Violence Exposure and Social Deprivation: Neural Connectivity Correlates and Protective Factors

by

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Dedication

To the families who participated the Fragile Families and Child Wellbeing Study. Thank you for sharing your lives for more than 20 years. This dissertation would not exist without them.

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Abstract

Dimensions of early adversity, such as violence exposure and social deprivation, may have different effects associated with socioemotional functioning in the developing brain and different factors may be protective. This dissertation examined the downstream effects of childhood violence exposure and social deprivation in data from the Fragile Families and Child Wellbeing Study at birth, and ages 1, 3, 5, 9, and 15 years. Study one examined the association between violence exposure, social deprivation, and amygdala-prefrontal cortex white matter connectivity, a crucial circuit for emotion regulation. High violence exposure coupled with high social deprivation related to less amygdala–OFC white matter connectivity. Violence exposure was not associated with white matter connectivity when social deprivation was at mean or low levels (i.e., relatively socially supportive contexts). Therefore, social deprivation may exacerbate the effects of childhood violence exposure on the development of white matter connections involved in emotion processing and regulation. Conversely, social support may buffer against them. Study two investigated the association between violence exposure, social deprivation, and adolescent resting-state functional connectivity in two resting-state networks involved in socioemotional functioning (salience network, default mode network) using a person-specific modeling approach. Childhood violence exposure, but not social deprivation, was associated with reduced adolescent resting-state density of the salience and default mode networks. A data-driven algorithm, blinded to childhood adversity, identified youth with heightened violence exposure based on resting-state connectivity patterns. Childhood violence exposure was associated with adolescent functional connectivity heterogeneity, which may reflect person-

specific neural plasticity and should be considered when attempting to understand the impacts of early adversity on the brain. Study three examined whether school connectedness was protective against violence exposure and social deprivation when predicting symptoms of internalizing and externalizing psychopathology and positive function and if school connectedness was uniformly protective against both dimensions of adversity. Results suggest that school connectedness is broadly related to better outcomes and may confer additional protection against social deprivation. These findings highlight the important role that the school environment can play for youth who have been exposed to adversity in other areas of their lives. Additionally, the interactive effect of school connectedness with social deprivation, but not violence exposure, supports modeling risk and resilience processes using dimensional frameworks to better identify specific groups of youth that may benefit from interventions that boost social connectedness at school in future research. Overall, this dissertation provides evidence for the complex and person-specific ways through which risk and resilience relate to development and points to considerations for future research. This research has implications for understanding how dimensions of adversity affect the brain and behavior during development and what factors can be protective, which can inform future neuroscience-informed policy interventions.

Chapter 1: Introduction

Early adversity is a potent and unfortunately common public health concern that has impacts on physical and mental health during childhood, adolescence, and throughout the lifespan (Green et al., 2010). Altered neural development is a hypothesized mechanism for the effects of early adversity (McLaughlin et al., 2014). This dissertation examines how childhood exposure to adversity shapes the adolescent brain using computationally sophisticated methods that more accurately assess neural development. Importantly, many children exposed to early adversity do not experience negative outcomes (Masten, 2001). Thus, this dissertation also aims to identify protective factors that promote resilience. This research has implications for understanding how dimensions of adversity affect the brain during development and what factors can be protective, which can inform future neuroscience-informed policy interventions.

Adverse childhood experiences (ACEs) consist of a broad set of negative exposures during childhood including physical and sexual abuse, emotional and physical neglect, violence in the home and neighborhood, institutional rearing, and chronic poverty (Felitti et al., 1998; Hughes et al., 2017). According to recent statistics, approximately 58% percent of adolescents experience one ACE, 1 in 4 children experience some form of child maltreatment, and approximately 40% are exposed to violence in the home or neighborhood (Finkelhor et al., 2015; McLaughlin, Greif Green, et al., 2012; Sacks & Murphy, 2018). These statistics are particularly alarming due to the associations of experiences of early adversity with a whole host of negative physical health outcomes, such as metabolic syndrome, heart disease, autoimmune conditions, and some cancers, as well as mental health outcomes, such as increased risk for internalizing

disorders (i.e., anxiety and depression), externalizing disorder, and substance use disorders (Green et al., 2010; Nusslock & Miller, 2016). This increased risk is present in childhood and persists through adolescence into adulthood (Green et al., 2010).

Exposure to early adversity impacts individual development in complex, multidimensional ways. Seminal theories of development and developmental psychopathology highlight the influence of the ongoing, reciprocal interactions between an individual and their environment, or context, throughout the lifespan in the development of adaptive and maladaptive outcomes (Bronfenbrenner & Morris, 2007; Cicchetti & Lynch, 1993; Masten & Cicchetti, 2010; Sameroff, 2010; Sroufe, 2009). Environmental risk and protective factors come from multiple, nested systems ranging from micro (e.g., parenting behaviors) to macro (e.g., societal attitudes and ideologies) (Bronfenbrenner & Morris, 2007). These contextual influences interact with individual-level biological and psychological variables to shape patterns of biological, socio-emotional and cognitive development (Masten & Cicchetti, 2010; Sroufe, 2009; Wiggins & Monk, 2013). The complex and probabilistic way through which adversity impacts the individual explains the existence of multiple different approaches that attempt to shed light on how early adversity shapes development.

The prevailing approach to studying early adversity has been the cumulative risk model (Evans et al., 2013; Felitti et al., 1998; Sameroff et al., 1987). In this approach, the number of ACEs that a person has experienced are tallied to create an index of cumulative risk across multiple domains. The hypothesized physiological mechanism through which adversity affects the brain and behavior is the body's general stress response, including the hypothalamic-pituitary-adrenal axis (HPA axis) (Evans & Kim, 2007; Koss & Gunnar, 2018). In the normal HPA axis response to stress, the hypothalamus releases corticotropin-releasing factor, which

binds to receptors in the pituitary gland releasing adrenocorticotropic hormone (ACTH). ACTH then binds to receptors in the adrenal gland which stimulates the release of cortisol, a commonly measured stress hormone (Hyman, 2009). The allostatic load model describes how this normal, healthy response to stress theoretically becomes harmful (McEwen, 1998). Allostasis is adaptation in the face of potentially stressful challenges which involves activation of neural, neuroendocrine and neuroendocrine-immune mechanisms. These allostatic processes are adaptive in many cases. However, with chronic exposure to multiple ACEs, allostatic systems may either be overstimulated or not perform normally, which has been termed *allostatic load*, or the price of adaptation. Over time, chronic high allostatic load is posited to lead to the negative health outcomes associated with early adversity (McEwen, 1998).

