The Childhood Origins of Intimate Partner Violence: The Role of Toxic Stress in Perpetuating the Intergenerational Transmission of Violence

by

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Abstract

The intergenerational transmission of violence describes a pattern of maltreatment that persists across an individual's life course and across multiple generations. People who experience violence in their family-of-origin are more likely to perpetrate intimate partner violence (IPV) in adulthood. While there are undoubtedly many factors that contribute to this complex phenomenon, the emerging field of toxic stress may illuminate mechanisms by which violence and abusive behaviors endure. Scientists across disciplines have found that repeated exposure to traumatic adversity early in life can cause a prolonged elevation and dysregulation of the body's stress response systems, resulting in a lasting legacy of developmental, neurological, behavioral and psychological maladies. The overarching purpose of this dissertation is to investigate the role of toxic stress in perpetuating the intergenerational transmission of violence. Guided by the conceptual model developed and described in the introductory chapter, I have conducted three studies using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) study, a longitudinal and nationally representative sample following American youths through young adulthood. In the first study, I developed a toxic stress response (TSR) index using exploratory and confirmatory factor analysis. The final index contained symptoms of depression, anxiety, emotional dysregulation, and anger. A low, significant chi-square and goodness of fit tests indicated that the model fit the data well (CFI of 0.997, a RMSEA of 0.07, CI 90% [0.045,0.097], and a CD of .90). I then validated the index by correlating TSR scores

with toxic stress exposures, such as childhood maltreatment and experiences of community violence. The correlations were mostly significant and all were in the expected direction. In the second study, employing structural equation modeling (SEM), I tested whether toxic stress response, as measured by the scale constructed in Study 1, mediated the relationship between toxic stress exposure (i.e., childhood maltreatment) and IPV perpetration. I tested this hypothesis in a subsample containing 1,000 participants who reported a history of childhood maltreatment and 2,000 participants who reported no such history. I found that childhood maltreatment is partially mediated by TSR. In the third study, I explored how resilience theory could improve our understanding of why some maltreated children grow up to perpetrate IPV while others do not. Supportive, caring relationships with adults appears to be one of the most consistent protective factors associated with resilience in the face of childhood maltreatment. Using multi-group analysis, I tested whether adult care moderated the mediational model tested in the second study. My analyses revealed that adult care had a moderating effect on the relationship between childhood maltreatment and TSR. For individuals who reported less adult care, the relationship between CM and TSR was stronger ($b=.27$; $p<0.001$) compared with individuals who reported more adult support ($b=.19$; $p<0.001$). These pathways significantly differed from each other across groups with a chi-square of 6.7 ($p<.01$). This dissertation adds to the existing literature base by: developing a new index of TSR that could help future researchers identify participants suffering adverse reactions from toxic stress exposure; illuminating a pathway by which violence is transmitted intergenerationally; and by identifying a resilience factor that may help break the cycle of violence.

CHAPTER 1

Toward an Ecological Model of the Intergenerational Transmission of Violence: Current Theories and New Directions

Introduction

While most maltreated children will not grow into violent adults (C.S. Widom, Czaja, & Dutton, 2014), childhood maltreatment has been identified as a risk factor for intimate partner violence (IPV) perpetration in adulthood (Capaldi, Knoble, Shortt, & Kim, 2012; Foshee, Bauman, & Linder, 1999; Gil-González, Vives-Cases, Ruiz, Carrasco-Portiño, & Álvarez-Dardet, 2008; Stith et al., 2000). This phenomenon in which family violence persists across generations is referred to as the Intergenerational Transmission of Violence (ITV), because a child that suffers violence at the hands of an adult caregiver or witnesses violence within the home is more likely to perpetrate violence against his or her own family members as an adult. One in 10 children bear witness to inter-parental aggression, 1 in 10 children suffer from physical abuse, and 1 in 16 children suffer from sexual abuse (U.S. Department of Health and Human Services, Administration for Children & Families, Office of Planning, Research & Evaluation, 2013). With these nontrivial incidence rates of childhood maltreatment, it is imperative that we understand this significant risk factor for IPV perpetration. Unearthing factors that differentiate childhood victims who become perpetrators from those who do not could significantly contribute to efforts aimed at reducing the incidence of IPV.

Empirical Findings on the Intergenerational Transmission of Violence

A 2008 review of ten research studies published between 1995 and 2004 indicated a consistent association between childhood experiences of violence and the occurrence of IPV (Gil-González et al., 2008). In their review of ITV literature, Delsol and Margolin (2004) determined that the percentage of men aggressing against their wives who reported any family-of-origin violence ranged from 55–70%, compared with 20–28% for men who did not commit violence against their wives. McKinney et al. (2009) found that men who reported having experienced moderate or severe physical abuse as children were at increased risk of perpetrating non-reciprocal violence against their female partners. Severe childhood physical abuse was associated with a two-fold increased risk of reciprocal IPV. The authors reported that women who reported any type of childhood family violence were more than 1.5 times as likely to engage in reciprocal IPV (McKinney, Caetano, Ramisetty-Mikler, & Nelson, 2009).

While longitudinal data regarding the intergenerational transmission of violence is limited, researchers have conducted prospective studies to investigate this phenomenon. Linder and Collins (2005) conducted a prospective study of 121 individuals followed from birth to 23 years of age. They found that people who experienced child maltreatment, witnessed parental IPV, and were subject to boundary violations by a parent (these include parental seductiveness or role reversal) reported higher levels of IPV as young adults. In a prospective cohort study, researchers looked at multiple forms of violent offending, including IPV, among survivors of child abuse and found that individuals with documented histories of abuse and neglect were significantly more likely to self-report IPV perpetration compared with matched controls (Milaniak & Widom, 2015). In a cross-sectional study, Jaffe et al. (1997) found that, among men who abuse their spouses, 75% observed violence between their own parents. Employing retrospective reporting, Roberts et al. (Roberts, Gilman, Fitzmaurice, Decker, & Koenen, 2010) found that witnessing

IPV as a child was correlated with IPV perpetration in adulthood. In sum, previous researchers have found that child maltreatment is a risk factor for IPV perpetration, though the strength of this association varies across studies. What is less clear is what mechanisms of violence transmission are at work.

Traumatic sequelae have been proposed as an important link between family-of-origin violence and adult IPV, both in perpetrators and victims. Experiences of childhood maltreatment are predictive of trauma symptoms and, in turn, trauma symptoms in adulthood may be associated with marital violence. The landmark 1998 Adverse Childhood Experiences (ACE) Study demonstrated a significant relationship between ten adverse childhood experiences—including sexual and physical child maltreatment and neglect--and future violence perpetration, among other unfavorable health and wellbeing outcomes (Felitti et al., 1998). In their analysis of the ACE data, Whitfield and colleagues (2003) reported that physical and sexual abuse as well as witnessing parental aggression against each other was associated with increased risk of IPV perpetration. In recent years scholars across multiple disciplines including Public Health, Biology, Nursing, and Developmental Psychology have built upon and expanded the ACE study findings in significant ways. Researchers have revealed that repeated and prolonged exposure to traumatic stressors, including childhood maltreatment, at a developmentally vulnerable age can yield an elevated and sustained activation of the body's stress response systems (Teicher, Anderson, Polcari, Anderson, Navalta, & Kim, 2003), a phenomenon referred to as Toxic Stress.

The field of toxic stress is comparatively new and the studies of toxic stress and its relationship to violence perpetration of any kind are scant. As such, I turn to the related constructs of trauma and resultant violent behavior.

Toxic Stress

Researchers have highlighted stress as a potent and often noxious force that can corrode mental and physical health, strain familial bonds, and even engender violence. People exposed to high levels of ongoing and traumatic stress early in life may result in psychological dysfunction. This ongoing stress, in turn, can have lasting neurobiological consequences well into adulthood, impeding healthy coping efforts and an individual's ability to manage their emotional arousal (De Bellis, 2001) and potentially making violent behavior toward a partner more likely. Moving forward, I will use the following definition of toxic stress:

> "Strong, frequent, and/or prolonged adversity—such as physical or emotional abuse, chronic neglect, caregiver substance abuse or mental illness, exposure to violence, and/or the accumulated burdens of family economic hardship—without adequate adult support. This kind of prolonged activation of the stress response systems can disrupt the development of brain architecture and other organ systems, and increase the risk for stress-related disease and cognitive impairment, well into the adult." ("Toxic Stress," n.d.)

The authors of a report issued by the American Academy of Pediatrics state that the advances in and cross-pollination across disparate fields such as neuroscience and developmental psychology have resulted in a "paradigm shift" (p. e232) in the way we understand and approach health and wellness (Shonkoff et al., 2012), resulting in toxic stress theory. Toxic stress research draws on multiple lines of inquiry and integrates findings across disciplines to help us forge a better understanding of how the individual interacts with, changes, and is changed by their environment. To wit, trauma symptoms associated with toxic stress encapsulate a larger symptom profile than what is described within Post-Traumatic Stress Disorder (PTSD). PTSD, the most thoroughly researched traumatic reaction, captures the symptom experience of people exposed to discrete traumas. Yet, researchers have found that those exposed to frequent and

repeated trauma, particularly as children, display a wider array of post-traumatic symptoms (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). Responses to traumatic stressors happen along a continuum of severity (Broman-Fulks, et al., 2006) and, beyond PTSD, a variety of negative outcomes may occur. Thus, broader consideration of traumatic experience and its fallout is warranted.

Throughout this dissertation, I move among disciplines with different preferred nomenclatures for the topics central to this discussion. Researchers from multiple disciplines have investigated the effects of early, profound, and prolonged stress on the body and mind. I will be drawing upon findings from the psychological literature on trauma and what several researchers have termed *complex-posttraumatic stress disorder* (C-PTSD) (e.g., Herman, 1997; van der Kolk, Pelcovitz, Roth, & Mandel, 1996), the literature on adverse childhood experiences (ACEs) (e.g., Anda et al., 2006; Felitti et al., 1998), and the toxic stress literature (Franke, 2014; "Toxic Stress," n.d.). While not synonymous, research on C-PTSD, ACEs and toxic stress have conceptual overlaps. The common thread among these distinct lines of research is the focus on the long-term consequences to individuals exposed to traumatic stress/adverse childhood experiences/toxic stressors at a developmentally vulnerable age. These areas of research also have important distinctions. ACE researchers, for example, have focused on a specific set of 10 childhood stressors (Felitti et al., 1998) and several adverse outcomes in adulthood that manifest as a result of exposure to those stressors. These outcomes include everything from chronic disease to behavioral health issues to economic consequences. C-PTSD researchers, on the other hand, are less restricted in their conceptualization of what constitutes a traumatic stressor; however, they limit their investigation of outcomes to the *psychological* consequences of ongoing stress. A toxic stress approach is broader in terms of both stressors and consequences

studied and does not require a clinical diagnosis like C-PTSD.

Theoretical Explanations for the Intergenerational transmission of violence

A variety of theories have been posited to explain the relationship between childhood maltreatment and adult IPV. Social Learning Theory (SLT), the idea that people learn behavior from observation (role modeling), has perhaps most often been employed to further elucidate the mechanisms by which violence is transmitted intergenerationally (Bandura & McClelland, 1977). SLT, however, has notable limitations. For example, SLT suggests that violence is a learned behavior and that children raised by violent adults are more likely to grow up to replicate the violence they witnessed or endured within their own home. Thus, boys' whose fathers physically abused them and their mothers would be more likely to grow up to physically abuse their wives and children. Yet, researchers have not found that type of abuse sustained in childhood (verbal, psychological, physical, sexual, emotional) predicts type of abuse perpetrated in adulthood. In their study looking at the relationship between maternally versus paternally perpetrated childhood maltreatment and how it is related to later dating violence perpetration, Dardis et al. (2013), found that among men, maternal neglect predicted physical partner violence and sexual abuse predicted psychological partner violence. Edwards et al (K. M. Edwards, Desai, Gidycz, & VanWynsberghe, 2009) examined verbal and physical IPV perpetration among college women and found that women who reported paternal physical abuse were more likely to engage in verbal IPV perpetration. SLT might also lead us to conclude that boys are more likely to mimic paternal behavior and girls more likely to reproduce maternal behavior. Here again, the extant literature seems to paint a more complicated picture. Researchers examining the influence of gender on the ITV have reported that abuse from the *opposite* gender parent is more predictive of IPV perpetration in adulthood (K. M. Edwards et al., 2009; Kaura & Allen, 2004). For

example, Dardis et al. (2013) found that, among women, sexual abuse perpetrated by their fathers predicted sexual partner violence perpetration later in life.

Another theory commonly used to explain the ITV is attachment theory (e.g., Babcock, Jacobson, Gottman, & Yerington, 2000). Attachment theorists assert that insecure parent-child attachment and unhealthy, negative ideas about self, others, and relationships are carried into adulthood, affecting their interpersonal behavior (e.g., Belsky, 1993). People displaying an anxious-ambivalent attachment style may be particularly prone to perpetrate violence against their partners. Anxious-ambivalent partners tend toward hyper-vigilance, distrust, jealousy, an outsized fear of rejection, and an intense and demanding need for extreme closeness. These partners may display poor emotional regulation, overreact to stressors, and resort to coercion or intimidation if they perceive a loss of control or power. Attachment theorists suggest that men who witnessed or experienced abuse as children form anxious-ambivalent attachments to their caregivers as well as internal working models that incorporate violence as part of family life and may be more likely to perpetrate IPV.

While attachment theory has provided many critical insights in the ITV, several drawbacks of this theory for explaining IPV are evident. Attachment theorists emphasize the primacy of parent-child interactions in early life, rather than examining attachment formation throughout childhood. Researchers frequently overlook the importance and influence of non-family authority figures (e.g., teachers), peers and other family members (e.g., siblings) to healthy childhood development. While the parent-child attachment bond may have been disrupted, the child may have become securely attached to another caretaking adult or older sibling. Lastly, attachment researchers often focus their inquiries on the mother-child dyad to the exclusion of

the father. Broader contextual factors that may strain the family system, such as neighborhood characteristics or poverty, are notably absent from attachment theory.

Lastly, explanations of the ITV built on family stress theory have been suggested. Family stress theory interprets family violence (i.e., child maltreatment and IPV) as the result of the interplay between partners as well as dynamics within a family system more broadly. Family stress theorists highlight the interplay between stress, resources, and perception, such that family violence is the consequence of accumulated stressors and the perception that one's resources are inadequate to handle said stressors. Resources herein typically refer to money, education, and employment status, though it has often been extended to include other forms of social capital (Rothwell & Han, 2010; Terrion, 2006). Accordingly, the increased levels of stress engendered by low socioeconomic status contribute to chronic relationship strain. Ongoing family violence therefore may be conflated with stressors resulting from insufficient resources (Fox, Benson, DeMaris, & Van Wyk, 2002).

However, while chronic strain engendered by the actual or perceived dearth of resources is surely a contributor to family violence, it is well documented that child maltreatment and IPV cut across all socioeconomic strata (though rates of family violence are more neighborhood among the economically disadvantaged) (Cunradi, Caetano, & Schafer, 2002; Farmer & Tiefenthaler, 1997; Fox et al., 2002). Individuals have innumerable ways, both adaptive and maladaptive, to deal with chronic strain; family stress theory does little to help us understand the etiology of violence specifically. Further, the mechanisms by which abused children grow into abusive adults are not well delineated in the family stress literature.

Child maltreatment is a risk factor for IPV, but it is far from deterministic. Existing theories have vastly deepened our understanding of the intergenerational transmission of

violence, however, they do not tell the full story. It is still unknown why some maltreated children grow up to be non-violent or how to effectively identify those at risk for violence perpetration. The growing body of interdisciplinary research into the developmental, neurological, emotional and psychological effects of early childhood adversity adds to the existing literature base and helps to provide a more complete accounting of the ITV.

Toxic Stress as Precursor to IPV Perpetration

Batterers are a heterogeneous population with diverse psychological profiles. Despite this diversity, researchers have successfully identified and catalogued frequently shared psychological--as well as behavioral, demographic, and environmental--characteristics of individuals who perpetrate violence against their partners. Many of the same psychological features associated with toxic stress have also been found in populations of batterers. People exposed to high levels of ongoing and traumatic stress early in life may result in psychological dysfunction that makes violent behavior toward a partner more likely. Researchers have found that symptoms of trauma mediate the relationship between childhood maltreatment and adult IPV perpetration (see Bell and Orcutt 2009; Taft et al. 2011, for reviews). Wolfe et al. (2004) conducted a longitudinal study and found that trauma symptoms partially mediated the relationship between child maltreatment and IPV perpetration among adolescents. The authors also reported that trauma-related anger predicted female-perpetrated IPV perpetration among female adolescents. Among female participants who reported perpetrating IPV against their partners within the past 6 months, Swan et al. (2005) found unique effects of three types of anger expression (externalized anger, internalized anger, and anger control) on composite IPV perpetration (i.e., combined sexual, psychological, and physical) were computed. Child

maltreatment was positively associated with trauma symptoms, which were in turn, positively associated with IPV perpetration.

Toward an Ecological Theory of the Intergenerational Transmission of Violence

Many theories applied to the intergenerational transmission of violence are somewhat incomplete because they often focus on only one ecological level of influence. Individual theories tend to ignore structural or environmental processes and social theories often ignore individual differences and psychological mechanisms. An ecological perspective that considers the mechanism that help explain how environmental influences operate to effect family and individual behaviors may provide useful insights for points of intervention that take into account multiple levels of influence.

I propose a conceptual model of the intergenerational transmission of violence (see Figure I.1), guided by the developmental-ecological theory (Bronfenbrenner, 1997) and the concept of Toxic Stress. Perpetrators of intimate partner violence are situated within the context of multiple, multi-tiered social systems that shape their health behaviors and outcomes across the lifespan. The model suggests that influences in childhood (distal factors) have effects on more recent factors (proximal influences), which have a more direct effect on IPV perpetration. Distal factors like community process and context and family factors and childhood experience on the left side of the model exert an indirect and weaker influence on IPV perpetration. Toxic stress response occurs in adulthood and exert a more direct influence on IPV perpetration than factors that happened earlier in people's lives. The dashed lines represent important though unmeasured/untested relationships. The solid lines represent relationships that I measure and test within the chapters of this dissertation. Briefly, community factors shape family functioning, which is linked to childhood maltreatment. Here, IPV perpetration is framed as the result of

toxic stress exposure early in life leading to the manifestation of a cluster of maladaptive psychological symptoms (referred to as "toxic stress response"), putting the individual at risk for partner violence perpetration. Risk of violence perpetration is exacerbated by the failure to develop effective strategies to cope with emotional upheaval and relationship conflict.

