, Hennen, Reich, Marino, & Vujanovic, 2002). Specifically, Litrownik and colleagues (2005) found that the preservation of ratings of severity within maltreatment types, may be optimal for assessing functional outcomes. And, Naar-King and colleagues (2002) found that severity predicted individual differences in internalizing

symptoms. It may be that cognitive ability plays a mediating role in the pathway between more severe maltreatment subtypes like physical abuse or neglect and psychosocial functioning, rather than in the pathway between cumulative exposure and psychosocial functioning. Of note, the findings on the importance of abuse severity and its impact on the predictive pathway between maltreatment and psychosocial outcomes have been mixed, with other studies finding a predictive effect of maltreatment on psychosocial outcomes above and beyond abuse severity (Herringa et al., 2013). Our findings seem to support that for psychosocial functioning broadly and for externalizing behavior problems specifically, cognitive ability does not significantly mediate this pathway and lends support to the argument that cognitive ability may serve as an explanatory variable for particular subtypes or severity levels of maltreatment.

Finally, given that our study was a prospective and parent report measure of ACE exposure, thus capturing risk for ACE exposure as well as parent report of likely ACE exposure, it cannot be ruled out that there may have been a significant mediating effect had our population examined children with substantiated ACE exposures, rather than those at risk for ACE exposure and those with parent report of ACE exposure. Specifically, we measure physical and sexual abuse prospectively and by parent report, and we measure physical and emotional neglect entirely by prospective report. Given the potential role of abuse severity mentioned above, it may be that are mediating variables were not significant because of study design.

Limitations and Future Directions

Our study has several strengths, including that the data are drawn from a diverse sample, and that the measure used to assess ACE exposures was found to be valid and reliable (Marie-Mitchell et al., In Progress). However, there were several limitations that must be considered, including the cross-sectional nature of the analysis, which limits our ability to draw conclusions about the causal nature of our variables. Another limitation was that psychosocial functioning was assessed using a parent report measure, rather than direct clinical assessment of the child's psychosocial functioning; using a parent-report measure of psychosocial functioning may not truly represent the child's psychosocial functioning, as it may over- or under-estimate the child's true psychosocial functioning. Additionally, a limitation may be that we used a combined prospective and parent-report measure of risk for ACE exposure, rather than direct ACE exposure assessed via substantiated investigation from a child abuse reporting agency. This may result in over or under identification of ACE exposures; however, research suggests that a combination of types of report (prospective and parent or self-report) is more likely to capture maltreatment exposure than self-report alone (Shaffer, Huston, & Egeland, 2008). Furthermore, the ACE measure was designed to capture whether or not particular ACE exposures occurred rather than assessing the impact of the exposure, the severity of the exposure, or the chronicity of the exposure; the bluntness of the instrument may limit some of the conclusions we can draw, as well as our ability to detect certain effects. Finally, for our exploratory aim, we had <60% chance to detect a truly significant effect of $r \square = .19$ at an α of .05, when N=99. Given this, the results of our exploratory aim and associated hypotheses should be interpreted with caution.

Future research should examine the relationship between substantiated ACE exposures and childhood psychosocial functioning in a longitudinal study of pediatric participants. This type of study would add further support for the association between ACEs and childhood psychosocial functioning. It would also allow researchers to draw causal conclusions and to better inform intervention studies. Future research should continue to examine the role of cognitive ability in the relationship between ACEs and psychosocial functioning to clarify whether or not this factor is significantly implicated in this relationship, or if it is only implicated for certain maltreatment subtypes and severity levels. Finally, future research should identify targeted interventions to decrease internalizing and externalizing symptoms in early childhood following ACE exposure, in order to reduce internalizing emotion problems and externalizing behavior problems in adolescence and internalizing and externalizing disorders in adulthood.

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APPENDIX A

Family Information Form

(Last, First	, Middle)	
 Child's Age (years):	3. Sex: 1 = Female	2 = Male
3 = Caucasian (White)		
4 = Hispanic/Latino		
5 = Native American		
6 = Other (Please specify:)	
Your Information 5. Full Name:		
(Last, First, Midd)	e)	
6. Age (years):	7. Sex: 1 = Female	2 - Male
8. Race: (Please circle all that apply)		
1 = African American (Black)		
2 = Asian/Pacific Islander		
3 = Caucasian (White)		
4 = Hispanic/Latino		
5 = Native American		
6 = Other (Please specify:)	
9a. Relationship to child: (Please circle one)		
1 = Biological parent		
2 = Step-parent		
3 = Adoptive Parent		
4 = Foster Parent		
5 = Other (Please specify:	×	_)
9b. If not natural parent, how many mont	hs or years have you live	d with this child?
(# of months)	(#0	of years)
0. Current marital status: (Please eirele one)		
1 - Married		
2 = Separated/Divorced		
3 = Widowed		
4 = Single/Never Married		