The cumulative risk framework has been instrumental in predicting which kids may be at an increased risk for negative outcomes and has underscored the importance of preventing exposure to early adversity when possible (Sameroff, 1999). However, it does not aim to identify the mechanism(s) through which early adversity can "get under the skin" and influence neural developmental (McLaughlin et al., 2014; Schilling et al., 2008). Additionally, there is considerable heterogeneity in the effect of early adversity on later physical and mental health outcomes (Hughes et al., 2017). Part of this heterogeneity in outcomes may be the result of treating all ACEs as the same and, perhaps, taking a more precise approach may help to parse not only the mechanisms of influence, but also the heterogeneity of effects.

Focusing on specific types or categories of experience is another approach to studying early adversity which take steps towards identifying mechanisms of influence on neural development. There are multiple ways to do this. One approach is to investigate a single type of adversity. For example, child neglect is hypothesized to affect how the ventral striatum functions

(Hanson, Hariri, et al., 2015) and child abuse is posited to alter the structure and function of brain regions, such as the medial prefrontal cortex (PFC) (Kaufman et al., 2000). However, this does not account for the complex, co-occurring nature of adversities. Additionally, these categorical studies are often conducted with samples where participants are preselected based on exposure to extreme adversity (i.e., institutional rearing) or a history a trauma-related psychopathology (i.e., PTSD) (Hyde et al., 2020; Teicher & Samson, 2016). Research in these samples is necessary and important; however, findings may not be generalizable to populations who experience less extreme, but more common, exposures to adversity (e.g., low-income families) (Hyde et al., 2020; Thapar & Rutter, 2019). Another way is to look at multiple categories of risk simultaneously (Trentacosta et al., 2013). This approach is designed to address multiple sources of exposure to early adversity in a single individual, but categorical exposures are often linked to distinct mechanisms and may miss common mechanisms that exist across multiple types of experiences (McLaughlin et al., in press). An approach that may provide specific insight into distinct and relatively global mechanisms of influence *and* account for the co-occurring, multidimensional nature of early adversity is a dimensional model of adversity (McLaughlin et al., 2014).

Theoretical Framework: Dimensional Model of Early Adversity

Dimensional models of adversity are a relatively recent approach where the complex experiences of early adversity are broken down into core underlying dimensions that have at least partially distinct impacts of development (McLaughlin et al., in press). In the dimensional model of adversity and psychopathology (DMAP), one of the first and more prominent dimensional models, adversity is broken down into the two correlated, but separable, dimensions of threat and deprivation (McLaughlin et al., 2014). Threat is defined as experiences that pose a

threat to an individual's physical integrity, and deprivation is defined as the absence of expected input and complexity in an individual's environment (Sheridan & McLaughlin, 2014). The threat and deprivation dimensions in the DMAP approach are hypothesized to influence brain development through both distinct and potentially more global mechanisms. Hypothesized mechanisms through which dimensions of adversity affect neural development include global stress mechanisms, such as upregulation of the HPA-axis, similar to cumulative risk models. However, dimensions of adversity are also hypothesized to influence development in more specific ways through experience-driven plasticity (McLaughlin et al., in press; Nelson & Gabard-Durnam, 2020). Threat is posited to influence the development of neural structure and function involving fear-learning and emotion regulation circuits including regions such as the amygdala, hippocampus, and ventromedial PFC. Alternatively, deprivation is posited to influence regions of the brain that are responsible for integrating complex social and cognitive stimuli including the PFC, superior/inferior parietal cortex, and superior temporal cortex (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). This theoretical framework emphasizes that, although these are theoretically distinct dimensions, these experiences frequently co-occur, so it is important to include both simultaneously in models predicting effects on the brain and behavior. Additionally, the DMAP framework acknowledges that these are likely not the only dimensions of early adversity and hypothesize that unpredictability of the environment may be another (McLaughlin et al., in press, 2014).

In work building on the DMAP approach, two dimensions of adversity, violence exposure and social deprivation, have been proposed to alter neural structure and function, cortisol function, and behavior in distinct ways (Hein et al., 2020; Peckins et al., 2019). The constructs included in these dimensions aim to account for multiple levels of proximity to the individual

(i.e., home, neighborhood) to more comprehensively index exposure (Figure 1.1.) (Bronfenbrenner & Morris, 2007; Sameroff, 2010). The violence exposure dimension, which is similar to the threat construct in DMAP, includes experiences of abuse, exposure to intimate partner violence in the home, and community violence (Hein et al., 2020). Social deprivation is a more specific construct than the general deprivation dimension in DMAP which includes experiences of neglect, a lack of intimate partner support for the mother in the home, and a lack of neighborhood cohesion (Hein et al., 2020). Social deprivation is posited to influence areas of the brain involved in reward processing, such as the ventral striatum (Hanson, Hariri, et al., 2015; Hein et al., 2020). Violence exposure in childhood, but not social deprivation, has been associated with a blunted amygdala response to threatening stimuli, a reduced habituation, or adaptation, of the amygdala's response to threatening stimuli, and blunted cortisol reactivity following a socially evaluated cold pressor task in adolescence (Hein et al., 2020; Peckins et al., 2019). This blunted amygdala response and cortisol reactivity diverges from extant literature on child abuse and general early adversity research which has largely found increased amygdala and cortisol reactivity with early adversity (for amygdala meta-analysis see Hein & Monk, 2016; for cortisol review see Koss & Gunnar, 2018). However, that may be due to differences in sample composition because the youth in both of these studies were exposed to relatively high levels of disadvantage. Previous research in samples exposed to higher levels of chronic adversity has found similar blunted amygdala reactivity (Gard et al., 2017; Holz et al., 2017). Additionally, the disparate findings may be due to a moderating variable in the environment, such as social support. Childhood social deprivation has been associated with blunted ventral striatum, an area of the brain involved in reward processing, to happy faces, a socially rewarding stimulus in adolescence (Hein et al., 2020). It has also been shown to moderate the association between