An important aspect of developmental-ecological theory is that the effect of major developmental influences, such as family functioning, are influenced by the sociological characteristics of the communities in which youth and families reside (Szapocznik and Coatsworth, ND; Tolan and Gorman-Smith, 1997). It is, therefore, important to consider the influence of the characteristics of the neighborhood when attempting to understand the impact of the family on risk of childhood maltreatment. Exposure to childhood maltreatment, which includes physical, sexual, and emotional abuse, is more prevalent in communities with high levels of concentrated disadvantage and crime (e.g., Aber, Bennett, Conley, & Li, 1997; Evans & Kim, 2007; Freisthler, 2004; Freisthler, Midanik, & Gruenewald, 2004; Zielinski & Bradshaw, 2006). Increased levels of stress engendered by living in impoverished communities contribute to chronic strain within families and impair family functioning (Patterson, 2002). Family functioning refers to the ways in which a family unit balances family demands (e.g., ongoing strain, acute stressors) with family capabilities (e.g., tangible psychosocial resources, coping strategies) (McCubbin & Patterson, 1983). In areas of high concentrated disadvantage, few or low paying employment opportunities may yield financial hardship that can threaten family stability and security. High crime rates also contribute to chronic and acute stress within communities, families and individuals (Raghavan, Mennerich, Sexton, & James, 2006). This chronic strain weakens transactions between couples as well as between parents and their children, thereby increasing the chances of exposure to childhood maltreatment (Zielinski &

Bradshaw, 2006). Chronic financial strain and concerns about physical safety could deplete emotional and psychological reserves, hampering the family's ability to appropriately deal with interpersonal stressors (Fox et al., 2002). Stressful family circumstances may overwhelm the parents' ability to parent effectively and who more often resort to physical punishment and emotional neglect (Zielinski & Bradshaw, 2006). Conversely, in communities with low concentrated disadvantage and crime the family's capabilities will be strengthened via provision of support, access to community resources. I suggest that families within communities characterized by high levels of disadvantage and crime are more likely to have greater demands and possess fewer resources to deal with those demands. Moreover, children from poorly adjusted families are more likely to be exposed to toxic stressors and less well equipped to cope with those stressors as adults.

Features of the community act upon family functioning by informing the strength and density of network ties. I suggest that children residing in communities characterized by dense network ties are afforded more opportunities to connect with non-parental adults who could potentially provide them with nurturing and/or mentorship. According to toxic stress researchers, the presence of caring adults and/or mentors may buffer children against toxic stressors, including maltreatment.

Childhood maltreatment is a toxic stressor and exposure to toxic stressors engenders toxic stress response. Based on a review of the extant literature, I suggest that toxic stress response manifests as a cluster of psychological symptoms including emotional dysregulation, anxiety, depression, and anger. I theorize that toxic stress response in people who experienced maltreatment as children renders them ill equipped to appropriately and healthfully negotiate relationship conflict. Victims of childhood maltreatment have lower levels of emotional

regulation--the utilization of efforts aimed at controlling or changing one's own emotional response to a stressful situation (Norman et al., 2012). They may become easily overwhelmed and overreact to minor stressors, have a hard time calming themselves down, and struggle with finding healthy ways to appropriately express anger (Foran & O'Leary, 2008; Norman et al., 2012; Stith, Smith, Penn, Ward, & Tritt, 2004). As a result, they are more likely to resort to violence when relationship conflict arises because they are less able to modulate their emotional state. Mood disorders, depression and anxiety in particular, have been found to be a consequence of childhood maltreatment (Cutajar et al., 2010; Whiting, Simmons, Havens, Smith, & Oka, 2009) and an antecedent to IPV perpetration (Birkley & Eckhardt, 2015; Norlander & Eckhardt, 2005).

Coping strategies moderate the relationship between toxic stress response and IPV. Coping generally refers to both adaptive and maladaptive strategies but given the overwhelming evidence pointing to the importance of alcohol abuse as a coping strategy among childhood survivors of abuse (Anda et al., 2006a; Cutajar et al., 2010; Norman et al., 2012) as well as a significant factor in IPV perpetration (Foran & O'Leary, 2008), I have limited my discussion of coping to alcohol abuse. After a stressor elicits an emotional response, the individual attempts to manage their thoughts and feelings through coping behaviors (Babcock et al., 2000). Researchers aver that individuals who have experienced chronic maltreatment more often employ maladaptive coping strategies including alcohol abuse (Benoit, Bouthillier, Moss, Rousseau, & Brunet, 2010). Alcohol abuse is hypothesized to influence IPV in a variety of ways. Alcohol abuse influences cognitive function and reduces self-control, rendering individuals less able to negotiate a non-violent resolution to relationship conflicts (Foran & O'Leary, 2008). Excessive drinking can also contribute to financial difficulties, which in turn can exacerbate stress levels

between partners ("WHO | Violence publications and resources," n.d.). Alcohol has been hypothesized as a leading contributing factor to IPV (Foran & O'Leary, 2008; Leonard & Senchak, 1996; Stuart, Moore, Kahler, & Ramsey, 2003). Two meta-analyses found significant associations between alcohol use and IPV (Foran & O'Leary, 2008; Moore et al., 2008), with other research identifying alcohol as an important risk factor for IPV perpetration (Stith et al., 2004).

In sum, this model proposes that concentrated disadvantage and community violence increase the likelihood of childhood maltreatment. Childhood maltreatment, experienced against a backdrop of neighborhood stressors, results in toxic stress, which has developmental, neurobiological ramifications resulting in toxic stress response.

Description of Dissertation Studies

Despite the relationship between early childhood maltreatment and adult IPV perpetration, the mechanisms, of violence transmission are not well understood. In the following three studies I employ the concept of toxic stress to guide my investigation into the intergenerational transmission of violence. I begin by developing an index of Toxic Stress Response. With this index I am attempting to capture the psychological symptoms that frequently arise after toxic stress exposure. In the second study, I test whether toxic stress response mediates the relationship between childhood toxic stressors (childhood maltreatment) and adult IPV perpetration. In the final study, I explore resiliency factors that moderate the mediated relationship between childhood toxic stressors and IPV perpetration.

Data are drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health) study, a longitudinal and nationally representative sample following American youths through young adulthood (Udry, Li, & Hendrickson-Smith, 2003). Add Health has four

waves of data collection completed thus far. My dissertation will utilize Waves I and IV only because I am interested in understanding childhood predictors or adult violence perpetration. Wave IV was carried out between January 2008 and February 2009. Survey data were collected using a 90-minute computer-assisted personal interview during in-home interviews. More information on the Add Health project is available from other sources (Blum, Beuhring, & Rinehart, 2000).

The following studies have been designed to investigate the central question: why do some maltreated children grow up to be violent adults while others do not? I hypothesize that toxic stressors and toxic stress response play a key role in propagating the intergenerational transmission of violence. I also suggest that there are promotive factors that buffer the individual against toxic stress, thereby helping to break the intergenerational transmission of violence. The three studies build on existing research and each other. In the first study I will construct a measure of toxic stress response. In the second study I will test whether toxic stress response mediates the relationship between childhood maltreatment and adult IPV perpetration. In the third study I will employ a resiliency framework to examine promotive factors that may buffer against toxic stress.

Study 1: Developing a Measure of Toxic Stress Response

Ongoing and profound stressful experiences can have neurobiological consequences that yield psychological changes. The responses to toxic stress manifest as a constellation of symptoms including emotional dysregulation, depression, anxiety, and anger (Cutajar et al., 2010; Hequembourg et al., 2006; Norlander & Eckhardt, 2004; Birkley and Eckhardt, 2015). Taken together, these impairments created in childhood may be an important link between childhood adversity and adult partner violence.

No measure of toxic stress response currently exists. In the first paper of my dissertation I will employ confirmatory factor analysis to develop a measure that assesses levels of toxic stress response. Convergent validity of the measure will also be evaluated by correlating mean TSR scores to other presumed toxics stressors, such as neighborhood disadvantage, family hardship and community violence. If the measure does in fact measure toxic stress response accurately, then I expect that scores on the Toxic Stress Response scale will increase concomitantly with frequency of other toxic stressors.

Study 2: The Traumatic Origins of Intimate Partner Violence Perpetration and the Mediating Role of Toxic Stress Response

Childhood maltreatment is a risk factor for IPV perpetration in adulthood, but the mechanisms of transmission are not well understood. The psychological sequelae associated with exposure to toxic stressors may be an important link between toxic stress exposure and IPV perpetration. Employing structural equation modeling (SEM), I will test whether toxic stress response, as measured by the scale constructed in Study 1, mediates the relationship between toxic stress exposure and IPV perpetration.

Study 3: The Traumatic Origins of Intimate Partner Violence Perpetration and the Mediating Role of Toxic Stress Response

For the final paper I will investigate what factors moderate the relationship between toxic stress response and IPV perpetration. Most children from violent homes do not become violent adults. Understanding what differentiates these non-violent individuals would help us understand how and where to intervene. To that end, resilience theory, defined as "the process of overcoming the negative effects of exposure, coping successfully with traumatic experiences, and avoiding the negative trajectories associated with risks" may provide crucial guidance

(Fergus & Zimmerman, 2005)(Fergus and Zimmerman, 2005, p. 399). This strengths-based approach focuses on identifying and understanding promotive factors—assets or resources-- that allow a person to thrive even under suboptimal circumstances. Regarding ITV, resilience theory could help us better understand what factors help children exposed to violence grow up have healthy, non-violent relationships. Researchers have identified a number of resiliency factors among youth including relationships with caring adults and mentorship (Sapienza & Masten, 2011). In the final study of my dissertation I will test whether the presence of caring adults moderates the relationship between toxic stressors and IPV perpetration.

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CHAPTER II

Developing a Measure of Toxic Stress Response

Introduction

Scholars across multiple disciplines have revealed that repeated and prolonged exposure to traumatic stressors at a developmentally vulnerable age can yield a lasting legacy of impairments across the lifespan (Anda et al., 1999, 2006a; Felitti et al., 1998; Mersky, Topitzes, & Reynolds, 2013; Sanchez & Pollak, 2009; Shonkoff et al., 2012; Teicher, 2002; Van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). While a certain amount of stress in childhood is normal and necessary for healthy development and survival, stress that is unrelenting, overwhelming, and un-buffered by a nurturing caregiver becomes toxic (Shern, Blanch, & Steverman, 2014). Stress triggers the body's stress-response system (also known as the flight or fight response), which is composed of a host of physiologic changes such as increased respiration and heart rate (Franke, 2014). In most cases, the stress-response is fleeting and the body returns to baseline soon after the perceived threat is removed. Yet, due the strength and chronicity of toxic stress, the body's fight or flight response remains activated resulting in the cumulative wear and tear on the brain and body over time (Shonkoff et al., 2012). Toxic stress early in life interferes with normal psychobiological development, causing a conversion from a brain primarily oriented toward learning (i.e., exploring, acquiring knowledge) to a brain that is primarily focused on survival (i.e., anticipating threat, mobilizing or conserving bodily resources) (Courtois & Ford,

2009).

Scientists have confirmed that toxic stress can lead to physical changes in the size and density of the brain, disrupt normal neurologic development, and damage other organ systems (Teicher, 2002). Because the brain is still developing, traumas occurring earlier in life have a greater potential to disrupt typical neurodevelopmental processes and produce long-term consequences. These neural changes lead to emotional problems and negatively affect mood, attention and inhibitory control (Shonkoff et al., 2009). Prolonged exposure to toxic stress throughout the life course can result in cell damage, causing a shorter lifespan as well as the development of chronic illnesses and disability (Shonkoff, 2012). Childhood toxic stressors have been causally linked with everything from poor school performance (Vaillancourt, Brittain, McDougall, & Duku, 2013) to substance use (cites?) to heart disease (Norman et al., 2012). Researchers of the landmark 1998 Adverse Childhood Experiences (ACE) Study revealed robust associations between childhood adversity and a multiplicity of poor health outcomes, including increased risk of mortality and many forms of morbidity such as autoimmune, liver, coronary, and pulmonary diseases (Dube et al., 2009; Felitti et al., 1998; Felitti & Anda, 2010). Further, the higher the number of adverse events the individual is exposed to, the worse their outcome (Felitti et al., 1998).

Sources of Toxic Stress

I use the term *toxic stressor* to refer to the adversity, trauma, or stress that is so profound as to alter the course of normal biopsychosocial development. Toxic stressors can include the ten ACEs included in the Adverse Childhood Experiences study (these include: physical abuse, emotional abuse, sexual abuse, physical neglect, emotional neglect, witnessing domestic violence, household mental illness, having an incarcerated parent, substance abuse by a member

of the household, and divorce or separation) (Felitti et al., 1998), but may also refer to other chronic trauma endured at a developmentally vulnerable period such as war, torture, and crippling poverty could all potentially be examples of toxic stressors. These toxic stressors have been referred to in other disciplines as chronic stressors, complex traumas, and adverse childhood experiences. What delineates toxic stress from tolerable or normal stress varies from person to person and depends on a variety of factors such as chronicity and severity of the stressor as well as protective factors (e.g., nurturing caregiver) available to the individual ("Toxic Stress," n.d.).

Exposure to trauma is not uncommon. Nearly 90% of individuals participating in the National Stressful Events Survey reported exposure to at least one traumatic event and 30% reported exposure to six traumatic events (Kilpatrick et al., 2013). Within the ACE study, nearly 40% of the baseline sample reported two or more ACEs and over 12% experienced four or more adverse events (Felitti et al., 1998). According to a national survey, nearly 61% of children in the United States have been exposed, either directly or indirectly, to violence (Finkelhor, Turner, Ormrod, & Hamby, 2009).

Sources of toxic stress are myriad and may include exposure to traumas both inside and outside the home. The original adverse childhood experiences study delineated ten relatively common traumatic events, all of which occur inside of the home (Felitti et al., 1998). Subsequent researchers have broadened the definition of what constitutes an adverse childhood experience by including traumas that occur outside of the home, such as community violence, and which are more common to low-income, racially diverse populations, such as the neighborhood disadvantage that may attend endemic poverty (Cronholm et al., 2015; Wade Jr. et al., 2016; Wade, Shea, Rubin, & Wood, 2014).

Child abuse is a relatively common and well-studied brand of adverse experience. According to the CDC, child abuse is "any act or series of acts of commission or omission by a parent or other caregiver (e.g., clergy, coach, teacher) that results in harm, potential for harm, or threat of harm to a child" (Leeb, 2008, pg. # 11). Child abuse, which encompasses physical, sexual, and emotional abuse and/or neglect, is relatively common in the United States. One in 10 children suffer from child abuse, 1 in 16 children suffer from sexual abuse, and almost 1 in 10 children are witnesses to family violence (U.S. Department of Health and Human Services, Administration for Children & Families, Office of Planning, Research & Evaluation, 2013). According to the ACE study of over 17,000 adults, over 30% of the sample reported physical abuse and nearly 20% reported sexual abuse as children (Felitti, Anda, Nordenberg, & Williamson, 1998). Among women, childhood maltreatment is by far the most common cause of traumatization (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In the general population, 17-33% report histories of abuse and these estimates increase to as high as 50% within clinic-based samples (Cloitre, Cohen, RE, & Han, 2001). Child abuse is highly correlated with depression, suicide, smoking, alcoholism, obesity, sexually transmitted infections, heart disease, stroke, and liver disease later in life (Felitti, Anda, Nordenberg, & Williamson, 1998).

Because toxic stress responses masquerade as other conditions, they are often missed in the clinical setting (Van der Kolk et al., 2005). This error in diagnosis overlooks the true cause of their pathology and undermines treatment efficacy. Further, individuals exposed to toxic stress are particularly vulnerable to perpetuating the cycle of violence, highlighting the importance of identifying patients as early as possible (Cook, et al., 2005). If we are able to effectively identify a consistent pattern of psychological symptoms that manifest subsequent to experiences of childhood adversity, we may be able to intervene before risky health behaviors are adopted. Yet,

no measure of toxic stress response that takes into account the multiple psychological consequences of toxic stress exposure currently exists.

Toxic Stress and the Developing Brain

When child abuse is repeated and prolonged, some individuals may experience lasting neurobiological consequences. Chronic trauma experienced at a young age leads to dysregulation of the autonomic nervous system and the limbic system. The hypothalamic-pituitary-adrenal axis (HPA-axis) controls reactions to stress and regulates numerous body processes, including emotion. When faced with a stressor, the HPA-axis acts upon a variety of hormones including epinephrine and norepinephrine (dictating the body's flight or fight response) and cortisol (regulating changes in the body due to stress, including immune function), (Gunner & Quevedo, 2007). When stressors become traumas, the levels of epinephrine and norepinephrine become chronically increased, resulting in damage to memory, rational thought, hypervigilance, and an inability to distinguish between dangerous versus benign signals. Corticosteroids become chronically lowed, reducing immune functioning. Traumatic exposure also impacts neurotransmitters, such as serotonin, dopamine, which impact mood.

Because the brain is still developing into early adulthood, traumas occurring from birth to approximately 25 years of age have the potential to disrupt typical neurodevelopmental processes and produce long-term consequences. Chronic abuse and multiple traumas have a greater neurobiological effect resulting in greater impairment to cognition, emotional regulation, and potential autoimmune disorders. Hippocampal shrinkage (loss of synaptic density) has been found in in children exposed to chronic early stress and maltreatment (Teicher, Anderson, Polcari, Anderson, Navalta, & Kim, 2003). The hippocampus is partially responsible for emotional regulation, which helps explain behavioral disinhibition among persons with trauma-

related disorders; memory storage and retrieval, which accounts for the amnestic and dissociative aspects of trauma response (Teicher, Anderson, Polcari, Anderson, Navalta, & Kim, 2003); and regulates the HPA-axis (Ford, 2009). Researchers in another study did MRIs on 44 children (average age of 12 years) with histories of abuse who met the diagnostic criteria for PTSD and compared them with 61 healthy, non-abused controls. Abused children emitted greater amounts of cortisol and epinephrine and norepinephrine compared with controls. Further, abused children had 7-8% less cerebral volumes (Cohen, Perel, DeBellis, Friedman, & Putnam, 1999). In short, traumatic experience has both psychological and neurobiological consequences and both age of onset duration of traumatic exposure likely play a role in how severe these consequences ultimately are.

Thus, toxic stress may result in lasting impairment to multiple domains of functioning, many of which have been correlated with IPV. Symptoms of chronic trauma include emotional dysregulation (e.g., overeating to minor stressors, difficulty calming down), anger, and disordered mood (e.g., depression and anxiety) (Luxenberg, Spinazzola, & van der Kolk, 2001; Herman, 1992). These impairments, created in childhood, may influence a person's ability to find and maintain a healthy partnership later in life.

Developing an Add Health Specific Index of Toxic Stress Response

The National Longitudinal Study of Adolescent to Adult Health study is the largest longitudinal study of adolescent health ever undertaken in the United States. Considered a "gold standard basic research" ("2016," n.d.), it has helped shape the conversation around human development and health by providing data for over 10,000 researchers who have published more than 5,000 articles (Joe, 2015). An Add Health-specific index of toxic stress response could help

future researchers deepen our collective understanding of this important phenomenon through the use of one of our most powerful and well-used existing datasets.

Researchers have suggested that toxic stress creates mental distress, which in turn increases the likelihood of adopting risky health behaviors, thereby increasing a person's risk of disease and death (CDC, 2017). Toxic stress affects multiple domains of functioning (for example, it has been linked to cognitive issues, such as impaired learning and memory; neurological deficiencies that may manifest as decreased synaptic density; and behavioral problems including poor impulse control and substance abuse). As such, retrofitting a measure to the Add Health data that captures the totality of these potential outcomes would be extremely difficult. However, researchers have uncovered evidence that individuals exposed to toxic stress may exhibit a distinctive cluster of *psychological symptoms* (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005) that could serve as an early indicator of serious maladaptive response to toxic stress exposure (Anda et al., 1999). I submit that a brief index focused exclusively on the psychological symptoms most often described in the literature could prove a reliable indicator of a pathological response to toxic stress exposure.