APPENDIX B

Whole Child Assessment for Ages 5-8 Years

Child's Name (first & last)	Date of Birth	D Female O Male	Today's Date	School/Grade in School
Person Completing Form © Relative © Friend © Other (specify)	Biological Pare Step Parent Adopted Parent Foster Parent	Regu	ar? □ Yes □ No	School Grades O Average or Better than average D Below average or Poor

ans	ld be healthy. You may skip any question if you do not know a wer. You may add comments to explain your answers. We will ess there's concern that your child is being hurt.	l keep this	information confi	dential,	Clinic Use Only:
1	Does your child eat breakfast every day?	Yes	Unsure	No	Nutrition
2	Does your child drink or eat 3 servings of calcium-rich foods daily, such as milk, cheese, yogurt, soy milk, or tofu?	Yes	Unsure	No	
3	Does your child eat fruits and vegetables at least 2 times per day?	Yes	Unsure	No	
4	Does your child cat high fat foods, such as fried foods, chips, ice cream, or pizza more than once per week?	No	Unsurg	Yes	
5	Does your child drink more than one small cup (4 - 6 oz.) of juice per day?	No	Unsure	Yes	
5	Does your child drink soda, juice drinks, sports drinks, energy drinks, or other sweetened drinks more than once per week?	No	Unsure	Yes	
7	In the past year, did you worry that your food would run out before you got money or Food Stamps to buy more?	Never	Sometimes	Often	1
\$	Does your child exercise or play sports most days of the week?	Yes	Unsure	No	Physical Activity
>	Does your child watch TV or play video games less than 2 hours per day?	Yes	Unsure	No	
10	Are you concerned about your child's weight?	No	A little	Yes	1
	Does your child have trouble falling asleep or staying asleep?	No	Sometimes	Yes	Sleep
12	Does your home have a working smoke detector?	Yes	Unsure	No	Safety
13	Have you turned your water temperature down to low-warm (less than 120 degrees)?	Yes	Unsure	No	
4	Does your home have the phone number of the Poison Control Center (800-222-1222) posted?	Yes	Unsure	No]
5	Do you always place your child in a booster seat in the back seat (or use a seat belt if your child is over 4'9")?	Yes	Unsure	No	
6	Does your child spend time near a swimming pool, river, or lake?	No	Unsure	Yes	
7	Does your child spend time in a home where a gun is kept?	No	Unsure	Yes	1
8	Does your child spend time with anyone who carries a gun, knife, or other weapon?	No	Unsure	Yes	
9	Does your child always wear a helmet when riding a bike, skateboard, or scooler?	Yes	Doesn't ride	No	
0	In the past year, have you felt safe in your relationship?	Yes	No partner	No	1
21	In the past year, have you and your partner fought a lot?	No	No partner	Yes	1
2	Has your child ever witnessed adults in the home pushing, hitting, kicking or physically threatening each other?	No	Unsure	Yes	

