POLYGENIC RESILIENCE ON THE ASSOCIATION BETWEEN CHILDHOOD MALTREATMENT, DELINQUENCY, AND VICTIMIZATION

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Eric M. Cooke

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DEDICATION

I dedicate this dissertation to my best friend, my partner, my world

Rachel Elysse O'Neil Cooke

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ABSTRACT

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Childhood maltreatment is a harmful form of interpersonal victimization associated with the development of maladaptive behavior throughout the life course. Within the context of criminology, childhood maltreatment has been linked to an increased likelihood of engaging in delinquent behavior and being the victim of a crime. However, not everyone who experiences victimization will develop maladaptive behaviors, engage in delinquency, or experience victimization. This subset of individuals are considered resilient to the adverse effects of childhood maltreatment. Studies have identified several factors that promote resilience in response to childhood maltreatment. Few studies have examined how polygenic scores influence resilience to childhood maltreatment in relation to delinquency and victimization later in life. The current dissertation seeks to address this gap by using latent growth modeling techniques to assess the moderating influence of polygenic scores as a source of resilience between childhood maltreatment, delinquent behavior, and criminal victimization in adolescence and young adulthood. Results indicate that the polygenic score for depression moderated the relationship between childhood maltreatment and delinquency while the polygenic score for extraversion moderated the relationship between childhood maltreatment and victimization. These findings have implications for the ecological transactional model as well as the differential susceptibility perspective.

KEY WORDS: Childhood maltreatment; Polygenic scores; Victimization; Delinquency; Longitudinal; Latent growth curve

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CHAPTER I

Introduction

Childhood maltreatment (CM) is a detrimental form of interpersonal violence involving one or more instances of abuse (i.e., physical, emotional, psychological, and/or sexual abuse) or neglect (i.e., physical, emotional, medical, and/or educational) perpetrated by a parent, caregiver, or other adult onto a child (i.e., someone under the age of 18). Worldwide estimates suggest that roughly a third of children will experience some form of CM while growing up (Hussey et al., 2006). Similar findings have been reported in the United States and show that approximately 25% of children under the age of 18 will experience one or more forms of CM (Finkelhor et al., 2015). The human and economic costs of CM are high. In 2018, there were approximately 678,000 substantiated cases of CM in the United States (Finkelhor et al., 2020; U.S. Department of Health & Human Services, 2018). The total costs to these victims including the loss of social, psychological, and economic capital are estimated to be over $2 trillion each year (Leung et al., 2020; Peterson et al., 2018).

Children who experience maltreatment are more likely to develop a range of negative health outcomes and dysfunctional psychopathological conditions including self-harm (Gibb et al., 2001), depression (Wright et al., 2009), alcohol abuse, poor impulse control (Hart et al., 1997), risky behaviors (Shaffer et al., 2009), suicide (Cassels et al., 2018), difficulties with interpersonal relationships (Dvir et al., 2014), and post-traumatic stress disorder (Thompson et al., 2000). Variation in the emergence and persistence of these psychopathological disorders and negative health consequences are influenced by the type, timing, severity, and chronicity of CM (Bousman et al., 2017;

Cowell et al., 2015). Research finds that children who experience more severe and frequent CM earlier in life are at an increased risk of developing dysfunctional behavioral and health adaptations to this early life adversity throughout adolescence and adulthood (Bousman et al., 2017; Cowell et al., 2015). It appears that frequent, severe, and early exposure to CM tends to "get under the skin" and results in individual differences in negative adaptive behavioral styles throughout the life course (Ioannidis et al., 2020; Domhardt et al., 2014; Scoglio et al., 2019; Yoon et al., 2019).

Two negative outcomes associated with exposure to CM, and relevant to the field of criminology, are delinquency and victimization. Over the past several decades a considerable amount of research has observed a link between CM exposure and later life delinquency and victimization (Banny et al., 2013; Barnes et al., 2009; Beaver et al., 2010; Connolly et al., 2015; Daigle et al., 2008; DeLisi et al., 2010; Duke et al., 2009; Fagan, 2005; Finkelhor et al., 2009; Hamilton et al., 2002; Hurren et al., 2017; Ireland et al., 2002; Maas et al., 2008; Malvaso et al., 2015; Pezzoli et al., 2019; Reckdenwald et al., 2013; Stewart et al., 2008; Thornberry et al., 2010; Tillyer et al., 2012; van der Put & de Ruiter, 2016; Widom & Brzustowicz, 2006; Widom et al., 2008; Wright & Fagan, 2013; Yoon et al., 2018). In their systematic review of 62 studies, Malvaso and colleagues (2015) found a consistent relationship between exposure to CM and offending behavior throughout the life-course. Similar results have been reported across studies examining the relationship between CM and different types of victimization including violent victimization (Tillyer, 2012), victimization within intimate relationships (Widom et al., 2008), and peer victimization (Yoon et al., 2018).

Collective research generally supports the relationship between CM and later negative outcomes including delinquency and victimization (Malvaso et al., 2015). However debate still exists regarding the strength, directionality, and causality of this effect as well as the role that type, timing, frequency, and severity of CM has on delinquent and recurring victimization (Ahmadabadi et al., 2018; Finkelhor et al., 2009; Hurren et al.., 2017; Malvaso et al., 2015). In sum, CM most likely influences delinquency and victimization, however, the *process* and *effect* of this relationship is still unclear (Hurren et al., 2017; Malvaso et al., 2015).

It could be the case that individuals who experience CM and engage in delinquency or experience recurring victimization later in life grow up in dangerous environments, or in families, that are dysfunctional and violent (Finkelhor et al., 2009). At the same time, children and adolescents who experience CM may display specific emotional or behavioral problems that increase their likelihood of engaging in delinquent activities and increase their risk of victimization (Finkelhor et al., 2009). Most likely it is a combination of both of these processes, whereby families pass on traits and environments to children who then interactively evoke and engage with proximal and distal social networks that increase their likelihood of being exposed to CM, engage in delinquency, and experience victimization later in life (Pezzoli et al., 2019).

Further conflating the process between CM, delinquency, and victimization is the role that type, timing, frequency, and severity of CM exposure has on delinquency and victimization throughout the life cycle. Some studies find that specific types of CM, like sexual abuse (Yoon et al., 2018), directly increase the likelihood for experiencing sexual and physical peer victimization while other forms of CM, like physical abuse (Yoon et

al., 2018), indirectly influence later victimization experiences through other factors such as internalizing or externalizing behaviors. Similar results have been reported when examining different types of CM and later delinquent behavior (Hurren et al., 2017; Maas et al., 2008). For Instance, in a cross-sectional sample of 13,613 juvenile offenders in the U.S., van der Put and colleagues (2015) reported that those who experienced physical abuse were more likely to engage in increased violent offending behavior. Conversely, other studies suggest that type of CM matters less than the timing, frequency, and severity of CM (van der Put & de Ruiter, 2016; Widom et al., 2008). This work examines how multiple forms of co-occurring CM influence delinquency and victimization. Findings suggest that co-occurring, persistent, and severe CM perpetrated by family members on a child or adolescent exert the largest effect on risk for delinquency and victimization (Duke et al., 2009; Hamilton et al., 2002; Hurren et al., 2017; Ireland et al., 2002; Stewart et al., 2008).

Taken together, it seems that the phenomena in which CM exposure influences delinquency and recurrent victimization is a dynamic process (Hurren et al., 2017). Multiple forms of chronic and severe CM occurring or co-occurring at vulnerable periods during childhood or early adolescence may exert direct effects on delinquency and vulnerability to victimization (Malvaso et al., 2015). Alternatively, co-occurring, frequent, severe, and early exposure to CM may indirectly influence delinquency and victimization vulnerability through a host of other factors such as delinquent peers, failure to form school attachments, poor family dynamics, and increased likelihood of risk seeking and behavioral problems (Banny et al., 2013; Pezzoli et al., 2019; Tillyer, 2012). Thus, CM may have a weaker direct effect on delinquency and victimization

vulnerability but a stronger indirect effect through multiple other individual, family, and environmental factors (Tillyer, 2012).

An additional caveat to the process of CM on later delinquency and victimization comes from the study of *resilience* (Luthar, 2003). A growing body of work reports that the association between CM and later life delinquency and victimization is not ubiquitous. Research shows that some children who are exposed to maltreatment display positive adaptations and do not develop dysfunctional behaviors such as engaging in delinquency or experiencing victimization (Luthar & Eisenberg, 2017; Masten & Coastworth, 1998). This type of positive adaptation in response to adversity like CM is operationally referred to as resilience.

The term resilience has been the subject of much debate regarding definitional considerations (Klika & Herrenkohl, 2013; Walsh et al., 2010). Early work in the resiliency literature conceptualized resilience as a fixed personality trait used to capture positive functioning across domain specific (i.e., individual, educational, family, and community) outcomes (Klika & Herrenkohl, 2013; Luthar et al., 2000). As the study of resilience to CM grew, however, developmental and longitudinal studies found that resilience is a flexible, multidimensional, and active process that shows a considerable amount of variation across the life-course (Bowes & Jaffee, 2013; Ungar et al., 2013). Results from these studies revealed that resilience is not a static personality trait, but rather a dynamic, multidimensional, and time dependent process influenced by multiple factors that vary across individual, families, and environments and are associated with positive adaptation after exposure to CM across the life cycle (Cicchetti & Blender, 2006; DuMont et al., 2007; Lösel & Bender, 2014).

In this regard, children who experience maltreatment can be resilient in some aspects of their life, but not others (Maples et al., 2014). To illustrate, a child who experiences maltreatment may go on to achieve good grades in school, but also be the victim of bullying. Alternatively, some children who experience CM at age 10 may not engage in delinquent activity at ages 11, 12, or 13, but then do engage in delinquency at age 15. The observed variation in adaptive responses to CM across the life-course is influenced by factors that increase vulnerability to early adversity and protective (i.e., resilient) factors that mitigate early exposure to adversity (Pérez-González et al., 2017). Factors associated with both vulnerability and resilience to CM include personality traits, relationships, and personal experiences that vary across individuals, families, and communities/environments (Cicchetti & Toth, 2005; Domhardt et al., 2014; Hébert et al., 2014; Holmes et al., 2015; Vanderbilt-Adriance et al., 2015).

In sum, the process by which CM exposure influences delinquency and victimization is dynamic. The process incorporates both direct and indirect individual, family, and environmental pathways from CM to delinquency and victimization across developmental stages (Stewart et al., 2008). In other words, CM may directly increase an individual's likelihood for engaging in delinquency and being victimized (Duke et al., 2009). Alternatively, CM may also indirectly influence delinquency and victimization through an increased propensity for developing dysfunctional individual-level traits (i.e., personality/temperament), negative peer and family networks, and an increased propensity for seeking out harmful environments (Tillyer, 2012).

Factors that promote resilience within the CM, delinquency, and victimization process may counterbalance the negative effects of CM by providing a means with which

individuals can positively adapt in response to CM experiences. Factors promoting resilience also vary at the environmental-, family-, and individual-level (Cicchetti, 2013). Thus, there is a synergistic, integrative, and counterbalancing process where CM directly and indirectly influences delinquency and victimization. Indirect associations on these outcomes involve negative adaptations within individuals, families, and environments. At the same time, positively adaptive environmental, familial, and individual factors may mitigate the negative outcomes associated with CM to promote resilience resulting in a decreased likelihood of engaging in delinquency and experiencing victimization.

The ecological-transactional model provides a theoretical framework for explaining the balance between CM exposure, delinquency, and victimization, as well as resilience to the negative effects of CM in relation to later delinquency and victimization (Belsky, 1980; Cicchetti & Lynch, 1993; Cicchetti, 2013). Rather than focusing on the etiology of CM perse, Cicchetti and colleagues sought to create an integrative model explaining *outcomes* associated with CM. The ecological-transactional model, then, examines the how a confluence of factors at the individual-, family-, community-, and cultural-levels influence the process of developmental outcomes of children who experience CM. Importantly, the theoretical framework of the ecological-transactional model identifies various systems (i.e., the macrosystem, exosystem, and microsystem) that interactively effect the ontogenetic development of children exposed to maltreatment in relation to violence later in life (Cicchetti & Lynch, 1993).

Within the ecological-transactional model (Cicchetti & Blender, 2006; Cicchetti & Lynch, 1993), traits associated with vulnerability and resilience to CM at the individual-level include variance in genetic haplotypes, neurobiological structures, and

personality/cognition (Chen et al., 2020; Howell & Miller-Graff, 2014; Yule et al., 2019). Family-level traits include family cohesion, parental/caregiver support, family attachment, and socioeconomic status (Yule et al., 2019). Community and environmental factors associated with vulnerability and resilience to CM include friendship networks, community attachment, school achievement and attachment, neighborhood organization, and residential mobility (Newsome & Sullivan, 2014; Yoon et al., 2020). Together, these individual-, family-, and community-level factors work in dynamic and integrative processes to influence vulnerability and resilience to CM across the life-course (Cicchetti & Lynch, 1993; Ioannidis et al., 2020).

The fields of psychology, epidemiology, and public health have generated a considerable amount of research examining factors associated with vulnerability and resilience in response to exposure to CM and relevant outcomes (Feder et al., 2019; Hunter et al., 2018; Jaffee et al., 2007). The field of criminology, however, has only begun to examine factors associated with resilience (i.e., positive adaptation) to CM exposure and later delinquency and victimization in adolescence and adulthood (Jaffee et al, 2007; Lösel & Bender, 2014; Newsome & Sullivan, 2014; Newsome et al., 2015; Sullivan & Newsome, 2015). This is a noteworthy limitation to our field. Understanding factors that may mitigate the effect that exposure to CM has on delinquency and victimization is paramount to the development and implementation of intervention and prevention strategies that can promote factors associated with resilience in order to foster prosocial positive behavioral adaptations (Feder et al., 2019; Hunter et al., 2018; Liu et al., 2020; Luthar & Eisenberg, 2017; Masten & Coatsworth, 1998; Osório et al., 2016; Tabibnia, 2020; Ungar, 2013).

Criminology needs more studies examining factors that promote resilience to CM exposure in relation to criminological outcomes, such as delinquency and victimization. Only a handful of studies have examined factors that promote resilience to CM in relation to these outcomes (Jaffee et al, 2007; Lösel & Bender, 2014; Newsome & Sullivan, 2014; Newsome et al., 2015; Sullivan & Newsome, 2015). Collectively, and despite their limitations, these studies do converge onto three major findings that can help guide future research in this area.

The first major finding is that factors that promote vulnerability and resilience to CM and later life outcomes vary across individuals, families, and communities (Jaffee et al, 2007; Lösel & Bender, 2014; Thibodeau et al., 2015). These findings are consistent with the ecological-transactional model of developmental outcomes associated with CM (Cicchetti & Blender, 2006; Cicchetti & Curtis, 2006; Cicchetti & Lynch, 1993; Cicchetti & Rogosch, 1996; Cicchetti & Toch, 2004) and suggest that individual- (e.g., personality traits, cognition and intelligence, neurobiological structures, and genetics), family- (e.g., parental support, family support, and family cohesion), and community-level (e.g., neighborhood stability, neighborhood violence, and peer networks) traits/characteristics interactively increase resilience to CM on outcomes associated with violence, antisocial behavior, and criminal offending later in life. Interestingly, though it appears that individual-, family-, and community-level traits differentially influence resilience to experiences of CM, a recent series of meta-analyses suggest that individual-level traits including *depression*, *self-esteem*, and *cognition/intelligence* may exert the largest influence on behaviorally adaptive outcomes (Nasvytiené et al., 2012).

The finding that differences in individual-level traits may be one of the most robust predictors of outcomes associated with resilience to CM exposure must be taken into consideration with the second major finding from this body of work, which concerns the heritability of resilience. Studies employing behavioral genetic biometric analysis of monozygotic (MZ) and dizygotic (DZ) twins suggest that resilience is a partly heritable trait with roughly 38% - 70% of the observed variance in resilient functioning attributable to additive genetic effects (A), 0% - 23% of the variance conditioned by the shared (C) environment, and the remaining 25% - 48% of the variance accounted for by the non-shared (E) environment and measurement error (Amstadter et al., 2014; Boardman et al., 2008; Newsome & Sullivan, 2014; Newsome et al., 2015; Waaktaar & Torgersen, 2011; Wolf et al., 2018).

The third major finding from the resilience literature builds off the observation that resilience to early life adversity is partly heritable by attempting to identify specific genes, or candidate genes, that may interact with early life adverse experiences (e.g., CM) to predict later outcomes including delinquency and criminal behavior (Sullivan & Newsome, 2015). Candidate gene association studies (CGAS) add to our understanding of the integrative nature between individuals and their environments by identifying how specific genes interact within adverse environments (GxE) to influence outcomes associated with exposure to CM. From an ecological-transactional perspective (Cicchetti et al., 2011; Cicchetti & Rogosch, 2012), CGASs of resilience can be framed within the diathesis-stress and differential susceptibility (Belsky & Pluess, 2013) models and provide a basis for exploring how genes moderate the direct effect of CM on delinquency and victimization (Cicchetti & Curtis, 2006).

Candidate gene association studies offer a progressive step toward explaining variation in behavioral outcomes associated with CM, however, there are some notable limitations to the CGAS approach (Elbau et al., 2019; Border & Keller, 2017; Duncan & Keller, 2011; Keller, 2014). By nature of the design, CGASs attempt to identify specific (i.e., single or only a few) genetic polymorphisms that are most likely to influence the neurobiological stress response systems. These stress sensitization genes typically involve the regulation of monoamine oxidase A (MAOA), dopamine (DA), serotonin (5-HT), and oxytocin (OXT). Often, CGASs explore the interaction between one genetic polymorphism and an environmental stressor to test the association between these GxE interactions in relation to a specific outcome of interest (e.g., Caspi et al., 2002). While this approach has been informative, it is limited in the sense that human behavior is complex, multifaceted, and is typically the resultant product of multiple genes exerting small effects on behavior over time (Belsky & Pluess, 2013). This concept, which is the Fourth Law of behavior genetics (Chabris et al., 2015; Plomin & Deary, 2015; Turkheimer, 2000), demonstrates the limited utility of CGASs. Indeed, candidate gene studies have received a substantive amount of criticism over the past decade (Elbau et al., 2019; Border & Keller, 2017; Duncan & Keller, 2011; Keller, 2014).

Technological innovations, reductions in the cost of genotyping, and a recognition of the limitations to the candidate gene approach prompted human molecular and quantitative genetic researchers to start using genome wide association studies (GWAS) to develop polygenic scores (PGSs) that capture the full range of variability in genes that each have a small effect on behavioral adaptations. Applied to the study of resilience from an ecological-transactional perspective, then, PGSs can potentially address the

inherent limitations to heritability and candidate gene studies by offering a more complete picture of additive genetic mechanisms involved in explaining individual-level genetic differences in resilient adaptation to adversity (Cicchetti & Toth, 2005). To date, the GWAS PGS design has not been applied to the study of resilience to CM exposure and outcomes associated with delinquency and victimization in adolescence and young adulthood.

Goals of the Current Dissertation

Exposure to CM is associated with a range of dysfunctional psychopathological and health adaptations throughout the life-course. Two of the most prominent negative behavioral outcomes associated with CM in the field of criminology are delinquency and victimization. However, not everyone who experiences CM goes on to engage in delinquency and experience recurring victimization; some individuals show resilience in response to CM and engage in little to no delinquency and experience few if any instances of victimization. Resilience to CM differs across individuals through traits and characteristics that vary at the individual-, family-, and environmental-level (Yoon et al., 2020). Thus, consistent with the ecological-transactional model (Cicchetti, 2013), individual differences in genes, neurobiology, personality/cognition, family and peer dynamics, and community and neighborhood structure influence the onset and development of resiliency in response to adversity.

Few studies have considered biological and genetic factors that promote resilience in response to exposure to CM in relation to outcomes associated with delinquency and victimization in adolescence and young adulthood. Furthermore, no study has examined the role that PGSs may have in operating as sources of individual-level genetic resilience

between CM exposure and decreases in delinquency and victimization experiences in adolescence and adulthood. In order to address these limitations and add to our understanding of the biological factors that may promote resilience to CM, the current dissertation examines how individual differences in PGSs moderate the relationship between CM exposure and outcomes associated with delinquency and victimization from adolescence to young adulthood. The specific PGSs examined as moderators in this study include PGSs for *major depression*, *extraversion*, and *educational attainment*.

These PGSs were chosen as potential genetic resilience promoting factors because several individual studies (Bolger & Patterson, 2003; Fritz et al., 2018; Jaffee, 2017; Maples et al., 2014; Zingraff et al., 1994) and a recent meta-analysis (Nasvytiené et al., 2012) identify depression, self-esteem, and intelligence/school performance as sources of individual-level trait specific measures that promote resilience to delinquency and victimization. In other words, when measured as a trait, research suggests that individuals with depression, lower self-esteem, and a lower ability toward problem-solving, cognition, and school performance are more vulnerable to engaging in higher levels of delinquency and experiencing more instances of victimization throughout the life-course (cite). The corollary of these trait-based continuums suggest that individuals presenting lower levels of depression, higher self-esteem, and an increased propensity towards problem-solving, cognitive ability, and educational attainment are less likely to engage in delinquency and experiences recurring victimization (Bolger & Patterson, 2003; Fritz et al., 2018; Jaffee, 2017; Maples et al., 2014; Nasvytiené et al., 2012; Zingraff et al., 1994). Of course, these studies are measuring depression, self-esteem, and educational attainment as traits, not polygenic scores. As such, there is a need to examine how genetic

load for developing these traits may act as genetic resilience promoting factors counterbalancing the negative effects of CM.

The current dissertation proceeds with two goals in mind. The first goal is to add to the literature on the ecological-transactional model to elucidate the individual-, familial-, and environmental-level processes associated with response to exposure to CM and later delinquent behavior and victimization. A longitudinal perspective is adopted in order to examine the time-dependent and dynamic nature of resilience in relation to positive adaptations following exposure to CM while accounting for genetics, personality traits, family dynamics, and neighborhood/environmental characteristics.

The second goal is to expand upon previous molecular genetic studies by adopting a moderation PGS design to assesses the moderating effect that three PGSs representing variation in the genetic development of depression, educational attainment, and extraversion have on the observed relationship between CM, delinquent behavior, and victimization. These three PGSs were chosen based on findings from meta-analysis, reviews, and empirical analyses assessing general resilience following exposure to CM (Bolger & Patterson, 2003; Fritz et al., 2018; Jaffee, 2017; Maples et al., 2014; Nasvytiené et al., 2012; Zingraff et al., 1994). Collectively, it appears that the individual-level cognitive personality traits involving depression, intelligence, and self-esteem exert the largest effect on general resilience following CM. As such, corollary PGSs of these traits (i.e., PGSs of depression, educational attainment, and extraversion) were selected for further analyses when considering resilient functioning to CM on outcomes associated with delinquency and victimization.

The major contribution of this dissertation is the addition of longitudinal quantitative genetic analyses examining individual differences in PGSs that promote resilience in response to exposure to CM and outcomes involving delinquency and victimization in adolescence and young adulthood. To this end, data from the first three waves of the National Longitudinal Study of Adolescent to Adult Health (Add Health) are analyzed. Results from this dissertation can help inform intervention and prevention strategies aimed at understanding factors that promote resilience from a multi-level and multisystemic biopsychosocial ecological-transactional approach.

Organization

This dissertation is divided into four main sections. Chapter II provides an overview of the current literature relevant to this dissertation. This chapter is divided into five subsections. The first subsection discusses the relationship between exposure to CM and delinquency and victimization in adolescence and young adulthood. This section pays attention to the fact that not all who are exposed to CM develop dysfunctional behavioral adaptations associated with delinquency and victimization. The next section builds from this observed variation by introducing the concept of resilience. Resilience is a complex construct and, therefore, requires a substantive overview of the definitional considerations of what constitutes resiliency following adversity. This section also adopts an operational definition of resilience that will be used throughout the remainder of this dissertation. The next section introduces individual-level traits associated with resilient functioning following CM exposure. Pertinent to this discussion are studies observing individual differences in traits associated with one's genotype, personality, and cognition in accordance with resilient functioning following adversity. This section also focuses on

limitations to current molecular genetic studies (i.e., candidate gene studies) and introduces the concept of GWAS and PGSs. Chapter II concludes with a summary of the literature and a presentation of the research questions for the current dissertation.

Chapter III provides a methodological overview of this dissertation. This includes a discussion of the Add Health sampling procedure as well as the construction of PGSs using the subsample of participants who provided genetic material at Wave IV. This section also provides an overview of the construction of the independent variable (i.e., childhood maltreatment) and the dependent variables including delinquency and victimization in adolescence and young adulthood. The individual-, family-, and neighborhood-level covariates are also discussed. This section concludes by providing an overview of the analytic procedure focusing on longitudinal analyses.

Chapter IV provides an overview of the analysis and results used to answer the research questions and hypotheses.

Chapter V discusses the findings and frames them within the context of the ecological-transactional, diathesis-stress, and differential susceptibility framework. This section also discusses limitations and directions for future research.

CHAPTER II

Literature Review

Exposure to CM and Later Life Delinquency and Victimization

Childhood maltreatment is one of the most serious forms of adverse childhood experiences (ACEs). Childhood maltreatment includes exposure to one or more forms of abuse and/or neglect. Abuse includes actions deliberately done to the child and can be sexual, physical, emotional, or psychological (Azar, 2002; Sedlak et al., 2010). Examples of sexual abuse are assault by penetration, non-penetrative sexual activities, watching or having a child watch others perform sexual acts, forcing a child to undress, encouraging sexually inappropriate behavior of a child, or grooming. Physical abuse involves instances of poisoning, hitting, biting, throwing, shaking, slapping, physical harm, and burning a child. Examples of emotional and psychological abuse includes ridiculing or silencing a child, shouting and threatening them, mocking them, limiting physical contact, preventing social interaction with peers, bullying, or engaging in emotional blackmail (Azar, 2002, Sedlack et al., 2010).

Neglect is the most common form of CM (Finkelhor et al., 2020) and involves the failure to meet a child's basic needs (Azar, 2002; Sedlack et al., 2010). There are several types of neglect including physical neglect, emotional neglect, educational neglect, and medical neglect. Physical neglect is a consistent pattern or failure to meet a child's basic physical needs such as providing food, shelter, and clothing. Emotional neglect involves a failure of or deprivation towards meeting a child's emotional needs such as allowing them to form positive attachments with adults. A failure to provide access to education is

considered educational neglect while a failure to provide adequate medical care is a representation of medical neglect (Barnett et al., 2005).

Reports by the U.S. Department of Health and Human Services (2018) National Child Abuse and Neglect Data Reporting System (NCANDS) indicate that there were an estimated 678,000 victims of child abuse and neglect in 2018. This equates to a rate of roughly 9.2 victims per 1,000 children in the US population (U.S. Department of Health and Human Services, 2018). Most maltreated children (~84.5%) are exposed to one form of CM with the remaining ~15.5% exposed to multiple forms of CM (Finkelhor et al., 2020; U.S. Department of Health and Human Services, 2018). Additionally, a large portion of children exposed to CM in 2018 were victims of neglect (~60.8%). Approximately 1,770 children who experienced one or more forms of CM died from their abuse (U.S. Department of Health and Human Services, 2018). The majority of CM perpetrators were parents (~77.5%). Most CM perpetrators were between the ages of 18 and 44 (~83.3%) and were female (~53.8%) relative to male (~45.3%).

Demographic characteristics linked to variation in CM experiences include gender, race/ethnicity, and age. Generally, males and females are equally likely to experience most forms of CM (Sedlack et al., 2010), however, females are more likely to experience sexual abuse compared to males (U.S. Department of Health and Human Services, 2018). Race and ethnicity are also important demographic characteristics to consider in relation to CM rates. According to the U.S. Department of Health and Human Services (2018), White, Hispanic, and Black children had the highest reported numbers of substantiated cases of CM in 2018. Black (14.0) and American Indian (15.2) children had the highest rates of maltreatment per 1,000 children. Children under the age of 1 are

the most vulnerable neglect and physical maltreatment with a substantial drop off in CM exposure in older children (U.S. Department of Health and Human Services, 2018).