violence exposure and adolescent cortisol reactivity such that, when social deprivation is high, compared to mean and low levels of social deprivation, the cortisol response is even more blunted (Peckins et al., 2019). Interestingly, the observed changes in brain function correlated with both violence exposure and social deprivation do not relate to adolescent psychopathology (Hein et al., 2020) suggesting that these neural changes may not necessarily be detrimental for adolescent outcomes.

Previous work examining the dimensional effects of childhood violence exposure and social deprivation on adolescent outcomes has provided important and useful insights, but also identify directions for future research. This work shows that childhood violence exposure and social deprivation are associated with differences in brain functions in single regions of interest (ROIs) and the stress response in distinct ways. Additionally, it shows that violence exposure and social deprivation can interact to predict adolescent outcomes meaning that the environment of adversity is likely unique for each individual and that multiple factors influence how adversity "gets under the skin". We do not know; however, how these dimensions of adversity influence how different regions of the brain are connected and communicating with each other. Given the lack of associations between the neural correlates of violence exposure and social deprivation and psychopathology, we also do not know what factors are promoting resilience and are helping the teens adapt to their environment of adversity. Perhaps given the interaction seen in Peckins et al. (2019), where low levels of social deprivation buffered against the effects of violence exposure, social support may play a role. This dissertation examines how childhood exposure to violence is associated with white matter connectivity between the amygdala and PFC, regions of the brain involved in socioemotional function and emotion regulation, and how social deprivation may influence observed associations (study 1). Additionally, this dissertation

examines how violence exposure and social deprivation are associated with differences in resting-state functional connectivity using a person-specific modeling approach which helps account for the unique way the individual is affected by the environment of adversity (study 2). Lastly, this dissertation aims to determine what factors may be protective against these dimensions of adversity to help promote resilience (study 3).

Theoretical Framework: The Brain as a System

Traditionally, the neural impact of early adversity has been evaluated by quantifying the task-dependent activation of a single ROI, such as the amygdala or the correlation of activation in a pair of ROIs, for example, the amygdala and the subgenual anterior cingulate cortex (ACC) (for review see McCrory et al., 2017). These methods are effective in identifying individual and group differences in localized areas of the brain, but these functional specialization approaches do not provide information on how different regions communicate and how that communication affects neural function (Stevens, 2009). The brain is a system where regions of the brain do not act in isolation, but rather interact with each other to influence perception, emotion, and cognition (Pessoa, 2018). The interconnections between elements of a system, in this case between neural ROIs, yield critical information about its purpose and function and may be more defining of the system than the elements themselves (Meadows, 2008). Thus, studying the connections between regions of the brain and how they are functionally communicating provides valuable information that is more than the sum of individual ROIs.

Neural connectivity can be studied at multiple scales which each provide unique insight into the brain. Analyses of full systems and reductionist approaches provide complimentary information and are characterized by their own strengths and weaknesses (Meadows, 2008). A more precise focus on the interconnections between a small number of ROIs can be studied with

more depth, while a focus on the connections within larger networks or the whole brain, provides a wider breadth of understanding about how the brain is structured and functions. Thus, the analysis of patterns of integration and interactions of networks of ROIs, on multiple scales, may provide a better representation of underlying neural activity than any single scale alone (Menon, 2011).

Connectivity between regions of the brain can be studied using several modalities. Two such modalities are diffusion magnetic resonance imaging (dMRI) to assess white matter connectivity and resting-state functional MRI (fMRI) to examine spontaneous, non-directed functional connectivity (Huettel et al., 2009). Each of these methods provide unique but complementing information regarding how regions of the brain are communicating.

White Matter Connectivity

White matter in the brain consists largely of myelinated axons (Le Bihan et al., 2001). When myelin, a fatty-sheath, coats axons, electrical action potentials travel at a more rapid pace, thus facilitating communication between neurons and, at a larger level, regions of the brain (Nave & Werner, 2014). This makes differences in white matter connectivity relevant for understanding neural function. Previous work has found that white matter connectivity between two regions of the brain is related to how those regions function (Goetschius et al., 2019; Hein et al., 2018; Swartz et al., 2013).

White matter connectivity can be measured using multiple approaches which each have strengths and weaknesses. Traditional tractography analysis, such as deterministic tractography, utilizes diffusion tensor imaging data to estimate white matter tracts based on diffusion tensors or metrics such as fractional anisotropy (FA) (Sherbondy et al., 2008). However, the specificity at which this task can be accomplished is not at the axonal level but rather is limited to voxel level

analyses. This leaves a degree of uncertainty in the estimated white matter connectivity that is not accounted for (Barbas, 2015; Behrens et al., 2007). While still limited to the voxel level, probabilistic tractography accounts for that uncertainty by creating a probability density function at each voxel that quantifies the probability of the white matter connectivity between a seed region and the rest of the brain (Behrens et al., 2007). Additionally, traditional tractography methods largely focus on large white matter bundles, such as the uncinate fasciculus. This provides important information regarding larger white matter tracts; however, it obscures the variation of white matter between specific structures (i.e., between the amygdala and specific regions of the PFC) which can give nuanced insight into the structure of the brain (Goetschius et al., 2019). Last, traditional tractography methods such as tract-based spatial statistics (TBSS) only analyze white matter that is common across all included participants in a "group mean FA skeleton" (S. M. Smith et al., 2006). This is a conservative approach to analyzing dMRI data that has advantages; however, it does not allow for the analysis of individual heterogeneity of white matter connectivity in the same way that probabilistic tractography does. Probabilistic tractography tracts white matter connectivity at the subject level, so the length and specific trajectory of white matter tracts are free to vary across individuals (Behrens et al., 2007), making probabilistic tractography potentially better able to model person-specific heterogeneity in white matter. This dissertation uses probabilistic tractography to map white matter connectivity between the amygdala and multiple regions of the PFC (study 1) – a circuit which underlies emotion processing and regulation (Phelps & LeDoux, 2005) that is posited to be influenced by exposure to early adversity (Gee et al., 2013).