Researchers have not attempted to examine how multiple indicators of psychological distress may combine to form an overall pattern of indicators indicative of toxic stress exposure within the Add Health dataset. Therefore, developing an index that accurately and reliably describes toxic stress response is necessary to test various hypotheses about the effects of toxic stress on human development. Taken together, these psychological impairments created in childhood may be an important link between childhood adversity and unfavorable health outcomes later in life.

Common Psychological Consequences of Toxic Stress Exposure

Exposure to early adversity has been consistently linked to depression (V. J. Edwards, Holden, Felitti, & Anda, 2003; Heim & Nemeroff, 2001; Putnam, 2003) and anxiety (Hovens et al., 2010; Kendler et al., 2000; Spinhoven et al., 2010). Childhood trauma not only increases the risk for major depression and anxiety but also predisposes those individuals to earlier onset, longer duration, and poorer response to treatments. Researchers looked at the associations of twelve childhood adversities with the first onset and persistence of anxiety and mood disorders. They found that childhood adversities associated with maladaptive family functioning (e.g. family violence and parental mental illness), were the strongest correlates of disorder onset and persistence throughout the life course (Green et al., 2010). Further, not only does the experience of multiple types of adversity increase the prevalence of depression and anxiety, the chronicity of the abuse effects prevalence as well (Cathy Spatz Widom, Czaja, & Dutton, 2008). Importantly, researchers across multiple studies have demonstrated that childhood, but not adult, sexual and physical abuse is strongly predictive of both chronic depression and anxiety (Browne & Finkelhor, 1986; MacMillan et al., 2001; Molnar, Buka, & Kessler, 2001).

Norman and colleagues (2012) conducted a meta-analysis of 124 studies and found statistically significant associations between physical abuse, emotional abuse, and neglect and depressive and anxiety disorders. More specifically, sufferers of emotional abuse had the highest likelihood of experiencing depression (OR=3.06), followed by neglect (OR=2.11) and then physical abuse (OR=1.54) (Norman et al., 2012). Maniglio published two separate systematic review of reviews on both depression (2010) and anxiety (2013). Maniglio found that across the four reviews examining depression, child sexual abuse was significantly associated with adult depression with consistently moderate effect sizes. Regarding anxiety, the author concluded that a significant relationship between child sexual abuse and adult anxiety existed; however, the

effect sizes across studies were small. Chen et al., (2010) conducted a meta-analysis of studies examining the correlation between childhood sexual abuse and adult mental health problems. They reported that a significant association between sexual abuse and a lifetime diagnosis of depression (OR= 2.66), suicide attempts (OR=4.14) and anxiety disorders (OR=3.09). Additionally, the role of childhood trauma in adult depression and anxiety has been corroborated by twin studies (Dinwiddie et al., 2000; Kendler et al., 2000; Nelson et al., 2002).

Researchers assessing the relationship between childhood adversity and adult health and emotional well-being among 2 waves of the ACE Study participants (n=17,337 HMO members) found that ACE scores had a strong graded relationship to depression and anxiety. Participants reporting one ACE were 1.5 times more likely to suffer depression and 1.2 times as likely to report suffering from anxiety. If they reported four or more ACEs they were more than three and a half times as likely to report depressed affect (Anda et al., 2006a) and approximately two and a half times more likely to report anxiety. Merskey et al. (2013) found a similar relationship between number of ACEs and likelihood of depression and anxiety among a large sample (n=1142) of underprivileged, racial and ethnic minority children in Chicago. Participants who reported two ACEs were over twice as likely to report having experienced frequent depressive episodes within the past month and almost two times as likely to report frequent anxiety within the past month. Those with 5 or more ACEs were over eight times as likely to have experienced frequent depression and more than four times as likely to have reported anxiety (Mersky et al., 2013).

Effective emotional-regulation requires the capacity to manage strong feelings, tolerate distress, and regulate emotionally-driven behavior (Murray, Rosanbalm, Christopoulos, & Hamoudi, 2015). Researchers have distinguished between two types of emotional regulation:

reactive/automatic and deliberate/effortful (Eisenberg & Spinrad, 2004; Philips, Ladouceur, & Drevets, 2008). Reactive emotional regulation involves automatic evaluations of and responses to encountered stimulants. Conversely, deliberate emotional regulation is slower and more reflective. Furthermore, emotional regulation is not a single process, but rather a collection of strategies--such as appraisal and suppression-- developed over the course of an extended period of time, from birth to young adulthood (Murray et al., 2015).

Individuals exposed to early, ongoing traumas are also less able to effectively regulate their emotions than those who have not been exposed to trauma (Franke, 2014; Van der Kolk et al., 2005). They may become easily overwhelmed and overreact to minor stressors, have a hard time calming themselves down, and struggle with finding healthy ways to appropriately express anger (Dankoski et al., 2006; Norman et al., 2012). Toxic stress is associated with loss of synaptic density in the hippocampus, thought to be the center of emotion (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012), and the medial prefrontal cortex, which mediates decision making (Euston, Gruber, & McNaughton, 2012). The degradation of these brain structures leads to impaired emotional regulation (McEwen & Gianaros, 2011).

Emotional dysregulation is common among survivors of child abuse. Chang et al. (2009) found a moderate, significant relationship between harsh parenting (which included physical, emotional, and verbal abuse) and emotional dysregulation. In a sample of 141 maltreated and 87 non-maltreated children, the maltreated participants displayed considerably more difficulty regulating their emotional arousal compared with the non-maltreated group (Shields & Cicchetti, 1998). Van der Kolk et al. (2005) found that the earlier the age of onset of child adversity, the more likely the individual would suffer from numerous post-traumatic symptoms, including increased levels of anger and decreased emotional regulation. Researchers have demonstrated

that emotional dysregulation mediates the relationship between childhood adversity and a variety of health-risk behaviors including drug and alcohol abuse (Berking et al., 2011), violent acting out (Davidson, Putnam, & Larson, 2000), and self-harm behaviors (Muehlenkamp, Kerr, Bradley, & Adams Larsen, 2010).

I fill this gap in the research literature through the development of a psychometrically sound multivariate scale that captures the complex psychological sequelae of toxic stress exposure. I will use both exploratory and confirmatory factor analysis to test and confirm the factor structure and evaluate the convergent validity of the scale by measuring the degree to which scores on the measure correlate with known causes of toxic stress including child abuse, neighborhood disadvantage, and community violence.

Methods

Sample

Data were drawn from the Adolescent Health (Add Health) study, a longitudinal and nationally representative sample following American youths through young adulthood (Udry, 2003). The study was designed to investigate adolescent health, mental health, neighborhood and school context, health behavior, and relationships, among other issues. Wave I of the data collection began in 1994-1995. Respondents were enrolled in 7th through 12th grades. In total, 132 schools in the nation were sampled by using stratified cluster sampling techniques. Over 90,000 students attending these schools completed the self-report questionnaire. To obtain more detailed information from respondents, a stratified subsample was selected and re-interviewed at home. In all, 20,745 adolescents were re-interviewed at home. Adolescents were asked about their family relationships, participation in unlawful or delinquent activity, and other issues related to adolescent development. Wave 2 of data collection was conducted with 14,738 of these

respondents in 1996. The third wave of data were collected in 2001-2002, when most participants had reached between 18 and 26 years of age. Overall, 15,197 participants were interviewed successfully. Wave IV was carried out between January 2008 and February 2009, at which point the Add Health sample was disseminated across the country with respondents living in all 50 states. Ninety-two point five percent of the sample was located and 80.3% of the eligible sample members were interviewed. The sample for Wave-IV was 15,701. Survey data were collected using a 90-minute computer-assisted personal interview during in-home interviews. Less sensitive questionnaire sections were administered with the assistance of an interviewer (Bearman et al., 1997; Blum, Beuhring, & Rinehart, 2000). The sample was divided into three equal, random samples to perform the EFA, the CFA and the convergent validity. See table II.1 for more detail concerning the makeup of the total sample and the EFA, CFA, and convergent validity sub-samples.

EFA Sample. A sample of 4,147 people was randomly selected without replacement from the original pool of Wave IV Add Health participants. The study sample included 1,976 men (45.5%) and 2,351 women (54.5%). The mean age was 28-years old with participants ranging in age from 24 to 34. Roughly 17% of the sample reported a high school degree and 26% of the sample reported a bachelor's degree as their highest level of education. Sixty-percent (2,610) of participants were white and 20.7% (900) were black. Approximately 17% reported earning less than $10,000 per year and 21.4% earned between $10,000 and $25,000 per year.

CFA Sample. A second random sample of 4,147 people was selected without replacement from the original pool of remaining? Wave IV Add Health participants. Participants who were selected for the EFA were not eligible to be selected for the CFA. This sample included 1,993 men (45.9%) and 2,351 women (54.1%). The mean age was 28-years old with

participants ranging in age from 24 to 34. Roughly 16% of the sample reported a high school degree and 27.1% of the sample reported a bachelor's degree as their highest level of education. Sixty percent (2,607) of participants were white and 21% (902) were black. Approximately 18% reported earning less than $10,000 per year and 21.3% earned between $10,000 and $25,000 per year.

Convergent Validity Sample. A third random sample of 4,147 people was selected without replacement from the original pool of remaining Wave IV Add Health participants. Participants who were selected for the EFA or CFA were not eligible to be selected to test convergent validity. This study sample included 1,979 men (XX%) and 2,366 women (XX%). The mean age was 28-years old with participants ranging in age from 24 to 33. About 16% of the sample reported a high school degree and 26.5% of the sample reported a bachelor's degree as their highest level of education. Sixty-one percent (2,660) of participants were white and 20.7% (898) were black. Approximately 18% reported earning less than $10,000 per year and 21.3% earned between $10,000 and $25,000 per year.

Initial Scale Construction

The Toxic Stress Response (TSR) measure was developed based on a thorough review of the empirical and conceptual toxic stress, traumatic stress, and developmental psychology literatures. The measure covers those domains of cognitive and mood disturbances most often associated with toxic stress exposure, including: depression, anxiety, emotional dysregulation, and anger. I created indexes (described below) by aggregating items within each domain to create 4 new variables for inclusion in the initial exploratory factor analysis (EFA).

Measures

Toxic Stress Response Scales

See Table II.2 for more detail on the Toxic Stress Response Scales.

Depression. Depression was assessed at Wave IV with five items taken from the CES-D. The items asked respondents to answer based on how they were feeling during the past seven days. Items included: "You were bothered by things that usually don't bother you", "You could not shake off the blues, even with help from your family and friends", You had trouble keeping your mind on what you were doing", "You felt depressed", "You felt sad". Responses were recorded on Likert-type scale ranging from 0 (never/rarely) to 3 (most of the time). The alpha was equal to 0.81.

Anxiety. Anxiety was assessed at Wave IV with four items including: "I worry about things", "I am not easily bothered by things" (reverse coded), "I get stressed out easily", "I don't worry about things that have already happened" (reverse coded). Responses ranged from 1 (strongly disagree) to 5 (strongly agree). Higher scores indicated more anxiety. The alpha was equal to 0.70.

Emotional Dysegulation. The Emotional regulation scale measured the volatility of the participants' mood across 3-items. The scale included the following items: "I have mood swings", "I get upset easily", and "I keep my cool" (reverse coded). Responses were recorded on a Likert-type scale ranging from 1 (strongly agree) to 5 (strongly disagree). Higher scores indicated higher levels of emotional dysregulation. The Emotional Dysregulation subscale demonstrated adequate reliability (alpha=0.85).

Anger. Anger was assessed with three variables: "I lose my temper", "I get angry easily", and "I rarely get irritated" (reverse coded). Responses were recorded on a Likert-type scale ranging from 1 (strongly agree) to 5 (strongly disagree). Higher scores indicated higher levels of anger. The Anger subscale demonstrated adequate reliability (alpha=0.74).

Convergent Validity Variables

Emotional Abuse. Emotional abuse was measured retrospectively at Wave IV with the single item: "Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 6 (more than ten times). The mean score was 1.35 with a standard deviation of 1.77.

Physical Abuse. Physical abuse was measured retrospectively at Wave IV with the single item: "Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 6 (more than ten times). The mean score was 0.43 with a standard deviation of 1.16.

Sexual Abuse. Sexual abuse was measured retrospectively at Wave IV with the single item: "How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 6 (more than ten times). The mean score was 0.14 with a standard deviation of 0.71.

Neighborhood Disadvantage. The neighborhood disadvantage scale included contextual variables measured at Wave I. The variables were: 1) the proportion of African Americans living in the county, 2) the proportion of female-headed households in the county, 3) the proportion of households with an annual income below $15,000, 4) the proportion of households receiving public assistance, and 5) the unemployment rate. Factor analysis revealed that all of the items loaded on a unitary factor (all factor loadings were above 0.78). I created an index by summing

the individual items together and dividing by the five. The scale ranged from 0 to 1 (mean=0.12; SD=0.11). Higher values represent more neighborhood disadvantage.

Family Hardship. Family hardship included items included a host of interpersonal difficulties experienced in childhood. All of the items were yes/no questions and were either asked at Wave I or retrospectively at Wave IV. Childhood adversities included parental mental or physical disability (Wave I), parental incarceration (Wave IV), the death of a family member or friend by suicide (Wave I), and family financial hardship (Wave IV). Items were summed yielding a scale that ranged from 0 to 4 with a mean of 0.48 and a standard deviation of 0.71.

Community Violence. Community violence was assessed at Wave I with five items that asked respondents to report whether they either were the victims of or witnesses to community violence over the past 12 months (yes/no). The items included: 1) you saw someone shot or stabbed; 2) someone pulled a knife on you; 3) someone shot you; 4) someone cut or stabbed you; and 5) you were jumped. Items were summed yielding a scale that ranged from 0 to 5 with a mean of 0.34 and a standard deviation of 0.76.

Analyses

All variable construction, standardization and inferential analyses was conducted in Stata, version 14.0. Exploratory factor analysis was conducted to determine whether individual variables coalesce to form a larger construct and generate factor loadings. Principle Component Analysis was used to explore if these variables have a common factor structure. An alpha score was generated to assess scale reliability.

After completing the EFA I drew a new random sample of 1,086 participants without replacement from Wave IV of the Add Health data and performed a confirmatory factor analysis on the TSR measure. The measurement model estimates the relationships between all of the

indicating variables that comprise the latent factor identified in the EFA (Vinokur, 2010). Within the measurement model, I began with the latent factor (TOXIC STRESS RESPONSE) and the specific indicator variables (depression, anxiety, emotional dysregulation, and anger). I then ran additional goodness-of-fit indices to see if the model fit better if error terms were allowed to co-vary.

To further validate the measure, I assessed convergent validity on a third sample of randomly selected Wave IV participants (n=4,147). That is, the degree to which individual's mean scores on the TSR measure correlates with their scores on items or measures that I would expect to cause toxic stress response. For the current study I assessed childhood maltreatment (emotional, physical, and sexual abuse) as measured retrospectively at Wave IV and TSR scores. I hypothesize that TSR scores will have a moderate, positive correlation with reported childhood maltreatment. I also examined whether exposure to neighborhood disadvantage, family dysfunction and community violence as measured in Wave I correlated with TSR scores. I predict that these neighborhood factors will have a small, positive correlation with TSR scores.

For the convergent validity checks, I created the TSR variable by summing the five standardized indicators (depression, anxiety, emotional dysregulation, and anger) together.

Results

My findings support a one-factor structure with adequate reliability and significant correlations with several variables hypothesized to engender toxic stress response. In the exploratory factor analysis items loaded onto a single factor with factor loading score of .68 or higher. Factor loadings changed between the EFA and CFA, decreasing for all constructs with the exception of emotional dysregulation, which increased. Correlations between TSR scores and convergent validity were small but significant and in the expected direction, with the exception

of neighborhood disadvantage, which was found to have a non-significant relationship to TSR and negative relationships to physical and sexual abuse.

Exploratory Factor Analysis. Factors with Eigenvalues greater than 1 were retained. The sample was adequate for EFA based on the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy (.72). The KMO statistic is a measure of the proportion of variance among variables that might be common variance. This statistic returns values between 0 and 1, with higher values indicating more sampling adequacy. Varimax rotation was performed to maximize the variance of the factor loadings (i.e., making the large loadings larger and the small loadings smaller) and improve the interpretability of the factors. Thus, a single factor, 4-item scale was retained. Factor loadings ranged from 0.68 (depression) to 0.89 (emotional dysregulation). See Table 1 for more detail about the factored scales.

Confirmatory Factor Analysis. Using maximum likelihood, 4 iterations were required to set the target model. For ease of interpretation, I standardized the factor loading values for each of the five variables. The factor loadings and standard errors were all significant ($p<.05$) and are reported in Table 2. The chi-square value was significant at 159.54, potentially suggesting that the model may not fit the data well. Yet, given that the chi-square goodness of fit test is particularly sensitive to large sample sizes, I ran additional goodness of fit statistics including the comparative fit index (CFI; Bentler, 1990), the root mean square error of approximation (RMSEA; Browne & Cudeck1993), and the coefficient of determination (CD). The CFI assesses fit relative to other models on a continuum ranging from entirely uncorrelated (0) to a saturated or perfect fit (1). A score of over 0.95 is often considered a good fitting model (Hu & Bentler, 1999). The RMSEA estimates the lack of fit in a model compared to a perfectly fitted model (Steiger, 2000). Values of 0.07 or less indicate well-fitted models and values larger

than 0.1 indicate models with a poor fit. The CD is the proportion of the variance in the dependent variable explained by the independent variables in the model. The current model has a CFI score of 0.97 and a RMSEA of 0.141, and a CD of 0.874.

Because the fit statistics indicated that the model may not be a good fit, I examined the modification indices. The modification statistics indicated omitted covariance between anxiety and depression. I re-specified the model to allow the error terms to vary among the variables. Once the model was re-run, the new model chi-square was significant at 20.3. The other fit indices were significantly improved as well with a CFI of 0.997, a RMSEA of 0.07, CI 90% [0.045,0.097], and a CD of .90.

Convergent Validity. I examined convergent validity by correlating TSR scores with experiences associated with toxic stress. Based on Cohen's (1988) guidelines, I report significant correlations of .10 as small, .30 as moderate, and .50 as large. Childhood emotional abuse correlated most strongly with TSR, albeit the relationship was small to moderate ($r=.20$; $p<.001$). Both childhood physical and sexual abuse had a small, significant, positive correlation with TSR. Contrary to expectation, community violence did not correlate significantly with TSR. I found a small, positive correlations between TSR and neighborhood disadvantage and between TSR and family hardship.

Discussion

People who suffer chronic, profound, traumatic stressors at a developmentally vulnerable age suffer a variety of adverse mental health outcomes. While these outcomes likely resemble and even co-occur with other psychological maladies (such as posttraumatic stress disorder), both clinicians and researchers have voiced the need to recognize the unique experiences and symptom profile of people who have been exposed to ongoing trauma in childhood (Cook et al.,

2017; Herman, 1997; Van der Kolk et al., 2005). For those who suffer a profusion of seemingly unrelated and unexplained mental health maladies, a history of toxic stress exposure could very well be the etiologic agent underlying their suffering. Because chronic trauma disorders may manifest as other conditions, they are often missed in the clinical setting. To help more accurately identify people within the Add Health dataset who may suffer adverse psychological problems in reaction to exposure to toxic stressors, I constructed and tested a measure of toxic stress response amongst randomly selected samples from Add Health's nationally representative dataset. The TSR scale measures the psychological sequelae of toxic stress exposure.