23	Has your child ever lived with a parent or other adult who pushed, kicked, physically hurt or threw something at the child?	Nu	t	June	Υœ	
24	Does your child have trouble with anger or get into fights with other children?	No		Unsure	Yes	
25	Has your child ever lived away from home for more than a month?	No	t	Jasure	Yes	
26	Has your child ever been bullied or felt unsafe at school or in your neighborhood (or been cyber-bullied)?	No			Yes	
27	Does your child brush and floss her/his teeth daily?	Yes	ī	Unsure	No	Dental
28	Do you feel your child is difficult to take care of?	Never	So	metimes	Often	Parenting
29	Do you swear at or insult your child?	Never	So	metimes	Often	Stress
30	Do you need to hit/spank your child?	Never	So	metimes	Often	
31	Are you currently living with a spouse or partner?	Yes	1	Unsure	No	
32	Are your child's parents separated, divorced, or not living together?	No	. 1	Unsure	Yes	
33	Did your child ever live with anyone who went to prison, jail or other correctional facility?	No		Unsure	Yes	
34	Do you have friends or family who help take care of your child?	Often	So	metimes	Never	
35	Does your family look out for each other, feel close to each other and support each other?	Often		metimes	Never	
36	Does your child often seem sad or depressed?	No	1	Unsure	Yes	Mental
37	Over the past 2 weeks, how often have you been bothered by any of the following problems?	Not at all	Several days	More than half the days	Nearly every day	Health
	A1. Little interest or pleasure in doing things A2. Feeling down, depressed, or hopeless	0	1	2	3	Total Part A:
	B1. Feeling nervous, anxious or on edge	ŏ	l i	2	3	
	B2. Not being able to stop or control worrying	0	l i -	2	3	Total Part B
38	Did your child ever live with anyone who was depressed, mentally ill or suicidal?	No		Unsure	Yes	
39	Does your child spend time with anyone who smokes?	No		Unsure	Yes	Substance
40	During the past three months, have you had more than 4 drinks containing alcohol in one day?	No		Unsure	Yes	Exposure
41	Did your child ever live with anyone who had a problem with drugs or alcohol?	No		Unisure	Yes	1
42	Do you know or are you concerned that your child was ever touched, or asked to touch, an adult or someone at least 5 years older sexually?	No		Unsure	Yes	Sexual Issue
43	Has a family member or contact had tuberculosis disease?	No		Unsure	Yes	Tuberculosi
44	Has a family member had a positive tuberculin skin test result?	No		Unsure	Yes	Risk
45	Was your child born in a high-risk country (countries other than the United States, Canada, Australia, New Zealand, or Western and North European countries)?	No		Unsure	Yes	
46	Has your child traveled (had contact with resident populations) to a high-risk country for more than 1 week?	No		Unsure	Yes	

47 Do you have a high school degree?	Yes	Unsure	No	Other
48 Do you have any other questions or concerns about your abild's health as behavior?	No	Unsure	Yes	Questions
child's health or behavior?	-			

Clinic Use Only Screened	Counseled	Referred	Provided Handout	Ordered Follow-up	Comments
Nutrition			0		
Physical Activity	D	0	0	0	1
o Sleep	0	0	0	0	
D Safety		0]
Dental Health	0	0	0	0	1
D Parenting Stress	D]
 Mental Health 	0				1
 Substances 	D	Ċ	0		3
Sexual	0	D	0	D	Patient Declined WCA.
Tuberculosis	0	0	D	D]
Other	0	0	D	D]
PCP's Signature	-	F	rint Name	-	Date

APPENDIX C

Participant #

Television and the second state of the second state

Pediatric Symptom Checkilst (PSC)

Emotional and obysical health go together in children. Because parents are often the first to notice a publicm with their child's behavior, emotions, or learning, you may help your child get the first cure possible by answering these quasitors. Please indicate which statement best describes your child.

Please mark under the Islading that best describes your child:

		Never	So wethers	Oilten
 Completins of actes and pains 		and a second second		
Spends more time alone	2			
 Tires easily, has little onergy 	3	NAME OF TAXABLE PARTY.		
 Fidgety, unable to st still 	4			
Has trouble with teacher	. ž. –			
Lett Interested in school	ő			
Acts as if driven by a moto:	7			
8. Daydroams too much	8			
0. Distracted easily	9			
10. Is afraid of new situations	10			
11. Feels sad, unhappy	11			
12. Is initiable, angry	12			
 Feels hopeiess 	1.3			
14. Has trouble concentrating	14			
15. Lass interested in triends	15			
fights with other children	16		1000	
17. Absent from school	125	A short of shorter		
 School grades dropping 	16	1 - 1 - 1 - 2 - 2 - 2 - 2 - 2 - 2 - 2 -	a contactor of	
19. Is down on him or herseli	19	management -		
20. Visits the doctor with doctor linding nothing wrong	20			-
 Has trouble sleoping 	21			
Worries a lot.	22			
 Wants to be with you more than before 	23			
14. Feels he or sho is bad	24			
 Takos unnecessary risks 	25			
%. Gets hurt frequently	26			
7. Seems to be having less fun	22			
Acts younger than children his or her ago	20			
9. Does not listen to rules	20			
0. Dots not show feelings	30			
 Does not understand other people's feelings 	31			
2. Teases others	32			
 Illemes others for his or her troubles 	33			
 Takes things that do not belong to him or her 	34			
5. Refuses to share		The second second	1.000	
	35			
otal score				
os your child have any emotional or behavioral problems for	which s	the or He needs help?	< 5 N	(3Y
there any services that you would like your child to receive it	or thes	e problems?	() N	() Y
w, who services?				