Resting State Functional Connectivity

Resting state functional connectivity (rsFC) examines neural activity in a non-goal directed state in terms of the integration and segregation of networks posited to underlie sensory, cognitive, emotive, and motor processes (Biswal et al., 2010; Finn et al., 2015). Analysis of rsFC is a powerful tool for understanding the human brain for multiple reasons. First, task-related increases in brain metabolism are generally small compared to the amount of energy used while the brain is at rest (Raichle & Mintun, 2006). Thus, studying rsFC may provide a richer source of variation in neural activity that is consuming much of the metabolic resources used by the brain (Fox & Greicius, 2010). Additionally, the relatively low cognitive demand and short duration of time in the scanner necessary for rsFC analyses make it a more feasible method for widespread use, including in pediatric and clinical populations (Fox & Greicius, 2010; Uddin & Menon, 2010). Last, rsFC networks have been reliably identified (Finn et al., 2015), even in children (Supekar et al., 2009; Thomason et al., 2011), are posited to be related to the functional architecture of the brain (Stevens, 2009), have been useful in predicting behavioral outcomes, such as general intelligence (e.g. Finn et al., 2015), and may be helpful in predicting the presence of psychopathology, such as depression (e.g. Greicius et al., 2007).

Much of the work with task-based fMRI focuses on group averages. Although this is helpful for examining gross trends in activation, the brain's functional organization varies in meaningful ways across the individual. Resting-state functional connectivity network analysis are capable of detecting person-specific modulations in the BOLD signal (Finn et al., 2015; Gates et al., 2014), highlighting the utility of using these networks as a tool for studying the individual. This dissertation focuses on two networks which have previously been associated with early adversity: the default mode network (DMN) and the salience network (SN) (study 2).

A description of these networks can be found below and their modulations in the context of early adversity can be found in the subsequent section.

Salience Network. The salience network is a neural network including the dorsal ACC, dorsolateral PFC, orbitofrontal cortex, insular cortex, and limbic structures such as the amygdala, substantia nigra, and ventral tegmental area (Menon, 2011; Seeley et al., 2007). It is involved in identifying and integrating relevant emotional, reward, interoceptive, and autonomic input to guide behavior. Through interaction with default mode and the central executive networks, the salience network acts in response to novel stimuli to transmit relevant information to other areas of the brain (Uddin, 2017a).

Default Mode Network. The default mode network is the most widely studied rsFC network and includes the posterior cingulate cortex, medial prefrontal cortex, hippocampus, angular gyrus, and the medial temporal lobe and is associated with self-referential introspective activity (Menon, 2011). This task-negative network is generally attenuated during cognitive tasks, typically in relation to the cognitive demands of the task. The DMN has also been linked to internal thought and memory retrieval, social-cognitive processes, reward-based decision making, and emotion regulation (Etkin et al., 2011; Menon, 2011; Rangel et al., 2008).

A Network Neuroscience Approach to Early Adversity

Swanson and Lichtman (2016, p. 197) in a review of network neuroscience state that, "the greatest challenge today is extracting knowledge and understanding of nervous system structure-function architecture from vast amounts of data." This statement highlights the complexity of getting an accurate picture of how the human brain works, but it can be argued that an even greater challenge is to understand how that structure-function architecture is shaped by the external social context within which the individual develops. Research on how early

adversity affects neural networks is a growing literature that includes work focused on both individual networks and whole brain functional connectivity. This dissertation is focused largely on socioemotional function, thus the network research reviewed here will focus on networks and interconnections associated with socioemotional function (i.e., emotion processing and regulation and reward processing).

Much of the socioemotional work on the association between early adversity and white matter in the brain has focused on the uncinate fasciculus – the major bundle of white matter connecting, among other regions, the PFC and subcortical regions (Olson et al., 2015). This work, although limited, has found that early adversity is related to weaker "structural integrity" of the uncinate fasciculus (UF) as measured using fractional anisotropy (FA) (Eluvathingal et al., 2006; Govindan et al., 2010; Hanson, Knodt, et al., 2015; Ho et al., 2017; M. J. Kim et al., 2019). Interestingly, this white matter connectivity has been shown to moderate associations between early adversity and internalizing disorders, such that in individuals with higher UF "structural integrity", there is not an association between early life stress and anxiety (M. J. Kim et al., 2019). Other white matter tracts of interest include the corpus callosum, inferior and superior longitudinal fasciculi. Research on these tracts has also largely found decreased "structural integrity" in individuals exposed to higher amounts of early adversity (Bick et al., 2015; Choi et al., 2012; Huang et al., 2012). Relatively little research on the association between early adversity and white matter connectivity has utilized more precise methods of white matter tracking, such as probabilistic tractography. One exception is a study focusing on child poverty that found decreased white matter connecting regions of the brain involved in socioemotional function, such as the amygdala and prefrontal cortex (D.-J. Kim et al., 2019).

Comparatively more research has focused on the effects of early adversity on rsFC. Connectivity involving SN has been a focus of rsFC research on early adversity. In individuals with exposure to child maltreatment, increased connectivity has been observed within the salience network, potentially reflecting an exaggerated salience attributed to typical events (Thomason & Marusak, 2017). This modulation in the SN has been related to psychopathology, such as anxiety disorders, and may partially explain the increased vulnerability for the development of anxiety disorders in youth exposed to childhood maltreatment (Marusak, Etkin, et al., 2015; McCrory et al., 2017; Uddin, 2017b). Enhanced SN segregation (increased connectivity within the SN) has also been associated with blunted reward sensitivity, a behavioral trait seen in the child maltreatment literature and may contribute to increased latent vulnerability for anxiety disorders, substance use, and depression (Marusak, Etkin, et al., 2015; McCrory et al., 2017). Early adversity has been shown to alter functional connectivity of limbic structures in the salience network, such as the amygdala, and the prefrontal cortex which mediated associations with internalizing disorders in adolescents (Herringa et al., 2013) and adults (Cisler et al., 2013).