U.S. National estimates identify that CM is a serious and pervasive form of victimization that results in harm and/or the death of a child (Finkelhor et al., 2020; U.S. Department of Health and Human Services, 2018).[1] What these national trends fail to capture, however, is the human cost of CM. Studies across multiple fields in the social and behavioral sciences find that children who are exposed to CM are more likely to develop dysfunctional psychopathological and health adaptations throughout the life course (Aas et al., 2020).

These negative behavioral and health adaptations include a range of psychological, emotional, and physical disorders (Ioannidis et al., 2020) and often result in the loss of an individual's social, economic, and psychophysiological capital (Leung et al., 2020; Peterson et al., 2018). The most common negative developmental outcomes associated with CM across psychopathological domains include depressive symptomatology (Sheerin et al., 2018), lower cognition and educational attainment (Breslau et al., 2013), decreased self-esteem (Bolger & Patterson, 2003), decreases in inhibitory control and working memory (Cowell et al., 2015), and post-traumatic stress disorder (Scoglio et al., 2019). The onset and growth of these negative developmental adaptations in response to CM exposure is highly dependent upon the type, timing, frequency, and severity of CM (Cowell et al., 2015; Morris et al., 2019). Oshri and colleagues (2018) explore this "developmental cascade" effect of type, timing, frequency,

[1] Official reported statistics like the NCANDS may be underestimating the true extent of abuse and neglect because of issues with reporting among children who cannot verbalize the abuse/neglect they are experiencing. Additionally, children may be deterred from or threatened by family members when reporting instances of CM.

and severity of exposure to CM in a sample of 1,461 individuals who were referred to child protective services (CPS). Using a Growth Mixture Modeling (GMM) approach to identifying individual differences in trajectories of CM exposure, they found that individuals who experienced more forms of trauma and increased harsh parental discipline were less likely to display positive adaptations regarding future orientation and success in the transition into adolescence and young adulthood. These results were also supported in cross-sectional analysis of 136 maltreated youth in relation to decreased working memory performance and inhibitory control in individuals exposed to earlier and more chronic forms of CM (Cowell et al., 2015). Taken together, it appears that adaptive behavioral and psychopathological responses to CM are highly variable. Individual differences in type, timing, frequency, and severity of CM exposure largely influence developmental outcomes in relation to dysfunctional psychopathological adaptations (Jaffee et al., 2017).

These findings can be extended beyond the field of psychology and public health and into the field of criminology in order to elucidate the process in which CM influences delinquency and victimization in adolescence and young adulthood (Beaver, 2008). Decades of research have observed associations between exposure to CM and delinquency and victimization later in life (Banny et al., 2013; Barnes et al., 2009; Beaver et al., 2010; Connolly et al., 2015; Daigle et al., 2008; DeLisi et al., 2010; Duke et al., 2009; Fagan, 2005; Finkelhor et al., 2009; Hamilton et al., 2002; Hurren et al., 2017; Ireland et al., 2002; Maas et al., 2008; Malvaso et al., 2015; Pezzoli et al., 2019; Reckdenwald et al., 2013; Stewart et al., 2008; Thornberry et al., 2010; Tillyer et al., 2012; van der Put & de Ruiter, 2016; Widom & Brzustowicz, 2006; Widom et al., 2008;

Wright & Fagan, 2013; Yoon et al., 2018). In one of the largest studies to date, Duke and colleagues (2009) used a nationally representative sample of 136,549 participants to examine the longitudinal relationship between CM and violence perpetration in adolescence. They found a robust link between physical abuse, sexual abuse by a family member, and sexual abuse by a non-family member in relation to increases in delinquent behavior, bullying, physical fighting, and dating violence in boys and girls. This finding is substantiated by two systematic reviews reporting consistent associations between CM and delinquency across 62 (Malvaso et al., 2015) and 12 (Maas et al., 2008) studies.

Studies examining the relationship between CM and victimization report results consistent with the CM and delinquency literature. Yoon et al. (2018) conducted a longitudinal study of 798 children examining the effects of physical and sexual abuse on adolescent physical and sexual peer victimization. Their results supported a direct link between sexual abuse and physical and sexual peer victimization. Physical abuse, on the other hand, was indirectly associated with both forms of peer victimization through individual and peer indicators. This is consistent with a cross-sectional study by Widom et al. (2008) who, in a sample of 892 children, found that those reporting a co-occurrence of multiple forms of maltreatment were more likely to experience increases in one time and recurrent forms of interpersonal victimization including physical and sexual assault as well as kidnapping and stalking.

The literature supports an association between CM and delinquency and victimization later in life (Malvaso et al., 2015). Where much uncertainty remains in the literature, however, is with the *process* by which CM influences delinquency and victimization across the life-course (Finkelhor et al., 2009) as well as the extent of the

effect that CM has on delinquency and victimization. Put differently, there is still much debate surrounding the direction, strength, and causal association between CM, delinquency, and victimization. Additionally, research needs to further consider how type, timing, frequency, and severity effects the process in which CM influences delinquency and victimization (Ahmadabadi et al., 2018).

Regarding the former process involving directionality, strength, and causality, studies assessing the relationship between CM, delinquency, and victimization that account for relevant covariates that vary at the individual-, familial-, and environmental-level find that the association between CM, delinquency, and victimization is rendered null once these covariates are included in the model (Malvaso et al., 2015; Tillyer, 2012). For example, Tillyer (2012) used a sample of 2,762 participants from the Add Health to examine if retrospective reports of maltreatment during childhood were associated with increased violent victimization during adolescence. Before accounting for individual, family, and environmental covariates, Tillyer (2012) reported a direct effect of CM on victimization in adolescence. After controlling for psychological vulnerability, self-esteem, delinquent peer association, and school truancy, Tyllier (2012) found that the direct effect of CM on victimization in adolescence was rendered non-significant. Instead, CM was found to indirectly influence increases in psychological vulnerability, low self-esteem, delinquent peer association, and school truancy which directly increased violent victimization later in life. Similar results were reported by Yoon et al. (2018) in a longitudinal sample of 798 children. Sexual abuse experiences directly increased physical and sexual abuse by peers while exposure to childhood physical abuse increased

internalizing and externalizing behaviors, which increased delinquent peer association, which increased sexual and physical victimization by peers.

These findings might be attributable to three effects identified by Finkelhor et al. (2009). The First effect is that children who experience maltreatment may be reared in dangerous environments. Second, that children who experience CM live in families that engage in victimization and violence. And third, that children who have behavioral or emotional problems may be more likely to experience CM as well as engage in delinquency and experience recurrent victimization.

The processes Finkelhor and colleagues (2009) are describing are more broadly conceptualized as gene-environment correlations (*r*GE) (Plomin et al., 1977; Pluess & Belsky, 2010) and specify how genes and environments that are shared among individuals and families can effect similar pathways to delinquency and victimization. It could be that parents pass down genes and expose children to environments that increase their likelihood for engaging in delinquency of experiencing victimization later in life (i.e., passive *r*GE). Within the context of this study, inherited genetic load for educational attainment could influence both delinquency and victimization, where those with a propensity for educational attainment may have a decreased susceptibility to engaging in delinquency and experiencing victimization within vulnerable environments. Genes and environments could also be functioning as an active *r*GE process where children actively seek out specific environments based on their genetic makeup. Individuals who are genetically susceptible to developing depression, for example, may seek out environments based on their behavior that increases their likelihood of experiencing victimization. Finally, children with genetic traits might evoke (i.e., evocative *r*GE)

responses from their environment. Children with a genetic likelihood for extraversion, for instance, might elicit responses from prosocial or promotive social networks to keep them from engaging in delinquency or experiencing victimization.

Theoretically, the pathways to delinquency and victimization may operate on the same continuum where genes and environments interactively influence the process by which individuals engage in delinquency and experience victimization. This cross-level interaction or correlation between genes and environments is consistent with the ecological-transactional model (Cicchetti & Toth, 2005) and with the literature documenting a significant amount of overlap in delinquent activity and victimization experiences (Widom, 1989). Theoretically then, the process for delinquency and victimization from an ecological-transactional biopsychosocial perspective may be the same.

Type, timing, frequency, and severity of CM have also been found to interactively influence delinquency and victimization later in life (Hurren et al., 2017; Stewart et al., 2008). Barnes et al. (2008), for instance, found that women who experienced childhood sexual abuse were more likely to report being sexually and physically revictimized in adulthood. Hamilton and colleagues (2002), on the other hand, found that recurrent maltreatment in childhood was associated with increased offending net of the type. Hurren et al. (2017) found, in a sample of 4,511 participants, that individuals who reported chronic and frequent maltreatment were more likely to offend. These results are consistent with Ireland et al. (2002) and Stewart et al. (2008). Largely, it appears that more frequent and severe exposure to co-occurring types of CM at developmentally sensitive periods of life (or turning points; Laub & Sampson, 2003) will exert both direct

and indirect influences on delinquency and victimization (Stewart et al., 2008). When considering the indirect influences that CM has on delinquency and victimization, it seems that CM increases the development of dysfunctional personality traits, negative peer and familial relationships, and increase exposure to vulnerable environments that increase one's propensity for both engaging in delinquency and experiencing victimization (Finkelhor et al., 2009; Pezzoli et al., 2019).

Exposure to co-occurring, frequent, and severe forms of CM at developmentally sensitive times in the life cycle can have both direct and indirect effects on delinquency and vulnerability for victimization. CM, then, influences developmentally relevant individual-, family-, and environmental-level interactions that occur in a dynamic process to increase the likelihood of engaging and delinquency and experiencing victimization (Cicchetti, 2013). Within this context, the ecological-transactional model provides a theoretical basis for explaining the observed relationship between CM exposure and later life delinquency and victimization.

Similar to Belsky's (1980) ecological model of the etiology of CM, Cicchetti and colleagues (see Cicchetti & Lynch, 1993; Cicchetti & Toth, 2004; Cicchetti, 2013) sought to examine CM from an ecological and transactional perspective. Compared to Belsky's (1980) work, however, Cicchetti and Lynch (1993) did not set out to explain the etiology of CM, rather, they wanted to explain developmental outcomes in individuals exposed to CM. In other words, they adopted an ecological-transactional approach to explain variation in outcomes associated with those who were exposed to CM. Specifically, they were interested in how community violence (i.e., environmental adversity) and CM

interact to influence individual variation in developmental outcomes throughout the life course (Cicchetti & Lynch, 1993).

There are four central system wide components to the ecological-transactional model (Cicchetti & Lynch, 1993). These include the macrosystem, exosystem, microsystem, and ontogenic development. From their perspective (Cicchetti & Lynch, 1993), the macrosystem operates at the broadest conceptual environmental level and includes culture, beliefs, and traditions that exist within families and communities. The exosystem is nested within the macrosystem and includes formal and informal structures (e.g., neighborhood interactions, social networks, availability of services, employment) that impact a person's proximal environment. Nested within both of these constructs in the microsystem. The microsystem represents the immediate family environment and includes interactions with parents, caregivers, relatives, and siblings. The ontogenic level includes individual level functioning such as the development of personality traits, attachment styles, and emotions (Cicchetti & Lynch, 1993).

Collectively, the ecological-transactional model represents a nested systems perspective where each level operates in cooperation and cross-talk with every other level. Stated differently, no one system (i.e., the macro-, exo-, micro-, or ontogenic-system) operates in seclusion of the other systems. Developmental outcomes in response to CM, then, are conditioned by factors that indirectly, directly, and interactively influence each other within and across systems over the life course. A noteworthy aspect of the ecological-transactional perspective is the recognition that human behavior is influenced across multiple individual and environmental levels. Later sections expand upon the ecological-transactional perspective and connect that model to more recent

conceptualizations of bottom-up and top-down processes that operate similar to the systems outlined by Cicchetti and Lynch (1993) and can better explicate the relationship between CM, delinquency, and victimization.

In sum, a convergence of findings from the literature on CM indicates that exposure to CM conditions negative behavioral adaptations throughout the life cycle. CM, then, can be conceptualized as an early life environmental factor that confers vulnerability to maladaptive behaviors later in life. Individuals exposed to co-occurring, frequent, and severe CM at developmentally sensitive periods are more likely to develop a range of dysfunctional personality and cognitive traits as well as negative peer, family, and environmental networks that increases susceptibility to delinquent behavior and interpersonal victimization. Alternatively, research also finds that not all individuals who are exposed to CM go on to engage in delinquency and experience victimization. In fact, many victims of CM go on to lead prosocial and healthy lives despite being exposed to co-occurring, frequent, and severe abuse. The observed variation in these outcomes suggests that individual differences exist in relation to susceptibility to the negative effects of CM. This concept, otherwise known as resilience, is explored in more detail in the next section.

Definitional Considerations of Resilience

In order to operationalize the process of resilience it is important to understand the concept of resilience from a historical perspective. Resilience was first observed by Garmezy (1974) and colleagues (see Cicchetti & Garmezy, 1993; Cicchetti, 2013) while examining the effect that early experiences of childhood stress had on the development and maintenance of psychopathology (e.g., schizophrenia and other mental health

disorders). Garmezy (1974) noticed that some children who experienced early stressors positively adapted to these experiences and developed no dysfunctional psychopathological profiles. In fact, many children who experienced early life trauma and stress went on to lead healthy lives with no instances of psychopathological maladaptation, thus, the abstract idea of resilience was born (Luthar, 2003; Masten & Powell, 1999; Luthar, 2003).

Following Garmezy's (1974) work, several researchers began examining psychological resilience following adversity (Cicchetti & Blender, 2006; Cicchetti & Curtis, 2006; Cicchetti & Garmezy, 1993; Cicchetti & Lynch, 1993; Cicchetti & Rogosch, 1996; Cicchetti, 2013; Kim & Cicchetti, 2003; Luthar & Cicchetti, 2000; Masten & Cicchetti, 2010; Masten & Tellgen, 2012) . These studies assessed several domain specific criteria involving early life stress while examining outcomes associated with maladaptive psychopathological dysfunction (Luthar, 2003). From this line of work emerged the static, or deterministic, concept of resilience (Luthar, 2003). This perspective posited that resilience was a fixed unidimensional personality trait that ensured optimal psychological adaptation in response to changing contextual circumstances or in resistance to the effects of adverse psychophysiological experiences (Nasvytiené et al., 2012).

The operationalization of resilience as a fixed, or static, personality trait was subject to much debate and criticism and ultimately abandoned with the advancement of research using longitudinal developmental contextual analyses that reported a significant amount of change in resilience in children exposed to adversity over time (Luthar, 2003; Nasvytiené et al., 2012; Walsh, 2010). Growing work in the area of childhood

development from a longitudinal perspective found that resilience was not a deterministic psychological personality trait (Cicchetti & Garmezy, 1993; Ben-David & Jonson-Reid, 2017; Luthar & Cicchetti, 2000). Rather, resilience was better conceptualized as a multidimensional and multi-determined process that influenced positive adaptive functioning at specific points during the life course (Cicchetti & Curtis, 2006; Fritz et al., 2018b; Kilka & Herrenkohl, 2013). Resilience to early adversity was found to vary over time and across developmental phases as a "complex dynamic system" that changed in response to fluctuations across resources and factors that increased vulnerability to context dependent environmental stressors (Cicchetti & Rogosch, 1996; Cicchetti, 2013; Ioannidis et al., 2020; Oshri et al., 2013; Ungar et al., 2013). Accordingly, children exposed to environmental adversity could be resilient in some aspects or points in time in their lives, but not others.

Framed this way, the concept of resilience has been further studied and found to account for variation in exposure to differing levels of environmental stress (Bennett et al., 2018 Collishaw et al., 2007). As discussed earlier, ACEs vary in the type, timing, frequency, and severity of exposure (Moreno-López et a., 2020) with co-occurring, frequent, and severe ACEs occurring at sensitive developmental periods resulting in an increased chance of developing maladaptive psychopathological disorders (Kalisch et al., 2017; Kelifa et al., 2020). This observed "dose response" stress sensitization effect was used to distinguish between two forms of resilience, namely, *emergent resilience* and *minimal-impact resilience* (Bonanno & Diminich, 2013; Bonanno, 2004). The former refers to positive adaptations in response to chronic early adversity and the latter involves

an immediate negative adaptational response to a less severe/frequent adversity followed by positive adaptation at an undetermined period later in life (Bennett et al., 2018).

Within the context of the current dissertation, CM is the equivalent of an early environmental adversity. As discussed in the previous section, exposure to CM is associated with dysfunctional psychopathological and criminal adaptations throughout the life course (Cichetti et al., 2011). Specifically, experiencing co-occurring, severe, and frequent CM at key points in the life-cycle is associated increased delinquency and victimization (Sullivan & Newsome, 2015). Clearly, exposure to CM can "get under the skin" (Chen et al., 2020) and influence delinquency and victimization later in life. However, not everyone exposed to CM will negatively adapt to these experiences (Choi et al., 2019). This suggests that resilience can be "skin deep" (Chen et al., 2020) and promote positive adaptation in the face of environmental adversity.

The process by which CM "gets under the skin" and resilience is "skin deep" are conceptually two different sides of the same ecological-transactional coin. As discussed previously, CM has direct effects on delinquency and victimization (Malvaso et al., 2015). At the same time, however, CM can indirectly increase delinquency and victimization through an ecological-transactional processes in which CM increases dysfunctional individual-, family-, and environmental-level processes (or systems) that each have small effects on the likelihood of engaging in delinquency or experiencing recurrent victimization (von Stumm & d'Apice, 2021). These dysfunctional behavioral outcomes are influenced by CM which increases negative adaptative outcomes of delinquency and victimization. Resilience, on the other hand, is "skin deep" in the sense that it represents the positive aspects of the ecological-transactional components in which

the environment, family, and individual traits promote positive adaptation in response to CM exposure thus decreasing the likelihood of engaging in delinquency or experiencing victimization (Cicchetti, 2013).

Though operationally defining resilience to CM can be difficult (Yoon et al., 2019), it is not impossible and must be done while considering relevant recommendations from the previous literature (Cicchetti & Garmezy, 1993; Luthar & Cicchetti, 2000; Luthar et al., 2000). Thus, this dissertation adopts Luthar et al.'s (2000) more modern definition of resilience as a *dynamic process encompassing positive adaptation within the context of significant adversity*. Significant adversity is exposure to CM. Positive adaptation will be evaluated as a decreased likelihood of engaging in delinquency and experiencing victimization following exposure to CM. Polygenic scores are the main factor promoting resilience. This dissertations tests how PGSs for depression, extraversion, and educational attainment promote positive adaptation as measured by decreased delinquency and victimization in response to retrospective accounts of early life CM. Therefore, individual differences in resilience will be assessed in two ways: first, as a quantitative genetic factor of resilience captured by PGSs, and second as a decrease in delinquency and victimization. PGS represent the biological process in which resilience to CM is achieved. This process is explored in more detail in the next section.

Individual Differences in Factors that Influence Resilience to Childhood Maltreatment

The ecological-transactional model is an integrative theoretical perspective that is uniquely situated to explain inter-individual variability in resilience following exposure to CM (Cicchetti & Blender, 2006; Cicchetti & Curtis, 2006; Cicchetti & Garmezy, 1993;

Cicchetti & Lynch, 1993; Cicchetti & Rogosch, 1996; Cicchetti, 2013; Luthar & Cicchetti, 2000; Masten & Tellegen 2012). As a reminder, the ecological-transactional model suggests that individual ontegenic developmental outcomes, such as resilience to adversity, are influenced by factors that vary and interact across micr-, macro-, and exo-systems (Cichetti & Lynch, 1993). Factors that vary among individuals involving genes (Aas et al., 2020; Cicchetti & Rogosch, 2012), neurobiological structures (Cicchetti et al., 2011; Osório et al., 2017), personality traits (Collishaw et al., 2007. Pérez-González et al., 2017) as well as family (Malvaso et al., 2016; Meng et al., 2018) and community/environmental (Fritz et al., 2018b; Masten & Cicchetti, 2010) characteristics can promote positive adaptation (i.e., resilience) in response to stress.

A hallmark of the ecological-transactional model is the recognition that resilience occurs as a process across multiple systems. These systems are providing constant feedback that reorganizes the function of genetic and neurobiological structures in response to environmental exposures that influences behaviorally adaptive outcomes (Cicchetti & Curtis, 2006). The ecological-transactional model (see Cicchetti & Lynch, 1993), as well as similar integrative models developed around the same time (see Belsky, 1980; Bronfenbrenner, 1992), operationalized these systems as an exo-system, meso-system, and micro-system. These conceptually operationalized definitions provided the basis of understanding how various processes across multiple-levels act as mitigating factors that condition individual differences in response to CM exposure. Recently, these system-specific definitional terms have been replaced with more current terminology to specify the genetic, neurobiological, familial, and environmental factors that interactively influence resilience to CM. More specifically, the exo-, macro-, and micro-system have

re-conceptualized as bottom-up (i.e., genes to environment) and top-down (i.e., environment to genes) systems (Ioannidis et al., 2020). Collectively, these bottom-up and top-down systems affect resilience in response to stress through time varying interactive processes otherwise referred to as interactive feedback loops (Ioannidis et al., 2020) or dynamic feedback systems (Cicchetti & Curtis, 2006).

Bottom-up systems (see Figure 1) involve the genetic regulation of the neurobiological stress response system which influences variation in personality traits that condition interactions between the immediate (e.g., friends and family) and ecological environment (e.g., community cohesion/support and cultural contextual effects). Conversely, top-down processes (see Figure 1) specify the way in which distal and proximal environments (e.g., friends, communities, cultures, and social services) influence individual differences in perceptions of these environments, which convey information to neurobiological structures that can alter gene expression (Ioannidis et al., 2020).

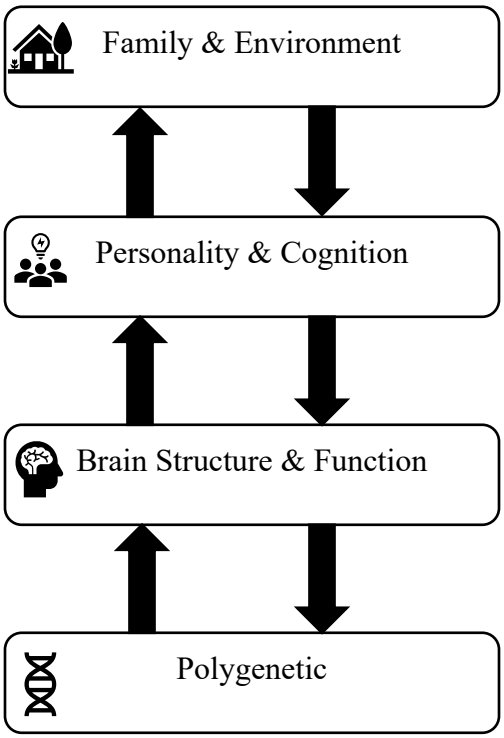
Together, bottom-up biological processes and top-down environmental/contextual exposures influence individual differences in negative and positive adaptation in a cyclical and time-dependent nature. Individuals who are more resilient to the effects of CM, therefore, are more likely to be carriers of genetic variants that decrease susceptibility of neurobiological stress response and increase positively adaptive psychological traits that foster constructive responses to distal and proximal stressors. Alternatively, positive distal and proximal familial and environmental stimuli may enhance positively adaptive personality traits, which then increases normative functionality of the neurobiological stress system which decreases the likelihood of

developing potentially negative behavioral psychopathological adaptations in response to early life CM.

Two concepts that are related to the bottom-up and top-down interactive process within the ecological-transactional model are the diathesis-stress and differential susceptibly biological moderation processes used to contextualize behavioral adaptations to early adversity (Belsky & Pluess, 2013; Elbau et al., 2019). The diathesis-stress model suggests that genetic susceptibility determines differences in an individual's threshold to adversity whereas the differential susceptibility framework posits that genotypes are relatively plastic and increase susceptibility to environmental adversity on a continuum rather than as a fixed indicator (Belsky & Pluess, 2013; Elbau et al., 2019). These two processes demonstrate that genes and the environment interact (GxE interaction) to influence individual behavioral outcomes. Clearly, the diathesis-stress and differential susceptibility models represent two competing explanations of the bottom-up and top-down GxE interactions between biological processes and environmental adversities (Masten & Tellegen, 2012). This dissertation will test the evidence for either the diathesis-stress or differential susceptibility GxE model in relation to outcomes associated with delinquency and victimization in those exposed to CM who display polygenetic profiles for the development of depression, extraversion, and educational attainment.

Figure 1

Bottom-up and Top-Down Processes



Note: Arrows pointing up represent bottom-up processes moving from polygenetic variation to family and environmental influences; Arrows pointing down represent top-down processes moving from family and environmental influences to polygenetic variation.

Factors associated with bottom-up and top-down systems have been applied to better understand resilient adaptations to CM in relation to outcomes associated with depression (Starr et al., 2020), bipolar disorder (Aas et al., 2020), obesity (Opel et al., 2019), educational attainment (Goltermann et al., 2020), cognition (Samplin et al., 2013), and familial/peer relationships (Azeredo et al., 2019). These include factors associated with individual differences in genetic expression (Amstadter et al., 2014; Belsky & Pluess, 2013), neurobiological structures (Bennett et al., 2018), personality/cognitive traits (Ben-David & Jonson-Reid, 2017), families (Dohmhardt et al., 2015), and communities (DuMont et al., 2007). Few studies, have examined bottom-up and top-

down systems that influence resilience in response to CM exposure and later delinquency and victimization. This is a notable limitation to the field of criminology. As such, this dissertation examines the polygenetic processes that promote resilience to CM exposure and delinquency and victimization in adolescence and young adulthood. The polygenetic factors expected to promote resilience to CM on delinquency and victimization includes PGSs capturing susceptibility to the development of depression, extraversion, and educational attainment. Thus, resilience promoting PGSs of depression, extraversion, and educational attainment are expected to moderate the relationship between CM and delinquency/victimization in adolescence and young adulthood. Additionally, these PGSs represent individual-level biological traits within the ecological-transactional model.

These three PGSs were selected given results from studies indicating that individual-level traits (i.e., personality) including depression, self-esteem, and cognition/intelligence may exert the largest effect on resilience in response to adversity in relation to antisocial and violent behavior (Bolger & Patterson, 2003; Fritz et al., 2018; Jaffee, 2017; Maples et al., 2014; Nasvytiené et al., 2012; Zingraff et al., 1994). This is also consistent with previous research suggesting that cognition/intellectual skills (Cicchetti & Curtis, 2006), depression (Masten & Tellegen, 2012), and self-esteem (Kim & Cicchetti, 2003) may influence resilience in response to exposure to adversity.

This section proceeds by providing an overview of the relevant behavioral genetic literature on the heritability of resilience and then discusses specific candidate genes associated with resilience to early life adversity. Limitations of the candidate gene approach are discussed before moving on to an overview of the more robust PGS method that can be used to measure biological resilience. Following this is a discussion of the

specific individual-level personality traits associated with resilience, which are then framed within the context of the current dissertation to identify specific PGSs associated with personality traits that will be analyzed in reference to outcomes linked with resilience to CM, delinquency, and victimization in adolescence and young adulthood.

The Heritability of Resilience

Behavioral genetic methods are useful for examining variance in the genetic and environmental effects on the expression of phenotypic traits (Plomin et al., 2009). By utilizing quasi-experimental designs of families, full/half siblings, and MZ/DZ twins, behavioral geneticists are able to parse out observed variance in phenotypic traits into latent domains that quantitatively estimate the amount of variance accounted for by additive (A) genetics, non-additive (D) genetics, the shared (C) environment, and the nonshared environment (E). To date, research reveals that all human behavior is partially explained by variation in genes, the shared environment, and the nonshared environment (Polderman et al., 2015). This finding is consistent with bottom-up ecological-transactional systemic processes where variation among genetic expression influences neurobiological and psychological adaptations.