Previous researchers have found evidence supporting the causal link between childhood adversity and each of the four constructs that comprise the TSR individually. I am not aware of any prior attempts at determining whether exposure to toxic stress reliably results in a psychological response characterized by the five sequelae presented here. Trauma specialist Judith Herman has voiced the need for a new diagnostic category that captures the symptom experiences of people who have suffered prolonged and repeated traumatic exposure. Purposing the term "Complex Post Traumatic Stress Disorder (C-PTSD), she grouped symptoms six clusters associated with chronic trauma. These include alterations in emotional regulation. She notes that people who suffer from C-PTSD frequently experience depression, anxiety, and substance abuse as well (Herman, 1997). Other trauma researchers concerned with the ramifications of ongoing childhood trauma have outlined a number of symptoms associated with chronic trauma exposure that tend to cluster together, including emotional dysregulation, hopelessness, mood disturbances (anxiety and depression) (Briere, Scott, & Weathers, 2005; Courtois & Ford, 2009).

I found small, significant correlations between child maltreatment and adult TSR. This is consonant with the previous research regarding the relationship between child abuse and adult psychopathology. A review of meta-analyses regarding childhood sexual abuse and adult psychopathological consequences reported small, significant correlations between sexual abuse and PTSD (0.20), anxiety (0.13), anger (0.18), and depression (0.22) (Hillberg, Hamilton-Giachritsis, & Dixon, 2011). Erickson et al., (2013), examined the relationship between child abuse and PTSD, anxiety and depression in adulthood. They found small, significant correlations between experiences of child abuse and symptoms of anxiety (0.18) and depression (0.21). The relationship between child abuse and PTSD was small and non-significant (Eriksson et al., 2013). Interestingly, I found that emotional abuse was more highly correlated with TSR compared to either physical or sexual abuse. This is in agreement with findings from past research. Lilly and colleagues (2014) examined emotional dysregulation and PTSD symptoms among an ethnically diverse, high-risk community sample who reported having experienced child abuse. Emotional abuse was more highly correlated with emotional dysregulation (0.30) and PTSD symptoms (0.31) compared to either physical or sexual abuse (Lilly, London, & Bridgett, 2014).

Despite the clear benefits of accurately identifying people suffering from toxic stress response, many challenges exist for developing such a measure. For one thing, the psychological effects of profound trauma accumulated over time may not easily fit into a single diagnostic syndrome (Briere & Spinazzola, 2009). The nature of the symptoms and of how they cluster may differ substantially from individual to individual depending upon the severity of toxic stressor, the timing and duration of the stressor, and the number of stressors encountered.

Further, the presence or absence of relevant social, biological or psychological factors could influence which symptoms manifest and cluster together.

This study has several limitations that should be noted. Some of the convergent validity variables, including all childhood abuse variables, were measured retrospectively. Researchers have conducted studies assessing the validity of self-report, retrospective reporting of child abuse. The authors found that the most common type of misreporting was underreporting (Fergusson, Horwood, & Woodward, 2000; Hardt & Rutter, 2004). Reasons for failing to disclose abuse could include discomfort with the interviewer, the desire to avoid the pain caused by remembering the abuse. It is also very possible that the respondent has simply forgotten that the abuse took place. Not only may the abuse have happened when the participant was very young and less likely to form memories, but researchers have found that survivors of childhood victimization experience memory impairment that inhibits their ability to recall the event (Glaser, 2000; Teicher, 2002). As a result, the correlations reported here may be smaller than they would be were the child abuse data collected at the time of the incident through court or police reports.

Given that the chi-square of the unmodified model was large and significant, the results from the CFA may suggest that this model is not ideally specified. The modified model, however, improved goodness-of-fit considerably. Many authors have cautioned against the practice of allowing measurement errors to correlate in order to improve model fit (e.g., Cortina, 2002; Kaplan, 1990; MacCallum, Tomarken & Waller, 2003). Landis et al. (2009) wrote, "to the degree that two residuals correlate, there is evidence that there exists a cause of both of the variables to which the residuals are attached but that is not specified in the model" (p. 17). That said, Landis et al. (2009) argue that the addition of model paths may be appropriate when there is

sufficient theoretical justification for doing so, such as situations when indicator variables share components. There is theoretical justification for allowing the error terms among depression and anxiety to co-vary.

Given the correlation between childhood adversity and intimate partner violence perpetration, future research should be focused on determining the extent to which TSR mediates the relationship between toxic stress exposure and behavioral risk behaviors, such as violence perpetration. Further, researching factors that mitigate the effects of TSR would aid in intervention and prevention efforts of long-term consequences to early childhood exposure. Lastly, because outcomes associated with toxic response vary widely, any measure designed to capture toxic stress response must include a wide range of symptoms. Determining the core features of TSR, versus associated features, is challenging. Similarly, differentiating between TSR and other, related conditions, such as posttraumatic stress disorder or borderline personality disorder, both of which are correlated with childhood trauma and share common symptom domains, is essential. Researchers should test the sensitivity and specificity of the measure to determine whether the measure is able to delineate between TSR and other related conditions.

Toxic stress exposure can trigger a cascade of negative consequences that may ultimately result in higher rates of morbidity and mortality. Previous researchers have suggested that psychological symptoms precede behavioral ones, such as substance abuse or violence perpetration, following toxic stress exposure. If this is, in fact, the case, then this work is a step toward better identifying individuals at a critical point of intervention.

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Table II.1: Comparing Characteristics Across Samples				
Individual Characteristics	**Total (N=12,441)**	**EFA Sample (N=4,147)**	**CFA Sample (N=4,147)**	**Validity Sample (N=4,147)**
Gender: N (%)				
Males	6,096 (49.1)	1,976 (45.5)	1,993 (45.9)	1,979 (45.6)
Females	6,332 (50.9)	2,369 (54.5)	2,351 (54.1)	2,366 (54.5)
Race: N (%)				
White	8,811 (71.7)	2,610 (60.1)	2,607 (60.0)	2,660 (61.2)
Black	1,799 (14.6)	900 (20.7)	902 (20.8)	898 (20.7)
AI/AN	127 (1.0)	53 (1.2)	51 (1.2)	50 (1.2)
Asian/Pacific Islander	413 (3.0)	275 (6.3)	269 (6.2)	258 (5.9)
Other	1,138 (9.3)	507 (11.7)	515 (11.9)	479 (11.0)
Age at Wave 4: Mean (SD)	28.4 (1.8)	28.4 (1.8)	28.4 (1.8)	28.4 (1.8)
Education at Wave 4: N (%)				
No HS Degree	997 (8.4)	313 (7.2)	311 (7.2)	356 (8.2)
High School Graduate	1,004 (16.8)	720 (16.6)	676 (15.6)	680 (15.7)
Some College/Vo-Tech	4,356 (36.7)	1,649 (38.0)	1,659 (37.8)	1,570 (36.1)
College/Vo-Tech Graduate	3,361 (26.4)	1,127 (25.9)	1,174 (27.1)	1,153 (26.5)
Some Grad/Professional School	558 (4.7)	204 (4.7)	191 (4.4)	199 (4.5)
Grad/Professional Degree	866 (7.3)	332 (7.6)	350 (8.1)	386 (8.9)
Income at Wave 4: N (%)				
0-10,000	2,138 (17.2)	689 (16.6)	724 (17.5)	725 (17.6)
10,000-25,000	2,646 (21.3)	888 (21.4)	880 (21.3)	878 (21.3)
25,000-50,000	5,008 (40.3)	1,671 (40.3)	1,675 (40.5)	1,662 (40.3)
50,000-75,000	1,820 (14.6)	630 (15.2)	573 (13.8)	617 (15.0)
75,000-100,000	491 (4.0)	169 (4.1)	171 (4.13)	151 (3.7)
<100,000	311 (2.5)	100 (2.4)	118 (2.9)	93 (2.3)
Childhood Abuse: N (%)				
Physical Abuse	2,044 (17.3)	780 (18.1)	784 (18.3)	767 (17.9)
Sexual Abuse	569 (4.8)	220 (5.1)	216 (5.0)	218 (5.1)
Emotional Abuse	5,542 (47.1)	2,024 (47.2)	2,020 (47.3)	2,039 (47.5)

Table II.2. Exploratory Factor Analysis Toxic Stress Response					
Scale	*Item*	*Factor Loadings*	*Range*	*Mean (SD)*	*Alpha*
Depression			**1-4**		**0.81**
	Bothered by things that usually don't bother you	0.71		1.51 (0.69)	
	Could not shake the blues	0.83		1.33 (0.64)	
	Had trouble keeping your mind on what you were doing	0.55		2.20 (0.88)	
	Felt depressed	0.84		1.37 (0.66)	
	Felt sad	0.82		1.57 (0.63)	
Anxiety			**1-5**		**0.70**
	Worry about things (reverse)	0.74		3.70 (0.98)	
	Not easily bothered by things	0.70		2.79 (0.98)	
	Get stressed out easily (reverse)	0.78		2.76 (1.02)	
	Worry about things that already happened	0.66		3.05 (1.05)	
Emotional Dysregulation			**1-5**		**0.85**
	Frequent mood swings	0.78		2.69 (1.08)	
	Get upset easily	0.85		2.56 (0.96)	
	Keep my cool (reverse coded)	0.77		2.16 (0.73)	
Anger					**0.74**
	Lose my temper	0.87		2.45 (0.97)	
	Get angry easily	0.71		2.57 (1.02)	
	I rarely get irritated (reverse coded)	0.85		2.97 (0.98)	

Table II.3. EFA and CFA Results (n=4,147)

	EFA			CFA	
Measure	**Factor Loadings**	**Mean (SD)**	**Range**	**Factor Loadings**	**Mean (SD)**
Depression	0.68	1.42 (0.45)	1-4	0.52*	1.44 (0.54)
Anxiety	0.73	3.07 (0.72)	1-5	0.62*	3.08 (0.73)
Emotional Dysregulation	0.78	2.59 (0.72)	1-5	0.92*	2.49 (0.75)
Anger	0.57	2.00 (0.58)	1-5	0.72*	2.67 (0.80)
CFA Fit Indices: X2=20.3, p<0.001; RMSEA=0.07; CFI=0.99; TLI=0.98; CD=0.78					

*p<0.001

Table II.4. Correlations Among Included Measures

	1	2	3	4	5	6	7
1. TSR	1	.20*	.14*	.10*	.08*	.02	.17*
2. Emotional Abuse		1	.48*	.18*	.09*	-.03*	.10*
3. Physical Abuse			1	.17*	.12*	-.04*	.09*
4. Sexual Abuse				1	.02*	.01	.06*
5. Community Violence					1	.10*	.13*
6. Neighborhood Disadvantage						1	.15*
7. Family Hardship/Loss							1

*p-value less than 0.05

CHAPTER III

The Traumatic Origins of Intimate Partner Violence Perpetration and the Mediating Role of Toxic Stress Response

Introduction

Intimate Partner Violence (IPV) is a pervasive public health problem that contributes to significant morbidity and mortality. The Centers for Disease Control defines IPV as "physical, sexual, or psychological harm by a current or former partner or spouse" ("Definitions|Intimate Partner Violence|Violence Prevention|Injury Center|CDC," n.d.). Approximately twenty-five percent of women and twenty percent of men in the United States have suffered severe intimate partner violence (WHO, 2013). Prevalence rates yielded from the National Intimate Partner and Sexual Violence Survey are estimated at more than 4.2 million physical assaults, rapes, and stalking perpetrated against women annually by an intimate partner and 3.2 million physical assaults, rapes, and stalking against men by an intimate partner (Kessler, Molnar, Feurer, & Appelbaum, 2001). The cost of IPV to the United States economy is over $8.3 billion. This figure includes medical care, mental health services, and lost productivity due to missed work (Max, Rice, Finkelstein, Bardwell, & Leadbetter, 2004). The cost of IPV is greater for female victims compared with males. In 2005, CDC researchers determined the average medical cost for women victimized by physical domestic violence was $958 compared to $387 for men; mental

health services costs for women was $207 compared to $80 for men; while productivity losses were similar at $257 for women and $224 for men (CDC, 2005). Of all women murdered in the US, approximately one-third are killed by intimate partners (CDC, 2008). Victims of IPV are more likely to suffer from depression, anxiety, and posttraumatic stress symptoms than non-victims, and substance use is estimated to be five times greater in women who have been abused compared with those who have not (Black, et al., 2011). While researchers have identified many risk and protective factors related to victimhood as well as consequences of IPV victimization, less attention has been devoted to the study of IPV perpetration. Consequently, we have significant gaps in our collective understanding regarding what underlying factors impel some people to aggress against their partners.

Researchers have demonstrated a growing interest in identifying and cataloguing the psychological, behavioral, and environmental characteristics of individuals who perpetrate violence against their partners. These investigations have revealed, in large part, that batterers are a heterogeneous population with diverse psychological profiles and demographic backgrounds; however, consistent risk factors have been revealed. One such risk is experiences of maltreatment in childhood. A comprehensive body of research has been amassed connecting childhood maltreatment with intimate partner violence (IPV) perpetration in adulthood, referred to as the Intergenerational Transmission of Violence (ITV) (D. G. Dutton, 2011; Ehrensaft et al., 2003; Harris & Dersch, 2001; Stith et al., 2000; White & Widom, 2003; Cathy Spatz Widom & Wilson, 2015; C.S. Widom et al., 2014). Childhood maltreatment is far from deterministic and most children who are abused will not grow up to abuse any member of their family. Within this study I aim add to the ITV evidence base by to test whether childhood maltreatment produces

symptoms of Toxic Stress Response (TSR) in some individuals, and whether those symptoms are a precursor to IPV.

Review of the Empirical Support for the Intergenerational Transmission of Violence

Swinford and colleagues (Swinford, DeMaris, Cernkovich, and Giordano; 2000) found that child abuse predicted IPV among participants in the Toledo Adolescent Relationship Study. In a 2005 study, individuals exposed to child abuse were at risk for IPV in romantic relationships (Linder and Collins, 2005). A prospective study conducted by Ehrensaft et al. (2003), childhood physical abuse was the most significant predictor of partner violence perpetration and injury to the victim. Similarly, White and Widom (2003) found that individuals reporting childhood maltreatment were significantly more likely to perpetrate violence against their partners compared to those with no history of abuse. In a case-control study, Widom et al. (2013) found that childhood neglect increased the risk of perpetrating physical injury to a partner, compared to matched controls. In a prospective cohort study, Milaniak and Widom (2015) matched 676 children (11-years old and younger) with documented histories of physical and sexual abuse and neglect with matched controls and assessed violence perpetration (criminal, IPV, and child abuse) in both groups as young adults. The authors reported that, compared to the control group, individuals with histories of child abuse and/or neglect were significantly more likely to perpetrate IPV. In fact, they were significantly more likely to be poly-violence perpetrators, perpetrating criminal violence, IPV, and child abuse.

Stith and colleagues conducted a meta-analysis of studies exploring the relationship between growing up in a violent home and perpetrating violence against a romantic partner. The authors concluded that a small to medium association exists between the experience of childhood maltreatment and adult IPV perpetration. Similarly, in an extensive systematic review of the

literature regarding risk factors for IPV, Capaldi and colleagues found a low to moderate significant association between family-of-origin abuse and IPV perpetration in adulthood (Capaldi et al., 2012). While childhood maltreatment appears to increase the risk of IPV perpetration, the mechanisms by which childhood trauma transforms into adult violence is not well understood.

Toxic Stress and Intimate Partner Violence

Numerous researchers have found that symptoms of trauma mediate the relationship between childhood maltreatment and adult IPV perpetration (see Bell and Orcutt 2009; Taft et al. 2011, for reviews). Wolfe et al. (2004) conducted a longitudinal study and found that trauma symptoms partially mediated the relationship between child maltreatment and IPV perpetration among adolescents. The authors also reported that trauma-related anger predicted female-perpetrated IPV perpetration among female adolescents. Among female participants who reported perpetrating IPV against their partners within the past 6 months, Swan et al. (2005) found unique effects of three types of anger expression (externalized anger, internalized anger, and anger control) on composite IPV perpetration (i.e., combined sexual, psychological, and physical) were computed. Child maltreatment was associated with more trauma symptoms, which were in turn, associated with IPV perpetration.

The same psychological features associated with toxic stress have also been found in populations of batterers. Childhood toxic stressors can lead to depression and anxiety in adulthood (Felitti & Anda, 2010; Horwitz, Widom, McLaughlin, & White, 2001). Researchers looking at the Adverse Childhood Experiences (ACE) data found that the link between ACEs and adult depression and anxiety was significant and graded, meaning that the more ACEs the individual reported the greater the symptom severity (Felitti & Anda, 2010; Mair, Cunradi, &

Todd, 2012). Besides being a consequence of childhood adversity, depression and anxiety are risk factors for both reciprocal (Melander, Noel, & Tyler, 2010) and unidirectional IPV (Lehrer, Buka, Gortmaker, & Shrier, 2006; Lipsky, Caetano, Field, & Bazargan, 2005). Ehrensaft and colleagues found psychiatric disorders in adolescence predicted partner violence in early adulthood (2004), while past-week depressive symptoms were associated with partner violence in a sample of adolescents. According to the Miller et al. (2011), nearly one-fifth of the risk for IPV perpetration in attributable to premarital mental health conditions.

Individuals who have experienced higher levels of childhood toxic stress may have a decreased capacity to negotiate relationship conflict (Millett, Kohl, Jonson-Reid, Drake, & Petra, 2013). Individuals exposed to ongoing maltreatment in childhood have lower levels of emotional regulation, the utilization of efforts aimed at controlling or changing one's own emotional response to a stressful situation (Norman et al., 2012). They may become easily overwhelmed and overreact to minor stressors, have a hard time calming themselves down, and struggle with finding healthy ways to appropriately express anger. Anger, often accompanied by romantic jealousy, is a common emotional antecedent to IPV (Foran & O'Leary, 2008; Norman et al., 2012; Stith et al., 2004). A 2012 review of the literature examining motivations for IPV found that nearly half of the studies included jealousy as a driver in relationship violence (Langhinrichsen-Rohling, Selwyn, & Rohling, 2012).

Emotional dysregulation appears to be an important mediator between toxic stress exposure and IPV perpetration. Norlander & Eckhardt (2004) conducted a meta-analysis of studies focused on the characteristics of male domestic abusers and found higher rates of anger and hostility among the abusers as compared to non-violent men. Birkley and Eckhardt (2015) conducted another meta-analysis wherein they revealed a moderate effect size between anger,

hostility, internalizing negative emotions and IPV perpetration. The authors found no differences in this relationship across sex of perpetrator, relationship type, or perpetrator population. Among male perpetrators of IPV mandated to group counseling, Eckhardt and colleagues (2008) reported that men with higher rates of anger perpetrated violence against their partners more often than those with lower levels of anger. Dutton (1995) found that emotional dysregulation, lack of behavioral control, and depression are all moderately correlated with IPV perpetration. Further, he demonstrated that men who physically abuse their partners report more such symptoms compared with non-violent men.