The DMN is another target for research examining the impact of early adversity. In adults exposed to child maltreatment, the DMN has exhibited altered connectivity including decreased segregation of DMN nodes and attenuated deactivation of the DMN during demanding cognitive tasks (Philip, Sweet, Tyrka, Price, Bloom, et al., 2013; Philip, Sweet, Tyrka, Price, Carpenter, et al., 2013). Impaired DMN deactivation is associated with major depression, which may provide insight into why early adversity is associated with an increased vulnerability for depression (McCrory et al., 2017; Menon, 2011). Decreased integration of the SN and DMN has been found in both children and adults exposed to early life adversity, which may reflect an inabili1ty

to disengage attention from salient stimuli leading to an increased vulnerability for the development of psychopathology (Marusak, Martin, et al., 2015; Teicher et al., 2014; Thomason & Marusak, 2017). Previous work has found that this disruption may be driven by the anterior and posterior insula (Marusak, Etkin, et al., 2015; McCrory et al., 2017; Menon, 2011).

Extant research aimed at understanding how early adversity affects white matter and functional connectivity is limited in multiple ways. First, much of the work has used retrospective reports of early adversity in cross-sectional studies (i.e., Hanson, Knodt, et al., 2015; Marusak, Etkin, et al., 2015). Second, relatively few studies of neural correlates of early adversity, let alone the neural connectivity correlates, have been done in samples experiencing higher levels of chronic adversity (for examples of work in samples with higher levels of adversity, see Gard et al., 2017; Hein, 2019; Holz et al., 2017). Last, no previous work on either structural or functional network connectivity has used the dimensional approach to early adversity which may better capture the heterogeneity of early adversity. This dissertation aims to address these gaps by examining how two dimensions of adversity, violence exposure and social deprivation, are prospectively associated with differences in white matter (study 1) and functional connectivity (study 2) using data from a longitudinal sample of youth who come from largely disadvantaged environments.

An understanding of how early adversity affects the structure and function of neural circuits can help create neuroscience-informed policies and interventions. Although effects of early adversity are frequently discussed in terms of their effects on physical and mental health outcomes, the mechanism through which they work may be neural (Dufford et al., 2019; Farah, 2018). With an appropriate research design, understanding neural correlates of early adversity can lead to the identification of neural mechanisms driving behavioral change. These

mechanisms can provide targets to help design and measure the efficacy of interventions (Farah, 2018). Neural markers can be more sensitive predictors than behavioral ones which may aid in early identification of those at risk for negative outcomes (Pavlakis et al., 2015). Additionally, evidence for differences in neural circuits based on exposure to early adversity that complement behavioral findings provide converging evidence for how the environment affects the brain (Farah, 2018). Multiple sources converging bolsters findings and provides more support for specific theories and interventions. The benefits of neuroimaging research are often discussed in terms of differences in single regions of the brain; however, as previously discussed, this may oversimplify the complexity of the brain. Thus, early adversity-related differences in neural circuits and networks may be of even more benefit to interventions and policy because they more accurately model the complex brain (Dufford et al., 2019).

An important caveat to discuss when studying the neural correlates of early adversity is that differences in the brain are not necessarily reflective of deficits or a "broken brain" (Tolwinski, 2019). The brain is remarkably plastic, so alterations in neural circuitry may reflect adaptations to the environment that improve outcomes in certain situations and promote resilience (McEwen, 2016; Teicher et al., 2016). Additionally, it is possible that differences in the brain that are associated with negative outcomes can be compensated for with environmental interventions and support. This is particularly the case if identified early in development when the brain is more plastic highlighting importance of research studying the effects of early adversity on development (McEwen, 2012, 2016).

Resilience to Early Adversity

Across the lifespan, both risk and protective factors interact within the individual to shape development (Cicchetti, 2010). These factors exist at multiple levels within the individual and

their environment (i.e., home, community, culture) and are person-specific as evidenced by multifinality in developmental outcomes in response to adversity (Cicchetti & Lynch, 1993; Cicchetti & Rogosch, 1996; Hughes et al., 2017). Contributing to individual differences in these effects is resilience, or having positive outcomes in the context of adversity (Masten, 2001). Factors promoting resilience include individual characteristics such as adaptable temperament, a positive view of the self, and hopefulness for the future. Outside of the individual, environmental factors such as high neighborhood quality, effective schools, and good health care seem to be protective. In addition, many aspects of the individual's social relationships appear to promote positive outcomes suggesting that the presence of social support is a strong predictor of resilience (Ceballo & McLoyd, 2002; Ozbay et al., 2007; Wright et al., 2013).

School connectedness, or how safe, happy, and secure a child feels in their school environment, is an aspect of social support that has been linked not only to improved academic outcomes, but also overall improved adaptive function (C. E. Foster et al., 2017). School connectedness as a buffer is rooted in the risk and resilience framework, which posits that factors that promote developmental competency may also promote resilience to early adversity (Luthar et al., 2000). Previous research has found connectedness to school to be protective against violence in the neighborhood (Hardaway et al., 2012). Additionally, it has been posited to compensate for lower support in other areas of a youth's life (e.g., the home or neighborhood) (Loukas et al., 2010). School connectedness may be a particularly advantageous target for interventions because it is a potentially socially modifiable factor (H. Foster & Brooks-Gunn, 2009; McNeely et al., 2002).