Resilient functioning is also a partially heritable trait, meaning that resilient behavioral adaptations in response to adversity are partly explained by individual differences in genetic expression and environmental exposure. In a sample of 7,500 MZ and DZ twins, for instance, Amstadter and colleagues (2014) found that roughly 53% of the observed variance in resilient functioning to the development of internalizing symptoms in response to stressful life events were accounted for by additive genetic effects. The remaining 47% of the variance was attributable to the nonshared

environment. This is consistent with, but slightly lower than, a study by Waaktaar & Torgersen (2011), which found that additive genetic sources of variance accounted for roughly 70% of the variance in trait resilience in a sample of 2,638 twins. The remaining 30% of trait resilience was accounted for by differences in the nonshared environment. Boardman and colleagues (2008) also found that resilience to maladaptive psychological functioning may be higher in men (52%) than women (38%) while Newsome and Sullivan (2014) reported that additive genetic influences accounted for roughly 38% of the observed variation in trait resilience in to cumulative risk and outcomes associated with delinquency. This finding is consistent with Wolf et al. (2018) who, in a sample of 3,318 MZ and DZ twins, reported that additive genetic effects were responsible for roughly 25% of the observed variance in the relationship between exposure to early environmental stress and later resilience to the development of PTSD.

Collectively, behavioral genetic biometric ACE models suggest that resilience is partially heritable with additive genetic effects accounting for between 25% and 70% of the variance in the expression of resilience with the remaining variation accounted for by differences in exposure to nonshared environmental factors (Amstadter et al., 2014; Boardman et al., 2008; Newsome & Sullivan, 2014; Newsome et al., 2015; Waaktaar & Torgersen, 2011; Wolf et al., 2018). One of the major limitations of behavioral genetic analysis, however, is that they do not identify specific genetic variants that may be involved in the observed individual differences in traits associated with resilience. This is an important limitation to address from an ecological-transactional perspective because understanding how specific genes influence responses to environmental adversities can

help explain the observed variance in inter-individual outcomes associated with resilience to engaging in delinquency and being victimized across the life-course.

Candidate Gene Association Studies

Candidate gene association studies (CGAS) attempt to expand upon behavioral genetic designs by using *a priori* procedures to identify specific genetic variants (e.g., single nucleotide polymorphisms, haplotypes, indels, copy number variations, and variable number tandem repeats) located along chromosomal pathways that influence the expression of psychopathological and behavioral traits in response to environmental adversity (Duncan & Keller, 2011). Typically, CGASs examine the effect that early life adversity (i.e., stress) has on the development of negative behavioral adaptations by assessing genetic variants that increase susceptibility to environmental exposures (GxE interaction). These studies generally examine genetic susceptibility of the neurobiological stress response system by examining genes that regulate MAOA, DA, 5-HT, and OXT (Bowes & Jaffee, 2013; Cicchetti & Rogosch, 2012).

CGASs assessing genetic sensitivity to environmental adversity are mixed (Duncan & Keller, 2011), but commonly indicate that genes associated with stress sensitization influence resilience in the face of exposure to adverse experiences. Thibodeau et al. (2019), for example, found that dopaminergic genotypes including the dopamine receptor D4 (DRD4), DRD2, and the dopamine transporter (DAT1) moderated the relationship between impulsivity and CM on antisocial behavior (ASB) in a sample of 1,012 children. Specifically, they found that the estimated effect of trait impulsivity on the relationship between CM and adolescent ASB was lower in participants with a decreased genetic susceptibility to the development of impulsivity.

Others have created candidate gene indexes – which combine several candidate genes into one measure – to examine the influence that multiple candidate genes have on resilience to CM (Bousman et al., 2017; Cicchetti & Rogosch, 2012; Keers & Pluess, 2017; Tung et al., 2018). Cicchetti and Rogosch (2012) created such an index by combining together the serotonin-transporter-linked polymorphic region (5HTTLPR), corticotropin releasing hormone receptor 1 (CRHR1), tyrosine aminotransferase (TAT), DRD4, and the oxytocin receptor (OXTR) as a cumulative assessment of genetic sensitivity in a sample of 595 children reporting low familial income. They found that children with higher genetic resilience (i.e., lower susceptibility) were more likely to positively adapt (measured as a composite of resilient functioning) in response to CM compared to those with greater genetic liability to environmental adversity. Similar results were reported by Keers and Pluess (2017), who created a cumulative candidate gene index from twelve previously identified candidate genes in a sample of 7,075 participants. They found that individuals with greater genetic sensitivity to psychological distress who experienced early environmental adversity in the form of low socioeconomic status (SES) were more susceptible to reporting low SES later in life. Conversely, participants conveying less genetic susceptibility to psychological distress, but who had also experienced low SES earlier in life, were less likely to report low SES in adulthood.

Studies using candidate gene designs have advanced our understanding of the complex bottom-up diathesis-stress and differential susceptibility GxE processes associated with resilience. However, CGASs have been criticized for failure to replicate and for limitations of their methodological design (Elbau et al., 2019; Border & Keller,

2017; Duncan & Keller, 2011; Keller, 2014). Regarding the former, several studies examining the role of genetic resilience to adversity failed to find a significant effect of specific candidate genes on behavioral adaptations (Cicchetti et al., 2011; Sullivan & Newsome, 2015). Sullivan and Newsome (2015) reported no direct effects on individuals with lower genetic susceptibility to risk and delinquency in a sample of 2,573 participants from the Add Health dataset. Using a cumulative genetic susceptibility index of DA functionality, Tung and colleagues (2018) reported no significant moderating effect between genetic resilience to DA regulatory functionality in a sample of 9,421 Add Health participants who experienced maltreatment in relation to reported parental closeness and friendship involvement. These are only two of the many instances where CGASs failed to replicate across similar samples, outcomes, and settings (Elbau et al., 2019; Border & Keller, 2017; Duncan & Keller, 2011; Keller, 2014).

Regarding the latter, CGAS designs violate one of the more modern assumptions in the field of human genetics concerning the Fourth Law of behavior genetics (Chabris et al., 2015; Plomin & Deary, 2015; Turkheimer, 2000). Indeed, with the mapping of the human genome and the growth of research examining the role that genes play in the development of several psychopathological adaptations (see section below concerning GWAS) came the observation that most human behavioral traits are influenced by several genes exerting small effects on behavioral adaptions over time (Chabris et al., 2015; Plomin & Deary, 2015; Turkheimer, 2000). Accordingly, the Fourth Law of human behavior genetics states that:

> A typical human behavioral trait is associated with very many genetic variants, each of which accounts for a very small percentage of the behavioral variability (Chabris et al., 2015 p. 305).

CGASs clearly violate this assumption as they include only one or a few gene variants instead of relying on large amounts of genetic information that collectively influence genetic susceptibility and behavioral adaptation in response to environmental adversity.

Genome Wide Association Studies and Polygenic Scores

Technological innovation in the field of human biological research since the 1980s has led to the creation and refinement of microarrays that are capable of quantitatively processing millions of genotypes in a relatively cost effective and time efficient manner (Plomin et al., 2009). The growth in the field of quantitative and molecular genetics was met with the observed Fourth Law of behavior genetics (stated above) and the relative abandonment of CGAS approaches in favor of the more robust GWAS. Unlike CGASs, which operate on *a priori* principals, GWAS are hypothesis-free and survey genetic variation across the entire genome to create a genetic profile of common disorders (Belsky & Harden, 2019).

Results from GWASs capture variation in genetic susceptibility to behaviorally adaptive traits across the entire genome, rather than one chromosomal line like in CGASs (Dudbridge, 2013). This is a significant improvement upon the CGAS approach as it allows for the creation of PGSs that capture variation across the genome in relation to a specific outcome of interest (Bogdan et al., 2018). Stated differently, genome wide variation captured in GWAS can be used to create PGSs which are genome wide cumulative indexes associated with genetic susceptibility to the development of a specific

behavioral trait (Bodgan et al., 2018). Calculating PGSs involves the rank ordering of genetic markers across the genome based on *p*-values. These ranks are then combined to represent a weighted sum of traits associated with allelic frequencies based on the top-ranking genetic markers gathered from a GWAS sample (Dudbridge, 2013). Thus, PGSs represent, the aggregate additive effects of several genetic variants (i.e., single nucleotide polymorphisms) on an adaptive trait (Bodgan et al., 2018).

From an ecological-transactional perspective, PGSs represent the additive genetic variation attributable to individual-level biological genetic substrates that influence vulnerability and resilience to early life adversities, such as CM. Thus, PGSs are resilience promoting or mitigating factors depending on where they fall on the additive genetic continuum. Individuals with a higher genetic susceptibility for developing depression may be less resilient in the face of CM. Conversely, individuals who have lower genetic susceptibility to developing depression may be more resilient to exposure to CM.

Studies assessing PGSs capturing genetic sensitivity to the development of psychopathological disorders in individuals exposed to CM generally find that PGSs can capture resilient functioning following maltreatment (Aas et al., 2020; Bousman et al., 2017; Fang et al., 2020; Galtermann et al., 2020; Yu et al., 2020). Using a PGS of MDD (MDD-PGS), Fang et al. (2020) found that individuals who were less susceptible to developing depression based on their MDD-PGS were less likely to develop trait depression in response to stress, demonstrating resilient adaptive functioning following environmental adversity. In one of the most robust studies of resilient adaptation following CM, Yu et al. (2020) genotyped 428 Chinese participants and constructed

PGSs of DA, 5HT, and OXT functionality to examine whether individuals with low or high genetic susceptibility to automatic negative thoughts who were exposed to CM would develop automatic negative thoughts (ANT). They found that individuals who experienced CM and displayed low DA, 5HT, and OXT PGS susceptibility were more resilient to developing ANT, meaning that they were less likely to develop automatic negative thinking styles in response to stress. However, not all studies are supportive of the effect of PGSs in predicting resilient functioning following environmental adversity. Bucknor and Derringer (in press) examined the relationship between stressful life events and negative automatic thoughts while examining the moderating role that 32 PGSs had on the resilient functioning following early life stress. Using a sample of 9,480 adults, Bucknor and Derringer found that only 4 PGSs (out of 32) – subjective wellbeing, neuroticism, depressive symptomatology, and educational attainment – were associated with a decreased likelihood of developing negative affectivity in individuals who experienced higher stressful life events. While Bucknor and Derringer's findings are a reminder of the limits of PGS research, they still point to the promising utility of the PGS approach.

Research should consider the effect that PGSs have on criminologically relevant outcomes in order to capture ecological-transactional bottom-up systems that contribute to the development of human behavior. Nevertheless, no studies, to date, have applied a PGS framework to elucidate the role of genetic resilience on the relationship between exposure to CM and adolescent/young adult delinquency and victimization. This dissertation will capture individual-level genetic factors that influence resilience from an ecological-transactional perspective by assessing how PGSs measuring genetic load for

depression, extraversion, and educational attainment moderate the relationship between CM exposure and delinquency and victimization in adolescence and young adulthood. Selection for these PGSs are discussed in the next section.

Personality Traits Associated with Resilience

PGSs are a novel step forward for aiding empirical understanding of the interactive GxE bottom-up diathesis-stress or differential susceptibility biological processes that influence resilient adaptation following adversity. Future research on individual differences on resilience to CM will need to consider PGSs within the context of corollary individual-level personality traits known to influence resilient functioning in response to CM exposure. Several personality and cognitive traits have been identified as factors that promote resilience following exposure to adversity. These include perceptions of control/self-control (Bolger & Patterson, 2003), self-esteem (Jaffee, 2017), conscientiousness (Chen et al., 2020), cognition and intelligence (Jaffee et al., 2017), spirituality (Howell & Miller-Graff, 2014), social skills, confidence, and empathy (Pérez-González et al., 2017), positive self-perceptions (Yule et al., 2019), and depression (Sheerin et al., 2018).

Despite the relatively large number of identified personality traits that influence resilient adaptation it does appear that a convergence in the literature identifies some traits to be more important than others (Nasvytiené et al., 2012; Yule et al., 2019; Fritz et al., 2018b; Meng et al., 2018). Nasvytiené and colleagues (2012) meta-analysis of 13 studies examining positive functioning in response to CM indicates that individual-level traits involving cognitive ability and positive self-esteem exerted the largest effect on positive functionality. This finding was supported by Yule et al. (2019) in a meta-analysis

of 71 cross-sectional and 47 longitudinal studies assessing factors promoting resilience in response to exposure to early childhood violence. Again, these researchers found that positive perceptions of the self (i.e., self-esteem) and self-regulation (i.e., self-control) increased resilience to violence.

Systematic reviews also provide evidence of individual-level personality traits that increase resilient adaptation in response to adversity. In their systematic review of 22 studies, Fritz et al. (2018b) found that high self-esteem and high mental flexibility increased resilience in response to sexual abuse. Meng et al. (2018) provided a rank ordered system for individual-level factors that were found to increase resilience to CM across 85 studies. In order, they found that coping skills, self-control, intelligence, education, and self-esteem were the most reported significant individual-level traits associated with resilience to CM.

Research Questions

Genetic susceptibility for the development of individual-level personality and cognitive traits may influence resilience to environmental adversity. Personality and cognitive traits that promote resilience in response to adversity in relation to outcomes associated with delinquency and victimization typically include self-esteem, cognitive problem solving, intelligence, and depression (Yule et al., 2019; Newsome et al., 2015; Newsome & Sullivan, 2014). Variation in genetic expressions that influence neurobiological functionality largely contribute to the etiology of personality and cognitive traits. This type of genetic susceptibility research has grown into a large-scale quantitative field that uses GWAS to develop PGSs representing genetic sensitivity to the

development of behaviorally adaptive traits. Collectively, PGSs represent the variation in genetic material that contributes to the development of personality and cognitive traits.

Studies have begun to use PGSs to capture biological resilience to early environmental adversity and later life outcomes. However, no studies, to date, have assessed the role that PGSs capturing biological variation in the development of relevant personality/cognitive have on resilience to CM and delinquency/victimization in adolescence and young adulthood. To address this gap, and to add to our understanding of genetic resilience from an ecological-transactional perspective, this dissertation examines how individual differences in PGSs influence resilience to CM on delinquency and victimization in adolescence and young adulthood. This dissertation specifically examines the role that PGSs capturing genetic load for the development of the personality/cognitive traits of depression, extraversion (i.e., a proxy of self-esteem), and educational attainment have on the association between CM, delinquency, and victimization. This dissertation proceeds with three research questions (RQ) and fourteen hypotheses (H) in order to better understand the effect that CM and PGSs capturing personality/cognitive traits associated with resilience to adversity have on delinquency and victimization in adolescence and young adulthood. A visual representation of the hypotheses accompanying each RQ are provided in Figure 2.

RQ1: Is CM associated with delinquency and victimization in adolescence and young adulthood?

RQ1H1: A greater exposure to CM will be associated with an increase in delinquency in adolescence and young adulthood.

RQ1H2: A greater exposure to CM will be associated with an increase in victimization in adolescence and young adulthood.

RQ2: Are PGSs for depression, extraversion, and educational attainment associated with variation in delinquency and victimization in adolescence and young adulthood?

RQ2H1a: PGSs for depression will be associated with increased delinquency in adolescence and young adulthood.

RQ2H1b: PGSs for depression will be associated with increased victimization in adolescence and young adulthood.

RQ2H2a: PGSs for extraversion will be associated with decreased delinquency in adolescence and young adulthood.

RQ2H2b: PGSs for extraversion will be associated with decreased victimization in adolescence and young adulthood.

RQ2H3a: PGSs for educational attainment will be associated with decreased delinquency in adolescence and young adulthood.

RQ2H3b: PGSs for educational attainment will be associate with decreased victimization in adolescence and young adulthood.

RQ3: Do PGSs representing susceptibility for the development of depression, extraversion, and educational attainment operate as sources of resilience to protect against the detrimental effects of CM on delinquency and victimization in adolescence and young adulthood?

RQ3H1a: Genetic susceptibility for depression will moderate the effect of CM on delinquency in adolescence and young adulthood such that

individuals who experience more CM, but have lower genetic susceptibility for depression, will report less delinquency in adolescence and young adulthood.

RQ3H1b: Genetic susceptibility to depression will moderate the effect of CM on victimization in adolescence and young adulthood such that individuals who experience more CM, but have lower genetic susceptibility for depression, will report less victimization in adolescence and young adulthood.

RQ3H2a: Genetic susceptibility to extraversion will moderate the effect of CM on delinquency in adolescence in young adulthood such that individuals who experience more CM, but have higher genetic susceptibility for extraversion, will report less delinquency in adolescence and young adulthood.

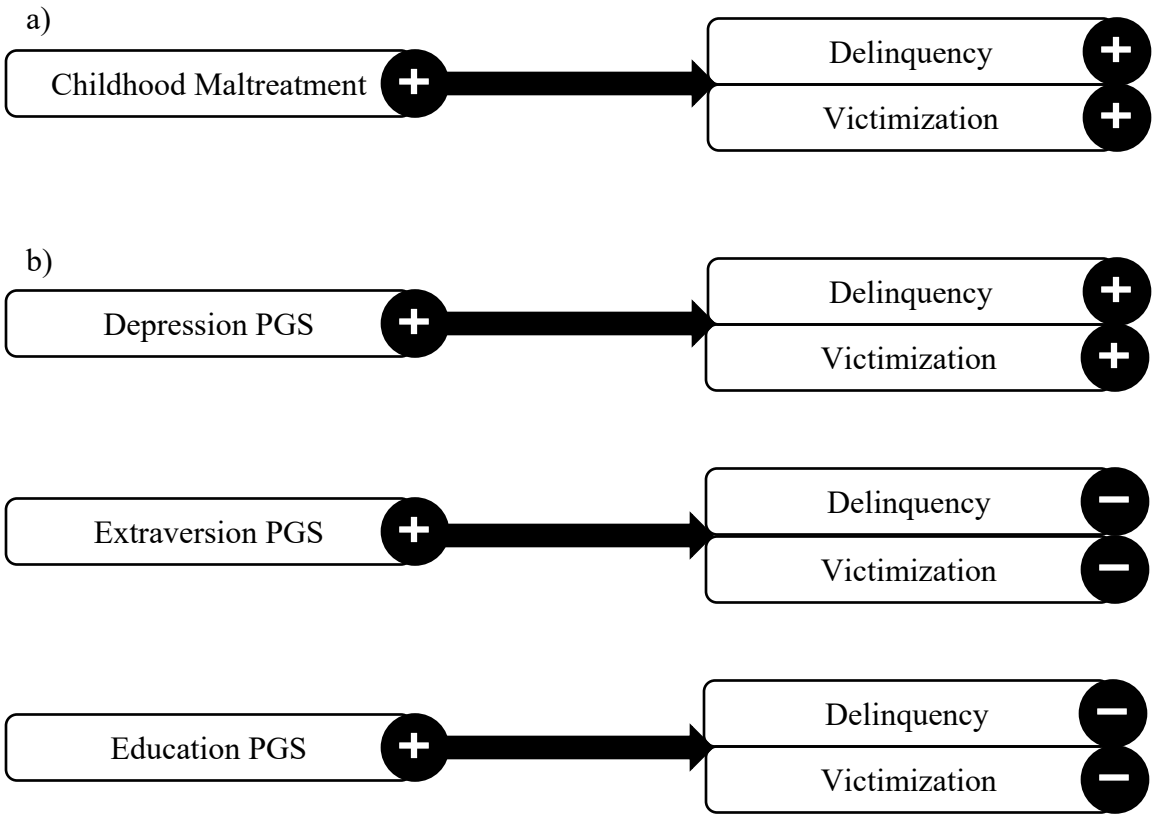
RQ3H2b: Genetic susceptibility to extraversion will moderate the effect of CM on victimization in adolescence in young adulthood such that individuals who experience more CM, but have higher genetic susceptibility for extraversion, will report less victimization in adolescence and young adulthood.

RQ3H3a: Genetic susceptibility to educational attainment will moderate the effect of CM on delinquency in adolescence and young adulthood such that individuals who experience more CM, but have higher genetic susceptibility for educational attainment, will report less delinquency in adolescence and young adulthood.

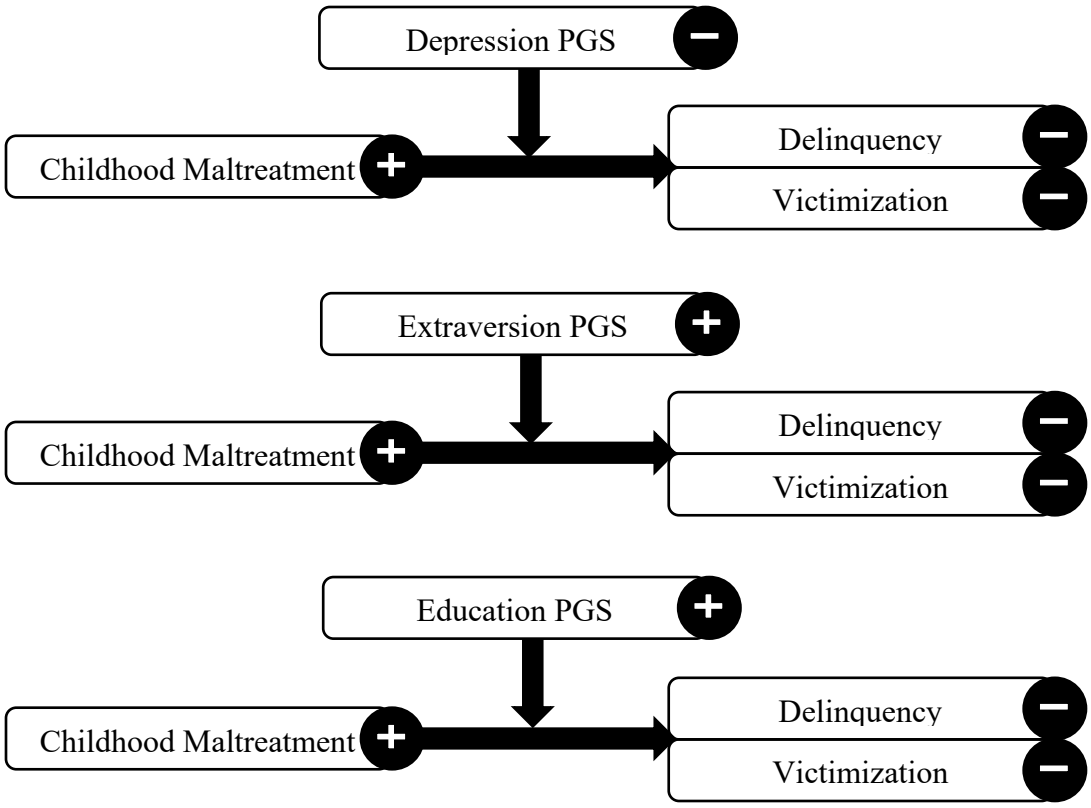
RQ3H3b: Genetic susceptibility to educational attainment will moderate the effect of CM on victimization in adolescence and young adulthood such that individuals who experience more CM, but have higher genetic susceptibility for educational attainment, will report less victimization in adolescence and young adulthood.

Figure 2

Visual Representation of Hypothesized Research Questions



(continued)



Note: Visual depictions of the hypothesized relationship between CM, PGSs, delinquency, and victimization; a) shows RQ1H1 and RQ1H2; b) shows RQ2H1a through RQ2H3b; b) shows RQ3H1a through RQ3H3b.

CHAPTER III

Methodology

Data

Data for this dissertation came from the National Longitudinal Study of Adolescent to Adult Health (Add Health). The Add Health study began in 1994 when roughly 90,000 American youths in grades 7 through 12 – selected through a stratified multi-stage cluster sampling technique of 132 schools – were asked to complete questionnaires and participate in interviews assessing multiple individual, familial, and environmental histories and experiences (Harris et al., 2006). Add Health employed a longitudinal design that has spanned five waves of data collection. Wave I (N = 90,118) began in 1994 and finished in 1995 and included participants in grades 7-12. In 1996, participants completed Wave II, which included roughly 15,000 participants from the original youth sample. Participants for Wave II were in grades 8 through 12. Wave III (N = 15,170 Wave I respondents) data collection took place between 2001 and 2002, when subjects were between 18 and 26 years of age. Wave IV (N = 15,701 Wave I respondents), collected from 2007-2008, assessed participants when they were between the ages of 24 and 32 years. Wave V, collected in 2017, was the most recent data collection. Participants for this wave were between the ages of 32 and 42. Access to data to support this dissertation was made available by Add Health personnel under the license of Dr. Danielle Boisvert. The Institutional Review Board at Sam Houston State University approved this project.

At Wave IV, respondents were asked to provide saliva samples for genotyping and biomarker analysis. Roughly 12,200 of the total Wave IV respondents (N = 15,701)

consented to provide information for long term genome-wide genotyping. Genome-wide data is available for 9,974 participants because approximately 2,226 respondents were removed from the GWAS analysis due to quality control measures. Polygenic scores were constructed for extraversion, depression, and educational attainment based on this sample of 9,974 respondents. More information on the construction of these measures is provided below. For the purpose of this dissertation, the current analyses rely on this subsample of 9,974 participants restricted to those of European ancestry (N = 5,728) given the need to construct PGSs within ancestral groups (Braudt & Harris, 2018).[2]

Measures

Measures used in this dissertation are outlined below. Table 1 provides descriptive statistics, fit indices, and internal consistency analyses for the measures. Confirmatory factor analysis was used to assess fit for many of the measures. Fit criteria were assessed using the Confirmatory Fit Index (CFI), Tucker Lewis Index (TLI), and root mean square error of approximation (RMSEA). Following Hu and Bentler (1999) good model fit is determined by a CFI > .90, TLI > .90, and RMSEA < .05. A measures internal consistency was assessed with Cronbach's alpha (α).

Delinquency

Delinquency was measured consistently with similar items across Waves I – V. The current analysis focuses on outcomes in Waves I – III when individuals were still considered to be in adolescence and early adulthood. Six items were used to measure delinquency and included questions asking respondents to indicate how often (0 = *never*;

[2] PGSs are constructed from GWASs that examine genetic variation across populations of individuals. Most participants of GWASs studies are from European ancestries, thus, it is inappropriate to include participants from other ancestral domains as GWASs may not capture the full range of variation in polygenetic scores.

3 = *five or more times*) they burglarized a building; sold drugs; stole something worth less than $50; used or threatened to use a weapon to get something from someone; damaged property; or stole something worth more than $50. Items were summed into an index of delinquency at Wave I, II, and III.

Victimization

Victimization experiences were also consistently assessed with four items at Waves I, II, and III. These items asked respondents to indicate how often (0 = *never*; 1 = *ate least once*), in the past 12 months, they had a knife or gun pulled on them; they were shot or stabbed; or they were jumped or beaten up. Items were summed to create an index of victimization experiences in adolescence and early adulthood.

Childhood Maltreatment

During Wave III, participants were asked to retrospectively report their experience of childhood and adolescent parental abuse. Childhood maltreatment was assessed with four items asking participants to indicate how often (0 = *never*; 4 = *six to ten times*), before 6th grade, they were left home alone when an adult should have been there; did not have their basic needs taken care of by a parent or caregiver; were slapped, hit, or kicked by parents or caregivers; or were touched in a sexual way by parents or caregivers. Items were summed to create an index of childhood maltreatment.

Table 1

Descriptive Statistics, Fit Indices, and Reliability Analyses for all Variables (N = 5,728).