Child maltreatment can result in an unstable and negative self-concept. Repeated exposure to violence, rejection, and neglect may internalize the explicit and implicit messages their caretakers are conveying (Cook, et al., 2005) (Herman, 1992) (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). Further, maltreatment often leads to a failure to achieve developmental benchmarks and age-appropriate competencies, which can, in turn make a child feel powerless and incompetent (Cook, et al., 2005). They may consider themselves ineffectual, helpless, and unable to affect change within their own lives (van der Kolk B. , 2005). Because children are dependent on their caretakers for their very survival, allying themselves with the perpetrator is an understandable strategy to ensure safety (Herman, 1992). As adults, these individuals may use violence in an attempt to feel powerful and exert control over their own lives.

In the first dissertation study I suggest that the responses to toxic stress may include a constellation of symptoms including depression, anxiety, emotional dysregulation, and anger. In the current study, I hypothesize that TSR mediates the relationship between exposure to childhood toxic stressors and perpetrating intimate partner violence (see Figure 1).

Methods

Sample

Data were drawn from the Adolescent Health (Add Health) study, a longitudinal and nationally representative sample following American youths through young adulthood (Udry, 2003). The study was designed to investigate adolescent health, mental health, neighborhood and school context, health behavior, and relationships, among other issues. Wave 1 of the data collection began in 1994-1995. Respondents were enrolled in 7th through 12th grades. In total, 132 schools in the nation were sampled by using stratified cluster sampling techniques. Over 90,000 students attending these schools completed the self-report questionnaire. To obtain more detailed information from respondents, a stratified subsample was selected and re-interviewed at home. In all, 20,745 adolescents were re-interviewed at home. Adolescents were asked about their family relationships, participation in unlawful or delinquent activity, and other issues related to adolescent development. Wave 2 of data collection was conducted with 14,738 of these respondents in 1996. The third wave of data were collected in 2001-2002, when most participants had reached between 18 and 26 years of age. Overall, 15,197 participants were interviewed successfully. Wave IV was carried out between January 2008 and February 2009, at which point the Add Health sample was disseminated across the country with respondents living in all 50 states. Over ninety percent of the sample was located and 80.3% of the eligible sample members were interviewed. The sample for Wave-IV was 15,701. Survey data were collected using a 90-minute computer-assisted personal interview during in-home interviews. Less sensitive questionnaire sections were administered with the assistance of an interviewer (Bearman et al., 1997; Blum, Beuhring, & Rinehart, 2000). For the current study I randomly

select 1000 participants who reported any child maltreatment at or above one standard deviation above the mean and then matched them with 2000 participants who never experienced any childhood maltreatment. Participants were matched on age, education, gender, race, and income. I used only those who were one standard deviation above the mean since such a large number of people reported at least one incidence of emotional abuse.

Measures (see Table III.1 for a more detailed description of the measures)

Dependent Variables—Intimate Partner Violence

Partner Physical Abuse. Partner physical abuse was assessed with one item measured at Wave IV, "How often (have/did) you (slapped/slap), hit, or (kicked/kick) {your partner}?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6 ("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.21, with a standard deviation of 0.75.

Partner Sexual Abuse. Partner sexual abuse was assessed with one item measured at Wave IV, "How often (have/did) you (insisted/insist) on or (made/make) {initials} have sexual relations with you when (he/she) didn't want to?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6 ("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.08, with a standard deviation of 0.51.

Threatened Partner Violence. Threatened partner abuse was assessed with one item measured at Wave IV, "How often (have/did) you threatened {your partner} with violence, pushed or shoved (him/her), or thrown something at (him/her) that could hurt?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6

("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.29, with a standard deviation of 0.87.

Independent Variables—Childhood Maltreatment

Emotional Abuse. Emotional abuse was measured retrospectively at Wave IV with the single item: "Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 1.08, with a standard deviation of 1.8.

Physical Abuse. Physical abuse was measured retrospectively at Wave IV with the item: "Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 0.50, with a standard deviation of 1.25.

Sexual Abuse. Sexual abuse was measured with the item: "How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 0.12, with a standard deviation of 0.63.

Mediating Variables—Toxic Stress Response

Toxic Stress Response. The Toxic Stress Response (TSR) scale developed in Chapter II is composed of four indicators: depression (4 items; alpha = 0.81), anxiety (4 items; alpha = 0.70), emotional dysregulation (7 items; alpha =0.85), and anger (3 items; alpha = 0.74).

Statistical Analysis

All variable construction and inferential analyses was conducted in Stata, version 13.0. In order to test the hypothesis that the relationship between childhood toxic stressors (CTS) and IPV is mediated by TSR, I used structural equation modeling (SEM) to test the hypothesized paths between these constructs (see Figure 1). SEM estimates simultaneous path coefficients between multiple predictors and outcome variables, thus testing a conceptual model in its entirety rather than piece by piece as would be the case with alternative methods such as path analysis (Kline, 2004). SEM also controls for any variance related to measurement error due to the fact that estimated paths are between latent factors composed of multiple indicators rather than between single indictors (Kline, 2004).

I first established that my measurement model adequately represented each latent factor. I validated the childhood maltreatment and intimate partner violence scales separately through confirmatory factor analysis and reliability testing. To do this, I examined whether emotional, physical and sexual childhood abuse fit together as a single childhood maltreatment factor through principle axis factoring (see Table III.4 for factor loadings). I then examined whether partner physical abuse, partner sexual abuse, and threat of partner violence fit together as a single intimate partner violence latent variable (see Table III.4). The TSR scale was validated through CFA in the first study of my dissertation.

The structural model and hypothesized path valances are represented in Figure III.1. Model parameter estimates are reported in standardized forms. I assessed the total and specific indirect pathways with parameter point estimates and their associated 95% confidence intervals (CI). If zero was not between the upper and lower bounds of the 95% confidence interval of the standardized specific direct and indirect effect, I determined it as a significant effect (Hayes, 2009). I also assessed the direct and total effects.

Goodness of fit of the model was assessed through evaluation of the Bentler Bonett Index which compares the chi-square of a null model to the proposed model (values of .9 or higher indicate strong model fit), and the Root Mean Square Error of Approximation (RMSEA) which is a metric based on the noncentrality parameter (values of .06 or less are strong fitting models) (Bauermeister, 2011). I then ran a series of Sobel tests to assess the magnitude of all indirect effects specified within the model (Kline, 2004). Finally, I reversed the directionality of the model and compared the models across all fit indices to examine which direction best fits the data (Kline, 2004).

Results

Table III.2 displays the sample characteristics. The study sample included 1,418 men (47.27%) and 1,582 women (52.73%). The mean age was about 28-years old with participants ranging in age from 24 to 34. Roughly 15% of the sample reported a high school degree and 20% of the sample reported a bachelor's degree as their highest level of education. About 60% (649) of participants were white and 21.2% (230) were black. Approximately 17% reported earning less than $10,000 per year and 40% earned between $25,000 and $50,000.

Table III.2 also compares study participants who reported having perpetrated any IPV in the past year with those who reported no IPV in the past year. Appropriate test statistics and p-values are included. Perpetrators were significantly more likely to be female (x^2 =24.16, $p<0.001$) with just over 19% of females reporting IPV perpetration compared to 12.54% of males. Among white respondents, approximately 14% reported IPV perpetration compared to 18.29% of black respondents (x^2=3.10; p=0.009). A higher percentage of respondents who reported no IPV had completed college or vocational school (27.25%) and held graduate degrees (8.99%) compared to those who had perpetrated IPV in the past year (6.22%). There was a

significant difference between the two groups regarding mean income, with respondent reporting no IPV earning, on average, $7,000 per year more than perpetrators (x^2=4.5; p<0.001). Regarding exposure to childhood maltreatment, I conducted t-tests comparing both groups of participants across the three types of abuse and found that perpetrators reported significantly more abuse across all three types (physical, sexual, and emotional) compared to non-perpetrators. For perpetrators, the mean score on the emotional abuse item was 1.00 (SD=1.66) compared to 0.40 (SD=1.14) for non-perpetrators (t=9.38, p<0.001). Perpetrators average scores for the sexual abuse item was over three times as high compared to non-perpetrators (0.29 and 0.09, respectively; t=-6.38, p<0.001). The average score for the emotional abuse item was 1.77 for perpetrators compared to 0.94 for non-perpetrators (t=-9.22, p<0.001).

Table III.3 displays the correlations between every variable within the model. All the correlations were significant--with the exception of partner sexual assault, which had non-significant relationships to anxiety, emotional dysregulation, anger, and childhood sexual abuse--and were in the expected directions.

Table III.4 displays the factors loadings, standard errors, and z-scores for the measurement model without the structural pathways. The measurement model included latent factors childhood maltreatment (CM), toxic stress response (TSR), and intimate partner violence (IPV). Childhood maltreatment is made up of manifest variables emotional abuse, physical abuse, and sexual abuse. All factor loadings were above .40 and significant at p<0.001. The four toxic stress response variables loaded above 0.50 and were significant at p<0.001. For the intimate partner violence variables, partner physical assault and partner threat had high, significant loadings (0.78 [p<0.001] and 0.85 [p<0.001], respectively). Partner sexual assault had a low, significant factor loading (0.17; p<0.001).

Table III.5 displays the results from the structural models as well as the fit indices and changes in chi-square as pathways between constructs are added. In Model 1, CM and TSR were correlated and there was a direct path from CM to IPV (b=0.26; chi-square=180.5; p<0.001). In Model 2, I added a second pathway between CM and TSR. The resultant fit indices remained the same with the inclusion of this path. In the third and final model (the full mediation model) I added a pathway between TSR and IPV (chi-square=130.9; p<0.001). The change in chi-square was significant at 49.6. In Model 3, I found a relationship between CM and IPV (b=0.26; p<0.001). I also found a significant relationship between CM and TSR (b=0.33; p<0.001) and between TSR and IPV (b=0.22; p<0.001). The RMSEA and CFI both indicated good fit with a RMSEA score below 0.05 (0.035) and the CFI of over 0.09 (0.987). Figure III.1 displays the model of standardized coefficients for the structural pathways.

Discussion

The relationship between childhood maltreatment and IPV perpetration has been well documented (e.g., Ehrensaft et al., 2003; Harris & Dersch, 2001; Cathy Spatz Widom & Wilson, 2015; C.S. Widom et al., 2014), however, the mechanism for why maltreatment might result in IPV perpetration is not well understood. Toxic stress has received mounting attention in recent years as both a consequence of ongoing trauma in childhood and a cause of myriad psychological, physical, and behavioral maladies in adulthood. To my knowledge, the psychological sequelae of toxic stress have not been examined as a possible mediator, linking experiences of childhood maltreatment with that of IPV perpetration. In this study I contribute to our understanding of the intergenerational transmission of violence by assessing whether toxic stress response mediates the relationship between childhood maltreatment and intimate partner violence. The current mediational model was tested in a nationally representative sample of

young adults. Participants exposed to childhood maltreatment were matched on demographic characteristics with participants who reported no exposure to childhood maltreatment. I found that, consistent with previous research, CM was a risk factor for IPV perpetration and that this relationship was partially mediated by TSR.

Findings from the current study indicate that childhood maltreatment, a form of childhood adversity, can yield a particular cluster of psychological symptoms. This results in individuals for whom these symptoms manifest with increased risk for perpetrating IPV. Researchers have found that ACEs influence the adoption of risky health behaviors (including IPV) through emotional and cognitive impairment (Felitti et al., 1998; Felitti & Anda, 2010; Mair et al., 2012). They have called for more research to better understand and more accurately characterize the link between ACEs and resulting maladaptive behaviors (Felitti et al., 1998; Felitti & Anda, 2010; Mair et al., 2012). My findings help address this gap by suggesting that ACEs may be a form of toxic stress that effects emotional and cognitive impairments (TSR) that make IPV more likely. In this way, if toxic stress symptoms manifest subsequent to exposure to ACEs, this could be an early indication of IPV perpetration.

While no previous study has assessed whether the toxic stress response mediate the relationship between CM and IPV, previous researchers have documented that symptoms included in the TSR index are separately associated with both childhood maltreatment and adult IPV. Toxic stress researchers have posited that toxic stress exposure may interfere with the development of healthy and adaptive emotion regulation by placing children in situations that elicit intense emotional reactions while simultaneously failing to teach them how to regulate emotional arousal and tolerate emotional discomfort (Cook et al., 2017; Murray et al., 2015; Shern et al., 2014). Consistent with this theoretical supposition, researchers have found that

maltreated children are significantly more likely than children not victimized to exhibit emotion dysregulation (Shipman, Edwards, Brown, Swisher, & Jennings, 2005; Shipman, Zeman, Penza, & Champion, 2000). Gratz and colleagues (Gratz, Paulson, Jakupcak, & Tull, 2009) found that emotion dysregulation was positively associated with the IPV perpetration among men and fully mediated the relationship between childhood maltreatment and the frequency of intimate partner abuse perpetration. Interestingly, the relationship between CM and IPV was not mediated by emotional dysregulation among women. The authors concluded that, "men's violent behavior in the context of an interpersonal relationship may function to regulate unwanted and/or overwhelming emotions associated with adverse childhood experiences" (pg. 77).

White and Widom (2003) found that anger mediated the relationship between childhood maltreatment IPV perpetration in adulthood. According to Pollack (2015), individuals exposed to childhood maltreatment are at risk for developing externalizing behavioral problems characterized by anger and/or aggression. In a 2014 study, Shackman and Pollak found that maltreated children displayed more negative emotions when confronted by an interpersonal stressor which was subsequently correlated with more anger (Shackman & Pollak, 2014). The researchers concluded that maltreatment causes children to more closely attend to emotional cues that undermine their capacity for emotional regulation and increase aggression.

Mood disorders, including depression and anxiety, are well-established correlates of both childhood maltreatment (Bernet & Stein, 1999; Gibb et al., 2007; Norman et al., 2012; Pollak, 2015) and IPV perpetration (Birkley & Eckhardt, 2015; Capaldi et al., 2012; Lipsky et al., 2005). The mediational role that mood disorders play, however, is not well understood. A meta-analysis by Birkley and Eckhardt (2015) found that IPV perpetration was associated with depression and anxiety. This is consistent with a previous literature review conducted by

Norlander and Eckhardt (Norlander & Eckhardt, 2005). In general, Riggs et al. (2000) found that men who are violent toward their partners endorse more depressive symptoms compared with non-violent men. Capaldi and colleagues (Capaldi et al., 2012) have suggested that mood disorders may indirectly increase the risk of IPV perpetration via its corrosive effects on the quality of the relationship and increase in conflict between partners.

Regarding perpetrator compared with non-perpetrators characteristics, I found that perpetrators were more likely to have less education and make less money (though this is likely confounded by gender) compared to those who reported no IPV perpetration. These findings are in keeping with prior researchers who have found that, while IPV perpetration cuts across demographic differences, low SES groups tend to bear a higher burden of IPV (Allen, Swan, & Raghavan, 2009; Capaldi et al., 2012; Cunradi et al., 2002; Fox et al., 2002; Langhinrichsen-Rohling et al., 2012; Stith et al., 2004). Similar to other studies examining gender differences in rates of battering, I found that women were more likely to report perpetrating IPV compared to men (Allen et al., 2009; Langhinrichsen-Rohling et al., 2012). Female-to-male IPV perpetration, however, tends to be in reaction to male violence (defensive) and much less severe compared to male-to-female IPV (Allen et al., 2009; Langhinrichsen-Rohling et al., 2012). Black participants reported higher rates of perpetration compared to white participants and this, too, is commensurate with previous studies (Langhinrichsen-Rohling et al., 2012; Stith et al., 2004).

Limitations and Directions for Future Research

Several limitations to this study are important to note. I measure exposure to toxic stressors at Wave I (and retrospectively at Wave IV) and I measure TSR at Wave IV. Measuring TSR closer to the exposure itself might better capture the psychological fallout of toxic stress exposure. Measuring TSR closer to exposure within this particular dataset, however, would

mean measuring these psychological symptoms with adolescents, which may, in and of itself, be confounding given the emotional volatility associated with that age group. Furthermore, researchers have found that the psychological fallout from childhood maltreatment is not time limited and lasts well into adulthood (Anda et al., 2006a; Bernet & Stein, 1999; Gibb et al., 2007).

Childhood maltreatment is measured retrospectively at Wave IV while the other exposure variables are measured at Wave I. While the Wave IV childhood maltreatment variables ask about the same time period as those assessed at Wave I, the retrospective nature of the survey questions introduces the possibility of recall bias. Importantly, however, some have argued that studies employing retrospective reporting are more accurate than prospective studies (Kendall-Tackett & Becker-Blease, 2004). This is due to the fact that, unlike other study populations, professionals who discover abuse amongst children are ethically and legally bound to report it to law enforcement and/or child protection agencies, which are then required to intervene. Therefore, children who have been identified as abuse victims have a higher likelihood of receiving intervention or treatment during childhood compared with children who are never identified as victims, which alters their psychological trajectory. Additionally, prospective studies likely miss cases of child abuse given how profoundly underreported this problem remains (Kendall-Tackett & Becker-Blease, 2004). The IPV perpetration measure relies on self-report. Given the sensitive nature of the topic it is possible that the responses may be biased due to social desirability. Yet IPV perpetration is likely underreported by participants, which would attenuate the relationship between childhood maltreatment and IPV rather than strengthen it, rendering these findings overly conservative.

Theories including Social Learning Theory, Attachment Theory, and Family Stress Theory have all contributed to our understanding of the intergenerational transmission of violence. Ideally, variables included in those theories such as role modeling, attachment style, or family hardship would be accounted for in a model of ITV. These variables were not included in the Add Health data and are therefore missing from my analyses. Future research that accurately measures and integrates elements of other ITV theories with toxic stress response would likely render a more complete picture of this phenomenon. Nevertheless, this research is one of the first studies to examine the psychological sequelae of toxic stress exposure in aggregate as a mediator linking CM and IPV. My results suggest that the inclusion of other theories, such as toxic stress, would help to further understand ITV.

The intergenerational transmission of violence may be more pronounced and intractable among low income and minority populations. Researchers have reported higher rates of both child maltreatment and IPV among racial/ethnic minorities and lower-income households. While the vast majority of toxic stress literature is focused on child abuse, toxic stressors also include environmental conditions such as community violence and neighborhood disadvantage (Shern et al., 2014). The ACEs study relied on data principally collected from white, middle- and upper-middle-class participants and focuses on experiences within the home. As a result, researchers have suggested that the ACEs included in the original measure may not adequately capture adversity among racially and socioeconomically diverse populations (Cronholm et al., 2015). Wade and colleagues (2014) collected qualitative data with Black and Latino youth to explore whether expanding the definition of an adverse childhood experience was warranted. They found support for the inclusion of neighborhood-level indicators such as community violence,

living in an unsafe neighborhood, and residential instability within the concept of 'childhood adversity' (Wade et al., 2014).

Conclusion. These limitations notwithstanding, this study makes several unique and significant contributions to the literature on IPV perpetration. First, child maltreatment is correlated with toxic stress response. Second, toxic stress response mediates the relationship between CM and intimate partner violence perpetration. Finally, people exhibiting TSR may be at an increased risk for perpetrating IPV.