Protective factors can work through different processes that are defined based on who they are protective for and how that protection differs across risk level (Luthar et al., 2000)

(Figure 1.2). They can be *promotive*, meaning that they promote positive outcomes for everyone in all situations (Sameroff, 2010). This would be observed statistically as a main effect of a certain factor. Additionally, these relationships can be more complex involving interactions between multiple variables. For example, a protective variable can be *protective-stabilizing*, where, in the context of increasing risk, having a certain attribute present results in no decrease in rates of the positive outcome (H. Foster & Brooks-Gunn, 2009; Luthar et al., 2000). Conversely, a protective variable can be *protective but reactive,* where it promotes positive outcomes, but less so as environmental risk or stress increases (Luthar et al., 2000; Proctor, 2006). Lastly, factors can be *protective-enhancing*, where having that attribute is associated with more positive outcomes as risk increases (Luthar et al., 2000). The different ways that protective factors can promote resilience highlight the multidimensional nature of adversity and the need to study them in multiple contexts and on multiple outcomes.

Current research on resilience, including the extant literature on the protective effects of school connectedness, is limited. First, it often takes a deficit approach, meaning that it is focused on alleviating or avoiding negative outcomes rather than promoting positive outcomes in a strengths-based perspective (Masten & Cicchetti, 2016). Additionally, we know that the environment of adversity is complex; however, much of the research on resilience treats adversity as either a single construct or focuses on a single category of adversity. Both of these methods may oversimplify the impact of early adversity and may obscure protective factors that more selectively promote resilience. This dissertation aims to overcome both of those limitations by examining how two dimensions of adversity, violence exposure and social deprivation, predict both positive and negative developmental outcomes and how school connectedness may influence those associations (study 3).

Specific Aims of this Dissertation

The purpose of this dissertation is to understand how early adversity shapes the brain and behavior and what factors in the environment protect against adversity. Much of the research examining this question has been done in white, middle-class samples using retrospective reports of early adversity (Dufford et al., 2019; Falk et al., 2013). More research is needed which prospectively asks these questions in samples that include families living in socioeconomically disadvantaged contexts, who are more at risk for exposure to violence and social deprivation (McLaughlin, Costello, et al., 2012; McLoyd, 1998).

All three chapters in this dissertation use data from the Fragile Families and Child Wellbeing Study (FFCWS) (Reichman et al., 2001). The FFCWS is a population-based sample of approximately 5000 children born in large US cities, with an oversample of non-marital births which led to inclusion of a large proportion of low-income and minority families (Reichman et al., 2001). These sample characteristics make the FFCWS an appropriate sample to examine how early adversity prospectively shapes development. In the FFCWS, data were collected from the mother, father (if available), the primary caregiver (most frequently the mother), and the focal child at the birth of the focal child, and again when the child was 1, 3, 5, 9, and 15 years of age through a combination of in-home and phone visits (*Public Data Documentation for FFCWS*, 2019). Variables used to measure early exposure to two dimensions of adversity, violence exposure and social deprivation, were calculated from FFCWS data from waves at ages 3, 5, and 9 years (Figure 1.1). At age 15, a subsample of 237 teens and their families from Detroit, Toledo, and Chicago, came to University of Michigan to participate in the Study of Adolescent Neural Development (SAND Subsample). Informed consent was provided by the parent/legal guardian

and informed assent from the subject (UM IRBMED: HUM00074392). Diffusion (dMRI - white matter connectivity) and resting-state MRI data were collected during this visit.

Study 1 (Chapter 2): Childhood violence exposure and social deprivation predict adolescent amygdala-orbitofrontal cortex white matter connectivity [SAND Subsample]

Aim 1: Determine whether childhood violence exposure is associated with adolescent amygdala-PFC (orbitofrontal and dorsomedial PFC) white matter function.

Aim 2: Determine whether childhood exposure to social deprivation moderates the association between violence exposure and adolescent white matter.

Study 2 (Chapter 3): Neural sequelae of early adversity: Childhood violence exposure predicts patterns of adolescent resting-state connectivity [SAND Subsample]

Aim 1: Determine if childhood exposure to violence or social deprivation are associated with network density of the salience and default mode networks in adolescence.

Aim 2: Determine if childhood exposure to violence or social deprivation are associated with node degree for 4 nodes in the salience network and 3 nodes in the default mode network (per hemisphere) in adolescence.

Aim 3: Determine if data driven subgroups can be identified in the adolescent resting-state MRI data and if subgroup membership is associated with childhood violence exposure or social deprivation.

Study 3 (Chapter 4): School connectedness as a protective factor against childhood exposure to violence and social deprivation: Evidence of resilience and risk from a longitudinal sample [Full FFCWS Sample]

Aim 1: Determine whether childhood exposure to violence and social deprivation predicts child or adolescent internalizing and externalizing psychopathology and adolescent positive function.

Aim 2: Determine whether school connectedness at either age 9 or 15 moderates observed associations between childhood dimensions of adversity and behavioral outcomes.