Variables	Mean (%)	*SD*	*Min. - Max.*	Cronbach's α	*CFI*	*TLI*	*RMSEA*
Main variables							
Delinquency$_{w1}$	.90	1.94	0 - 18	.71	.93	.90	.04
Delinquency$_{w2}$	.67	1.64	0 - 18	.66	.95	.91	.03
Delinquency$_{w3}$	.47	1.32	0 - 13	.61	.95	.92	.02
Victimization$_{w1}$	.25	.64	0 - 4	.60	.97	.99	.02
Victimization$_{w2}$	.18	.56	0 - 4	.60	.99	.97	.02
Victimization$_{w3}$	.09	.38	0 - 4	.50	.94	.81	.03
Childhood maltreatment	2.12	3.01	0 - 20	.50	.94	.82	.06
Extraversion - PGS	0.00	1.00	-3.37 - 4.55	—	—	—	—
Major depressive disorder - PGS	0.00	1.00	-4.17 - 3.50	—	—	—	—
Educational attainment - PGS	0.00	1.00	-3.41 - 4.23	—	—	—	—
Individual-level covariates							
Self-esteem	24.57	3.52	6 - 30	.85	.98	.97	.05
Depression	10.55	7.38	0 - 52	.87	.92	.91	.05
Intelligence	104.95	11.80	18 - 138	—	—	—	—
Problem solving	24.42	3.42	8 - 35	.60	.97	.96	.04
Education level							
No high school[†]	(6.70)	—	—	—	—	—	—
Highschool/GED	(62.20)	—	—	—	—	—	—
College	(14.50)	—	—	—	—	—	—
Graduate/Professional degree	(0.30)	—	—	—	—	—	—
Family-level covariates							
Family SES	51.53	50.22	0 - 999	—	—	—	—
Parental incarceration							

(continued)

	Mean (%)	*SD*	*Min. - Max.*	Cronbach's α	*CFI*	*TLI*	*RMSEA*
No parent in jail†	(77.00)	—	—	—	—	—	—
One parent in jail at least once	(13.50)	—	—	—	—	—	—
Both parents in jail at least once	(1.40)	—	—	—	—	—	—
Parental attachment	9.62	.87	2 - 10	.52	.99	.99	.01
Parental engagement	33.28	5.18	10 - 40	.86	.98	.96	.05
Parental supervision	1.74	1.50	0 - 7	.62	.85	.80	.05
Community-level covariates							
Community attachment	2.30	.95	0 - 3	.58	.99	.99	.01
Neighborhood safety							
No†	(6.90)	—	—	—	—	—	—
Yes	(93.00)	—	—	—	—	—	—
Neighborhood decay							
Very poorly kept†	(.90)	—	—	—	—	—	—
Poorly kept	(4.60)	—	—	—	—	—	—
Fairly well kept	(22.50)	—	—	—	—	—	—
Very well kept	(36.60)	—	—	—	—	—	—
Demographics							
Sex							
Male†	(34.30)	—	—	—	—	—	—
Female	(52.90)	—	—	—	—	—	—

Notes: SD = Standard deviation; *Min.* = Minimum; *Max.* = Maximum; *CFI* = Confirmatory fit index; *TLI* = Tucker-Lewis index; *RMSEA* = Root mean square error of approximation; PGS = Polygenic score; SES = Socioeconomic status (measured in 1,000's); † = comparison group.

Polygenic Scores

Braudt and Harris (2018) followed Dudbridge's (2013) procedure for calculating PGSs from the Add Health GWA information. As stated earlier in the review, PGSs represent a rough indicator of additive genetic traits calculated as the weighted sum of the regression coefficient taken for each single nucleotide polymorphism (SNP) from an independent GWAS assessing the frequency of trait specific allelic phenotypes for the same SNP in the complete genome-wide data. The raw PGS for depressive symptomatology for each individual, i, is calculated as:

$$PGS_{DEPi} = \sum_{j=1}^{k} \beta jSNPij$$

Where, SNP_{ij} is the allelic frequency of the j^{th} SNP for the i^{th} individual and β_j is the estimated association between SNP j and the susceptibility to depression as reported in the GWAS summary statistics based on an independent sample. The raw PGSs are then calculated and standardized within ancestry groups. This is done to account for between and within group population stratification differences. Thus, the PGSs for extraversion, depressive symptomatology, and educational attainment represent the genetic score associated with the development of extraversion, depression, and number of years of education completed. These three PGSs were chosen for this analysis because they are the closest corollaries of the individual-level personality traits associated with self-esteem, depression, and intelligence/cognition provide by the GWAS Add Health data.

Covariates

The current dissertation examines the individual-level bottom-up genetic systems that promote resilience in response to CM on outcomes associated with delinquency and

victimization in adolescence and young adulthood. Even though this dissertation examines PGSs and CM experiences on delinquency and victimization, it is important to account for covariates at the individual-, family-, and environmental-levels that may explain portions the observed differences in individual resilience to CM and later delinquency and victimization. As such, this dissertation controls for a range of individual, family, community/environmental, and demographic variables.

Individual-Level Covariates

Self-esteem was measured at Waves I with a six item indicator asking respondents to rate their level of agreement (1 = *strongly disagree*; 5 = *strongly agree*) with the following questions: you have lots of good qualities; you have a lot to be proud of; you do everything just about right; you feel socially accepted; you feel loved and wanted; you like yourself as you are. Items were summed to provide an index of self-esteem.

Depression was measured at Wave I with a nineteen-item questionnaire. Items asked participants to indicate (0 = *never/*rarely; 3 = *most/all of the time*) if they had ever been bothered by things; had poor appetites; had the blues; thought they were just as good as other people; had trouble keeping their mind focused; felt depressed; were too tired to do things; were hopeful about the future; felt their life had been a failure; were fearful; were happy; talked less than usual; felt lonely; felt people were unfriendly to them; enjoyed their life; felt sad; felt people disliked them; found it hard to start doing things; and felt their life was not worth living. Items were summed to create an index of depression. Four items (i.e., felt just as good as other people; felt hopeful about the future; felt happy; and enjoyed life) were reverse coded.

The Add Health Peabody Picture Vocabulary Test (PPVT), which is a shortened version of the PPVT-Revised, was used to measure participant *intelligence* at Wave I. The test required participants to select the best illustration of a word that was read to them by an interviewer. Higher scores equate to higher verbal intelligence which has been used as a proxy of intelligence in other studies (Newsome & Sullivan, 2014; Newsome et al., 2016).

During Wave I surveys, respondents were asked seven questions gauging their *problem-solving* capabilities. These questions asked respondents to record their level of agreement (1 = *strongly disagree*; 5 = *strongly agree*) with the following situations: accomplish things through hard work; get upset by difficult problems; rely on gut feelings; research solutions to a problem; think there are many approaches to solving a problem; use rational decision making when approaching a problem; evaluate the outcome of a decision you made. Responses were reverse coded such that higher scores indicate higher problem-solving mindsets. Items were summed into a cumulative problem-solving index.

Participants *education level* was assessed by capturing, at Wave III, their highest level of education which included: no high school education; a high school GED; an undergraduate college education; or graduate/professional degrees.

Family-Level Covariates

The current dissertation controls for variation at the family-level by examining family socioeconomic status (SES), parental incarceration, attachment to parents, parental engagement, and parental supervision.

Family SES was measured during the Wave I parent interviews and represents parents/caregiver's income in the thousands of dollars.

Parent incarceration was measured at Wave IV and included indicators of whether a participant's mom, dad, or both parents had ever been in jail. The measure was recoded for analysis to indicate if a participant had: never had a parent in jail, had one parent in jail at least once, or had both parents in jail at least once.

At Wave I, respondents were asked to report on various indicators of their relationship with their parents and caregivers. *Parental attachment* was captured with two items asking participants how much (1 = *not at all*; 5 = *very much*) their mother/mother caregiving figure; father/or father caregiving figure cares about them. Higher levels indicate higher perceived parental attachment. *Parental engagement* was assessed with eight items asking participants (1 = *strongly disagree*; 5 = *strongly agree*) if their mother was warm and loving; encouraged independence; discussed ethics; had good communication; and if they had a good relationship. Items also asked respondents to indicate if their father was warm and loving; had good communication; and if they had a good relationship. Items were summed into an index with higher values equaling higher perceived parental engagement. *Parental supervision* was measured with seven items asking respondents (0 = *yes*; 1 = *no*) if they were allowed to make their own decisions on weekend curfews; friend groups; clothing; amount of time they watched tv; the types of tv programs they could watch; their weekday bedtime; and their diets. Items were again summed to indicate higher values associated with more parental supervision.

Community-Level Covariates

The current dissertation also controlled for several community- and neighborhood-level covariates including attachment to the community, neighborhood safety, and neighborhood physical decay.

Community attachment was measured at Wave I with three items asking participants (0 = *false*; 1 = *true*) if they know most of the people in their neighborhood; stop and talked to their neighbors in the past month; and if neighbors looked out for each other. Higher summed index values are indicative of a higher perceived attachment to the community.

Neighborhood safety and *neighborhood decay* were measured with one item each. These indicators asked respondents if they felt safe in their neighborhood (i.e., *yes* and *no*) and what the condition of their neighborhood buildings were (i.e., *very poorly kept*, *poorly kept*, *fairly well kept*, and *very well kept*).

Demographics

Demographics for this dissertation include sex. All analyses were conducted in European ancestries; thus race/ethnicity was not included as a covariate.

Plan of Analysis

The analytic plan was divided into a series of four interrelated steps to examine the moderating effect that PGSs on the relationship between CM and outcomes associated with delinquency and victimization in adolescence and young adulthood.

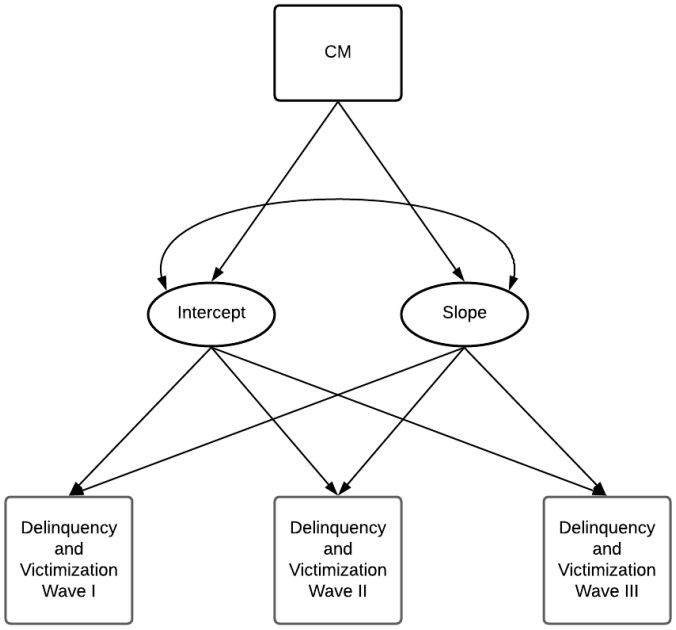
The first step was to fit unconditional (i.e., baseline) latent growth curve (LGC) models to Wave I – III delinquency and victimization measures to test whether growth in these outcomes is best modeled as a linear or non-linear trajectory. To do this, an

unrestricted latent model was fitted first. This model allows the parameters between the intercept and slope to vary freely on the outcome measures at Waves I, II, and III. After fitting the unrestricted unconditional model to the outcomes, the next step was to fit a series of increasingly restricted nested models assessing levels and growth in these outcomes over time. Using a chi-square comparison test (Byrne, 2012; Muthén & Muthén, 2010), the restricted models were compared to the baseline model with free parameters to determine the best fitting model for change in delinquency and victimization over time.

After determining the best fitting unconditional model for delinquency and victimization, the second step in the analysis was to test the relationship outlined in RQ1 by fitting a conditional LGC to assess the relationship between CM, delinquency, and victimization in adolescence and young adulthood. A visual representation of the hypothesized model is provided in Figure 3. Analysis first tested the direct effect that CM has on delinquency and victimization and then incorporated covariates to see if this direct effect persists with the additional of relevant individual-, family-, and community-level control variables.

Figure 3

Hypothesized Latent Growth Curve Model for RQ1

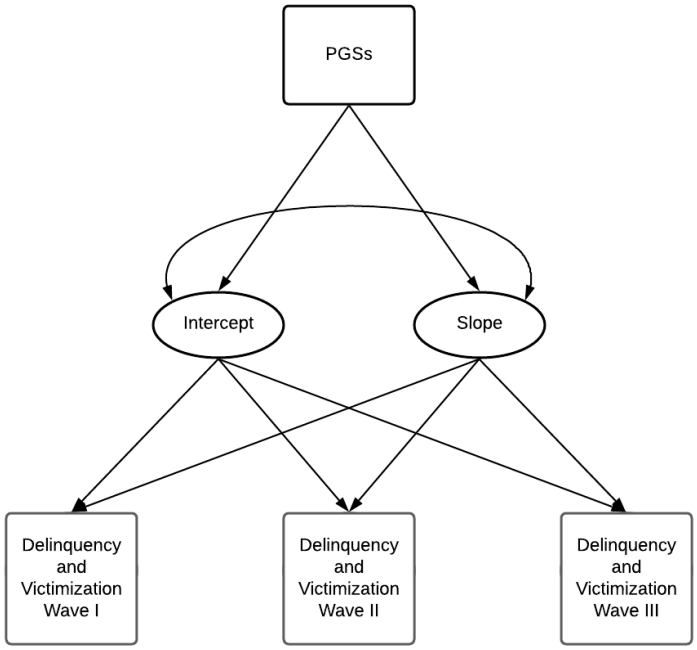


Note: CM = Childhood Maltreatment; All models account for covariates.

The third step in the analysis was to fit similar LGC models to assess the relationship that PGSs have on delinquency and victimization in adolescence and young adulthood as outlined in RQ2 (see Figure 4). Models assessed main effects of each PGS on delinquency/victimization as well as the remaining effect after controlling for relevant covariates.

Figure 4

Hypothesized Latent Growth Curve Model for RQ2

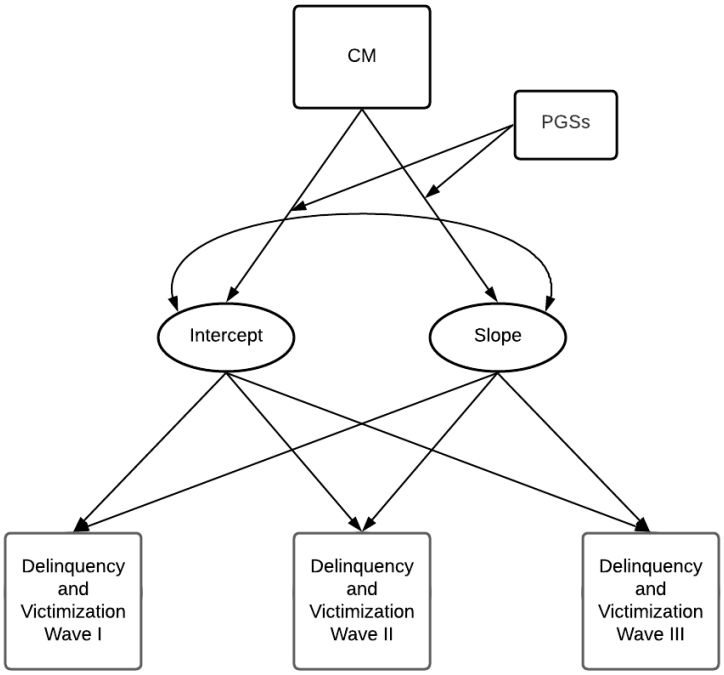


Note: PGS = Polygenic Score; All models account for covariates.

The final analyses fit several moderation LGC models to test the moderating effect that PGSs have on the relationship between CM, delinquency, and victimization as proposed in RQ3 (see Figure 5). Several analyses also accounted for individual, family, and environmental covariates. All analyses were conducted in M*plus* Version 8.4 (Muthén & Muthén, 1998-2017) using the robust maximum likelihood (MLR) estimator to account for missing and non-normal data (Byrne, 2012; Grimm & Ram, 2009; Ram & Grimm, 2007).

Figure 5

Hypothesized Latent Growth Curve Model for RQ3



Note: CM = Childhood Maltreatment; PGS = Polygenic Score; All models account for covariates.

CHAPTER IV

Results

Results for the first set of analyses assessing the best fitting unconditional LGC model for delinquency and victimization are shown in Table 2. The linear baseline model is a freely estimated model with no restrictions on the intercept and slope parameters. Model fit was good for delinquency (x^2 = 1.31, *df* = 2, *CFI* = .99, *TLI* = .99, *RMSEA* = .01) and moderate for victimization (x^2 = 52.61, *df* = 2, *CFI* = .86, *TLI* = .79, *RMSEA* = .07). The linear nested model is similar to the baseline model except the intercept and slope parameters have been fixed. The model fit indices for delinquency (x^2 = .70, *df* = 3, *CFI* = .99, *TLI* = .99, *RMSEA* = .01) and victimization (x^2 = 20.14, *df* = 3, *CFI* = .95, *TLI* = .95, *RMSEA* = .03) were both good. Chi-square difference tests (Muthén & Muthén, 2010) indicated that the nested comparison model was a better fit than the baseline model (T = .0016, *df* = 1, *p* = .97) meaning that growth in delinquency and victimization is best represented by a linear change in which the slope and intercept parameters are fixed. Non-linearity of the data was assessed by fitting a latent basis LGC model to both delinquency and victimization. The difference between this and the linear model is that a latent basis model allows for freely estimated parameters in the time points between the first and last data collection periods. Model fitting for the latent basis estimation was good for delinquency (x^2 = 1.31, *df* = 2, *CFI* = .99, *TLI* = .99, *RMSEA* = .01) and moderate for victimization (x^2 = 52.64, *df* = 2, *CFI* = .86, *TLI* = .79, *RMSEA* = .07). The shape of the freely estimated growth parameter for the latent basis model, however, was similar to the baseline linear model which indicates that a linear model was the better fitting model to the data. Thus, the nested linear model for delinquency and

Table 2

Model Fit Comparisons for LGC Models of Delinquency and Victimization.

	Delinquency Models						Victimization Models					
Model	x^2	*df*	Scaling Factor	*CFI*	*TLI*	*RMSEA*	x^2	*df*	Scaling Factor	*CFI*	*TLI*	*RMSEA*
Linear baseline	1.31	2	2.36	.99	.99	.01	52.61	2	1.39	.86	.79	.07
Linear nested	**.70**	**3**	**4.44**	**.99**	**.99**	**.01**	**20.14**	**3**	**3.65**	**.95**	**.95**	**.03**
Latent basis	1.31	2	2.36	.99	.99	.01	52.64	2	1.39	.86	.79	.07

Notes: x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; All models were tested with the chi-square difference test for the robust maximum likelihood (MLR) estimator; The chi-square difference test for the linear nested model for delinquency was T = .0016, *df* = 1, *p* = .97; The chi-square difference test for the linear nested model for Victimization was T = .053; *df* = 1; *p* = .82; Non-significant *p*-values for the chi-square difference tests indicate that the model can be retained in comparison to the baseline model; Bold = best fitting models.

victimization was retained for subsequent conditional LGC analyses with main independent variables and covariates.

Findings for the linear LGC model of delinquency are shown in Table 3. Model 1 shows results for the unconditional nested linear growth model without any independent variables or covariates. In other words, Model 1 shows the findings for the initial levels (i.e., intercept) and growth (i.e., slope) in delinquency over time without including any predictors or covariates. The mean level of delinquency was significant (*Coeff.* = .94, *SE* = .03, *ES* = .60, *95% CI* = .90, .98, $p < .01$) indicating that the average participant had a delinquency score of .94 which decreased by .18 points per wave as indicated by the significant mean slope parameter (*Coeff.* = -.18, *SE* = .01, *ES* = -.40, *95% CI* = -.20, -.16, $p < .01$). The variances represented the interindividual variation from the average. Thus, participants significantly differed in both their mean levels (*Coeff.* = 2.47, *SE* = .21, *ES* = 1.00, *95% CI* = 2.13, 2.81, $p < .01$) and growth (*Coeff.* = .22, *SE* = .04, *ES* = 1.00, *95% CI* = .15, .29, $p < .01$) in delinquency from Wave I to Wave III. The significant covariance or correlation between the intercept and slope parameters (*Coeff.* = -.70, *SE* = .08, *ES* = -.95, *95% CI* = -.83, -.57, $p < .01$) indicates that participants mean levels of delinquency were negative correlated with change in delinquency over time. Overall, the significant findings for this model suggest that including predictors and covariates would be appropriate for estimating factors that may account for variation in the levels and change in delinquency over time.

Table 3

Latent Growth Curve Models Examining the Relationship between Childhood Maltreatment and Delinquency.

	Model 1					Model 2					Model 3				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the outcome and childhood maltreatment															
$I_{delinquency}$	.94**	.03	.60	.90, .98	.01	—	—	—	—	—	—	—	—	—	—
$S_{delinquency}$	-.18**	.01	-.40	-.20, -.16	.01	—	—	—	—	—	—	—	—	—	—
Childhood maltreatment	—	—	—	—	—	2.14**	.05	.70	2.06, 2.22	.01	2.15**	.05	.71	2.07, 2.23	.01
Variances and covariances for the outcome and childhood maltreatment															
$I_{delinquency}$	2.47**	.21	1.00	2.13, 2.81	.01	—	—	—	—	—	—	—	—	—	—
$S_{delinquency}$	.22**	.04	1.00	.15, .29	.01	—	—	—	—	—	—	—	—	—	—
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.95	-.83, -.57	.01	-.69**	.08	-.95	-.82, -.56	.01	-.57**	.07	-1.00	-.67, -.45	.01
Childhood maltreatment	—	—	—	—	—	9.25**	.40	1.00	8.53, 9.88	.01	9.26**	.40	1.00	8.63, 9.88	.01
Intercepts															
$I_{delinquency}$	—	—	—	—	—	.77**	.04	.49	.72, .83	.01	4.165**	.68	2.70	3.05, 5.28	.01
$S_{delinquency}$	—	—	—	—	—	-.17**	.02	-.36	-.19, -.14	.01	-1.49**	.30	-3.20	-1.98, -.98	.01
Means for covariates															
Self-esteem	—	—	—	—	—	—	—	—	—	—	24.46**	.05	7.00	24.37, 24.54	.01
Depression	—	—	—	—	—	—	—	—	—	—	10.65**	.12	1.43	10.47, 10.82	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	105.33**	.17	9.01	105.04, 105.61	.01
Problem solving	—	—	—	—	—	—	—	—	—	—	24.42**	.05	7.10	24.34, 24.48	.01
Education level	—	—	—	—	—	—	—	—	—	—	1.12**	.01	2.18	1.10, 1.13	.01
Family SES	—	—	—	—	—	—	—	—	—	—	51.84**	.81	1.00	50.49, 53.17	.01
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.20**	.01	.43	.17, .19	.01
Parental attachment	—	—	—	—	—	—	—	—	—	—	9.60**	.02	10.81	9.57, 9.62	.01
Parental engagement	—	—	—	—	—	—	—	—	—	—	32.94**	.09	6.37	32.78, 33.07	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	1.40**	.02	1.09	1.35, 1.41	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	2.20**	.01	2.23	2.12, 2.17	.01
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.93**	.01	3.60	.92, .93	.01
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	3.50**	.01	5.02	3.43, 3.47	.01
Female	—	—	—	—	—	—	—	—	—	—	1.60**	.01	3.06	1.51, 1.54	.01
Variances for covariates															
Self-esteem	—	—	—	—	—	—	—	—	—	—	12.30**	.30	1.00	11.80, 12.73	.01
Depression	—	—	—	—	—	—	—	—	—	—	55.00**	1.60	1.00	52.31, 57.49	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	136.49**	3.10	1.00	131.41, 141.57	.01
Problem solving	—	—	—	—	—	—	—	—	—	—	11.81**	.27	1.00	11.36, 12.26	.01
Education level	—	—	—	—	—	—	—	—	—	—	.26**	.01	1.00	.25, .27	.01

(continued)

Parameter	Model 1					Model 2					Model 3				
	Coeff.	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Family SES	—	—	—	—	—	—	—	—	—	—	2735.04**	497.40	1.00	1916.86, 3553.20	.01
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.20**	.01	1.00	.16, .17	.01
Parental attachment	—	—	—	—	—	—	—	—	—	—	.80**	.05	1.00	.71, .86	.01
Parental engagement	—	—	—	—	—	—	—	—	—	—	26.72**	.80	1.00	25.49, 27.94	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	1.60**	.05	1.00	1.52, 1.68	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	.94**	.02	1.00	.91, .96	.01
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.07**	.01	1.00	.06, .07	.01
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	.47**	.01	1.00	.45, .50	.01
Female	—	—	—	—	—	—	—	—	—	—	.25**	.01	1.00	.25, .25	.01
Regression Parameters on $I_{delinquency}$ for childhood maltreatment and covariates															
Childhood maltreatment	—	—	—	—	—	.08*	.01	.15	.06, .10	.01	.04**	.01	.08	.02, .06	.01
Self-esteem	—	—	—	—	—	—	—	—	—	—	-.01	.01	-.01	-.03, .01	.58
Depression	—	—	—	—	—	—	—	—	—	—	.04**	.01	.17	.03, .04	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	.01**	.01	.07	.01, .01	.01
Problem solving	—	—	—	—	—	—	—	—	—	—	-.07**	.01	-.16	-.09, -.06	.01
Education level	—	—	—	—	—	—	—	—	—	—	-.17**	.06	-.05	-.27, -.07	.01
Family SES	—	—	—	—	—	—	—	—	—	—	.01	.01	.03	.01, .01	.27
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.11	.09	.03	-.03, .25	.20
Parental attachment	—	—	—	—	—	—	—	—	—	—	.01	.05	.01	-.08, .10	.83
Parental engagement	—	—	—	—	—	—	—	—	—	—	-.04**	.01	-.14	-.06, -.03	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	-.06**	.02	-.05	-.10, -.03	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	.04	.03	.22	-.01, .08	.20
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.02	.12	.01	-.16, .19	.86
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	-.08	.05	-.03	-.16, .01	.12
Female	—	—	—	—	—	—	—	—	—	—	-.72**	.06	-.23	-.82, -.62	.01
Regression Parameters on $S_{delinquency}$ for childhood maltreatment and covariates															
Childhood maltreatment	—	—	—	—	—	-.01	.01	-.04	-.02, .01	.21	.01	.01	.04	-.01, .02	.23
Self-esteem	—	—	—	—	—	—	—	—	—	—	.01*	.01	.08	.01, .02	.04
Depression	—	—	—	—	—	—	—	—	—	—	-.01**	.01	-.23	-.02, -.01	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	.01	.01	-.10	-.01, .01	.73
Problem solving	—	—	—	—	—	—	—	—	—	—	.02**	.01	.11	.01, .02	.01
Education level	—	—	—	—	—	—	—	—	—	—	.01	.03	.01	-.03, .05	.73
Family SES	—	—	—	—	—	—	—	—	—	—	.01	.01	-.01	.01, .01	.76
Parental incarceration	—	—	—	—	—	—	—	—	—	—	-.04	.04	-.03	-.14, .03	.37
Parental attachment	—	—	—	—	—	—	—	—	—	—	.01	.02	.01	-.04, .04	.92
Parental engagement	—	—	—	—	—	—	—	—	—	—	.02**	.01	.17	.01, .02	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	.04**	.01	.12	.03, .06	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	-.01	.01	-.02	-.03, .01	.58

(continued)

	Model 1					Model 2					Model 3				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.01	.02	.01	-.07, .09	.25
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	.03	.02	.04	-.01, .06	.25
Female	—	—	—	—	—	—	—	—	—	—	.12**	.03	.13	.07, .17	.01
Fit Statistics															
R^2 Intercept			—					.02					.19		
R^2 Slope			—					.01					.21		
x^2/df			362.81/3.00					572.27/6.00					1650.53/48		
CFI			.99					.99					.34		
TLI			.99					.99					.68		
RMSEA			.01					.01					.05		

Notes: *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation.

Table 3 also shows results from the conditional linear LGC model including the main effect of CM on delinquency (Model 2) and the effect of CM on delinquency while controlling for relevant covariates (Model 3). Looking first at Model 2, participants significantly differed in their mean levels (*Coeff.* = 2.14, *SE* = .05, *ES* = .70, *95% CI* = 2.06, 2.22, $p < .01$) and interindividual variation (*Coeff.* = 9.25, *SE* = .40, *ES* = 1.00, *95% CI* = 8.53, 9.88, $p < .01$) in experiences of CM. Childhood maltreatment was also significantly associated with higher levels of delinquency (*Coeff.* = .08, *SE* = .01, *ES* = .15, *95% CI* = .06, .10, $p < .01$) such that individuals who reported more experiences of CM were more likely to engage in higher levels of delinquency at Wave I. This relationship is shown in Figure 5 and demonstrates that participants scoring 1 standard deviation above the mean in CM had higher initial levels of delinquency at Wave I. Childhood maltreatment was not significantly associated with growth in delinquency between Waves I, II, and III. The intercept values for delinquency represent the delinquency score and rate of change in delinquency after controlling for experiences of CM. Therefore, after controlling for CM, individuals had mean delinquency levels of .77 (*SE* = .04, *ES* = .49, *95% CI* = .72, .83, $p < .01$) with a slower .17 (*SE* = .02, *ES* = -.36, *95% CI* = -.19, -.14, $p < .01$) rate of change over time.