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Table III.1: Demographic Characteristics of IPV Perpetrators and Non-Perpetrators					
Individual Characteristics	**Total (n=3,000)**	**IPV (n=466)**	**No IPV (n=2,426)**	**Test value**	**P-value**
Gender: N (%)				24.16	0.00
Males	1,418 (47.27)	170 (12.54)	1,186 (87.46)		
Females	1,582 (52.73)	296 (19.27)	1,240 (80.73)		
Race: N (%)				3.10	0.009
White	1,857 (61.90)	253 (14.03)	1,550 (86.07)		
Black	596 (19.87)	103 (18.29)	460 (81.71)		
AI/AN	29 (0.97)	3 (0.10)	26 (89.66)		
Asian/Pacific Islander	188 (6.27)	42 (24.14)	132 (75.86)		
Other	330 (11.00)	65 (20.12)	258 (79.87)		
Age at Wave 4: Mean (SD)	28.45 (1.78)	28.35 (1.86)	28.47 (1.76)	1.39	0.16
Education at Wave 4: N (%)				20.94	0.004
No HS Degree	206 (6.87)	41 (8.80)	154 (6.36)		
High School Graduate	474 (15.80)	85 (18.24)	364 (15.00)		
Some College/Vo-Tech	1,153 (38.43)	198 (42.48)	917 (37.79)		
College/Vo-Tech Graduate	783 (26.10)	98 (21.03)	661 (27.25)		
Some Grad/Professional	128 (4.27)	15 (3.22)	112 (4.62)		
Grad/Professional Degree	256 (8.53)	29 (6.22)	218 (8.99)		
Income: Mean (SD)	34,516 (30,110)	28,857 (26,377)	35,873 (30,902	4.5	0.00
Childhood Abuse: Mean (SD)					
Physical Abuse	0.49 (1.25)	1.00 (1.66)	0.40 (1.14)	-9.38	0.00
Sexual Abuse	0.12 (0.63)	0.29 (1.00)	0.09 (0.53)	-6.38	0.00
Emotional Abuse	1.08 (1.08)	1.77 (2.07)	0.94 (1.17)	-9.22	0.00

Table III. 2. Description of Measures

Latent Construct	Variable	M	SD	Range	Skewness	Kurtosis
Child Maltreatment						
	Emotional Abuse	1.08	1.8	0-5	1.29	2.99
	Sexual Abuse	0.12	0.63	0-5	6.09	41.4
	Physical Abuse	0.50	1.25	0-5	2.64	8.89
TSR						
	Depression	1.50	0.49	0-4	1.60	6.50
	Anxiety	3.05	0.73	0-5	-0.05	2.77
	Emotional Dysregulation	2.45	0.74	0-5	0.48	3.07
	Anger	2.64	0.80	0-5	0.34	2.71
IPV						
	Physical Assault	0.21	0.75	0-6	4.15	21.39
	Sexual Assault	0.08	0.51	0-6	7.79	70.9
	Threat	0.29	0.87	0-6	3.32	14.44

Table III. 3. Correlations among study variables

	1	2	3	4	5	6	7	8	9	10
(1) Depression	1.0									
(2) Anxiety	0.42*	1.0								
(3) Emotional Dysregulation	0.42*	0.60*	1.0							
(4) Anger	0.32*	0.52*	0.74*	1.0						
(5) Emotional Abuse	0.18*	0.17*	0.15*	0.12*	1.0					
(6) Sexual Abuse	0.12*	0.09*	0.09*	0.08*	0.23*	1.0				
(7) Physical Abuse	0.11*	0.07*	0.09*	0.11*	0.54*	0.20*	1.0			
(8) Physical Assault Partner	0.19*	0.15*	0.18	0.17*	0.14*	0.13*	0.13*	1.0		
(9) Sexual Assault Partner	0.07*	0.01	0.03	0.04	0.06*	0.01	0.08*	0.13*	1.0	
(10) Threaten Partner	0.20*	0.16*	0.21*	0.21*	0.14*	0.11*	0.16*	0.67*	0.17*	1.0

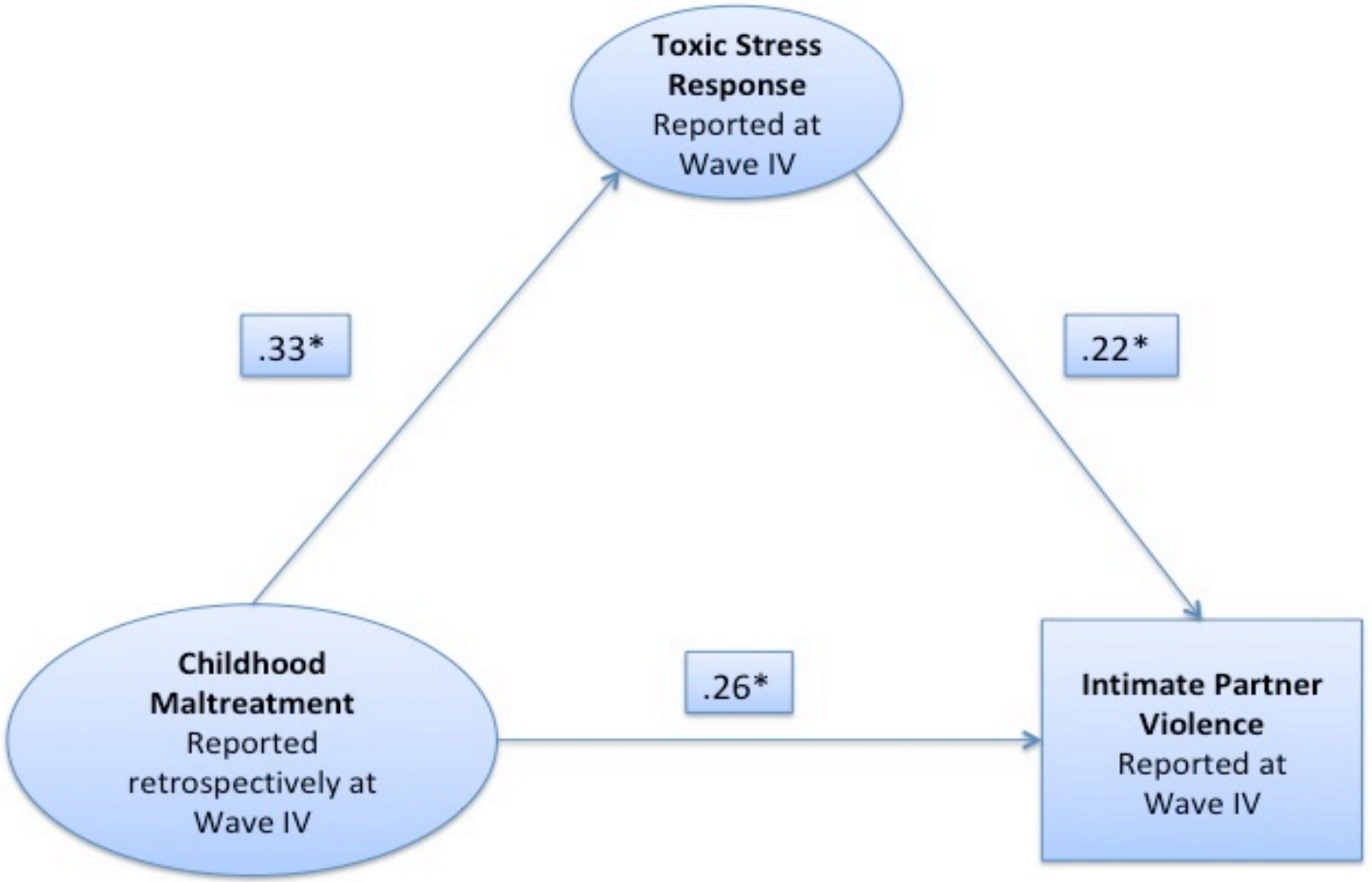
*p<0.05

Table III.4. Measurement Model Factor Loadings, Standard Errors, and Z-Scores			
Measurement Model	Factor Loadings	SE	Z-score
Childhood Maltreatment→			
Emotional Abuse	0.56*	0.04	14.01
Physical Abuse	0.48*	0.04	12.27
Sexual Abuse	0.42*	0.03	13.45
Intimate Partner Violence Perpetration→			
Physically assaulted partner	0.78*	0.02	36.24
Sexually assaulted partner	0.17*	0.02	8.60
Threatened partner with violence	0.85	0.02	37.65
Toxic Stress Response→			
Depression	0.54*	0.03	21.12
Anxiety	0.79*	0.04	30.96
Emotional Dysregulation	0.78*	0.03	30.64
Anger	0.66*	0.03	26.40

*$p<0.001$

Table III. 5. Structural Models and Fit Indices as Pathways Between Constructs are Added								
	b	SE	Z	X^2	$X^2 \Delta$	RMSEA, 90% CI	CFI	TLI
Model 1				180.5		0.042 [0.036,0.048]	0.980	0.970
CM→ IPV	0.26*	0.02	10.87					
Model 2				180.5	0	0.042 [0.036,0.048]	0.980	0.970
CM→ IPV	0.22*	0.02	9.48					
CM→ TSR	0.26*	0.05	10.71					
Model 3				130.9	49.6	0.035 [0.029,0.041]	0.987	0.979
CM→IPV	0.26*	0.04	7.29					
CM→TSR	0.33*	0.03	10.06					
TSR→IPV	0.22*	0.03	8.01					

Figure III.1. Model displaying standardized coefficients for structural pathways.



*p<0.001

Chapter IV

Resilience and Toxic Stress: Examining the Influence of Caring Adults on the Relationships between Childhood Maltreatment, Toxic Stress and Intimate Partner Violence Perpetration

Introduction

Intimate partner violence (IPV) is a serious and preventable public health concern (Sinha, 2013; World Health Organization, 2014). A consistent risk factor for IPV perpetration is exposure to maltreatment in childhood (Smith et al., 2011; Renner & Whitney, 2012; Millett et al., 2013). Pathways between childhood maltreatment to IPV perpetration, hereafter referred to as the intergenerational transmission of violence (ITV), are many and complex, necessitating a multilevel approach to investigating and disrupting this cycle (Patel, 2011; Baker, Cunningham & Harris, 2011; Hill & Thies, 2010; Murphy, 2010; WHO, 2006). The intergenerational transmission of violence has been observed in numerous studies across diverse populations (Cathy Spatz Widom & Wilson, 2015). Theories of attachment, social learning, and family stress have all been employed to help us better understand how and why children exposed to violence are more likely to grow up to be violent adults (Babcock et al., 2000; Cochran, Sellers, Wiesbrock, & Palacios, 2011; Fox et al., 2002; McCubbin & Patterson, 1983). More recently, researchers have increased focus on the ways in which childhood adversity can engender stress

so profound that it catalyzes a cascade of health and mental health issues across the lifespan, including a pattern of psychological sequelae that may increase a person's risk for perpetrating relationship violence (Mair et al., 2012). Viewing ITV through the lens of toxic stress offers new insights into how victimized children grow into violent adults in addition to highlighting critical points of intervention. Yet, most children from violent homes do not become violent adults. In fact, correlations between childhood maltreatment and IPV perpetration are typically found to be low to moderate (Cathy Spatz Widom & Wilson, 2015), suggesting that the effects of childhood maltreatment are being buffered by other factors.

Resilience theory provides a conceptual framework for studying the factors that may buffer the effects of risks for adverse outcomes. Resilience is defined as "the process of overcoming the negative effects of exposure, coping successfully with traumatic experiences, and avoiding the negative trajectories associated with risks" may provide crucial guidance (Fergus & Zimmerman, 2005, pg. 399). Thus, it refers to the positive functioning of individuals who experience hardship and thrive despite the obstacles they face. This strengths-based approach focuses on identifying and understanding which assets or resources allow a person to thrive even in the face of toxic stressors.

Early work on resilience focused on innate, individual level assets that helped to determine whether a person faced with hardship would flourish or not (Richardson, 2002). This conceptualization of resilience tended to view assets as intractable and inherent; as something a person was either born with or not. Later, researchers expanded the notion of resiliency to include resources (i.e., social and environmental factors) that an individual could leverage to overcome adversity (Fergus & Zimmerman, 2005). Researchers have also begun to regard

resilience as a dynamic process, whereby qualities that promote resilience could be developed (Richardson, 2002). Fergus and Zimmerman (2005) have suggested that employing a resilience framework moves the emphasis of public health research and intervention away from a singular focus on risk and toward identifying the assets and resources that promote healthy development in spite of risk (Zimmerman, 2013).

A number of individual and family characteristics have been identified by researchers as promoting resilience and buffering toxic stress. Children with resilience have been identified as having the following characteristics: higher IQ, easy temperament, a perception of competence, a positive self-concept, a realistic sense of control of the situation, empathy, and social problem solving skills (Bowman, Dukes, & Moore, 2012; Franke, 2014). Afifi and MacMillon (2011) reviewed the literature on protective factors associated with resilience after childhood maltreatment. They determined that, regarding individual assets, ego resilience, positive self-esteem, easy child temperament, and daily living skills (personal, domestic, and community adaptive functioning skills) were all protective against the negative consequences of child abuse. Family conditions that contribute to resilience include parents who are responsive, supportive, and provide firm boundaries but are not controlling (CITES). The family structure, positive relationships between parents, family cohesion, stimulating environments, social support, and adequate income also help provide an environment where individuals thrive (Benzies & Mychasiuk, 2009).

Few researchers, however, have studied how and whether resilience can be cultivated. In one longitudinal study examining resilience factors among children exposed to abuse and neglect, DuMont and colleagues (DuMont, Widom, & Czaja, 2007) found that 50% of

participants who were resilient in adolescence were found to be resilient in young adulthood. Conversely, of those found to have low resilience in adolescence, 89% remained low in resilient in adulthood. Within the context of this study, few people were able to move from low to high resilience; however, it would appear that resilience can be cultivated and can grow over time.

Supportive, caring relationships with adults appears to be one of the most consistent protective factors associated with resilience in the face of childhood maltreatment. A stable relationship with a non-violent parent or other significant caregiver has consistently been found to be an important protective factor in alleviating the effects of trauma and distress (Graham-Bermann, DeVoe, Mattis, Lynch, & Thomas, 2006; Mullender et al., 2002), In fact, one author concluded in her review of the literature that the presence of caring, supportive adults in the lives of maltreated children is the most important protective factor (Osofsky, 1999). In a prospective study of resilience in young children transitioning to primary school in the context of cumulative adversity, Miller-Lewis et al. (2013) found that higher quality child-parent and child-teacher relationships were associated with better mental health at time one. Higher quality child-parent relationships predicted more resilient mental health outcomes two years later (Miller-Lewis, Searle, Sawyer, Baghurst, & Hedley, 2013). Carroll and colleagues examined toxic stress exposure in the form of childhood maltreatment and allostatic load among 756 participants in the Coronary Artery Risk Development in Young Adults Study. The authors reported that higher amounts of childhood abuse and lower levels of parental warmth was associated with increased allostatic load in young and middle adulthood. Further, participants who reported having had a loving and affectionate parent were somewhat buffered from the negative effects of childhood abuse on allostatic load. Conversely, individuals who reported experiencing emotional or physical abuse as a child with little or no parental love and affection were more at risk for high

allostatic load (Carroll et al., 2013). Collishaw et al. looked at the relationship between a variety of protective factors and mental health disorders and suicidal ideation in a community sample of abused and non-abused participants. Perceived good parental care was significantly associated with resilience in both abused and non-abused participants (Collishaw et al., 2007).

Luthar has asserted that quality relationships are "critical for achieving and sustaining resilient adaptation" (Luthar, 2006, p. 780). Positive relationships with caring adults appear to both reduce the effects of exposure to risk and reduce a child's exposure to risk factors in the first place (Moore, 2013). For abused children, the presence of at least one stable and loving caregiver can significantly buffer the ill effects of maltreatment (Doll, Jones, Osborn, Dooley, & Turner, 2011). In the absence of such a caregiver, other positive supportive relationships, whether they be with a mentor, teacher, or other non-parental adult, offers protection as well. (Reed-Victor, 2008).

Adverse outcomes resulting from toxic stress are believed to manifest as a result of "increased sensitivity to both psychological and physiologic stress with a decreased resources for social and psychological support to help with stress coping skills" (Franke, 2014, p. 393). Chen et al. (2013) examined the effects of toxic stressors on immune activation and systemic inflammation among adults who reported significant adversity in childhood. They found that maternal warmth significantly buffered the effects of toxic stress (Chen et al., 2010). Further, researchers have found that maternal warmth is correlated with a more positive response to therapies (Carroll et al., 2013; Chen et al., 2010) and both maternal warmth and paternal protection are predictive of better treatment response among maltreated children (Johnstone et al., 2013).

While the protective effects of caring adults in the lives of individuals who have experienced childhood maltreatment is well studied, we do not have a full understanding of how caring adults influences the manifestation of toxic stress response nor on the relationship between toxic stress response and IPV perpetration. Within this study I am also attempting to shed light on at what point within the cycle of violence the presence of caring adults is most efficacious by examining whether caring adults mitigates the relationship between childhood maltreatment and toxic stress response as well as the relationship between toxic stress response and intimate partner violence, or just one relationship or the other. To help address the gaps in our understanding regarding why some maltreated children grow up to be violent adults while others do not, I will look at the unique influence of caring adults on the relationship between toxic stress exposure and IPV as mediated by TSR.

Methods

Sample

Data were drawn from the Adolescent Health (Add Health) study, a longitudinal and nationally representative sample following American youths through young adulthood (Udry, 2003). The study was designed to investigate adolescent health, mental health, neighborhood and school context, health behavior, and relationships, among other issues. Wave 1 of the data collection began in 1994-1995. Respondents were enrolled in 7th through 12th grades. In total, 132 schools in the nation were sampled by using stratified cluster sampling techniques. Over 90,000 students attending these schools completed the self-report questionnaire. To obtain more detailed information from respondents, a stratified subsample was selected and re-interviewed at

home. In all, 20,745 adolescents were re-interviewed at home. Adolescents were asked about their family relationships, participation in unlawful or delinquent activity, and other issues related to adolescent development. Wave 2 of data collection was conducted with 14,738 of these respondents in 1996. The third wave of data were collected in 2001-2002, when most participants had reached between 18 and 26 years of age. Overall, 15,197 participants were interviewed successfully. Wave IV was carried out between January 2008 and February 2009, at which point the Add Health sample was disseminated across the country with respondents living in all 50 states. Over ninety percent of the sample was located and 80.3% of the eligible sample members were interviewed. The sample for Wave-IV was 15,701. Survey data were collected using a 90-minute computer-assisted personal interview during in-home interviews. Less sensitive questionnaire sections were administered with the assistance of an interviewer (Bearman et al., 1997; Blum, Beuhring, & Rinehart, 2000). For the current study I randomly select 1000 participants who reported any child maltreatment at or above one standard deviation above the mean and then matched them with 2000 participants who never experienced any childhood maltreatment.

Measures (see Table III.1 for a more detailed description of the measures)

Dependent Latent Variable—Intimate Partner Violence (Wave IV)

Partner Physical Abuse. Partner physical abuse was assessed with one item measured at Wave IV, "How often (have/did) you (slapped/slap), hit, or (kicked/kick) {your partner}?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6 ("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.21 (STD = 0.75).