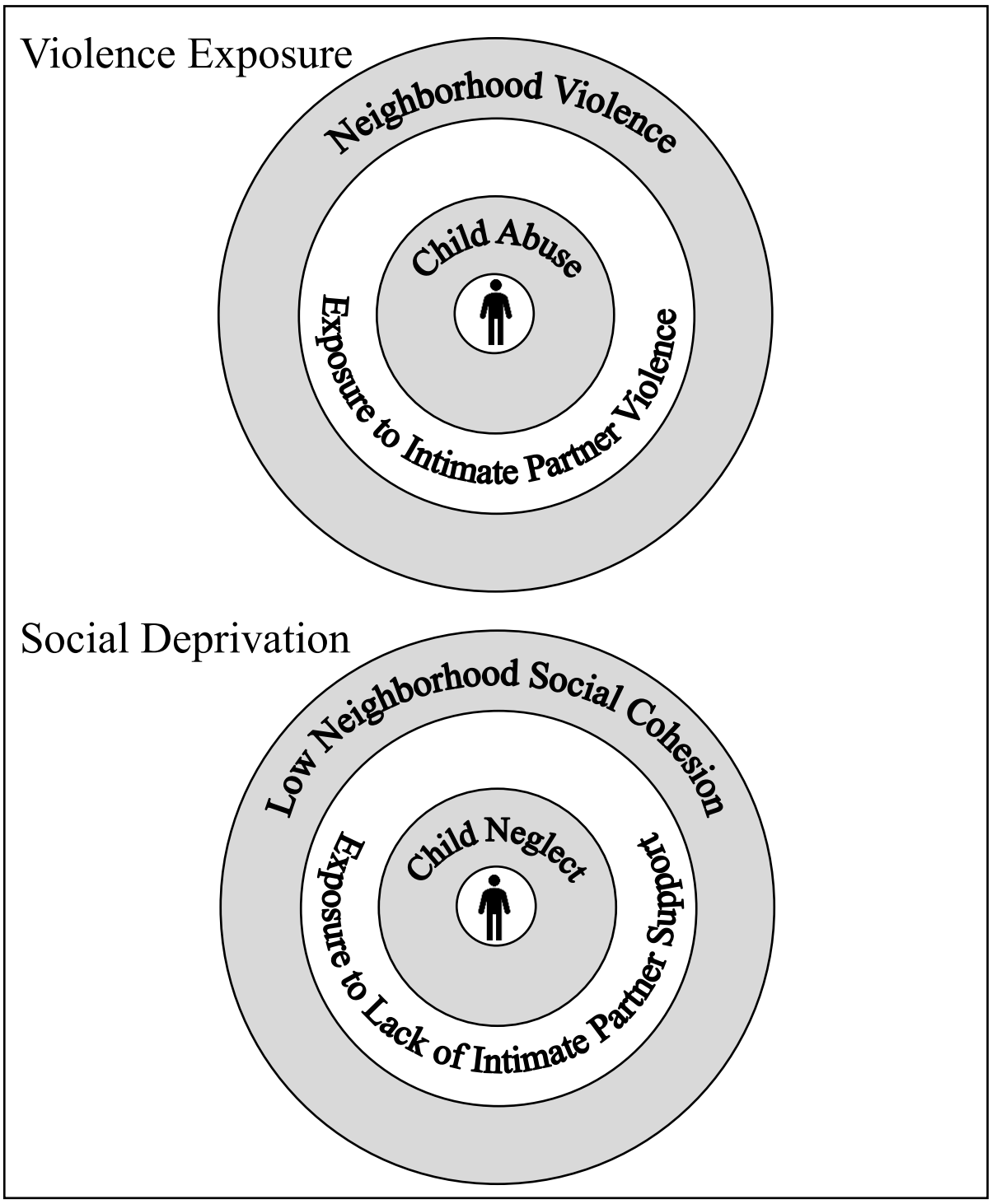


Figure 1.1: Violence exposure and social deprivation conceptual figure

Composite scores for these dimensions were created from data collected from the FFCWS study waves at ages 3, 5, 9.

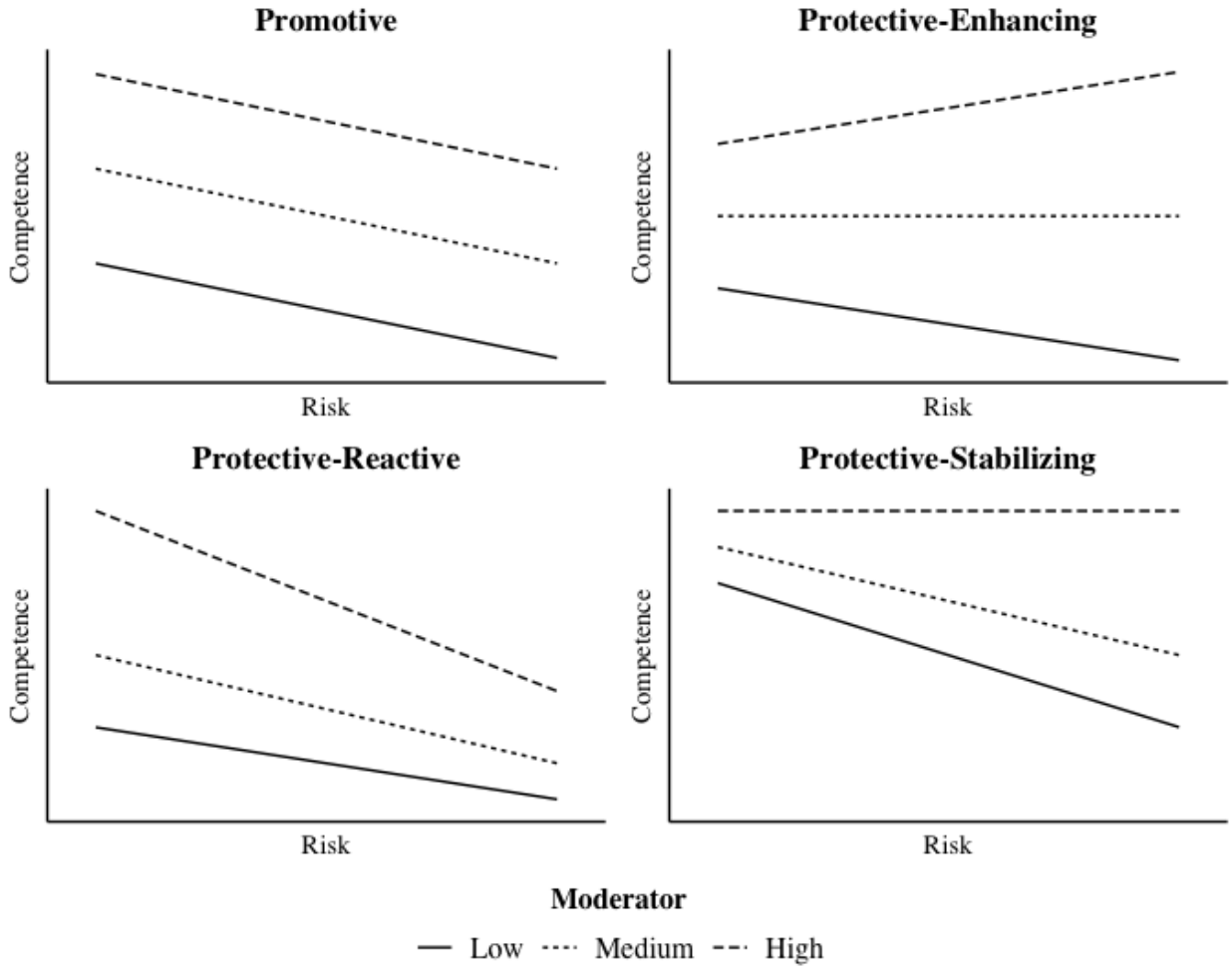


Figure 1.2: Protective processes conceptual figure

Hypothetical plots representing the different definitions of protective processes.