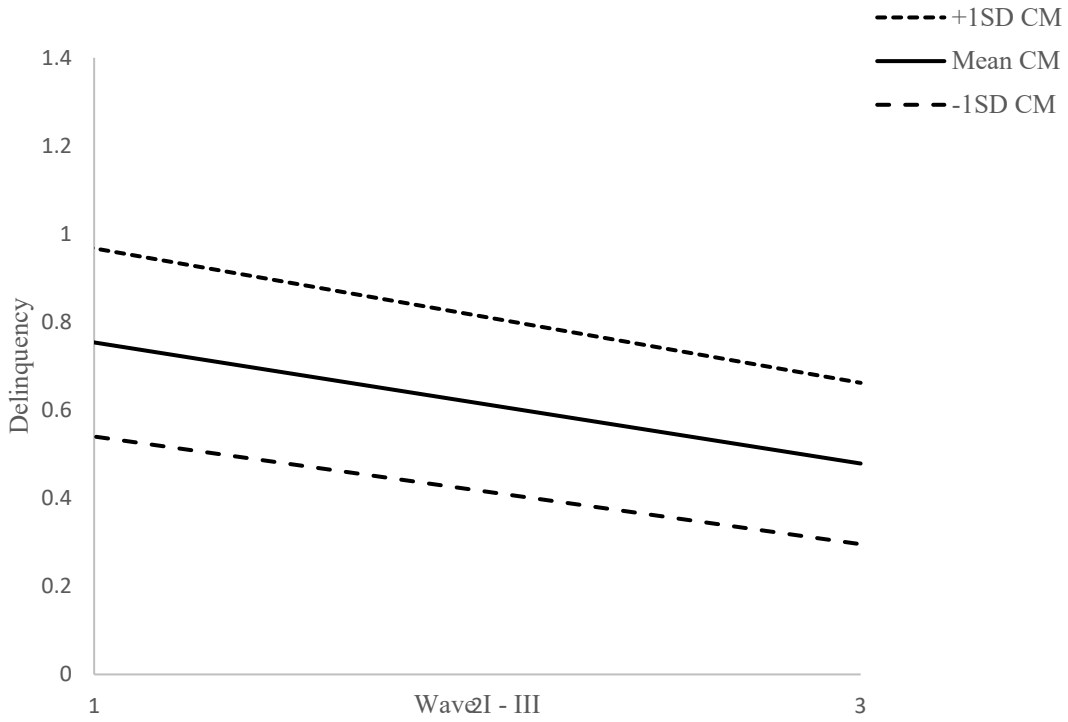
Model 3 provides the results for the relationship between CM and delinquency while controlling for all covariates. Mean levels and interindividual variation was significant for CM and all covariates. Consistent with the previous model, CM had a significant effect on mean levels of delinquency while controlling for relevant covariates (*Coeff.* = .04, *SE* = .01, *ES* = .08, *95% CI* = .02, .06, $p < .01$). Participants with higher

levels of depression (*Coeff.* = .04, *SE* = .01, *ES* = .17, *95% CI* = .03, .04, $p < .01$) and higher intelligence (*Coeff.* = .01, *SE* = .01, *ES* = .07, *95% CI* = .01, .01, $p < .01$) reported engaging in more delinquency at Wave I while those who had higher problem-solving capabilities (*Coeff.* = -.07, *SE* = .01, *ES* = -.16, *95% CI* = -.09, -.06, $p < .01$), a higher reported education level at Wave III (*Coeff.* = -.17, *SE* = .06, *ES* = -.05, *95% CI* = -.27, -.07, $p < .07$), increased parental engagement (*Coeff.* = -.04, *SE* = .01, *ES* = -.14, *95% CI* = -.06, -.03, $p < .01$) and parental supervision (*Coeff.* = -.06, *SE* = .02, *ES* = -.05, *95% CI* = -.10, -.03, $p < .01$) were less likely to have higher initial delinquency levels. Compared to males, females were also less likely to report higher levels of delinquency at Wave I (*Coeff.* = -.72, *SE* = .06, *ES* = -.23, *95% CI* = -.82, -.62, $p < .01$).

Childhood maltreatment was not significantly associated with growth in delinquency over time, however, those with higher self-esteem (*Coeff.* = .01, *SE* = .01, *ES* = .08, *95% CI* = .01, .02, $p = .04$), more problem-solving capabilities (*Coeff.* = .02, *SE* = .01, *ES* = .11, *95% CI* = .01, .02, $p < .01$), higher parental engagement (*Coeff.* = .02, *SE* = .01, *ES* = .17, *95% CI* = .01, .02, $p < .01$) and supervision (*Coeff.* = .04, *SE* = .01, *ES* = .12, *95% CI* = .03, .06, $p < .01$), and females (*Coeff.* = .12, *SE* = .03, *ES* = .13, *95% CI* = .07, .17, $p < .01$) had a greater rate of change in delinquency over time. Those who reported experiencing more depression (*Coeff.* = -.01, *SE* = .01, *ES* = -.23, *95% CI* = -.02, -.01, $p < .01$) had a lower rate of change in their initial levels of delinquency over time.

Figure 6

Regression Parameter for Childhood Maltreatment on the Intercept of Delinquency



Notes: SD = standard deviation; CM = childhood maltreatment; Delinquency is the regression score for the mean levels of delinquency.

Table 4 shows the results of the linear LGC models assessing the relationship between PGSs for extraversion, depression, and educational attainment on delinquency. Models 1, 3, and 5 show the main effects of each PGS on delinquency while models 2, 4, and 6, show the main effect while controlling for all covariate. Results for the means, variances, and regression parameters for covariates remained the same across all delinquency models. As can be seen, there were no significant effects of any of the PGSs on delinquency.

Table 4

Latent Growth Curve Models Examining the Relationship between PGSs and Delinquency

	Model 1					Model 2				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	-.01	.01	-.01	-.03, .01	.60	-.01	.01	-.01	-.03, .02	.60
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-1.00	-.83, -.60	.01	-.56**	.07	-0.96	-.68, -.45	.01
Extraversion - PGS	1.00**	.02	1.00	.96, 1.03	.01	1.00**	.02	1.00	.96, 1.03	.01
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{delinquency}$	.94**	.03	.60	.89, .98	.01	4.74**	.68	2.78	3.27, 5.50	.01
$S_{delinquency}$	-.18**	.01	-.40	-.20, -.16	.01	-1.43**	.30	-3.08	-1.93, -.94	.01
Regression Parameters on $I_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	-.01	.03	-.01	-.05, .04	.80	.01	.02	.01	-.04, .04	.96
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	.01	.01	.02	-.01, .03	.40	.01	.01	.02	-.01, .03	.48
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.01					.18		
R^2 Slope			.01					.21		
x^2/df			601.20/6					1643.47/48		
CFI			.99					.42		
TLI			.99					.73		
RMSEA			.01					.04		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

(continued)

	Model 3					Model 4				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.02, .02	.99	.01	.01	.01	-.02, .02	.99
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.95	-.90, -.60	.01	-.60**	.07	-.96	-.68, -.45	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.99**	.02	1.00	.96, 1.03	.01	.99**	.02	1.00	.96, 1.03	.01
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{delinquency}$	.94**	.03	.60	.90, .98	.01	4.38**	.67	2.80	3.30, 5.50	.01
$S_{delinquency}$	-.18**	.01	-.40	-.20, -.16	.01	-1.44**	.31	-3.09	-1.94, -.95	.01
Regression Parameters on $I_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	-.02	.03	-.01	-.07, .02	.40	-0.03	0.03	-.02	-.07, .01	0.24
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.02	-.01, .03	.60	.01	.01	.02	-.01, .03	.50
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.01					.18		
R^2 Slope			.01					.21		
x^2/df			574.22/6					1626.21/48		
CFI			.99					.41		
TLI			.99					.72		
RMSEA			.01					.04		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

(continued)

Parameter	Model 5 *Coeff.*	*SE*	*ES*	*95% CI*	*p*	Model 6 *Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.01	.01	.01	-.01, .03	.52	.01	.01	.01	-.01, .03	.52
Variances and covariances for the outcome and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.95	-.83, -.57	.01	-.60**	.07	-.96	-.68, -.45	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	1.01**	.02	1.00	.97, 1.04	.01	1.01**	.02	1.00	.97, 1.04	.01
Intercepts										
$I_{delinquency}$	.94**	.03	.60	.90, .98	.01	4.54**	.70	2.83	3.33, 5.56	.01
$S_{delinquency}$	-.18**	.01	-.40	-.20, -.16	.01	-1.43**	.30	-3.06	-1.92, -.93	.01
Regression Parameters on $I_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.02	.03	.01	-.03, .06	.50	.04	.03	.03	-.01, .09	.14
Regression Parameters on $S_{delinquency}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.02	.01	.04	-.01, .04	.20	.01	.01	.01	-.02, .03	.64
Fit Statistics										
R^2 Intercept			.01					.19		
R^2 Slope			.01					.21		
x^2/df			593.96/6					1639.51/48		
CFI			.99					.40		
TLI			.99					.70		
RMSEA			.01					.04		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

Table 5 shows results for the moderation linear LGC models estimating the moderating effect that PGSs have on the relationship between CM and delinquency. Childhood maltreatment had consistently significant effects on levels of delinquency in the main effects (*Coeff.* = .24, *SE* = .04, *ES* = .20, *95% CI* = .17, .30, $p < .01$) and covariate models (*Coeff.* = .12, *SE* = .04, *ES* = .08, *95% CI* = .06, .12, $p < .01$). Looking at Models 3 and 4, the PGS for major depressive disorder significantly moderated the relationship between childhood maltreatment and growth in delinquency (*Coeff.* = .03, *SE* = .02, *ES* = .06, *95% CI* = .01, .06, $p = .04$). Specifically, as visualized in Figure 7, participants with a greater genetic likelihood for the development of major depressive disorder who experienced greater levels of CM had a significantly greater rate of change in their delinquency over time. Thus, individuals who experienced more CM engaged in higher initial levels of delinquency over time. This relationship was then moderated by major depressive disorder PGSs such that individuals who experienced greater levels of CM and who were genetically susceptible for developing major depressive disorder engaged in slightly more delinquent activities over time.

Table 5

Moderation LGC Estimating the effect of PGSs on Childhood Maltreatment and Delinquency

	Model 1					Model 2				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .01	.85	.01	.02	.01	-.02, .03	.72
Extraversion - PGS	-.01	.01	-.01	-.03, .02	.60	-.01	.01	-.01	-.03, .02	.60
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.01	.02	.01	-.02, .03	.74	.01	.02	.01	-.02, .03	.73
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.95	-.82, -.56	.01	-.60**	.07	-.97	-.70, -.50	.01
Childhood maltreatment	1.00**	.04	1.00	.93, 1.07	.01	1.00**	.04	1.00	.94, 1.07	.01
Extraversion - PGS	.99**	.02	1.00	.96, 1.03	.01	1.00**	.02	1.00	.96, 1.03	.01
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.98**	.07	1.00	.90, 1.09	.01	.98**	.07	1.00	.90, 1.09	.01
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{delinquency}$	.94**	.03	.60	.89, .98	.01	4.30**	.67	2.71	3.14, 5.40	.01
$S_{delinquency}$	-.18**	.01	-.40	-.21, -.16	.01	-1.50**	.30	-3.13	-2.00, -1.00	.01
Regression Parameters on $I_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.23**	.04	.15	.17, .30	.01	.12**	.04	.08	.06, .12	.01
Extraversion - PGS	-.01	.03	-.01	-.05, .03	.80	.01	.02	.02	-.04, .04	.99
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.03	.04	.02	-.02, .09	.34	.01	.03	.01	-.04, .07	.71
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.02	.02	-.04	-.05, .01	.21	.02	.02	.04	-.01, .05	.24
Extraversion - PGS	.01	.01	.02	-.01, .03	.40	.01	.01	.02	-.01, .03	.50
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.01	.02	.01	-.03, .03	.98	.01	.02	.01	-.02, .03	.70
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.02					.19		
R^2 Slope			.01					.22		
x^2/df			834.78/12					1700.33/54		
CFI			.99					.40		
TLI			.99					.71		
RMSEA			.01					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

(continued)

	Model 3					Model 4				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .03	.90	.01	.02	.01	-.02, .03	.72
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.02, .02	.98	.01	.01	.01	-.02, .02	.98
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.01	.02	.01	-.02, .03	.67	.01	.01	.01	-.02, .03	.70
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.96	-.90, -.60	.01	-.60**	.07	-.97	-.68, -.45	.01
Childhood maltreatment	1.00**	.04	1.00	.93, 1.07	.01	1.01**	.04	1.00	.94, 1.07	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.99**	.02	1.00	.96, 1.03	.01	.99**	.02	1.00	.96, 1.03	.01
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.97**	.07	1.00	.86, 1.09	.01	.97**	.07	1.00	.86, 1.09	.01
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{delinquency}$	.94**	.03	.60	.90, .98	.01	4.25**	.70	2.71	3.14, 5.40	.01
$S_{delinquency}$	-.18**	.01	-.40	-.20, -.16	.01	-1.50**	.30	-3.14	-1.97, -.97	.01
Regression Parameters on $I_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.24**	.04	.20	.17, .30	.01	.12**	.04	.08	.06, .18	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	-.02	.03	-.02	-.07, -.02	.40	-.03	.03	-.02	-.07, .01	.24
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	-.02	.04	-.01	-.08, .04	.62	-.02	.04	-.01	-.08, .04	.54
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.02	.02	-.05	-.05, .01	.19	.02	.02	.04	-.01, .05	.26
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.02	-.01, .03	.60	.01	.01	.02	-.01, .03	.50
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.03*	.02	.06	.01, .06	.04	.03*	.02	.06	.01, .05	.04
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.02					.19		
R^2 Slope			.01					.22		
x^2/df			777.11/12					1662.45/54		
CFI			.99					.40		
TLI			.99					.74		
RMSEA			.01					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

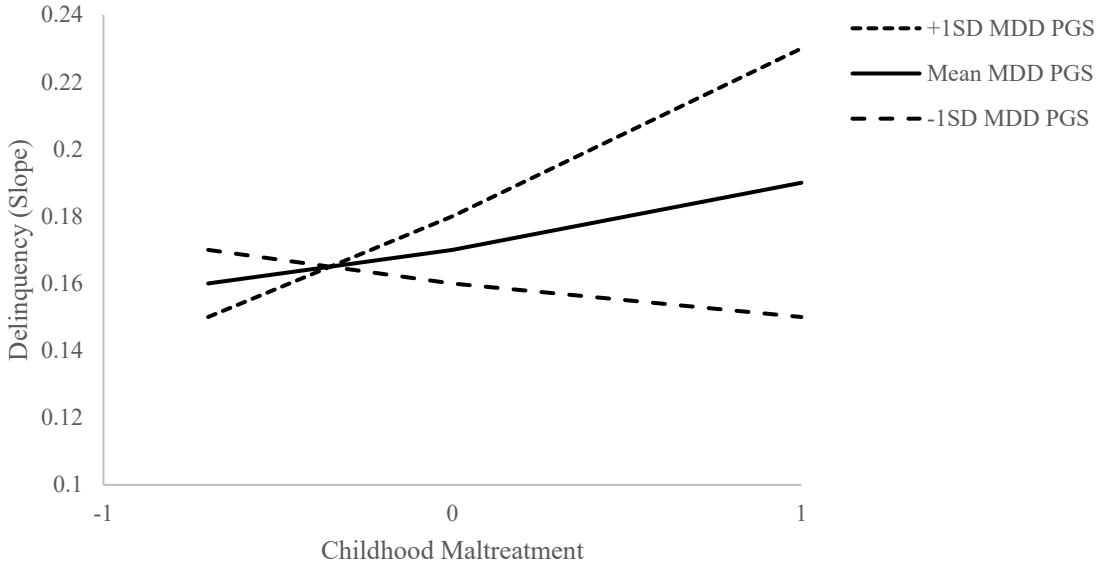
(continued)

	Model 5					Model 6				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .03	.85	.01	.02	.01	-.02, .03	.71
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.01	.01	.01	.01, .03	.52	.01	.01	.01	-.01, .03	.52
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	-.03	.02	-.03	-.06, -.01	.08	-.03	.01	-.03	-.06, -.01	.08
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{delinquency}$ <-> $S_{delinquency}$	-.70**	.08	-.96	-.82, -.56	.01	-.60**	.07	-.97	-.70, -.50	.01
Childhood maltreatment	1.00**	.04	1.00	.93, 1.07	.01	1.01**	.04	1.00	.94, 1.07	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	1.01**	.02	1.00	.97, 1.04	.01	1.01**	.02	1.00	.97, 1.04	.01
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	1.06**	.08	1.00	.93, 1.20	.01	1.06**	.08	1.00	.93, 1.20	.01
Intercepts										
$I_{delinquency}$	.94**	.03	.60	90, .98	.01	4.33**	.70	2.78	3.21, 5.44	.01
$S_{delinquency}$	-.18**	.01	-.40	-.21, -.16	.01	-1.50**	.31	-3.12	-1.96, -.96	.01
Regression Parameters on $I_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.24**	.04	.15	.17, .30	.01	.12**	.04	.08	.06, .18	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.02	.03	.02	-.02, .07	.38	.04	.03	.03	-.01, .09	.14
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	-.01	.03	.01	-.05, .05	.98	.01	.03	.01	-.05, .05	.96
Regression Parameters on $S_{delinquency}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.02	.02	-.04	-.05, .01	.23	.02	.02	.04	-.01, .05	.23
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.02	.01	.04	-.01, .04	.20	.01	.01	.01	-.02, .03	.61
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	-.01	.02	-.02	-.04, .02	.52	-.01	.02	-.02	-.03, .02	.52
Fit Statistics										
R^2 Intercept			.02					.19		
R^2 Slope			.01					.21		
x^2/df			835.50/12					1699.53/54		
CFI			.99					.40		
TLI			.99					.73		
RMSEA			.01					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{delinquency}$ = intercept for delinquency; $S_{delinquency}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

Figure 7

Moderating Effect of the MDD PGS on the Relationship between Childhood Maltreatment and Delinquency



Notes: SD = standard deviation; MDD PGS = major depressive disorder polygenic score.

Results for the linear LGC models for victimization are shown in Table 6. Model 1 provides estimates for the linear growth model of victimization without any predictor variables or covariates. Means for the intercept and slope in victimization suggest that participants had an average victimization score of .25 (*SE* = .01, *ES* = .51, *95% CI* = .24, .27, $p < .01$) which decreased .04 (*SE* = .01, *ES* = -.53, *95% CI* = -.04, -.04, $p < .01$) points per wave. The variances, again, represent the interindividual variation from the average levels. Results show that participants significantly differed in their mean levels (*Coeff.* = .24, *SE* = .02, *ES* = 1.00, *95% CI* = .22, .27, $p < .01$) and growth in victimization (*Coeff.* = .01, *SE* = .01, *ES* = 1.00, *95% CI* = .01, .01, $p < .01$) over each wave of data. The significant correlation between the intercept and slope (*Coeff.* = -.05,

SE = .01, *ES* = -1.23, *95% CI* = -.05, -.04, $p < .01$) suggest that participants victimization scores are negatively related to victimization over time.

Model 2 includes CM in the analysis. As can be seen, participants significantly varied in their mean levels (*Coeff.* = 2.14, *SE* = .05, *ES* = .70, *95% CI* = 2.06, 2.23, $p < .01$) and interindividual deviation (*Coeff.* = 9.25, *SE* = .40, *ES* = 1.00, *95% CI* = 8.63, 9.87, $p < .01$) in CM. Results from the regression parameters show that participants with more experiences of CM experienced higher levels of victimization (*Coeff.* = .02, *SE* = .01, *ES* = .13, *95% CI* = .02, .03, $p < .01$) with a decrease in the growth in victimization across each wave (*Coeff.* = -.01, *SE* = .01, *ES* = -.10, *95% CI* = -.01, -.01, $p = .02$). Figure 8 provides a visual representation of this relationship. The intercept parameters represent the initial levels (*Coeff.* = .20, *SE* = .01, *ES* = .42, *95% CI* = .19, .22, $p < .01$) and change (*Coeff.* = -.04, *SE* = .01, *ES* = -.50, *95% CI* = -.04, -.03, $p < .01$) in victimization for participants after controlling for experiences of CM.

Table 6

Latent Growth Curve Models Examining the Relationship between Childhood Maltreatment and Victimization

	Model 1					Model 2					Model 3				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the outcome and childhood maltreatment															
$I_{victimization}$	.25**	.01	.51	.24, .27	.01	—	—	—	—	—	—	—	—	—	—
$S_{victimization}$	-.04**	.01	-.53	-.04, -.04	.01	—	—	—	—	—	—	—	—	—	—
Childhood maltreatment	—	—	—	—	—	2.14**	.05	.70	2.06, 2.23	.01	2.15**	.05	.71	2.07, 2.23	.01
Variances and covariances for the outcome and childhood maltreatment															
$I_{victimization}$	.24**	.02	1.00	.22, .27	.01	—	—	—	—	—	—	—	—	—	—
$S_{victimization}$	.01**	.01	1.00	.01, .01	.01	—	—	—	—	—	—	—	—	—	—
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.23	-.05, -.04	.01	-.05**	.01	-1.23	-.05, -.04	.01	-.04**	.01	-1.30	-.04, -.03	.01
Childhood maltreatment	—	—	—	—	—	9.25**	.40	1.00	8.63, 9.87	.01	9.26**	.40	1.00	8.64, 9.88	.01
Intercepts															
$I_{victimization}$	—	—	—	—	—	.20**	.01	.42	.19, .22	.01	1.40**	.20	2.82	1.04, 1.71	.01
$S_{victimization}$	—	—	—	—	—	-.04**	.01	-.50	-.04, -.03	.01	-.30**	.06	-3.47	-.40, -.17	.01
Means for covariates															
Self-esteem	—	—	—	—	—	—	—	—	—	—	24.50**	.05	7.00	24.37, 24.54	.01
Depression	—	—	—	—	—	—	—	—	—	—	10.65**	.2	1.44	10.47, 10.82	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	105.33**	.17	9.02	105.04, 105.61	.01
Problem solving	—	—	—	—	—	—	—	—	—	—	24.41**	.05	7.10	24.34, 24.48	.01
Education level	—	—	—	—	—	—	—	—	—	—	1.12**	.01	2.17	1.10, 1.13	.01
Family SES	—	—	—	—	—	—	—	—	—	—	51.86**	.82	1.00	50.49, 53.17	.01
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.20**	.01	.43	.17, .19	.01
Parental attachment	—	—	—	—	—	—	—	—	—	—	9.60**	.02	10.81	9.57, 9.62	.01
Parental engagement	—	—	—	—	—	—	—	—	—	—	32.94**	.09	6.40	32.78, 33.07	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	1.40**	.02	1.09	1.35, 1.41	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	2.15**	.01	2.22	2.12, 2.17	.01
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.93**	.01	3.60	.92, .93	.01
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	3.45**	.01	5.04	3.43, 3.47	.01
Female	—	—	—	—	—	—	—	—	—	—	1.53**	.01	3.05	1.51, 1.54	.01
Variances for covariates															
Self-esteem	—	—	—	—	—	—	—	—	—	—	12.27**	.30	1.00	11.80, 12.73	.01
Depression	—	—	—	—	—	—	—	—	—	—	54.96**	1.60	1.00	52.31, 57.49	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	136.24**	3.08	1.00	131.41, 141.57	.01
Problem solving	—	—	—	—	—	—	—	—	—	—	11.81**	.27	1.00	11.36, 12.26	.01
Education level	—	—	—	—	—	—	—	—	—	—	.26**	.01	1.00	.25, .27	.01

(continued)

	Model 1					Model 2					Model 3				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Family SES	—	—	—	—	—	—	—	—	—	—	2751.82**	498.83	1.00	1916.86, 3553.20	.01
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.20**	.01	1.00	.16, .17	.01
Parental attachment	—	—	—	—	—	—	—	—	—	—	.79**	.05	1.00	.71, .86	.01
Parental engagement	—	—	—	—	—	—	—	—	—	—	26.67**	.75	1.00	25.49, 27.94	.01
Parental supervision	—	—	—	—	—	—	—	—	—	—	1.60**	.05	1.00	1.52, 1.68	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	.94**	.02	1.00	.91, .96	.01
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.07**	.01	1.00	.06, .07	.01
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	.47**	.01	1.00	.45, .50	.01
Female	—	—	—	—	—	—	—	—	—	—	.25**	.01	1.00	.25, .25	.01
Regression Parameters on $I_{victimization}$ for childhood maltreatment and covariates															
Childhood maltreatment	—	—	—	—	—	.02**	.01	.13	.02, .03	.01	.01*	.01	.06	.01, .02	.02
Self-esteem	—	—	—	—	—	—	—	—	—	—	.01	.01	.04	.01, .01	.07
Depression	—	—	—	—	—	—	—	—	—	—	.01**	.01	.20	.01, .01	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	.01	.01	-.01	-.01, .01	.98
Problem solving	—	—	—	—	—	—	—	—	—	—	-.01*	.01	-.04	-.01, -.01	.04
Education level	—	—	—	—	—	—	—	—	—	—	-.08*	.02	-.08	-.11, -.04	.01
Family SES	—	—	—	—	—	—	—	—	—	—	.01	.01	-.02	.01, .01	.09
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.02	.02	.02	-.02, .06	.44
Parental attachment	—	—	—	—	—	—	—	—	—	—	-.01	.02	-.02	-.03, .02	.60
Parental engagement	—	—	—	—	—	—	—	—	—	—	-.01*	.01	-.07	-.01, -.01	.03
Parental supervision	—	—	—	—	—	—	—	—	—	—	-.03**	.01	-.08	-.04, -.02	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	.02**	.01	.05	.01, .04	.01
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	-.15**	.04	-.08	-.22, -.08	.01
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	-.08**	.02	-.12	-.12, -.05	.01
Female	—	—	—	—	—	—	—	—	—	—	-.30**	.02	-.30	-.32, -.30	.01
Regression Parameters on $S_{victimization}$ for childhood maltreatment and covariates															
Childhood maltreatment	—	—	—	—	—	-.01*	.01	-.10	-.01, -.01	.02	.01	.01	-.01	-.01, .01	.74
Self-esteem	—	—	—	—	—	—	—	—	—	—	-.01	.01	-.03	-.01, .01	.51
Depression	—	—	—	—	—	—	—	—	—	—	-.01**	.01	-.21	-.01, -.01	.01
Intelligence	—	—	—	—	—	—	—	—	—	—	.01	.01	.02	.01, .01	.52
Problem solving	—	—	—	—	—	—	—	—	—	—	.01	.01	.03	-.01, .01	.38
Education level	—	—	—	—	—	—	—	—	—	—	.01	.01	.07	-.01, .02	.06
Family SES	—	—	—	—	—	—	—	—	—	—	.01*	.01	.06	.01, .01	.03
Parental incarceration	—	—	—	—	—	—	—	—	—	—	.01	.01	.02	-.01, .02	.68
Parental attachment	—	—	—	—	—	—	—	—	—	—	.01	.01	.02	-.01, .01	.80
Parental engagement	—	—	—	—	—	—	—	—	—	—	.01	.01	.09	.01, .01	.10
Parental supervision	—	—	—	—	—	—	—	—	—	—	.01**	.01	.14	.01, .01	.01
Community attachment	—	—	—	—	—	—	—	—	—	—	-.01	.01	-.05	-.01, .01	.12

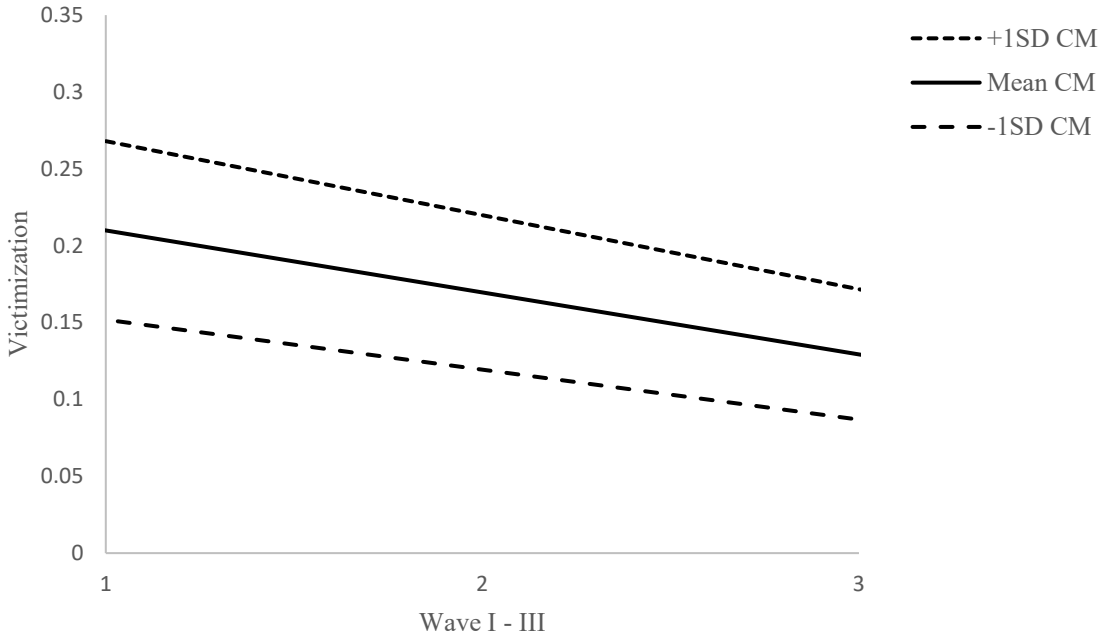
(continued)

	Model 1					Model 2					Model 3				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Neighborhood safety	—	—	—	—	—	—	—	—	—	—	.03*	.01	.10	.01, .05	.02
Neighborhood decay	—	—	—	—	—	—	—	—	—	—	.02**	.01	.16	.01, .03	.01
Female	—	—	—	—	—	—	—	—	—	—	.05**	.01	.30	.04, .06	.01
Fit Statistics															
R^2 Intercept			—					.02					.18		
R^2 Slope			—					.01					.24		
x^2/df			368.47/3					552.15/6					1502.10/48		
CFI			.95					.96					.30		
TLI			.95					.95					.64		
RMSEA			.03					.03					.05		

Notes: *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation.