Partner Sexual Abuse. Partner sexual abuse was assessed with one item measured at Wave IV, "How often (have/did) you (insisted/insist) on or (made/make) {initials} have sexual relations with you when (he/she) didn't want to?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6 ("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.08 (SD = 0.51).

Threatened Partner Violence. Threatened partner abuse was assessed with one item measured at Wave IV, "How often (have/did) you threatened {your partner} with violence, pushed or shoved (him/her), or thrown something at (him/her) that could hurt?" Respondents reported the frequency of IPV perpetration on 6-point Likert scale ranging from 0 ("never") to 6 ("more than 20 times"). Respondents who refused to answer the question were treated as missing. The average score on this item was 0.29 (SD = 0.87).

Independent Latent Variable—Childhood Maltreatment (retrospectively at Wave IV)

Emotional Abuse. Emotional abuse was measured retrospectively at Wave IV with the single item: "Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 1.08 (STD = 1.8).

Physical Abuse. Physical abuse was measured retrospectively at Wave IV with the item: "Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?" Responses were recorded on a

Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 0.50 (STD = 1.25).

Sexual Abuse. Sexual abuse was measured with the item: "How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?" Responses were recorded on a Likert-type scale ranging from 0 (never) to 5 (more than 10 times). The average score on this item was 0.12 (STD = 0.63).

Mediating Latent Variable—Toxic Stress Response (Wave IV)

Toxic Stress Response. The Toxic Stress Response (TSR) measure developed in Chapter II is composed of four indicators: depression (4 items; alpha = 0.81), anxiety (4 items; alpha = 0.70), emotional dysregulation (7 items; alpha =0.85), and anger (3 items; alpha = 0.74). In this analysis, TSR is treated as a latent variable. See Chapter II for a more complete description of the toxic stress response scale.

Moderating Variable—Caring Adults (Wave I)

Caring Adults. The caring adults scale includes four items: how much do adults about you, how much do your parents care about you, how much does your family understand you, does your family pay attention to you. The four items were summed and averaged. Factor analysis of the items revealed that all items loaded onto a single factor and all factor loadings were .67 or higher. The mean score on the scale was 4.1 (STD = .65; skewness = -.84; kurtosis=3.85). The scale had an alpha of .79.

Statistical Analysis

All variable construction and inferential analyses was conducted in Stata, version 15.0. The sample was dichotomized into two groups based on participants' responses to the caring adults scale. Those who scored higher than .25 of one standard deviation above the median were

placed in the high adult care group and those who scored lower than .25 of one standard deviation below the median were placed in the low adult care group. Participants who scored within .25 plus or minus the median score were dropped from the sample. The total analytic sample was 2,244.

Using the measurement model estimated in Chapter III with the full sample, I conducted multiple-group confirmatory factor analysis to assess measurement equivalence across the high and low groups. These tests directly compare factor loadings between groups. I then conducted multi-group structural equation modeling to test for the moderating effects of caring adults. Model parameter estimates are reported in both unstandardized and standardized forms. Inferences about total and specific indirect pathways were assessed with parameter point estimates and their associated 95% confidence intervals (CI). If zero was not between the upper and lower bounds of the 95% confidence interval of the standardized specific direct and indirect effect, we concluded that there was a significant effect (Hayes, 2009).

Goodness-of-fit of the model was assessed through evaluation of the Bentler Bonett Index which compares the chi-square of a null model to the proposed model (values of .9 or higher indicate strong model fit), and the Root Mean Square Error of Approximation (RMSEA) which is a metric based on the noncentrality parameter (values of .06 or less are strong fitting models) (Bauermeister, 2011). I then ran a series of Sobel tests to assess the magnitude of all indirect effects specified within the model (Kline, 2004). Finally, I reversed the directionality of the model and compared the models across all fit indices to examine which direction best fits the data (Kline, 2004).

Results

Descriptive Statistics. Table IV.1 describes the total sample as well as the sample broken out into those who reported low adult caring (low care) and those who reported high adult caring (high care). Comparisons of descriptive statistics between low care and high care groups were conducted using t-tests or chi-square tests.

The total sample included more females (53.1%) compared to males (46.9%). Over 60% of the sample was white and 20% were black. Participants were 28 years old on average and reported a mean income of $34,515 annually. About 17% of the sample had achieved a high school degree as their highest level of education and about one-quarter were graduates of college or a vocational degree program. The average score on the childhood maltreatment scale was 0.51 (SD=0.82), the TSR average score was 2.4 (SD=0.56) and the IPV perpetration average for the total sample was 0.28 (SD=0.71). All demographic variables were controlled for in the final analysis.

I found several significant differences between the low care and high care groups. The low care group reported less educational attainment compared to the high care group (chi-square=-3.9; $p<0.001$). Participants in the low care group were slightly younger ($t=6.3$; $p<0.001$) and had lower proportion of black participants and a higher proportion of Asian participants (chi-square=13.9, $p<0.05$). The TSR scores for low care participants were significantly higher compared to the high care group ($t=6.8$; $p<0.001$). Participants in the low care group were significantly more likely to report having experienced childhood maltreatment ($t=7.9$; $p<0.0010$ and IPV perpetration ($t=4.9$; <0.001) compared to those in the high care group. I did not find any significant differences between the groups regarding gender or income.

Correlations among study variables. Table IV.2 displays the correlations among the study variables. All correlations were significant with several exceptions. Physical partner

assault in the IPV perpetration latent variable did not significantly correlate with emotional dysregulation in the TSR latent variable. Sexual partner assault in the IPV perpetration latent variable did not significantly correlate with anxiety, emotional dysregulation, or anger in the TSR latent variable or with sexual abuse in the childhood maltreatment latent variable.

Multiple group SEM

Measurement model. Two measurement models were estimated: one model's parameters were constrained to be equal across support groups, and the other model's parameters were allowed to differ between groups. I found a significant difference in overall model fit between the fully constrained measurement model and the freely estimated measurement model (LR chi-square=66.0; p<.001). The following fit statistics were generated for the fully constrained model: chi-square=498.9, p < .001; CFI=.93; TLI=.93; RMSEA=.07, 90% CI [.06, .72]; SRMR=.095. I tested for invariance of parameters across groups and relaxed the identified constraints accordingly. This process resulted in a final model that was partially constrained and that was a good fit to the data: (chi-square=251.4, p=.001.; CFI=.97; TLI =.97; RMSEA=.04, 90% CI [.038, .05]; SRMR=.04). All indicator variables loaded on all factors significantly at p<0.001. Several factor loadings differed between groups. Depression had a .90 loading in the high care group versus .84 in the low support care group. In the low care group, emotional abuse loaded onto the childhood maltreatment latent variable at .81 compared to .79 in the high care group. Finally, child sexual abuse loaded onto the childhood maltreatment latent variable at .35 in the low care group versus .26 in the high care group.

Structural model. Three structural paths were added to the model and then estimated across low and high adult care groups. The group goodness-of-fit indicated a good fit to the data (chi-square=251.4, p<0.001; $SRMR_{group1}$=0.049, $SRMR_{group2}$=0.031; CD_{group1}=0.76,

DC_{group2}=0.74) Overall results are consistent with those from the full sample model; however, the analyses revealed that adult care has a moderating effect on the relationship between CM and TSR. For individuals who reported less adult care, the relationship between CM and TSR was stronger (b=.27; p<0.001) compared with individuals who reported more adult support (b=.19; p<0.001). These pathways significantly differed from each other across groups with a chi-square of 6.7 (p<.01). Adult support had a marginally moderating effect on the relationship between childhood maltreatment and IPV, though this effect was not significant (chi-square=2.8, p=.09). Figure IV.1 displays the structural pathways between the latent constructs in the model between low and high care groups.

Discussion

The intergenerational transmission of violence describes a pattern of violence that persists across the lifespan and across generations, but it is far from deterministic. While some children from violent homes will grow up to perpetrate violence themselves, many will find ways to overcome their past and grow into non-violent adults, suggesting that other factors may create turning points in this trajectory. I found support for the buffering effects of a caring adult suggesting that not all youth who experience serious risk, including maltreatment, experience negative outcomes (Collishaw et al., 2007; DuMont et al., 2007).. This finding supports resiliency theory because the presence of caring adults moderated the relationship between childhood maltreatment and toxic stress. These findings suggest that the ill effects of toxic stress exposure are attenuated by the presence of caring adults.

My finding that caring adults buffers the negative psychological consequences of child abuse is consonant with previous research. Caring adults can be conceptualized as a form of social support and the social support has consistently found to be an important correlate of

resilience (Glanz, Rimer, & Viswanath, 2008). Social support, broadly speaking, has been found to moderate the relationship between childhood maltreatment and IPV. Social support, both prior to and immediately following a traumatic event, has been shown to be an enormously influential protective factor against the development of adverse psychological symptoms (Robinaugh et al., 2011). According to trauma experts, the concept of group solidarity and the feelings of connectedness, affirmation, and the reconstruction of a fragmented sense-of-self engendered thereby, is the strongest antidote to traumatic experience (Herman, 1997). Individuals living within a context of strong family ties and community cohesion are better situated and more apt to cope effectively with trauma and post-trauma responses. A meta-analysis of trauma studies found that 40% in the variance in PTSD severity was accounted for by social support, by far the strongest predictor considered (Brewin, Andrews, & Valentine, 2000). Social support is most often thought to intervene between the stressor and the stress reaction, thereby attenuating or obviating a pathological response (Glanz et al., 2008).

The finding that caring adults does not moderate the direct relationship between CM and IPV is interesting given the previous literature. For example, Harris and Dertch (2001) investigated factors that helped children from violent homes grow up to be non-violent adults. They found that the presence of a caring adult was perceived by the study participants as essential in their successfully breaking the cycle of violence in their own lives (Harris & Dersch, 2001). It is possible that the presence of caring adults in childhood has an indirect effect on risk of IPV perpetration by helping maltreated individuals cultivate the ability to access support when necessary across the lifespan. When people who experienced maltreatment in childhood are successful at accessing support in adulthood, they are less likely to abuse their partners (Thornberry et al., 2013). In a large longitudinal study, Thornberry and colleagues found that

adults with a history of child maltreatment were significantly less likely to abuse either their partners or their children if they had current access to stable, safe, and caring relationships with other adults (Jaffee et al., 2013). Researchers examined factors that break the intergenerational cycle of violence longitudinally amongst individuals who had participated in a long-term residential care program as children and adolescents. They found that rates of IPV in adulthood were significantly less among those who stayed in residential care compared to both the control group and the national average. The authors concluded that the access to caring, supportive adults and the skills acquired as a result to forge healthy interpersonal relationships had long-term beneficial consequences for the treatment group (Huefner et al., 2007).

Limitations. Several limitations of the study should be noted. Regarding the maltreatment variables, neither age of onset nor severity were measured; only that the abuse occurred before the participant's 18th birthday and the number of times the abuse occurred. We know from previous studies that age of onset and severity matter in terms of the degree of impact the traumatic exposure will ultimately have. A more complete understanding of the importance of caring adults on toxic stress exposure and response would be gleaned were the traumatic exposure variable more thoroughly measured because it would allow for the controlling of potential confounders. For example, my findings that caring adults moderated the relationship between CM and TSR may have been confounded by the severity or age of onset of abuse, if caring adults was highly correlated with either variable.

Secondly, TSR and IPV perpetration were measured concurrently, so we cannot determine whether TSR preceded IPV perpetration or whether the reverse is actually true. Therefore, it is impossible to make any definitive statements regarding when caring adults are

most effective in the lives of maltreated individuals. Future research should examine TSR closer to the time of toxic stress exposure. Despite the potential confounding, theory

Lastly, the caring adults scale was dichotomized at plus/minus .25 of one standard deviation below and above the median. Dichotomizing was necessary to conduct the multi-group analysis, however, there is a cost to dichotomization. Namely, participants were excluded if they were too close to the median to ensure that the groups were adequately dissimilar resulting in a loss of data. However, the sample size was large enough to support such a loss.

Conclusion. Identifying factors that help individuals exposed to child maltreatment break the cycle of violence in their own lives can inform intervention design and clinical practice. Caring adults did not moderate the relationship between toxic stress response and IPV perpetration nor did it significantly moderate the direct relationship between childhood maltreatment and IPV perpetration. These findings point to the possibility that interventions with caring adults designed to disrupt the cycle of violence may be more effective prior to the onset of toxic stress response symptoms. Given that brain plasticity and age are inversely related, it makes sense that earlier intervention efforts would be more effective. The presence of caring adults buffers children against the negative effects of maltreatment, mitigates the risk of toxic stress response and disrupts the intergenerational transmission of violence. Interventions that increase a child's exposure to caring adults, such as mentorship (Molnar, Cerda, Roberts, & Buka, 2008) and home visiting (Garner, 2013) programs, could diminish the risk of TSR and, ultimately, IPV.

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Table IV.1. Demographic Characteristics for Total, Low Care, and High Care Sample					
Individual Characteristics	**Total (n=2,244)**	**Low Care (993)**	**High Care (1,251)**	**t/X^2**	**p-value**
Gender: N (%)				0.002	n.s.
Males	1,052 (46.9)	465 (46.8)	587 (46.9)		
Females	1,192(53.1)	528 (53.2)	664 (53.1)		
Race: N (%)				13.0	<0.05
White	1,381 (61.5)	610 (61.4)	771 (61.6)		
Black	446 (19.9)	175 (17.6)	271 (21.7)		
AI/AN	25 (1.1)	12 (1.2)	13 (1.0)		
Asian/Pacific Islander	145 (6.5)	80 (8.1)	65 (5.2)		
Other	247 (11.0)	116 (11.7)	131 (10.8)		
Age at Wave 4: Mean (SD)	28.5 (1.8)	28.7 (1.7)	28.2 (1.9)	6.3	<0.001
Education at Wave 4: N (%)				-3.9	<0.001
No HS Degree	155 (6.9)	85 (8.6)	70 (5.6)		
High School Graduate	370 (16.5)	162 (16.3)	208 (16.6)		
Some College/Vo-Tech	878 (39.1)	416 (41.9)	462 (36.9)		
College/Vo-Tech Graduate	573 (26.1)	241 (24.3)	332 (26.5)		
Some Grad/Professional	95 (4.2)	33 (3.3)	62 (4.9)		
Grad/Professional Degree	173 (7.7)	56 (5.6)	117 (9.4)		
Income: Mean (SD)	34,515 (30,109)	33,129 (30,838)	35,426 (30,899)	-1.7	n.s.
Childhood Abuse: Mean (SD)	0.51 (0.82)	0.68 (0.89)	0.39 (0.80)	7.9	<0.001
Toxic Stress Response	2.4 (0.56)	2.5 (0.56)	2.3 (0.49)	6.8	<0.001
Intimate Partner Violence	0.28 (0.71)	0.38 (0.89)	0.22 (0.64)	4.9	<0.001

Table IV. 2. Correlations among study variables

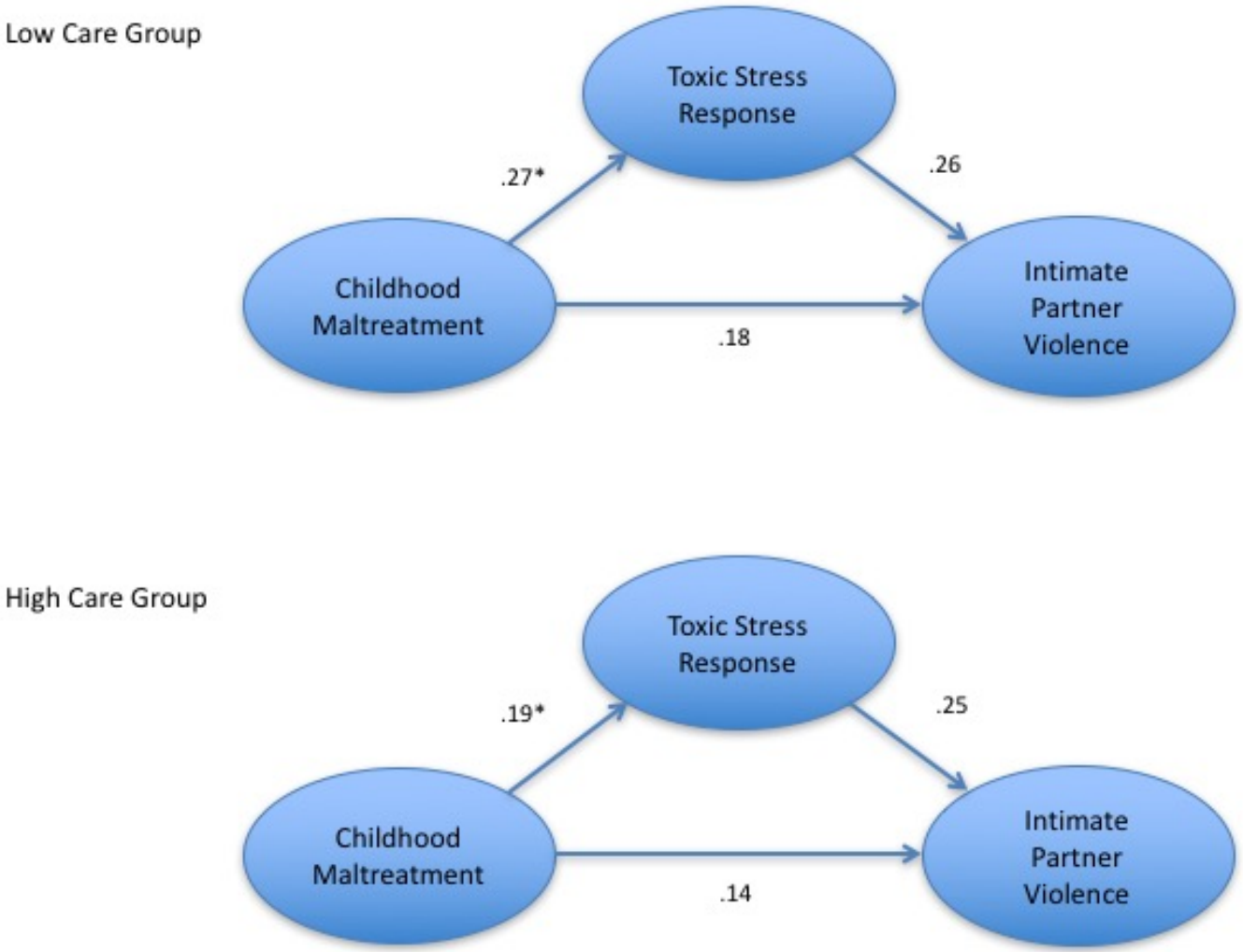
	1	2	3	4	5	6	7	8	9	10
(1) Depression	1.0									
(2) Anxiety	0.42*	1.0								
(3) Emotional Dysregulation	0.42*	0.60*	1.0							
(4) Anger	0.32*	0.52*	0.74*	1.0						
(5) Emotional Abuse	0.18*	0.17*	0.15*	0.12*	1.0					
(6) Sexual Abuse	0.12*	0.09*	0.09*	0.08*	0.23*	1.0				
(7) Physical Abuse	0.11*	0.07*	0.09*	0.11*	0.54*	0.20*	1.0			
(8) Physical Assault Partner	0.19*	0.15*	0.18	0.17*	0.14*	0.13*	0.13*	1.0		
(9) Sexual Assault Partner	0.07*	0.01	0.03	0.04	0.06*	0.01	0.08*	0.13*	1.0	
(10) Threaten Partner	0.20*	0.16*	0.21*	0.21*	0.14*	0.11*	0.16*	0.67*	0.17*	1.0

*$p<0.05$

Table IV.3. CFA Factor Loadings for Low and High Adult Care Groups		
	Low (n=993)	High (n=1,251)
Childhood Maltreatment		
Emotional Abuse	0.79	0.80
Physical Abuse	0.67	0.71
Sexual Abuse	0.33	0.27
Intimate Partner Violence Perpetration		
Physically assaulted partner	0.77	0.77
Sexually assaulted partner	0.18	0.18
Threatened partner with violence	0.86	0.86
Toxic Stress Response		
Depression	0.85	0.87
Anxiety	0.71	0.71
Emotional Dysregulation	0.84	0.84
Anger	0.73	0.73

**Note: all loading are significant at $p<0.001$

Figure IV.1. Model displaying the structural pathways and coefficients for both low and high parental care groups.