Figure 8

Regression Parameter for Childhood Maltreatment on the Intercept of Victimization



Notes: SD = standard deviation; CM = childhood maltreatment.

Model 3 of Table 6 shows results for the linear LGC models on the relationship between CM and victimization while accounting for all covariates. Looking to the regression parameters, it seems that those with higher reported experiences of CM had higher initial levels of victimization (*Coeff.* = .01, *SE* = .01, *ES* = .06, *95% CI* = .01, .02, *p* = .02), however, CM no longer had a significant effect on growth in victimization over time after controlling for relevant covariates. Increases in depression were associated with increased victimization (*Coeff.* = .01, *SE* = .01, *ES* = .20, *95% CI* = .01, .01, *p* < .01). Higher problem-solving (*Coeff.* = -.01, *SE* = .01, *ES* = -.04, *95% CI* = -.01, -.01, *p* = .01), educational attainment (*Coeff.* = -.01, *SE* = .02, *ES* = -.08, *95% CI* = -.11, -.14, *p* < .01), parental engagement (*Coeff.* = -.01, *SE* = .01, *ES* = -.07, *95% CI* = -.01, -.01, *p* = .03) and supervision (*Coeff.* = -.03, *SE* = .01, *ES* = -.08, *95% CI* = -.04, -.02, *p* < .01), as

well as increased perceptions of neighborhood safety (*Coeff.* = -.15, *SE* = .04, *ES* = -.08, *95% CI* = -.22, -.08, $p < .01$) and less neighborhood decay (*Coeff.* = -.08, *SE* = .02, *ES* = -.12, *95% CI* = -.12, -.05, $p < .01$) were associated with lower mean levels of victimization. Females, compared to males, reported lower initial levels of victimization (*Coeff.* = -.30, *SE* = .02, *ES* = -.30, *95% CI* = -.32, -.30, $p < .01$).

Depression was associated with a reduced rate of change in victimization over time (*Coeff.* = -.01, *SE* = .01, *ES* = -.21, *95% CI* = -.01, -.01, $p < .01$). Family SES (*Coeff.* = .01, *SE* = .01, *ES* = .06, *95% CI* = .01, .01, $p = .03$), parental supervision (*Coeff.* = .01, *SE* = .01, *ES* = .14, *95% CI* = .01, .01, $p < .01$), neighborhood safety (*Coeff.* = .03, *SE* = .01, *ES* = .10, *95% CI* = .01, .05, $p = .02$) and less neighborhood decay (*Coeff.* = .02, *SE* = .01, *ES* = .16, *95% CI* = .01, .03, $p < .01$) were associated with an increased rate of change in victimization over time. Compared to males, females also had a slightly increased rate of change in victimization over time (*Coeff.* = .05, *SE* = .01, *ES* = .30, *95% CI* = .04, .06, $p < .01$).

Taken into context with the initial regression parameters on the intercept of victimization, this indicates that individuals with higher depression had higher initial levels of victimization with a reduced change in the rate of victimization over time. Those with higher parental supervision had lower levels of initial victimization and a greater rate of change in their victimization over time. Participants who reported higher perceived neighborhood safety and less decay had lower levels of initial victimization with a greater rate of change in victimization over time. Females also reported lower initial levels of victimization which reduced slightly more than males over time.

Table 7 shows results from the linear LGC models assessing the relationship between the PGSs for extraversion, major depressive disorder, and educational attainment on victimization. Here again, Models 1, 3, and 5 show results for the main effects of each PGS on victimization while Models 2, 4, and 6 show the effect while controlling for covariates. Means and variances of the covariates remained the same across models. Models 5 and 6 show that the PGS for educational attainment was associated with lower initial levels (Model 5 *Coeff.* = -.04, *SE* = .01, *ES* = -.08, *95% CI* = -.05, -.02, $p < .01$; Model 6 *Coeff.* = -.02, *SE* = .01, *ES* = -.04, *95% CI* = -.03, -.01, $p = .04$) of victimization and, in the main effects model, a reduction in the rate of change (*Coeff.* = -.01, *SE* = .01, *ES* = .09, *95% CI* = .01, .01, $p < .01$) in victimization. Thus, those with a genetic susceptibility for higher educational attainment reported lower initial levels of victimization (see Figure 9).

The moderation linear LGC models estimating the moderating effect that PGSs have on the relationship between CM and victimization are shown in Table 8. Model 1 shows that the PGS for extraversion significantly moderated the effect of CM on victimization (*Coeff.* = .02, *SE* = .01, *ES* = .05, *95% CI* = .01, .05, $p = .04$). Figure 10 provides a visual representation of this moderation. Specifically, individuals who experienced CM were more likely to report higher levels of initial victimization if they exhibited a greater propensity for the development of traits associated with extroversion. This relationship, however, was not significantly associated with the rate of change in victimization over time. No other significant moderating effects were reported.

Table 7

Latent Growth Curve Models Examining the Relationship between PGSs and Victimization

	Model 1					Model 2				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	-.01	.01	-.01	-.03, .02	.60	-.01	.01	-.01	-.03, .02	.60
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.23	-.05, -.04	.01	-.04**	.01	-1.30	-.04, -.03	.01
Extraversion - PGS	1.00**	.02	1.00	.96, 1.03	.01	1.00**	.02	1.00	.96, 1.03	.01
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{victimization}$	.30**	.01	.51	.24, .27	.01	1.43**	.20	2.92	1.10, 1.80	.01
$S_{victimization}$	-.04**	.01	-.53	-.04, -.04	.01	-.30**	.06	-3.51	-.40, -.20	.01
Regression Parameters on $I_{victimization}$ for PGSs and covariates										
Extraversion - PGS	.01	.01	.03	.01, .03	.09	.01	.01	.03	.01, .03	.10
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{victimization}$ for PGSs and covariates										
Extraversion - PGS	-.01	.01	-.05	-.01, .01	.13	-.01	.01	-.04	-.01, .01	.14
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.01					.18		
R^2 Slope			.01					.25		
x^2/df			580.32/6					1514.94/48		
CFI			.96					.35		
TLI			.95					.70		
RMSEA			.03					.04		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

(continued)

	Model 3					Model 4				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.02, .02	.99	.01	.01	.01	-.02, .02	.99
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.23	-.05, -.04	.01	-.04**	.01	-1.30	-.04, -.03	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.99**	.02	1.00	.96, 1.03	.01	.99**	.02	1.00	.96, 1.03	.01
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{victimization}$	.30**	.01	.51	.24, .25	.01	1.43**	.20	2.91	1.10, 1.78	.01
$S_{victimization}$	-.04	.01	-.53	-.04, -.04	.01	-.30**	.06	-3.50	-.40, -.20	.01
Regression Parameters on $I_{victimization}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.01, .02	.41	.01	.01	.01	-.01, .02	.50
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{victimization}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.01, .01	.80	.01	.01	.01	-.01, .01	.74
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.01					.18		
R^2 Slope			.01					.25		
x^2/df			588.06/6					1518.56/48		
CFI			.96					.40		
TLI			.95					.70		
RMSEA			.03					.04		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

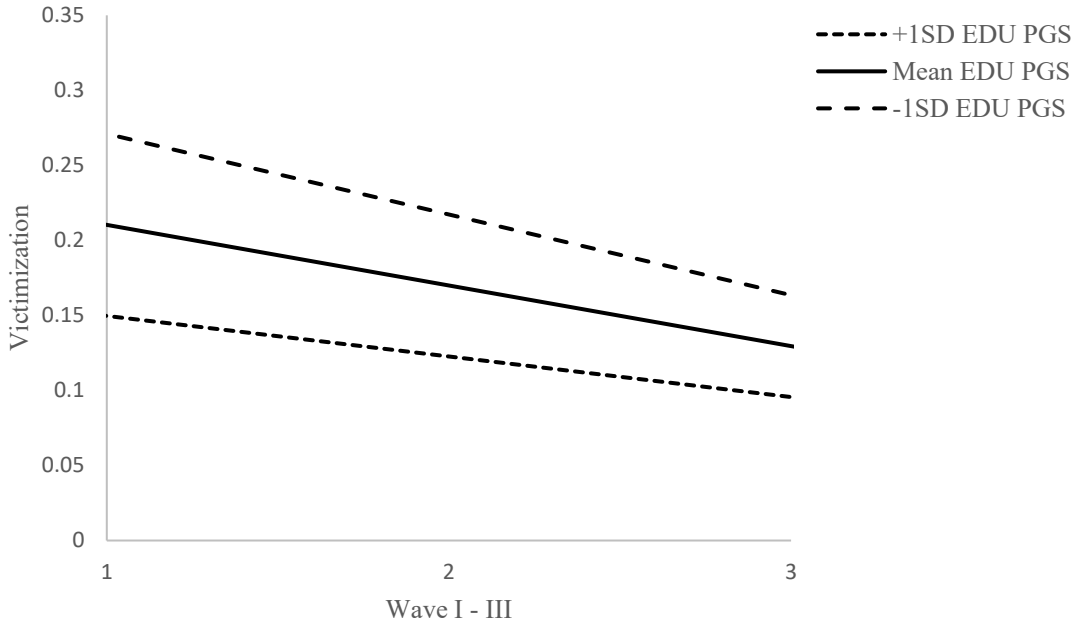
(continued)

	Model 5					Model 6				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for the PGSs										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.01	.01	.01	-.01, .03	.52	.01	.01	.01	-.01, .03	.52
Variances and covariances for the outcome and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.23	-.05, -.04	.01	-.04**	.01	-1.30	-.04, -.03	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	1.01**	.02	1.00	.98, 1.04	.01	1.01**	.02	1.00	.97, 1.04	.01
Intercepts										
$I_{victimization}$	.24**	.02	.51	.23, .30	.01	1.41**	.20	2.90	1.07, 1.74	.01
$S_{victimization}$	.01**	.01	-.53	-.05, -.04	.01	-.30**	.06	-3.50	-.40, -.17	.01
Regression Parameters on $I_{victimization}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	-.04**	.01	-.08	-.05, -.02	.01	-.02*	.01	-.04	-.03, -.01	.04
Regression Parameters on $S_{victimization}$ for PGSs and covariates										
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	-.01**	.01	.09	.01, .01	.01	.01	.01	.04	-.01, .01	.23
Fit Statistics										
R^2 Intercept			.01					.18		
R^2 Slope			.01					.25		
x^2/df			597.09/6					1518.20/48		
CFI			.97					.30		
TLI			.95					.70		
RMSEA			.03					.05		

Notes: PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

Figure 9

Regression Parameter for the Educational Attainment PGS on the Intercept of Victimization



Notes: SD = standard deviation; EDU = educational attainment; PGS = polygenic score

Table 8

Moderation LGC Estimating the effect of PGSs on Childhood Maltreatment and Victimization

	Model 1					Model 2				
Parameter	*Coeff.*	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .03	.82	.01	.02	.01	-.02, .03	.70
Extraversion - PGS	-.01	.01	-.01	-.03, .02	.60	-.01	.01	-.01	-.03, .02	.60
Major depressive disorder - PGS	—	—		—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.01	.02	.01	-.02, .03	.70	.01	.02	.01	-.02, .03	.70
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.30	-.05, -.04	.01	-.04**	.01	-1.31	-.04, -.03	.01
Childhood maltreatment	1.01**	.04	1.00	.93, 1.07	.01	1.00**	.04	1.00	.94, 1.07	.01
Extraversion - PGS	1.00**	.02	1.00	.96, 1.03	.01	1.00*	.02	1.00	.96, 1.03	.01
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	1.00**	.07	1.00	.90, 1.09	.01	.98**	.07	1.00	.87, 1.09	.01
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{victimization}$	.30**	.01	.51	.24, .26	.01	1.41**	.20	2.89	1.07, 1.74	.01
$S_{victimization}$	-.04**	.01	-.53	-.04, -.03	.01	-.30**	.06	-3.52	-.40, -.18	.01
Regression Parameters on $I_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.07**	.01	.13	.05, .09	.01	.03*	.01	.06	.01, .05	.02
Extraversion - PGS	.01	.01	.03	.01, .03	.17	.01	.01	.03	-.01, .03	.12
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	.02*	.01	.05	.01, .05	.04	.02	.01	.04	.01, .04	.08
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.01*	.01	-.10	-.01, -.01	.02	-.01	.01	-.01	-.01, .01	.77
Extraversion - PGS	-.01	.01	-.05	-.01, .01	.14	-.01	.01	-.04	-.01, .01	.15
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	-.01	.01	-.07	-.01, .01	.09	-.01	.01	-.06	-.01, .01	.12
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.02					.18		
R^2 Slope			.02					.25		
x^2/df			718.02/12					1499.30/54		
CFI			.98					.30		
TLI			.97					.70		
RMSEA			.02					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

(continued)

Parameter	Model 3					Model 4				
	Coeff.	*SE*	*ES*	*95% CI*	*p*	*Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .03	.81	.01	.02	.01	-.02, .03	.70
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.02, .02	.99	.01	.01	.01	-.02, .02	.98
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.01	.02	.01	-.02, .03	.64	.01	.01	.01	-.02, .03	.65
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.30	-.05, -.04	.01	-.04**	.01	-1.31	-.04, -.03	.01
Childhood maltreatment	1.00**	.04	1.00	.93, 1.07	.01	1.00**	.04	1.00	.94, 1.07	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	1.00**	.02	1.00	.96, 1.03	.01	.99**	.02	1.00	.96, 1.03	.01
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.97**	.07	1.00	.86, 1.09	.01	.97**	.07	1.00	.86, 1.09	.01
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Intercepts										
$I_{victimization}$	.30**	.01	.51	.24, .26	.01	1.41**	.20	2.89	1.07, 1.74	.01
$S_{victimization}$	-.04**	.01	-.53	-.04, -.03	.01	-.30**	.06	-3.52	-.40, -.18	.01
Regression Parameters on $I_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.07**	.01	.13	.05, .09	.01	.03*	.01	.06	.01,.05	.02
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.01, .02	.42	.01	.01	.01	-.01, .02	.50
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.01	.01	.01	-.02, .02	.83	.01	.01	.01	-.02, .02	.98
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Regression Parameters on $S_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.01**	.01	-.10	-.01, -.01	.01	-.01	.01	-.02	-.01, .01	.72
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	.01	.01	.01	-.01, .01	.80	.01	.01	.01	-.01, .01	.74
Educational attainment - PGS	—	—	—	—	—	—	—	—	—	—
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	.01	.01	.02	-.01, .01	.70	.01	.01	.02	-.01, .01	.63
CM x educational attainment	—	—	—	—	—	—	—	—	—	—
Fit Statistics										
R^2 Intercept			.02					.18		
R^2 Slope			.01					.25		
x^2/df			739.16/12					1508.28/54		
CFI			.98					.30		
TLI			.97					.71		
RMSEA			.02					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

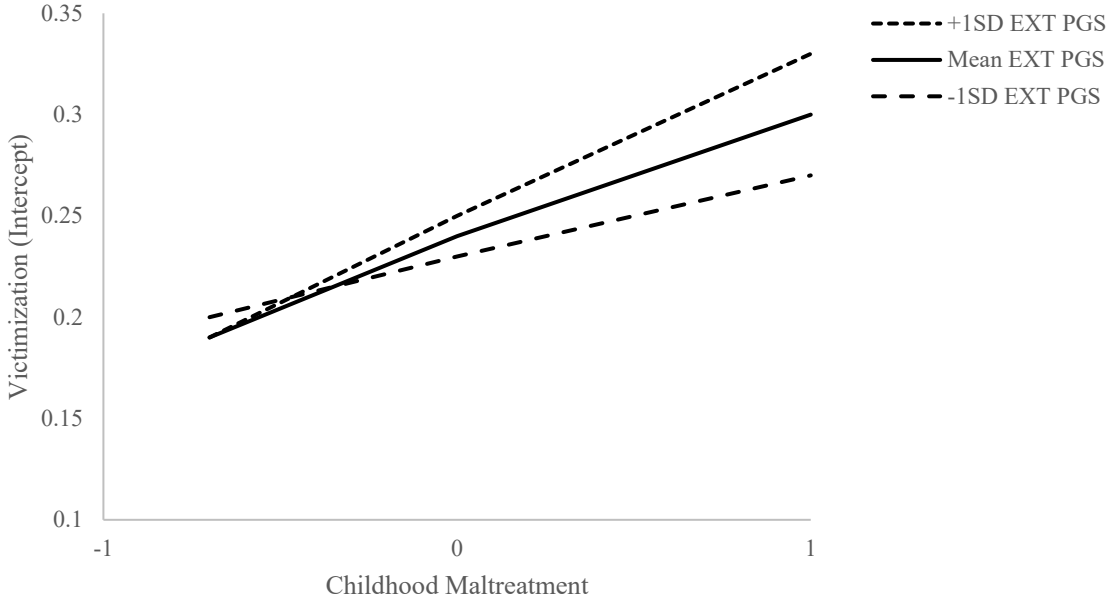
(continued)

Parameter	Model 5 *Coeff.*	*SE*	*ES*	*95% CI*	*p*	Model 6 *Coeff.*	*SE*	*ES*	*95% CI*	*p*
Means for childhood maltreatment and PGSs										
Childhood maltreatment	.01	.02	.01	-.02, .03	.82	.01	.02	.01	-.02, .03	.70
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	.01	.01	.01	-.01, .03	.52	.01	.01	.01	-.01, .03	.52
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	-.03	.01	-.03	-.06, -.01	.08	-.03	.02	-.03	-.06, -.01	.08
Variances and covariances for the outcome, childhood maltreatment, and PGSs										
$I_{victimization}$ <-> $S_{victimization}$	-.05**	.01	-1.30	-.05, -.04	.01	-.04**	.01	-1.31	-.04, -.03	.01
Childhood maltreatment	1.00**	.04	1.00	.93, 1.07	.01	1.00**	.04	1.00	.93, 1.07	.01
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	1.01**	.02	1.00	.97, 1.04	.01	1.00**	.02	1.00	.97, 1.04	.01
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	1.06**	.08	1.00	.93, 1.20	.01	1.06**	.08	1.00	.93, 1.20	.01
Intercepts										
$I_{victimization}$	.30**	.01	.51	.24, .26	.01	1.41**	.20	2.89	1.07, 1.74	.01
$S_{victimization}$	-.04**	.01	-.53	-.04, -.03	.01	-.30**	.06	-3.52	-.40, -.18	.01
Regression Parameters on $I_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	.07**	.01	.13	.04, .09	.01	.03*	.01	.06	.01. .05	.02
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	-.04**	.01	-.07	-.05, -.02	.01	-.02*	.01	-.04	-.03, -.01	.04
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	-.01	.01	-.01	-.02, .02	.85	-.01	.01	-.01	-.02, .02	.90
Regression Parameters on $S_{victimization}$ for childhood maltreatment, PGSs, and covariates										
Childhood maltreatment	-.01*	.01	-.10	-.01, -.01	.02	-.01	.01	-.02	-.01, .01	.73
Extraversion - PGS	—	—	—	—	—	—	—	—	—	—
Major depressive disorder - PGS	—	—	—	—	—	—	—	—	—	—
Educational attainment - PGS	-.01**	.01	.09	.01, .01	.01	.01	.01	.04	-.01, .01	.23
CM x extraversion	—	—	—	—	—	—	—	—	—	—
CM x major depressive disorder	—	—	—	—	—	—	—	—	—	—
CM x educational attainment	.01	.01	.01	-.01, .01	.96	.01	.01	.01	-.01, .01	.95
Fit Statistics										
R^2 Intercept			.02					.18		
R^2 Slope			.02					.24		
x^2/df			765.90/12					1523.60/54		
CFI			.97					.30		
TLI			.96					.70		
RMSEA			.02					.04		

Notes: CM = childhood maltreatment; PGS = polygenic score; *Coeff.* = unstandardized parameter coefficient; *SE* = standard error; *ES* = standardized parameter coefficient (effect size); *95% CI* = 95% confidence interval; *p* = *p*-value; $I_{victimization}$ = intercept for delinquency; $S_{victimization}$ = slope for delinquency; R^2 = proportion of variance explained; x^2 = Chi-square; *df* = degrees of freedom; *CFI* = Confirmatory Fit Index; *TLI* = Tucker-Lewis Index; *RMSEA* = Root mean square error of approximation; Models 2, 4, and 6 show the results with covariates included.

Figure 10

Moderating Effect of the EXT PGS on the Relationship between Childhood Maltreatment and Victimization



Notes: SD = standard deviation; EXT = extraversion; PGS = polygenic score.

CHAPTER V

Discussion

This dissertation adds to a growing body of literature examining how polygenetic scores condition adaptive responses to childhood maltreatment on outcomes associated with delinquency and victimization from adolescence to young adulthood. Situated within the ecological-transactional model, this dissertation explored the moderating role of three PGS capturing genetic load for depression, extraversion, and educational attainment on the relationship between CM and later life delinquency and victimization. These PGSs represented the biological resilience promotive factor hypothesized to influence positive adaptation to early exposure to CM as measured by a decreased likelihood of engaging in delinquency and being exposed to recurrent victimization through the life-course. This chapter proceeds by (1) summarizing the results for each of the proposed research questions and hypotheses, (2) situating the findings within the broader context of the ecological-transactional, diatheses-stress, and differential susceptibility models, and (3) discussing limitations and directions for future research.

The Effect of Childhood Maltreatment on Delinquency and Victimization

The first research question (RQ1) asked whether CM would be associated with delinquency and victimization in adolescence and young adulthood. Consistent with the previous literature in this area (Banny et al., 2013; Barnes et al., 2009; Beaver et al., 2010; Connolly et al., 2015; Daigle et al., 2008; DeLisi et al., 2010; Duke et al., 2009; Fagan, 2005; Finkelhor et al., 2009; Hamilton et al., 2002; Hurren et al., 2017; Ireland et al., 2002; Maas et al., 2008; Malvaso et al., 2015; Pezzoli et al., 2019; Reckdenwald et al., 2013; Stewart et al., 2008; Thornberry et al., 2010; Tillyer et al., 2012; van der Put &

de Ruiter, 2016; Widom & Brzustowicz, 2006; Widom et al., 2008; Wright & Fagan, 2013; Yoon et al., 2018), it was hypothesized that individuals who reported greater exposure to CM would be at an increased likelihood of engaging in delinquency and experiencing victimization across time points. The results largely confirmed this hypothesis. Individuals who reported experiencing more CM engaged in higher levels of delinquency and were more likely to report being victims over time even after controlling for relevant covariates.

Previous literature has documented that CM may have both direct and indirect effects on delinquency and victimization (Tillyer, 2012; Hurren et al., 2017; Malvaso et al., 2015; Yoon et al., 2018). While the current dissertation did not test the indirect or mediating effects that CM has on delinquency and victimization through various individual-, familial, and environmental-level factors, the results support the possibility for this relationship. For example, the effect that CM had on both delinquency and victimization was nearly half, yet still significant, after including relevant covariates into the model. This suggests that CM may be influencing variation in these individual-, family-, and environmental-level covariates. Results from the covariate model examining delinquency indicated that individuals with higher reported depression engaged in more delinquency. It could be the case that exposure to CM indirectly increases the likelihood of developing depression, which then increases reported engagement in delinquent activities. This would be consistent with the ecological-transactional model of offending behavior (Cicchetti, 2013) as it shows that environmental exposures influence the expression of individual-level traits and complex behavioral outcomes. Future research

will have to parse out the mediating and moderating role of various individual, familial, and environmental factors between CM, delinquency, and victimization vulnerability.

The Effect of PGSs on Delinquency and Victimization

The second research question (RQ2) sought to examine whether PGSs representing genetic load for the development of depression, extraversion, and educational attainment would be associated with delinquency and victimization in adolescence and young adulthood. Studies using candidate gene (Boisvert et al., 2012; Cicchetti & Rogosch, 2012; Cicchetti et al., 2011; Wells et al., 2017) and multilocus genetic profiles (Beaver, 2008; Keers & Pluess, 2017; Sullivan & Newsome, 2015; Thibodeau et al., 2015; Yu et al., 2020) have reported mixed findings regarding the role of specific genetic haplotypes on delinquency and victimization. This is most likely due to issues with the candidate gene approach (Elbau et al., 2019; Border & Keller, 2017; Duncan & Keller, 2011; Keller, 2014). In order to address these limitations, a polygenic approach was adopted where PGSs representing plasticity for developing depression, extraversion, and educational attainment were examined in relation to later delinquency and victimization.

The selected polygenic scores represent the continuum of genetic load hypothesized to promote genetic resilience to engaging in delinquency and experiencing victimization. Thus, variation across polygenic scores represent a continuum of resilience and vulnerability. It was hypothesized, for example, that individuals with a higher genetic likelihood of developing depression would engage in more delinquency and experience more victimization. Here, genetic load for an increased likelihood of developing depression is one side of the continuum where people more likely to develop depression

are assumed to be vulnerable to other outcomes like delinquency and victimization. Alternatively, individuals with a decreased genetic likelihood for developing depression are considered to be more resilient, meaning they are less likely to engage in subsequent dysfunctional behavior, such as engaging in delinquency or experiencing victimization. In short, polygenic load represents a quantifiable genetic profile of the resilience-vulnerability continuum whereby individuals falling closer to one side of the continuum are more resilient to CM, while those on the other side are more vulnerable.

Building from this, the hypotheses corresponding to RQ2 were threefold. First, it was expected that individuals higher on the genetic continuum for developing depression would be *more likely* to engage in delinquency and experience victimization. Second, it was expected that individuals higher on the continuum for extroversion would be *less likely* to engage in delinquency and experience victimization. Finally, it was hypothesized that individuals higher on the genetic continuum for educational attainment would be *less likely* to engage in delinquency and experience victimization. A decreased likelihood of developing depression, increased likelihood for being extroverted, and increased likelihood for higher educational attainment are the genetically informed resilience promoting factors.

Results from models assessing the relationship between PGSs for depression, extraversion, and educational attainment on delinquency and victimization reported no significant main effects of each PGS on the outcomes apart from educational attainment and victimization. The lack of significant effects between PGSs, delinquency, and victimization is not entirely surprising and fairly consistent with previous literature (Belsky & Harden, 2019; Ionnadis et al., 2020; Sullivan & Newsome, 2015). Broadly,

these findings support the idea that PGSs may not directly affect complex behavioral outcomes such as delinquency and victimization. This is consistent with broader perspectives in the ecological-transactional (Cicchetti, 2013), diathesis-stress, and differential susceptibility models (Belsky & Pluess, 2013) which suggest that genes, alone, are only one aspect of the integrative, dynamic, and time-dependent process by which complex behaviors manifest. Indeed, genes interact within proximal and distal environmental contexts to influence behavioral adaptations and outcomes (Cicchetti, 2013; Ionnadis et al., 2020). This is explored more in the next section.

Results indicated that genetic load for higher educational attainment was associated with a decrease in the initial levels of victimization with no significant effect on the growth in victimization over time. This finding is somewhat unique in that it documents, perhaps for the first time, that polygenic scores indicative of a being higher on the genetic continuum for increased educational attainment may act as a resilience promoting factor to vulnerability for victimization. In other words, individuals who are genetically more likely to attain a higher education may be less likely to be victimized in adolescence. The literature in this area has reported intelligence, problem solving, and education level as individual-level factors that protect against negative outcomes, such as delinquency and victimization (Domhardt et al., 2014; Meng et al., 2018; Newsome & Sullivan, 2014), however, this may be the first study to assess this at a quantitative genetic level as measured by PGSs.

Childhood Maltreatment and Polygenic Resilience on Delinquency and Victimization

The final question for this dissertation asked if PGSs for depression, extraversion, and educational attainment moderated the relationship between CM, delinquency, and victimization in adolescence and young adulthood. As a reminder, the PGSs represent the continuum of genetic likelihood for developing depression, extraversion, and educational attainment. Within the current framework, this continuum is conceptualized as the factor promoting resilience to CM where resilience (or positive adaptation) is measured as a decrease in the likelihood of engaging in delinquency and experiencing victimization. Individuals who have a lower genetic load for developing depression are hypothesized to have a greater likelihood for genetic resilience to CM as measured by decreases in delinquency and victimization from adolescence to young adulthood. Those with a higher genetic load for extraversion are thought to be more resilient to CM as indicated by a decrease in delinquency and victimization. Similarly, those with a higher genetic load for educational attainment are expected to be resilient to CM as measured by a decreased likelihood of engaging in delinquency and experiencing victimization.

Polygenic scores for depression moderated the relationship between CM and delinquency such that those who had a lower genetic load for developing depression and were exposed to CM were less likely to engage in delinquency across adolescence and adulthood. This finding supports the hypothesis that a lower genetic susceptibility for depression promotes positive adaptation in those who experienced CM as measured by a decreased likelihood of engaging in delinquency in adolescence and adulthood. A decrease in genetic vulnerability for developing depression, therefore, is a possible

resilience promoting genetic factor to CM and later life delinquency. This is consistent with research examining trait depression as an individual-level indicator of resilience (Bousman et al., 2017; Fang et al., 2020; Rodman et al., 2019) and suggests that CM, depression, and delinquency are intricately intertwined.

With regards to victimization in adolescence and adulthood, it appears that the PGS for extraversion moderated the effect of CM on initial levels of victimization in adolescence. Put differently, those who experienced more CM that had a decreased genetic propensity for being extroverts reported fewer initial instances of victimization in adolescence. This contradicts the proposed hypothesis as well as research suggesting that higher self-esteem may promote resilience to the negative effects of CM (Fritz et al., 2018; Jaffee, 2017; Maples et al., 2014). It is also possible that extraversion and self-esteem are two entirely different constructs with varyingly different additive genetic profiles. This is a concept that future research will need to consider and examine.

Implications for Theory and Research

This dissertation was framed within the ecological-transactional model (Cicchetti & Lynch, 1993) and also has implications for the diathesis-stress and differential susceptibility (Belsky & Pluess, 2013) perspectives. As a reminder, the ecological-transactional model suggests that various systems – also conceptualized as top-down and bottom-up processes within this dissertation – interactively influence the process by which exposure to CM effects complex behavioral outcomes such as delinquency and victimization.

Results from this dissertation provide initial support for the ecological-transactional model by examining how polygenetic profiles promote resilience to CM and

later life outcomes involving delinquency and victimization. Polygenic scores, within the ecological-transactional model (Cicchetti & Curtis, 2006), are conceptualized as individual-level biological traits that condition responses to the environmental exposure of CM and later outcomes of delinquency and victimization. Polygenic scores representing load for depression and extroversion were found to act as promotive factors that condition inter-individual resilience to the exposure of CM on delinquency and victimization later in life. These findings begin to disentangle the process by which genes can mitigate or exacerbate the effect of CM on behaviorally complex outcomes involving delinquency and victimization. Polygenetic profiles may be acting as one aspect of the bottom-up factors that contribute to the developmental cascade in which CM effects delinquency and victimization (Masten & Cicchetti, 2010).

This dissertation also found that several individual-, family-, and environmental-level measures influenced initial levels and change in delinquency and victimization over time. These included individual-level trait measures of depression, problem solving, and level of education. Family-level indicators involving parental engagement and supervision as well as neighborhood contextual measures including community attachment, neighborhood safety, and neighborhood decay. The effect that these covariates had on delinquency and victimization provide robust support for the need to examine theoretical framework like the ecological-transactional model (Yoon et al., 2020).

This dissertation also informs the diathesis-stress and differential susceptibility perspectives. These biopsychosocial models provide two competing explanations of the way in which genes moderate responses to environmental exposures. The diathesis-stress

model posits that genes are relatively fixed in their ability to mitigate responses to adverse environments such that inter-individual variability to environments will meet a threshold of positive functionality. Conversely, the differential susceptibility perspective suggests that genes are rather plastic and increase vulnerability or resilience to environmental adversity on a continuum as opposed to a fixed indicator (Belsky & Pluess, 2013; Elbau et al., 2019). Results from this PGS GxE analyses seem to support the differential susceptibility model. Polygenic load for a decreased likelihood of developing depression, for example, mitigated the likelihood of delinquent activity in those who experienced CM. The moderating effect that the PGS for depression had on the change (slope) of delinquency in those who did or did not experience varying levels of CM suggests that genetic load and environmental contexts are not fixed, rather they are plastic and exist on a continuum.

The results presented herein provide a basis for research to begin to apply the principles of the ecological-transactional model to further examine processes in which CM influences delinquency and vulnerability to victimization either directly or indirectly through the individual, family, and environmental systems (Finkelhor et al., 2009). Research need also be cognizant of the way in which genetic traits, which are passed through families, influence the likelihood of experiencing CM as well as the way in which CM influences the modulation of genetic, neurobiological, and tertiary personality architectures (Finkelhor et al., 2009; Pezzoli et al., 2019; Ioannidis et al., 2020). This is especially relevant to understanding how the intergenerational transmission of victimization affects genetic structuring which can condition parent-child relationships (Yoon et al., 2020).

Limitations and Implications for the Future

There were three limitations to this dissertation. The first limitation involved measurements of childhood maltreatment, delinquency, and victimization. Several studies identify the need to parse out the type, timing, frequency, and severity of CM into separate measures as opposed to aggregate constructs (Ahmadabadi et al., 2018; Finkelhor et al., 2009; Hurren et al.., 2017; Malvaso et al., 2015). The measure of CM used in this dissertation did not allow for disaggregation into these separate factors. This dissertation was able to capture timing, severity, and frequency of CM, however, this measure was based on retrospective self-report accounts which have received substantive criticism (Malvaso et al., 2015). Similar critiques have been leveled against aggregate measures of delinquency and victimization (Yoon et al., 2018; Yoon et al., 2020), which I was not able to account for in this study. Future work should examine how variation across the type, timing, frequency, and severity of CM differentially influences variation across the type, timing, frequency, and severity of delinquency and victimization across the life-course.

The second limitation involved internal issues with the data, specifically, the use of ancestral domains and PGSs. Analysis for this dissertation were based on a subset of a nationally representative sample take from the Add Health. Additionally, this sample was then reduced to those who provided genetic data and were from European ancestry due to the way in which GWAS are used to calculate PGSs (Braudt & Harris, 2018). This means that the sample was not nationally representative nor was it diverse across ancestries. Therefore, these findings are only relevant to those of European ancestry within a specific subset of the United States population.

The final limitation builds from the previous by recognizing the inability to generalize across demographic characteristics such as gender and race/ethnicity. Findings indicated a significant effect of gender in the models; however, gender stratified analyses were not conducted. Similarly, since the data was limited to those of European ancestry, there is no way to account for diversity across race/ethnicity. Future research will need to examine the applicability of these findings across diverse samples and demographic characteristics.

Despite these limitations, this dissertation provides fruitful insight into the complex, dynamic, and time dependent genetic and environmental interplay that PGSs and CM have on delinquency and victimization in adolescence and young adulthood. Future research in this, and broader areas in criminology, need to frame their work within ecological-transactional perspectives in order to account for the direct, indirect, and interactive effects that individual, family, and environmental factors have on complex behavioral outcomes. Similar to the fourth law of human behavior genetics (Chabris et al., 2015; von Stumm & d'Apice, 2021), research in this area is likely to find that several proximal and distal environmental factors each have a small effect on the way in which individuals navigate their lives and display positive and negative behavioral adaptations to trauma across the life course.

REFERENCES

Aas, M., Bellivier, F., Bettella, F., Henry, C., Gard, S., Kahn, J. P., ... & Jamain, S. (2020). Childhood maltreatment and polygenic risk in bipolar disorders. *Bipolar Disorders*, *22*(2), 174-181.

Afifi, T. O., & MacMillan, H. L. (2011). Resilience following child maltreatment: A review of protective factors. *The Canadian Journal of Psychiatry*, *56*(5), 266-272.

Ahmadabadi, Z., Najman, J. M., Williams, G. M., Clavarino, A. M., d'Abbs, P., & Abajobir, A. A. (2018). Maternal intimate partner violence victimization and child maltreatment. *Child Abuse & Neglect*, *82*, 23-33.

Amstadter, A. B., Myers, J. M., & Kendler, K. S. (2014). Psychiatric resilience: Longitudinal twin study. *The British Journal of Psychiatry*, *205*(4), 275-280.

Azar, S. T. (2002) *Parenting and Child Maltreatment*. In Bornstein (Eds.) *Handbook of Parenting Volume 4 Social Conditions and Applied Parenting*, 360.

Azeredo, A., Moreira, D., Figueiredo, P., & Barbosa, F. (2019). Delinquent behavior: Systematic review of genetic and environmental risk factors. *Clinical Child and Family Psychology Review*, *22*(4), 502-526.

Banny, A. M., Cicchetti, D., Rogosch, F. A., Oshri, A., & Crick, N. R. (2013). Vulnerability to depression: A moderated mediation model of the roles of child maltreatment, peer victimization, and 5-HTTLPR genetic variation among children from low-SES backgrounds. *Development and Psychopathology*, *25*(3), 599–614.

Barnes, J. E., Noll, J. G., Putnam, F. W., & Trickett, P. K. (2009). Sexual and physical revictimization among victims of severe childhood sexual abuse. *Child Abuse & Neglect*, *33*(7), 412-420.

Barnett, O., Miller-Perrin, C. L., & Perrin, R. D. (2005). *Family Violence across the Lifespan: An Introduction*. Sage Publications, Inc.

Beaver, K. M., DeLisi, M., Vaughn, M. G., & Wright, J. P. (2010). The intersection of genes and neuropsychological deficits in the prediction of adolescent delinquency and low self-control. *International Journal of Offender Therapy and Comparative Criminology*, *54*(1), 22-42.

Beaver, K. M. (2008). The interaction between genetic risk and childhood sexual abuse in the prediction of adolescent violent behavior. *Sexual Abuse*, *20*(4), 426-443.

Belsky, J. (1980). Child maltreatment: An ecological integration. *American Psychologist*, *35*(4), 320.

Belsky, D. W., & Harden, K. P. (2019). Phenotypic annotation: Using polygenic scores to translate discoveries from genome-wide association studies from the top down. *Current Directions in Psychological Science*, *28*(1), 82-90.

Belsky, J., & Pluess, M. (2013). Beyond risk, resilience, and dysregulation: Phenotypic plasticity and human development. *Development and Psychopathology*, *25*(2), 1243-1261.

Ben-David, V., & Jonson-Reid, M. (2017). Resilience among adult survivors of childhood neglect: A missing piece in the resilience literature. *Children and Youth Services Review*, *78*, 93-103.

Bennett, J. M., Rohleder, N., & Sturmberg, J. P. (2018). Biopsychosocial approach to understanding resilience: Stress habituation and where to intervene. *Journal of Evaluation in Clinical Practice*, *24*(6), 1339-1346.

Boardman, J. D., Blalock, C. L., & Button, T. M. (2008). Sex differences in the heritability of resilience. *Twin Research and Human Genetics*, *11*(1), 12-27.

Boisvert, D., Wright, J. P., Knopik, V., & Vaske, J. (2012). Genetic and environmental overlap between low self-control and delinquency. *Journal of Quantitative Criminology*, *28*(3), 477-507.

Bogdan, R., Baranger, D. A., & Agrawal, A. (2018). Polygenic risk scores in clinical psychology: Bridging genomic risk to individual differences. *Annual Review of Clinical Psychology*, *14*, 119-157.

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events?. *American Psychologist*, *59*(1), 20-28.

Bonanno, G. A., & Diminich, E. D. (2013). Annual research review: Positive adjustment to adversity–trajectories of minimal–impact resilience and emergent resilience. *Journal of Child Psychology and Psychiatry*, *54*(4), 378-401.

Border, R., & Keller, M. C. (2017). Commentary: Fundamental problems with candidate gene-by-environment interaction studies–reflections on Moore and Thoemmes. *Journal of Child Psychology and Psychiatry*, *58*(3), 328-330.

Bousman, C. A., Gunn, J. M., Potiriadis, M., & Everall, I. P. (2017). Polygenic phenotypic plasticity moderates the effects of severe childhood abuse on

depressive symptom severity in adulthood: A 5-year prospective cohort study. *The World Journal of Biological Psychiatry*, *18*(1), 75-81.

Bowes, L., & Jaffee, S. R. (2013). Biology, genes, and resilience: Toward a multidisciplinary approach. *Trauma, Violence, & Abuse*, *14*(3), 195-208.

Braudt, D. B., & Harris, K. M. (2018). Polygenic scores (PGSs) in the National Longitudinal Study of Adolescent to Adult Health (Add Health)-Release 1. Chapel Hill, NC: Carolina Population Center.

Breslau, N., Chen, Q., & Luo, Z. (2013). The role of intelligence in posttraumatic stress disorder: Does it vary by trauma severity?. *PLoS One*, *8*(6), e65391.

Bronfenbrenner, U. (1992). *Ecological Systems Theory*. Jessica Kingsley Publishers.

Bucknor, B., & Derringer, J. (in press). Current Polygenic Scores Fail to Capture Resilience. Under Review.

Byrne, B. M. (2013). *Structural Equation Modeling with Mplus: Basic Concepts, Applications, and Programming*. Routledge.

Campbell-Sills, L., Cohan, S. L., & Stein, M. B. (2006). Relationship of resilience to personality, coping, and psychiatric symptoms in young adults. *Behaviour Research and Therapy*, *44*(4), 585-599.

Carmody, D. C., & Washington, L. M. (2001). Rape myth acceptance among college women: The impact of race and prior victimization. *Journal of Interpersonal Violence*, *16*(5), 424-436.

Cassels, M., van Harmelen, A. L., Neufeld, S., Goodyer, I., Jones, P. B., & Wilkinson, P. (2018). Poor family functioning mediates the link between childhood adversity

and adolescent nonsuicidal self-injury. *Journal of Child Psychology and Psychiatry*, *59*(8), 881-887.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., ... & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*(5582), 851-854.

Chabris, C. F., Lee, J. J., Cesarini, D., Benjamin, D. J., & Laibson, D. I. (2015). The fourth law of behavior genetics. *Current Directions in Psychological Science*, *24*(4), 304-312.

Chen, E., Yu, T., Siliezar, R., Drage, J. N., Dezil, J., Miller, G. E., & Brody, G. H. (2020). Evidence for skin-deep resilience using a co-twin control design: Effects on low-grade inflammation in a longitudinal study of youth. *Brain, Behavior, and Immunity*, *88*, 661-667.

Choi, K. W., Stein, M. B., Dunn, E. C., Koenen, K. C., & Smoller, J. W. (2019). Genomics and psychological resilience: A research agenda. *Molecular Psychiatry*, *24*(12), 1170-1778.

Cicchetti, D. (2013). Annual research review: Resilient functioning in maltreated children–past, present, and future perspectives. *Journal of Child Psychology and Psychiatry*, *54*(4), 402-422.

Cicchetti, D., & Blender, J. A. (2006). A multiple-levels-of-analysis perspective on resilience: implications for the developing brain, neural plasticity, and preventive interventions. *Annals of the New York Academy of Sciences*, *1094*(1), 248-258.

Cicchetti, D., & Curtis, J. (2006). *The Developing Brain and Neural Plasticity: Implications for Normality, Psychopathology, and Resilience*. In Cohen &

Cicchetti (Eds.). *Developmental Psychopathology Volume 2: Developmental Neuroscience*.

Cicchetti, D., & Garmezy, N. (1993). Prospects and promises in the study of resilience. *Development and Psychopathology*, *5*(4), 497-502.

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry*, *56*(1), 96-118.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*(4), 597-600.

Cicchetti, D., & Rogosch, F. A. (2012). Gene by environment interaction and resilience: Effects of child maltreatment and serotonin, corticotropin releasing hormone, dopamine, and oxytocin genes. *Development and Psychopathology*, *24*(2), 411-427.

Cicchetti, D., Rogosch, F. A., & Oshri, A. (2011). Interactive effects of corticotropin releasing hormone receptor 1, serotonin transporter linked polymorphic region, and child maltreatment on diurnal cortisol regulation and internalizing symptomatology. *Development and Psychopathology*, *23*(4), 1125-1138.

Cicchetti, D., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology*, *1*, 409-438.

Connolly, E. J., Schwartz, J. A., Nedelec, J. L., Beaver, K. M., & Barnes, J. C. (2015). Different slopes for different folks: Genetic influences on growth in delinquent peer association and delinquency during adolescence. *Journal of Youth and Adolescence*, *44*(7), 1413-1427.

Cowell, R. A., Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2015). Childhood maltreatment and its effect on neurocognitive functioning: Timing and chronicity matter. *Development and Psychopathology*, *27*(2), 521-533.

Daigle, L. E., Beaver, K. M., & Hartman, J. L. (2008). A life-course approach to the study of victimization and offending behaviors. *Victims and Offenders*, *3*(4), 365-390.

DeLisi, M., Drury, A. J., Kosloski, A. E., Caudill, J. W., Conis, P. J., Anderson, C. A., ... & Beaver, K. M. (2010). The cycle of violence behind bars: Traumatization and institutional misconduct among juvenile delinquents in confinement. *Youth Violence and Juvenile Justice*, *8*(2), 107-121.

Domhardt, M., Münzer, A., Fegert, J. M., & Goldbeck, L. (2015). Resilience in survivors of child sexual abuse: A systematic review of the literature. *Trauma, Violence, & Abuse*, *16*(4), 476-493.

Dudbridge, F. (2013). Power and predictive accuracy of polygenic risk scores. *PLoS Genet*, *9*(3), e1003348.

Duke, N. N., Pettingell, S. L., McMorris, B. J., & Borowsky, I. W. (2010). Adolescent violence perpetration: Associations with multiple types of adverse childhood experiences. *Pediatrics*, *125*(4), e778-e786.

DuMont, K. A., Widom, C. S., & Czaja, S. J. (2007). Predictors of resilience in abused and neglected children grown-up: The role of individual and neighborhood characteristics. *Child Abuse & Neglect*, *31*(3), 255-274.

Duncan, L. E., & Keller, M. C. (2011). A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *American Journal of Psychiatry*, *168*(10), 1041-1049.

Dvir, Y., Ford, J. D., Hill, M., & Frazier, J. A. (2014). Childhood maltreatment, emotional dysregulation, and psychiatric comorbidities. *Harvard Review of Psychiatry*, *22*(3), 149-161.

Elbau, I. G., Cruceanu, C., & Binder, E. B. (2019). Genetics of resilience: Gene-by-environment interaction studies as a tool to dissect mechanisms of resilience. *Biological Psychiatry*, *86*(6), 433-442.

Fagan, A. A. (2005). The relationship between adolescent physical abuse and criminal offending: Support for an enduring and generalized cycle of violence. *Journal of Family Violence*, *20*(5), 279-290.

Fang, Y., Scott, L., Song, P., Burmeister, M., & Sen, S. (2020). Genomic prediction of depression risk and resilience under stress. *Nature Human Behaviour*, *4*(1), 111-118.

Feder, A., Fred-Torres, S., Southwick, S. M., & Charney, D. S. (2019). The biology of human resilience: Opportunities for enhancing resilience across the life span. *Biological Psychiatry*, *86*(6), 443-453.

Finkelhor, D., Saito, K., Jones, L. (2020). Updated trends in childhood maltreatment, 2018. *Crimes Against Children Research Center*.

Finkelhor, D., Ormrod, R., Turner, H., & Holt, M. (2009). Pathways to poly-victimization. *Child Maltreatment*, *14*(4), 316-329.

Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2015). Prevalence of childhood exposure to violence, crime, and abuse: Results from the national survey of children's exposure to violence. *JAMA Pediatrics*, *169*(8), 746-754.

Folger, S. F., & Wright, M. O. D. (2013). Altering risk following child maltreatment: Family and friend support as protective factors. *Journal of Family Violence*, *28*(4), 325-337.

Forsman, M., & Långström, N. (2012). Child maltreatment and adult violent offending: Population-based twin study addressing the 'cycle of violence'hypothesis. *Psychological Medicine*, *42*(9), 1977-1983.

Fritz, J., Fried, E. I., Goodyer, I. M., Wilkinson, P. O., & van Harmelen, A. L. (2018a). A network model of resilience factors for adolescents with and without exposure to childhood adversity. *Scientific Reports*, *8*(1), 1-13.

Fritz, J., de Graaff, A. M., Caisley, H., Van Harmelen, A. L., & Wilkinson, P. O. (2018b). A systematic review of amenable resilience factors that moderate and/or mediate the relationship between childhood adversity and mental health in young people. *Frontiers in Psychiatry*, *9*, 1-17.

Garmezy, N. (1974). Children at risk: The search for the antecedents of schizophrenia. *Schizophrenia Bulletin*, *8*, 14–90.

Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., Donovan, P., ... & Tierney, S. (2001). History of childhood maltreatment, negative cognitive styles, and episodes of depression in adulthood. *Cognitive Therapy and Research*, *25*(4), 425-446.

Goltermann, J., Redlich, R., Grotegerd, D., Dohm, K., Leehr, E. J., Böhnlein, J., ... & Repple, J. (2020). Childhood maltreatment and cognitive functioning: The role of depression, parental education, and polygenic predisposition. *Neuropsychopharmacology*, *0*, 1-9.

Grimm, K. J., & Ram, N. (2009). Nonlinear growth models in Mplus and SAS. *Structural Equation Modeling*, *16*(4), 676-701.

Hamilton, C. E., Falshaw, L., & Browne, K. D. (2002). The link between recurrent maltreatment and offending behaviour. *International Journal of Offender Therapy and Comparative Criminology*, *46*(1), 75-94.

Harris, K. M. (2013). The add health study: Design and accomplishments. *Chapel Hill: Carolina Population Center, University of North Carolina at Chapel Hill*, 1-22.

Hart, S. N., Binggeli, N. J., & Brassard, M. R. (1997). Evidence for the effects of psychological maltreatment. *Journal of Emotional Abuse*, *1*(1), 27-58.

Hébert, M., Langevin, R., & Charest, F. (2014). Factors associated with resilience in preschoolers reporting sexual abuse: A typological analysis. *International Journal of Child and Adolescent Resilience*, *2*(1), 46-58.

Holmes, M. R., Yoon, S., Voith, L. A., Kobulsky, J. M., & Steigerwald, S. (2015). Resilience in physically abused children: Protective factors for aggression. *Behavioral Sciences*, *5*(2), 176-189.

Howell, K. H., & Miller-Graff, L. E. (2014). Protective factors associated with resilient functioning in young adulthood after childhood exposure to violence. *Child Abuse & Neglect*, *38*(12), 1985-1994.

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1-55.

Hunter, R. G., Gray, J. D., & McEwen, B. S. (2018). The neuroscience of resilience. *Journal of the Society for Social Work and Research*, *9*(2), 305-339.

Hurren, E., Stewart, A., & Dennison, S. (2017). Transitions and turning points revisited: A replication to explore child maltreatment and youth offending links within and across Australian cohorts. *Child Abuse & Neglect*, *65*, 24-36.

Hussey, J. M., Chang, J. J., & Kotch, J. B. (2006). Child maltreatment in the United States: Prevalence, risk factors, and adolescent health consequences. *Pediatrics*, *118*(3), 933-942.

Ioannidis, K., Askelund, A. D., Kievit, R. A., & Van Harmelen, A. L. (2020). The complex neurobiology of resilient functioning after childhood maltreatment. *BMC Medicine*, *18*(1), 1-16.

Ireland, T. O., Smith, C. A., & Thornberry, T. P. (2002). Developmental issues in the impact of child maltreatment on later delinquency and drug use. *Criminology*, *40*(2), 359-400.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Polo-Tomas, M., & Taylor, A. (2007). Individual, family, and neighborhood factors distinguish resilient from non-resilient maltreated children: A cumulative stressors model. *Child Abuse & Neglect*, *31*(3), 231-253.

Jaffee, S. R. (2017). Child maltreatment and risk for psychopathology in childhood and adulthood. *Annual Review of Clinical Psychology*, *13*, 525-551.

Kalisch, R., Baker, D. G., Basten, U., Boks, M. P., Bonanno, G. A., Brummelman, E., ... & Geuze, E. (2017). The resilience framework as a strategy to combat stress-related disorders. *Nature Human Behaviour*, *1*(11), 784-790.

Keers, R., & Pluess, M. (2017). Childhood quality influences genetic sensitivity to environmental influences across adulthood: A life-course Gene× Environment interaction study. *Development and Psychopathology*, *29*(5), 1921-1933.

Kelifa, M. O., Yang, Y., Carly, H., Bo, W., & Wang, P. (2020). How adverse childhood experiences relate to subjective wellbeing in college students: The role of resilience and depression. *Journal of Happiness Studies*, *0*, 1-21.

Keller, M. C. (2014). Gene× environment interaction studies have not properly controlled for potential confounders: The problem and the (simple) solution. *Biological Psychiatry*, *75*(1), 18-24.

Kim, J., & Cicchetti, D. (2003). Social self-efficacy and behavior problems in maltreated and nonmaltreated children. *Journal of Clinical Child and Adolescent Psychology*, *32*(1), 106-117.

Klika, J. B., & Herrenkohl, T. I. (2013). A review of developmental research on resilience in maltreated children. *Trauma, Violence, & Abuse*, *14*(3), 222-234.

Leung, D. Y., Chan, A. C., & Ho, G. W. (2020). Resilience of emerging adults after adverse childhood experiences: A qualitative systematic review. *Trauma, Violence, & Abuse*, 1-19.

Liu, J. J., Ein, N., Gervasio, J., Battaion, M., Reed, M., & Vickers, K. (2020). Comprehensive meta-analysis of resilience interventions. *Clinical Psychology Review*, *82*, 101919.

Lösel, F., & Bender, D. (2014). Aggressive, delinquent, and violent outcomes of school bullying: Do family and individual factors have a protective function?. *Journal of School Violence*, *13*(1), 59-79.

Luthar, S. S., & Cicchetti, D. (2000). The construct of resilience: Implications for interventions and social policies. *Development and Psychopathology*, *12*(4), 857.

Luthar, S. S., & Eisenberg, N. (2017). Resilient adaptation among at-risk children: Harnessing science toward maximizing salutary environments. *Child Development*, *88*(2), 337-349.

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, *71*(3), 543-562.

Luthar, S. S. (2003). *Resilience and Vulnerability: Adaptation in the Context of Childhood Adversity*. Cambridge University Press, Cambridge, UK.

Maas, C., Herrenkohl, T. I., & Sousa, C. (2008). Review of research on child maltreatment and violence in youth. *Trauma, Violence, & Abuse*, *9*(1), 56-67.