*Indicates pathways that are significantly different from each other
Low: SRMR=0.068; CD=0.75
High: SRMR=0.77; CD=0.74

Chapter V

Summary

People who experience violence in their family of origin are more likely to grow up to perpetrate intimate partner violence (IPV), a phenomenon referred to as the intergenerational transmission of violence (ITV). Child maltreatment is common in the United States. Researchers from the Adverse Childhood Experiences (ACE) study reported that over 17,000 adults (over 30% of the sample) reported physical abuse and nearly 20% reported sexual abuse as children (Felitti, Anda, Nordenberg, & Williamson, 1998), making it a potentially significant risk factor for IPV. Theories of attachment, social learning, and family stress have all been employed to help us better understand how and why children exposed to violence are more likely to grow up to be violent adults (Babcock et al., 2000; Cochran et al., 2011; Fox et al., 2002; McCubbin & Patterson, 1983). There has been an increased focus on the ways in which childhood trauma can engender stress so profound that it affects health and mental health across the lifespan, including a pattern of psychological sequelae that may increase a person's risk for perpetrating relationship violence (Mair et al., 2012). Childhood adversity triggers a cascade of negative consequences, making up a causal chain that links early childhood traumatic exposure to psychological problems to maladaptive coping behaviors and then, ultimately, to increased levels of morbidity and mortality (Anda et al., 1999; Arias, 2004; Mair et al., 2012). More recently, scientists across

disciplines have found that repeated exposure to traumatic adversity early in life causes a prolonged elevation and dysregulation of the body's stress response systems, resulting in a lasting legacy of developmental, neurological, behavioral and psychological maladies (De Bellis, 2001), a phenomenon known as toxic stress. Within the context of this dissertation I have examined whether (1) toxic stress response manifests as a cluster of psychological symptoms; (2) toxic stress response mediates the relationship between child maltreatment and intimate partner violence; and (3) the presence of caring adults moderate the negative effects of toxic stress on toxic stress response or intimate partner violence.

Summary of Results

Chapter II: Developing a Measure of Toxic Stress Response. In Chapter II, I developed an Adolescent Health (Add Health) specific index of toxic stress response. This index assesses a cluster of psychological symptoms identified in the toxic stress, childhood maltreatment, and trauma literatures as commonly occurring after exposure to early, chronic, and profound adversity. The index includes emotional dysregulation, depression, anxiety and anger. Results from the exploratory and confirmatory factor analyses indicated that the model fit the data well. To test the index's content validity, I correlated scores from the TSR index with experiences that I hypothesized would likely yield a toxic stress response. These experiences included childhood maltreatment (emotional, physical and sexual abuse), community violence, community disadvantage (levels of poverty at the community level), and family hardship (e.g., parental incarceration, financial difficulties, death of family member). The correlations between TSR and hypothesized toxic stressors were significant and in the expected directions, though small. Unsurprisingly, the correlations were larger for individual and family level exposures (childhood maltreatment and family hardship) compared with community level exposures (community

violence and disadvantage). There are several reasons why these correlations may not have been larger. Perhaps the most notable being the time point at which TSR was assessed. I constructed the TSR index with data from Wave IV of the Add health survey, when the participants were in their 20s and early 30s. While I believe that the psychological fallout from childhood trauma persists well into adulthood, the relationship between toxic stress exposure and toxic stress response in adulthood would likely be attenuated. Emotional dysregulation and anger were not measured at earlier waves within the dataset and thus, constructing the TSR index in Wave IV was the only option available. That said, the small, significant correlations between child maltreatment and adult TSR found in my study is consonant with much of the previous research regarding the relationship between child abuse and adult psychopathology (Anda et al., 2006b; Arias, 2004; B. Bradley et al., 2011; R. G. Bradley et al., 2008; Capaldi & Clark, 1998; Gibb et al., 2007).

The unique contributions of this chapter include the creation of a toxic stress measure. This measure is Add Health specific, however, future research should attempt to tailor the measure for other populations and in other research settings. Given the simplicity and brevity of the index, it has the potential to be an efficient way to identify people suffering from toxic stress symptoms with low respondent burden. This study builds on past research by reviewing the psychological symptoms associated with toxic stress exposure and aggregating them into a single index. The current study supports the theoretical framework employed by the researchers in the ACE study. ACE researchers conceptualized the risk associated with childhood adversity across the life course as a pyramid building from traumatic exposure to disrupted neurodevelopment to social, emotional, cognitive impairment to the adoption of risky health behavior to disease and finally to early death. This study provides support for the pathway between traumatic exposure

(childhood maltreatment) to risky health behaviors (IPV perpetration) through neurodevelopmental disruption (toxic stress) and emotional/cognitive impairment (TSR). It is notable that the data for this dissertation is drawn from the National Longitudinal Study of Adolescent to Adult Health study, the largest longitudinal study of adolescent health ever undertaken in the United States. An Add Health-specific index of toxic stress response could help future researchers deepen our collective understanding of this important phenomenon through the use of one of our most powerful and well-used existing datasets.

Chapter III: The Traumatic Origins of Intimate Partner Violence Perpetration and the Mediating Role of Toxic Stress Response. In Chapter III, I sought to investigate the mechanisms by which violence is transmitted intergenerationally. There appears to be substantial overlap between the psychological sequelae of prolonged childhood exposure and the psychological precursors to IPV perpetration (Babcock et al., 2000; Baker et al., 2016; Birkley & Eckhardt, 2015; B. Bradley et al., 2011; R. G. Bradley et al., 2008; Gibb et al., 2007; Mair et al., 2012). Previous researchers have noted that IPV perpetrators were more likely to report experiences of abuse and exhibit symptoms of traumatization compared to non-perpetrators (M. A. Dutton, 2009; Scott & Babcock, 2010). I hypothesized that the symptoms caused by toxic stress—depression, anxiety, emotional dysregulation, and anger-- may increase a person's risk for perpetrating IPV. Specifically, I tested whether toxic stress response mediates the relationship between childhood maltreatment and adult IPV perpetration using structural equation modeling. I tested this hypothesis in a subsample containing 1,000 participants who reported a history of childhood maltreatment and 2,000 participants who reported no such history. I found that childhood maltreatment is partially mediated by TSR, however, the direct pathway between child maltreatment and IPV perpetration remained significant. I also compared IPV perpetrators

versus non-perpetrators on demographic characteristics and found that perpetrators were more likely to be low income, female, and Black. These findings are in keeping with prior researchers who have found that, while IPV perpetration cuts across demographic differences, low SES groups tend to bear a higher burden of IPV (Allen et al., 2009; Capaldi et al., 2012; Cunradi et al., 2002; Fox et al., 2002; Langhinrichsen-Rohling et al., 2012; Stith et al., 2004). Previous research has also found higher rates of female perpetrated IPV, however, it is worth noting that female-to-male IPV perpetration tends to be in reaction to male violence (defensive) and much less severe compared to male-to-female IPV(Allen et al., 2009; Langhinrichsen-Rohling et al., 2012).

This chapter offers the unique contribution of employing the TSR as a mediator between childhood maltreatment and IPV perpetration. These findings help us to better understand the psychological fallout of childhood exposure to toxic stressors as well as the mechanisms by which violence passes from one generation to the next. Numerous prior researchers have posited that psychological symptoms mediate the relationship between childhood maltreatment and IPV perpetration (e.g., Baker et al., 2016; Birkley & Eckhardt, 2015; Mair et al., 2012; Stith et al., 2004). This study builds on that research by identifying a specific cluster of psychological symptoms hypothesized to accompany toxic stress exposure and demonstrating the mediating role TSR plays in the intergenerational transmission of violence. Further, in Chapters III and IV, I employ structural equation modeling (SEM) to map the relationships between study variables. SEM is ideal for such an analysis because it estimates simultaneous path coefficients between multiple predictors and outcome variables, thus testing a conceptual model in its entirety at once rather than piece by piece. Additionally, SEM has the added benefit of controlling for any

variance related to measurement error due to the fact that estimated paths are between latent factors comprised of multiple indicators rather than between single indictors.

Chapter IV: Resilience and Toxic Stress: Examining the Influence of Caring Adults on the Relationships between Childhood Maltreatment, Toxic Stress and Intimate Partner Violence Perpetration. In Chapter IV, I explored how resilience theory could improve our understanding of why some maltreated children grow up to perpetrate IPV while others do not. Resilience theory, defined as "the process of overcoming the negative effects of exposure, coping successfully with traumatic experiences, and avoiding the negative trajectories associated with risks" may provide crucial guidance (Fergus & Zimmerman, 2005). Supportive, caring relationships with adults appears to be one of the most consistent protective factors associated with resilience in the face of childhood maltreatment. A stable relationship with a non-violent parent or other significant caregiver has consistently been found to be an important protective factor in alleviating the effects of trauma and distress (Graham-Bermann, DeVoe, Mattis, Lynch, & Thomas, 2006; Mullender et al., 2002). In the final study of my dissertation, I found that the presence of caring adults moderated the relationship between childhood maltreatment and toxic stress response but not the relationship between toxic stress response and intimate partner violence or the direct pathway between childhood maltreatment and intimate partner violence. Although the effects were reduced for respondents with a caring adult, these adults did not eliminate entirely the deleterious effects of childhood maltreatment. Nevertheless, these findings suggest that caring adults play a role in reducing the negative effects of childhood maltreatment on adult IPV.

One of the overarching objectives of this dissertation project was to view toxic stress through a lens of resilience, rather than from a standpoint of deficiency. A resilience lens helps us to not simply identify what is going wrong, but to pinpoint and build upon what is going right. Toward this end, I have identified a possible point of intervention, caring adults, to buffer children against abuse.

Limitations

Several limitations to this study are important to note. In *Chapter II*, some of the convergent validity variables, including all childhood abuse variables, were measured retrospectively. Researchers have conducted studies assessing the validity of self-report, retrospective reporting of child abuse. The authors found that the most common type of misreporting was underreporting (Fergusson et al., 2000; Hardt & Rutter, 2004). Reasons for failing to disclose abuse could include discomfort with the interviewer or the desire to avoid the pain caused by remembering the abuse. It is also very possible that the respondent has simply forgotten that the abuse took place. Not only may the abuse have happened when the participant was very young and less likely to form in memory, but researchers have found that survivors of childhood victimization experience memory impairment that inhibits their ability to recall the event (Glaser, 2000; Teicher, 2002). As a result, the correlations reported here may be smaller than they would be were the child abuse data collected at the time of the incident through court or police reports.

Given that the chi-square of the unmodified model was large and significant, the results from the CFA may suggest that this model is not ideally specified. The modified model, however, improved goodness-of-fit considerably. Many authors have cautioned against the practice of allowing measurement errors to correlate in order to improve model fit (e.g., Cortina,

2002; Kaplan, 1990; MacCallum, Tomarken & Waller, 2003). Landis et al. (2009) wrote, "to the degree that two residuals correlate, there is evidence that there exists a cause of both of the variables to which the residuals are attached but that is not specified in the model" (p. 17). That said, Landis et al. (2009) argue that the addition of model paths may be appropriate when there is sufficient theoretical justification for doing so, such as situations when indicator variables share components, as is the case with depression and anxiety.

In *Chapter III*, I measure exposure to toxic stressors at Wave I (and retrospectively at Wave IV) and I measure TSR at Wave IV. Measuring TSR closer to the exposure itself might better capture the psychological fallout of toxic stress exposure. Measuring TSR closer to exposure within this particular dataset, however, would mean measuring these psychological symptoms with adolescents, which may, in and of itself, be confounding given the emotional volatility associated with that age group. Furthermore, researchers have found that the psychological fallout from childhood maltreatment is not time limited and lasts well into adulthood (Anda et al., 2006a; Bernet & Stein, 1999; Gibb et al., 2007).

Childhood maltreatment is measured retrospectively at Wave IV while the other exposure variables are measured at Wave I. While the Wave IV childhood maltreatment variables ask about the same time period as those assessed at Wave I, the retrospective nature of the survey questions introduces the possibility of recall bias. Importantly, however, some have argued that studies employing retrospective reporting are more accurate than prospective studies (Kendall-Tackett & Becker-Blease, 2004). This is due to the fact that, unlike other study populations, professionals who discover abuse among children are ethically and legally bound to report it to law enforcement and/or child protection agencies, which are then required to intervene. Therefore, children who have been identified as abuse victims have a higher likelihood of

receiving intervention or treatment during childhood compared with children who are never identified as victims, which alters their psychological trajectory. Additionally, prospective studies likely miss cases of child abuse given how profoundly underreported this problem remains (Kendall-Tackett & Becker-Blease, 2004). The IPV perpetration measure relies on self-report. Given the sensitive nature of the topic it is possible that the responses may be biased due to social desirability. Yet IPV perpetration is likely underreported by participants, which would attenuate the relationship between childhood maltreatment and IPV rather than strengthen it, rendering these findings overly conservative.

In *Chapter IV,* neither age of onset nor severity were measured for the maltreatment variables; only that the abuse occurred before the participant's 18th birthday and the number of times the abuse occurred. We know from previous studies that age of onset and severity matter in terms of the degree of impact the traumatic exposure will ultimately have. A more complete understanding of the importance of caring adults on toxic stress exposure and response would be gleaned were the traumatic exposure variable more thoroughly measured because it would allow for the controlling of potential confounders. For example, my findings that caring adults moderated the relationship between CM and TSR may have been confounded by the severity or age of onset of abuse, if caring adults was highly correlated with either variable. Secondly, as with the previous two studies, TSR and IPV perpetration were measured concurrently, so we cannot determine whether TSR preceded IPV perpetration or whether the reverse is actually true. Therefore, it is impossible to make any definitive statements regarding when caring adults are most effective in the lives of maltreated individuals. Lastly, the caring adults scale was dichotomized at plus/minus 0.25 of one standard deviation below and above the median. Dichotomizing was necessary to conduct the multi-group analysis, however, there is a cost to

dichotomization. Namely, participants were excluded if they were too close to the median to ensure that the groups were adequately dissimilar. This resulted in dropping some respondents from the analysis, however, the sample size was large enough to support such a loss while maintaining sufficient statistical power to detect effects.

Implications for Health Promotion

The majority of the scholarship and theory about trauma and trauma response has been, up until recently, focused on individuals who experienced a single traumatic event or a time-bounded series of traumatic events (e.g., rape, combat). The event or events are aberrant, representing a significant departure from and disruption to their day-to-day lives. In contrast, toxic stress (i.e., prolonged exposure to trauma at a young age)--such as child maltreatment--can interfere with a person's emotional, psychological, social, and physical development and results in a distinct symptom profile that differs from posttraumatic stress disorder or other, better understood trauma related disorders (Luxenberg, Spinazzola, & van der Kolk, 2001). Trauma researchers and clinicians aver that among those who suffer a profusion of seemingly unrelated and unexplained mental, emotional, behavioral and somatic difficulties, chronic trauma in childhood could very well be the etiologic agent underlying their suffering (Cook, et al., 2005; van der Kolk B. , 2005). For example, significant correlations have been found between the experience of toxic stress exposure and depression, suicide, smoking, alcoholism, obesity, sexually transmitted infections, heart disease, stroke, liver disease, and intimate partner violence (Felitti, Anda, Nordenberg, & Williamson, 1998). Additionally, those exposed to toxic stressors in childhood are more likely to experience future traumas as adults (Briere & Spinazzola, 2009). Given the findings that intimate partner violence is far from the only long-term consequence of toxic stress, a brief index focused exclusively on the psychological symptoms most often

described in the literature that are theorized to manifest before the physical symptoms (Arias, 2004) could prove a reliable indicator of other pathological responses to early and prolonged childhood trauma.

Another major implication of this research is the finding that presence of caring adults buffers children against the negative effects of maltreatment, mitigates the risk of toxic stress response and disrupts the intergenerational transmission of violence. Interventions that increase a child's exposure to caring adults, such as mentorship (Molnar et al., 2008) and home visiting (Garner, 2013) programs, could help diminish the risk of TSR and, ultimately, IPV.

Implications for Future Research. There are other, unaccounted for avenues by which child maltreatments acts on IPV perpetration. Theories including Social Learning Theory, Attachment Theory, and Family Stress Theory have all contributed to our understanding of the intergenerational transmission of violence. Ideally, variables included in those theories such as role modeling, attachment style, or family hardship would be accounted for in a comprehensive model of ITV. These variables were not measured in the Add Health data and are therefore missing from my analyses. Future research that accurately measures and integrates elements of other ITV theories with toxic stress response would likely render a more complete picture of this phenomenon. Nevertheless, this research is one of the first studies to examine the psychological sequelae of toxic stress exposure in aggregate as a mediator linking CM and IPV.

Future research should attempt to validate the TSR index in other populations and examine TSR in reaction to other kinds of toxic stressors. For example, the intergenerational transmission of violence may be more pronounced and intractable among low income and minority populations. Researchers have reported higher rates of both child maltreatment and IPV among racial/ethnic minorities and lower-income households. While the vast majority of toxic stress

literature is focused on child abuse, toxic stressors also include environmental conditions such as community violence and neighborhood disadvantage (Shern et al., 2014). The ACEs study relied on data principally collected from white, middle- and upper-middle-class participants and focuses on experiences within the home. As a result, researchers have suggested that the ACEs included in the original measure may not adequately capture adversity among racially and socioeconomically diverse populations (Cronholm et al., 2015). Wade and colleagues (2014) collected qualitative data with Black and Latino youth to explore whether expanding the definition of an adverse childhood experience was warranted. They found support for the inclusion of neighborhood-level indicators such as community violence, living in an unsafe neighborhood, and residential instability within the concept of 'childhood adversity' (Wade et al., 2014). Future studies could determine whether the TSR index is valid with different populations and whether it captures toxic stress response resulting from a diversity of stressors.

The intergenerational transmission of violence describes a pattern of violence that persists across the lifespan and across generations. But it is a cycle that can be, and often is, broken. Most children from violent homes will find ways to overcome their past and grow into non-violent adults. However, a sizeable minority of them will not. Within this dissertation I have contributed to the knowledge base by helping to recognize child abuse victims who are at greater risk for perpetrating IPV in adulthood, clarifying the mechanisms by which violence is transmitted from one generation to the next, and identifying resilience factors that buffer abuse victims against toxic stress response.

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