Malvaso, C. G., Delfabbro, P., & Day, A. (2018). The maltreatment–offending association: A systematic review of the methodological features of prospective and longitudinal studies. *Trauma, Violence, & Abuse*, *19*(1), 20-34.

Manchikanti Gómez, A. (2011). Testing the cycle of violence hypothesis: Child abuse and adolescent dating violence as predictors of intimate partner violence in young adulthood. *Youth & Society*, *43*(1), 171-192.

Maples, L. A., Park, S. S., Nolen, J. P., & Rosén, L. A. (2014). Resilience to childhood abuse and neglect in college students. *Journal of Aggression, Maltreatment & Trauma*, *23*(10), 1001-1019.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491-495.

Masten, A. S., & Coatsworth, J. D. (1998). The development of competence in favorable and unfavorable environments: Lessons from research on successful children. *American Psychologist*, *53*(2), 205.

Masten, A. S., & Powell, L. (2003). A resilience framework for research, Policy. In S.S. Luthar (Eds.), *Resilience and Vulnerability: Adaptation in the Context of Childhood Adversities*, (pp. 1-29). Cambridge University Press.

Masten, A. S., & Tellegen, A. (2012). Resilience in developmental psychopathology: Contributions of the project competence longitudinal study. *Development and Psychopathology*, *24*(2), 345-361.

McEwen, B. S., Bowles, N. P., Gray, J. D., Hill, M. N., Hunter, R. G., Karatsoreos, I. N., & Nasca, C. (2015). Mechanisms of stress in the brain. *Nature Neuroscience*, *18*(10), 1353-1363.

Meng, X., Fleury, M. J., Xiang, Y. T., Li, M., & D'arcy, C. (2018). Resilience and protective factors among people with a history of child maltreatment: A systematic review. *Social Psychiatry and Psychiatric Epidemiology*, *53*(5), 453-475.

Moreno-López, L., Ioannidis, K., Askelund, A. D., Smith, A. J., Schueler, K., & Van Harmelen, A. L. (2020). The resilient emotional brain: A scoping review of the medial prefrontal cortex and limbic structure and function in resilient adults with a history of childhood maltreatment. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *5*(4), 392-402.

Morris, M. C., Marco, M., Maguire-Jack, K., Kouros, C. D., Bailey, B., Ruiz, E., & Im, W. (2019). Connecting child maltreatment risk with crime and neighborhood disadvantage across time and place: A Bayesian spatiotemporal analysis. *Child Maltreatment*, *24*(2), 181-192.

Muthén, L.K. and Muthén, B.O. (1998-2017). Mplus User's Guide. Eighth Edition. Los Angeles, CA: Muthén & Muthén.

Muthén, L. K., & Muthén, B. (2010). Growth modeling with latent variables using Mplus: Introductory and intermediate growth models. *Mplus Short Course Topic 3*.

Nasvytienė, D., Lazdauskas, T., & Leonavičienė, T. (2012). Child's resilience in face of maltreatment: A meta-analysis of empirical studies. *Psychology*, *46*(46), 7-26.

Newsome, J., & Sullivan, C. J. (2014). Resilience and vulnerability in adolescents: Genetic influences on differential response to risk for delinquency. *Journal of Youth and Adolescence*, *43*(7), 1080-1095.

Newsome, J., Vaske, J. C., Gehring, K. S., & Boisvert, D. L. (2016). Sex differences in sources of resilience and vulnerability to risk for delinquency. *Journal of Youth and Adolescence*, *45*(4), 730-745.

Opel, N., Redlich, R., Repple, J., Kaehler, C., Grotegerd, D., Dohm, K., ... & Leehr, E. J. (2019). Childhood maltreatment moderates the influence of genetic load for obesity on reward related brain structure and function in major depression. *Psychoneuroendocrinology*, *100*, 18-26.

Oshri, A., Duprey, E. B., Kogan, S. M., Carlson, M. W., & Liu, S. (2018). Growth patterns of future orientation among maltreated youth: A prospective examination of the emergence of resilience. *Developmental Psychology*, *54*(8), 1456.

Osório, C., Probert, T., Jones, E., Young, A. H., & Robbins, I. (2017). Adapting to stress: Understanding the neurobiology of resilience. *Behavioral Medicine*, *43*(4), 307-322.

Pérez-González, A., Guilera, G., Pereda, N., & Jarne, A. (2017). Protective factors promoting resilience in the relation between child sexual victimization and internalizing and externalizing symptoms. *Child Abuse & Neglect*, *72*, 393-403.

Peterson, C., Florence, C., & Klevens, J. (2018). The economic burden of child maltreatment in the United States, 2015. *Child Abuse & Neglect*, *86*, 178-183.

Pezzoli, P., Antfolk, J., Hatoum, A. S., & Santtila, P. (2019). Genetic vulnerability to experiencing child maltreatment. *Frontiers in Genetics*, *10*, 852.

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*(2), 309.

Plomin, R., & Deary, I. J. (2015). Genetics and intelligence differences: Five special findings. *Molecular Psychiatry*, *20*(1), 98-108.

Plomin, R., Haworth, C. M., & Davis, O. S. (2009). Common disorders are quantitative traits. *Nature Reviews Genetics*, *10*(12), 872-878.

Polderman, T. J., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, *47*(7), 702-709.

Powell, D. R. (1999). *Early childhood development*. In A. J. Reynolds, H. J. Walberg, & R. P. Weissberg (Eds.), *Promoting positive outcomes: Issues in children's and families' lives* (pp. 45–71). Washington, DC: Child Welfare League of America

Ram, N., & Grimm, K. (2007). Using simple and complex growth models to articulate developmental change: Matching theory to method. *International Journal of Behavioral Development*, *31*(4), 303-316.

Reckdenwald, A., Mancini, C., & Beauregard, E. (2013). The cycle of violence: Examining the impact of maltreatment early in life on adult offending. *Violence and Victims*, *28*(3), 466-482.

Rodman, A. M., Jenness, J. L., Weissman, D. G., Pine, D. S., & McLaughlin, K. A. (2019). Neurobiological markers of resilience to depression following childhood maltreatment: The role of neural circuits supporting the cognitive control of emotion. *Biological Psychiatry*, *86*(6), 464-473.

Samplin, E., Ikuta, T., Malhotra, A. K., Szeszko, P. R., & DeRosse, P. (2013). Sex differences in resilience to childhood maltreatment: Effects of trauma history on hippocampal volume, general cognition and subclinical psychosis in healthy adults. *Journal of Psychiatric Research*, *47*(9), 1174-1179.

Scoglio, A. A., Kraus, S. W., Saczynski, J., Jooma, S., & Molnar, B. E. (2019). Systematic review of risk and protective factors for revictimization after child sexual abuse. *Trauma, Violence, & Abuse*, 1-13.

Sedlak, A. J., Mettenburg, J., Basena, M., Peta, I., McPherson, K., & Greene, A. (2010). *Fourth national incidence study of child abuse and neglect (NIS-4)*. Washington, DC: US Department of Health and Human Services, *9*.

Shaffer, A., Yates, T. M., & Egeland, B. R. (2009). The relation of emotional maltreatment to early adolescent competence: Developmental processes in a prospective study. *Child Abuse & Neglect*, *33*(1), 36-44.

Sheerin, C. M., Lind, M. J., Brown, E. A., Gardner, C. O., Kendler, K. S., & Amstadter, A. B. (2018). The impact of resilience and subsequent stressful life events on MDD and GAD. *Depression and Anxiety*, *35*(2), 140-147.

Starr, L. R., Stroud, C. B., Shaw, Z. A., & Vrshek-Schallhorn, S. (2020). Stress sensitization to depression following childhood adversity: Moderation by HPA axis and serotonergic multilocus profile scores. *Development and Psychopathology*, 1-15.

Stewart, A., Livingston, M., & Dennison, S. (2008). Transitions and turning points: Examining the links between child maltreatment and juvenile offending. *Child Abuse & Neglect*, *32*(1), 51-66.

Sullivan, C. J., & Newsome, J. (2015). Psychosocial and genetic risk markers for longitudinal trends in delinquency: An empirical assessment and practical discussion. *Criminal Justice Studies*, *28*(1), 61-83.

Tabibnia, G. (2020). An affective neuroscience model of boosting resilience in adults. *Neuroscience & Biobehavioral Reviews*. Journal Pre-Proof.

Thibodeau, E. L., Cicchetti, D., & Rogosch, F. A. (2015). Child maltreatment, impulsivity, and antisocial behavior in african-american children: Moderation effects from a cumulative dopaminergic gene index. *Development and Psychopathology*, *27*(4-2), 1621.

Thibodeau, E. L., Masyn, K. E., Rogosch, F. A., & Cicchetti, D. (2019). Child maltreatment, adaptive functioning, and polygenic risk: A structural equation mixture model. *Development and Psychopathology*, *31*(2), 443.

Thompson, M. P., Kaslow, N. J., Lane, D. B., & Kingree, J. B. (2000). Childhood maltreatment, PTSD, and suicidal behavior among African American females. *Journal of Interpersonal Violence*, *15*(1), 3-15.

Thornberry, T. P., Henry, K. L., Ireland, T. O., & Smith, C. A. (2010). The causal impact of childhood-limited maltreatment and adolescent maltreatment on early adult adjustment. *Journal of Adolescent Health*, *46*(4), 359-365.

Tillyer, M. S. (2015). The relationship between childhood maltreatment and adolescent violent victimization. *Crime & Delinquency*, *61*(7), 973-995.

Tung, I., Noroña, A. N., & Lee, S. S. (2019). Childhood maltreatment affects adolescent sensitivity to parenting and close friendships in predicting growth in externalizing behavior. *Development and Psychopathology*, *31*(4), 1237-1253.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*(5), 160-164.

Ungar, M., Ghazinour, M., & Richter, J. (2013). Annual research review: What is resilience within the social ecology of human development?. *Journal of Child Psychology and Psychiatry*, *54*(4), 348-366.

Ungar, M. (2013). Resilience after maltreatment: The importance of social services as facilitators of positive adaptation. *Child Abuse & Neglect*, *37*(2-3), 110-115.

United States. (2010). CAPTA Reauthorization Act of 2010: Report (to accompany S. 3817). Washington, D.C.: U.S. G.P.O.

U.S. Department of Health & Human Services (2020). *Child Maltreatment 2018*. Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau. Retrieved from: https://www.acf.hhs.gov/cb/research-data-technology/statistics-research/child-maltreatment.

van der Put, C. E., & De Ruiter, C. (2016). Child maltreatment victimization by type in relation to criminal recidivism in juvenile offenders. *BMC Psychiatry*, *16*(1), 1-9.

van der Put, C. E., Lanctôt, N., De Ruiter, C., & Van Vugt, E. (2015). Child maltreatment among boy and girl probationers: Does type of maltreatment make a difference in offending behavior and psychosocial problems?. *Child Abuse & Neglect*, *46*, 142-151.

van der Werff, S. J., Pannekoek, J. N., Veer, I. M., van Tol, M. J., Aleman, A., Veltman, D. J., ... & van der Wee, N. J. (2013). Resilience to childhood maltreatment is associated with increased resting-state functional connectivity of the salience network with the lingual gyrus. *Child Abuse & Neglect*, *37*(11), 1021-1029.

Vanderbilt-Adriance, E., Shaw, D. S., Brennan, L. M., Dishion, T. J., Gardner, F., & Wilson, M. N. (2015). Child, family, and community protective factors in the development of children's early conduct problems. *Family Relations*, *64*(1), 64-79.

von Stumm, S., & d'Apice, K. (2021). From genome-wide to environment-wide: Capturing the environome. *Perspectives on Psychological Science*, 1745691620979803.

Waaktaar, T., & Torgersen, S. (2012). Genetic and environmental causes of variation in trait resilience in young people. *Behavior Genetics*, *42*(3), 366-377.

Walsh, W. A., Dawson, J., & Mattingly, M. J. (2010). How are we measuring resilience following childhood maltreatment? Is the research adequate and consistent? What is the impact on research, practice, and policy? *Trauma, Violence, & Abuse*, *11*(1), 27-41.

Wells, J., Armstrong, T., Boisvert, D., Lewis, R., Gangitano, D., & Hughes-Stamm, S. (2017). Stress, genes, and generalizability across gender: Effects of MAOA and stress sensitivity on crime and delinquency. *Criminology*, *55*(3), 548-574.

Widom, C. S. (1989). The cycle of violence. *Science*, *244*(4901), 160-166.

Widom, C. S., & Brzustowicz, L. M. (2006). MAOA and the "cycle of violence:" Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry*, *60*(7), 684-689.

Widom, C. S., Czaja, S. J., & Dutton, M. A. (2008). Childhood victimization and lifetime revictimization. *Child Abuse & Neglect*, *32*(8), 785-796.

Wright, M. O. D., Crawford, E., & Del Castillo, D. (2009). Childhood emotional maltreatment and later psychological distress among college students: The mediating role of maladaptive schemas. *Child Abuse & Neglect*, *33*(1), 59-68.

Wolf, E. J., Miller, M. W., Sullivan, D. R., Amstadter, A. B., Mitchell, K. S., Goldberg, J., & Magruder, K. M. (2018). A classical twin study of PTSD symptoms and resilience: Evidence for a single spectrum of vulnerability to traumatic stress. *Depression and Anxiety*, *35*(2), 132-139.

Wright, E. M., & Fagan, A. A. (2013). The cycle of violence in context: Exploring the moderating roles of neighborhood disadvantage and cultural norms. *Criminology*, *51*(2), 217-249.

Yoon, S., Howell, K., Dillard, R., McCarthy, K. S., Napier, T. R., & Pei, F. (2019). Resilience following child maltreatment: Definitional considerations and developmental variations. *Trauma, Violence, & Abuse*, 1-19.

Yoon, S., Maguire-Jack, K., Knox, J., & Ploss, A. (2020). Socio-ecological predictors of resilience development over time among youth with a history of maltreatment. *Child Maltreatment*, 1077559520981151.

Yoon, D., Yoon, S., Park, J., & Yoon, M. (2018). A pernicious cycle: Finding the pathways from child maltreatment to adolescent peer victimization. *Child Abuse & Neglect*, *81*, 139-148.

Yu, M., Huang, L., Mao, J., & Luo, S. (2020). Childhood maltreatment, automatic negative thoughts, and resilience: The protective roles of culture and genes. *Journal of Interpersonal Violence*, 1-22.

Yule, K., Houston, J., & Grych, J. (2019). Resilience in children exposed to violence: A meta-analysis of protective factors across ecological contexts. *Clinical Child and Family Psychology Review*, *22*(3), 406-431.

Zingraff, M. T., Leiter, J., Johnsen, M. C., & Myers, K. A. (1994). The mediating effect of good school performance on the maltreatment-delinquency relationship. *Journal of Research in Crime and Delinquency*, *31*(1), 62-91.

VITA

Eric M. Cooke
College of Criminal Justice
Department of Criminal Justice and Criminology
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Education

2021 Ph.D., Criminal Justice and Criminology, Sam Houston State University
Dissertation: *Polygenic Resilience on the Association between Childhood Maltreatment, Delinquency, and Victimization*
Chair: Dr. Eric J. Connolly

2016 M.A., Criminal Justice and Criminology, Sam Houston State University
Thesis: *A Multi-Group Confirmatory Factor Analysis of the Reactive-Proactive Aggression Questionnaire in a Sample of Young Adults*
Chair: Dr. Todd Armstrong

2012B.A., Miami University, Psychology

Research and Teaching Interests

- Biopsychosocial Criminology
- Life-Course Criminology
- Quantitative Research Methods
- Victimology

Peer Reviewed Articles

Armstrong, T., Wells, J., Boisvert, D., Lewis, R., **Cooke, E.**, Woeckener, M., & Kavish, N. (2021). An Exploratory Analysis of Testosterone, Cortisol, and Aggressive Behavior Type in Men and Women. *Biological Psychology,* Accepted for publication.

Connolly, E. J., Hayes, B. E., Boisvert, D. L., & **Cooke, E. M.** (2021). Intimate Partner Victimization and Depressive Symptoms: Approaching Causal Inference Using a Longitudinal Twin Design. *Journal of Quantitative Criminology*. Accepted for publication.

Cooke, E. M., Lewis, R. H., Hayes, B. E., Bouffard, L. A., Boisvert, D. L., Wells, J., Kavish, N., Woeckener, M., & Armstrong, T. (2020). Examining the Relationship between Victimization, Psychopathy, and the Acceptance of Rape Myths. *Journal of Interpersonal Violence*. Ahead of Print. 0.1177/0886260520966669.

Cooke, E. M., Connolly, E. J., Boisvert, D. L., Armstrong, T. A., Kavish, N., Lewis, R. H., Wells, J., Woeckener, M., & Harper, J. (2020). Examining How Testosterone and Cortisol Influence the Relationship between Strain, Negative Emotions, and Antisocial Behavior: A Gendered Analysis. *Crime & Delinquency*, *66*, 1470-1501.

Connolly, E. J., **Cooke, E. M.**, Beaver, K. M., & Wyatt, B. (2020). Do Developmental Changes in Impulsivity and Sensation Seeking Uniquely Predict Violent Victimization? A Test of the Dual Systems Model. *Journal of Criminal Justice*, *66*, 101639.

Armstrong, T. A., Boisvert, D. L., Wells, J., Lewis, R. H., & **Cooke, E. M.**, & Woeckener, M. (2020). Assessing Overlap between Self-Control and Psychopathy: A Consideration of the Grasmick Self-Control Scale and the Levenson Self-Report Psychopathy Scale. *Journal of Criminal Justice*, *70*, 101725.

Cooke, E. M., & Zhang, Y. (2019). A Multilevel Analysis of Factors Related to Business Robbery Clearance Rates in a Large City in Texas. *Policing: An International Journal*, *43*, 213-228.

Kavish, N., Boisvert, D.L., **Cooke, E. M.**, Lewis, R. H., Woeckener, M., Wells, J., & Armstrong, T. A. (2020). Further Evaluating the Associations between Psychopathic Traits and Symptoms of PTSD and Depression. *Journal of Personality Disorders, 34,* 467-479.

Armstrong, T. A., Wells, J., Boisvert, D. L., Lewis, R. H., **Cooke, E. M.**, Woeckener, M., & Kavish, N. (2018). Skin Conductance, Heart Rate, and Aggressive Behavior Type. *Biological Psychology, 141,* 44-51.

Cooke, E. M., Armstrong, T. A., Boisvert, D. L., Wells, J., Lewis, R. H., Hughes-Stamm, S., & Gangitano, D. (2018). The Relationship between the MAOA-uVNTR Polymorphism, Delinquent Peer Affiliation, and Antisocial Behavior with a Consideration of Sex Differences. *Psychiatric Quarterly*, *89*, 841-853.

Connolly, E. J., Kavish, N., & **Cooke, E. M.** (2018). Testing the Causal Hypothesis that Repeated Bullying Victimization Leads to Lower Levels of Educational Attainment: A Sibling-Comparison Analysis. *Journal of School Violence*, *18*, 272-284.

Kavish, N., Boisvert, D. L., Wells, J., Lewis, R. H., **Cooke, E. M.**, Woeckener, M., & Armstrong, T. A. (2018). On the Associations between Indicators of Resting Arousal Levels, Physiological Reactivity, Sensation Seeking, and Psychopathic Traits. *Personality and Individual Differences*, *141*, 218-225.

Woeckener, M., Boisvert, D. L., **Cooke, E. M.**, Kavish, N., Lewis, R. H., Wells, J., Armstrong, T. A., Connolly, E. J., & Harper, J. (2018). Parental Rejection and Antisocial Behavior: The Moderating Role of Testosterone. *Journal of Criminal Psychology*, *8*, 302-313.

Articles Under Review

Cooke, E. M., Connolly, E. J., Boisvert, D. L., & Hayes, B. E. A Systematic Review of Biological Mechanisms and Interpersonal Victimization: Understanding the Implications of Biopsychosocial Research for Victimization Research. Revise and resubmit.

Randa, R., **Cooke, E. M.**, Boisvert, D. L., & Vaske, J. (2020). Vulnerability, Risky Lifestyles, and Victimization: A Gender Based Sibling Differences Approach. Revise and resubmit.

Wells, J., Armstrong, T. A., **Cooke, E. M.**, & Boisvert, D. L. (2020). Psychopathy and Aggression: Dimensionality of Psychopathy and Reactive/Proactive Aggression across Gender. Under review.

Woeckener, M., Lewis, R. H., Wells, J., Boisvert, D. L., Connolly, E. J., **Cooke, E. M.**, Kavish, N., & Armstrong, T. A. (2020). Does Heart Rate Reactivity Moderate the Influence of Emotional Child Abuse on Violent Behavior? A Gender-Based Analysis. Revise and resubmit.

Articles in Progress

Chumchal, M. J., Connolly, E. J., **Cooke, E. M.**, Boisvert, D. L., Wells, J., Lewis, R. H., Armstrong, T. A. (2020). Focusing on the Trees to Understand the Forest: Examining How Different Components of Self-Control and Psychopathy are Associated with Antisocial Behavior.

Comer, B. P., Wells, J., Boisvert, D. L., & **Cooke, E. M.** (2020). Consequences of Gender-Based Abuse: Trauma Symptomology among Victims/Survivors of Sexual Assault and Intimate Partner Violence.

Connolly, E. J. & **Cooke, E. M.** (2020). Gene x Environment Interaction between Gang Membership and Developmental Patterns of Desistance.

Connolly, E. J., **Cooke, E. M.**, & Smith, W. (2020). Are the Pathways to Criminal Justice Contact the Same for Males and Females? A Test of Competing Developmental Cascade Models.

Connolly, E. J., Jackson, D. B., & **Cooke, E. M.** (2020). Restless Sleep, Sleep Duration, and Delinquency: Unraveling Associations Using Sibling Comparisons.

Cooke, E. M., Boisvert, D. L., Armstrong, T., Kavish, N., Lewis, R. H., Wells, J., Woeckener, M., & Harper, J. (2020). Testing a Dual Hormone Model of

Psychopathy and Rape Myth Acceptance: Examining the Relationship between Psychopathy, Testosterone, and Cortisol on Rape Myth Acceptance.

Cooke, E. M., & Connolly, E. J. (2020). Using Bayesian Methods to Expand the Evidence on the Relationship between Dimensions of Self-Control and Victimization.

Kelly, S., Connolly, E. J., & **Cooke, E. M.** (2020). Genetic and Environmental Influences on Parenting and Risky Sexual Behavior in Adolescents.

Perez, K., Boisvert, D. L., & **Cooke, E. M.** (2020). The Influence of Prenatal Androgen Exposure on Psychopathy.

Royle, M. L., Boisvert, D. L., Armstrong, T., Wells, J., Lewis, R. H., & **Cooke, E. M.** (2020). The Relationship Between Heart Rate Reactivity, Antisocial Behavior, and Alcohol Use.

Book Chapters

Wells, J., **Cooke, E. M.**, & Marshall, E. (2020). *The Cambridge Handbook of Evolutionary Perspectives on Sexual Psychology: Female Sexual Offending* (T. K. Schakelford, Ed.). Cambridge University Press, Cambridge, UK.

Editorials

Goodson, A., **Cooke, E. M.**, & Hinojosa, I. (2016, October 11). Countering ignorance with evidence: The invisible nature of sexual assault and rape culture. *The Houstonian*. Retrieved from http://houstonianonline.com/2016/10/11/countering-ignorance-with-evidence-the-invisible-nature-of-sexual-assault-and-rape-culture/.

Grants and Sponsored Research

2019-2020

Co-PI, *Sexual Aggression and Biological Markers: An Investigation of Pornography Use, Sexual Aggression, and Biological Factors*. PI: Holly Miller. Internal Sam Houston State University grant. Not funded.

PI, *Risk and Resiliency Factors for Violent Criminal Behavior across the Life Course for Males and Females*. National Institute of Justice (NIJ-2019-90134-TX-R2), $68,991. Not funded.

PI, *Risk and Resiliency Factors for Violent Criminal Behavior across the Life Course for Males and Females*. Bureau of Justice Statistics (BJS-2019-16229), $40,000. Not funded.

2017-2018

PI, *Risk and Resiliency Factors for Violent Criminal Behavior across the Life Course for Males and Females*. National Institute of Justice (NIJ-2018-90082-TX-R2), $32,000. Not funded.

Interviewer, *Measuring the Effects of Correctional Officer Stress on the Well-Being of the Officer and the Prison Workplace and Developing a Practical Index of Officer Stress for Use by Correctional Agencies*. PI: John Hepburn; Co-PI/Texas Site Coordinator: Melinda Tasca; Co-PI/Texas Site Co-Coordinator: H. Daniel Butler. National Institute of Justice (Award No. 2014-IJ-CX-0026), $666,268. Funded.

2015-2016

Co-PI, *Student Wave III Data Collection*. PI: Todd Armstrong & Danielle Boisvert. Co-PI: Jessica Wells; Richard Lewis; Matthias Woeckener. Sam Houston State University Office of Research and Sponsored Programs, $50,000. Funded.

Interviewer, *Gangs on the Street, Gangs in Prison: Their Nature, Interrelationship, Control, and Re-Entry*. PI: Scott Decker; Co-PI: David Pyrooz. National Institute of Justice, Research on Gangs and Gang Violence (2014-MU-CX-0111), $840,807. Funded.

Academic Presentations

Cooke, E. M., Connolly, E. J., (November, 2020). Using Bayesian Methods to Expand the Evidence on the Relationship between Dimensions of Self-Control and Victimization. Washington, DC.

Cooke, E. M., Boisvert, D. L., Connolly, E. J. (November, 2019). Risk and Resilience for Criminal Behavior Across the Life Course. American Society of Criminology. San Francisco, CA.

Cooke, E. M. & Zhang, Y. (March, 2019). A Multilevel Analysis of Factors Related to Business Robbery Clearance Rates in a Large City in Texas. Baltimore, MD.

Cooke, E. M., Connolly, E. J., Boisvert, D. L., Armstrong, T. A., Lewis, R. H., Kavish, N., Woeckener, M., Wells, J., & Harper, J. (November, 2018). A Biosocial Test of General Strain Theory: How Testosterone and Cortisol Influence the Relationship between Strain and Anger on Antisocial Behavior. American Society of Criminology. Atlanta, GA.

Cooke, E. M. (November, 2017). The Relationship between Psychopathic Personality Traits and Rape Myth Acceptance across Gender. American Society of Criminology. Philadelphia, PA.

Cooke, E. M., Armstrong, T. A., & Wells, J. (November, 2016). Gene-Environment Interactions: The Relationship between MAOA Polymorphism, Peer Affiliation, and Antisocial Behavior. American Society of Criminology. New Orleans, LA.

Cooke, E. M., & Armstrong, T. A. (November, 2015). Validation of an Aggression Measurement Tool across Gender and Ethnicity. American Society of Criminology. Washington, D.C.

Teaching Experience

Undergraduate

2020-2021

Introduction to Methods of Research

2019-2020

Introduction to Methods of Research

2018-2019

Introduction to Methods of Research

Academic Awards

2018-2019

Graduate Studies General Scholarship, Sam Houston State University

2017-2018

Recipient of Rolando, Josefa, and Jocelyn del Carmen Criminal Justice Scholarship, Sam Houston State University

Recipient of Summer Research Fellowship, Department of Criminal Justice and Criminology, Sam Houston State University

2016-2017

Recipient of Summer Research Fellowship, Department of Criminal Justice and Criminology, Sam Houston State University

Professional Development

2020-2020

Data Science: R Basics, edX: HarvardX

Introduction to Bayesian Statistics, University of Texas at Austin

2019-2020

ACUE Course in Effective Teaching Practices, Sam Houston State University
2018-2019

Annual SHSU Teaching and Learning Conference, Sam Houston State University
Teaching Online with Blackboard Certification, Sam Houston State University

Professional Memberships

Academy of Criminal Justice Sciences
American Society of Criminology
Division of Biopsychosocial Criminology
Division of Developmental/Life-Course Criminology
Division of Victimology
Behavior Genetics Association
Biosocial Criminology Association

Ad Hoc Reviewer

Criminal Justice and Behavior
International Journal of Offender Therapy and Comparative Criminology
Journal of Criminal Justice Education
Psychoneuroendocrinology