Chapter 2[1]: Childhood Violence Exposure and Social Deprivation Predict Adolescent Amygdala-Orbitofrontal Cortex White Matter Connectivity

Childhood adversity is common and predicts a host of negative mental and physical health outcomes (Sacks & Murphy, 2018). Such experiences also shape the neural circuitry underlying emotion processing and regulation (Hein & Monk, 2017). Here, we examined a predominantly low-income sample of adolescents who have been followed since birth to better understand how specific dimensions of early adversity prospectively shape adolescent white matter connectivity between the amygdala and subregions of the prefrontal cortex (PFC), as well as the association between this white matter and amygdala reactivity during socioemotional processing.

Examining dimensions of adversity, that are modeled separately from socioeconomic status, may elucidate how complex experiences influence the brain and may contribute to negative consequences (Amso & Lynn, 2017; McLaughlin et al., 2014). Previous research highlighted two core dimensions – threat and deprivation – that have roots in neurobiological research (McLaughlin et al., 2014). Further, behavioral research using this dimensional framework found that modeling the dimensions as cumulative exposure compared to a binary incidence variable (i.e., whether the person had experienced threat or deprivation) better predicted child outcomes (Wolf & Suntheimer, 2019). The present study examined two similar dimensions: (1) violence exposure and (2) social deprivation. Violence exposure is characterized

[1] Chapter 2 corresponds to Goetschius et al., 2020 published in *Developmental Cognitive Neuroscience*

by witnessing or being the victim of home and community violence. Social deprivation is defined as a lack of home and community emotional support (Hein, 2019). These dimensions exist on continua: violence exposure ranges from low (i.e., safety) to high and social deprivation from low (i.e., high levels of home/neighborhood support) to high (i.e., lack of support). Violence exposure is posited to alter regions of the brain involved in fear learning and emotion regulation, including the amygdala and PFC (McLaughlin et al., 2014). Compounding the stress of violence exposure, co-occurring social deprivation may exacerbate the effects of violence exposure and, conversely, low social deprivation (i.e., social support) may act as a buffer (Sheridan et al., 2018; Sonuga-Barke et al., 2010).

Diffusion MRI (dMRI) work in this area is limited and has yielded mixed results (McLaughlin et al., 2019). Moreover, to date, the potential effects of different types of adversity (i.e., threat versus deprivation) on white matter connectivity have not been investigated simultaneously within the same analyses to understand how these complex exposures shape the brain. Retrospective reports of early social deprivation (i.e., neglect) have been associated with decreased strength of structural connections between the amygdala and PFC (Hanson, Knodt, et al., 2015; Ho et al., 2017). Additionally, one study found that retrospective reports of threat, specifically, trauma were associated with increased strength of the uncinate fasciculus (Gur et al., 2019), the major bundle of white matter connecting the PFC and subcortical regions (Olson et al., 2015). However, reported studies have also found null effects of threat, deprivation, or mixed exposures on the fronto-amygdala white matter (Bick et al., 2015; Dennison et al., 2016; Park et al., 2016). The vast majority of existing dMRI work examining adversity, though not all (D.-J. Kim et al., 2019), has used diffusion tensor imaging (DTI) (Hanson, Knodt, et al., 2015; Ho et al., 2017) which measures bundles of white matter in aggregate. Much of the DTI literature on

adversity has focused on the uncinate. However, DTI does not permit precise mapping of white matter tracts between specific structures, such as the amygdala and particular PFC subregions.

Probabilistic tractography uses dMRI to precisely map white matter tracts between structures (Behrens et al., 2007). This method, in a smaller subset of the current sample, showed that amygdala white matter connectivity with the orbitofrontal cortex (OFC – Brodmann's Area (BA) 47, 11), dorsomedial PFC (BA10), and subgenual cingulate (BA25) was greater than amygdala white matter connectivity with other PFC regions, such as the dorsal anterior cingulate and dorsolateral PFC (Goetschius et al., 2019). Non-human primate studies also provide support for specific amygdala connectivity with the OFC, dmPFC, and subgenual cingulate (Ray & Zald, 2012; Zikopoulos et al., 2017). Additionally, our previous work revealed that adolescents with less white matter connectivity between the amygdala and the OFC (right BA47, left BA11) and dmPFC (bilateral BA10), but not the dorsolateral PFC, anterior cingulate, or subgenual cingulate, showed greater amygdala activation to threatening faces (Goetschius et al., 2019). Thus, the OFC, dmPFC, and subgenual cingulate seem to be well-connected via white matter to the amygdala. Additionally, amygdala–OFC and amygdala–dmPFC connectivity may play an important role in emotion processing and regulation; however, the effect of dimensional adversity on this white matter has not yet been examined.

Building on Goetschius et al. (2019), we used probabilistic tractography to assess whether violence exposure across childhood (ages 3, 5, 9 years) predicted adolescent (15-17 years) amygdala–PFC white matter connectivity with a focus on OFC, dmPFC, and subgenual cingulate subregions in a longitudinal, sample with a substantial representation of African American and low-income participants — populations that are underrepresented in neuroimaging research (Falk et al., 2013). We also examined whether the degree of social deprivation in

childhood predicted adolescent amygdala–PFC white matter microstructure through interaction with violence exposure. We hypothesized the following: childhood violence exposure would be associated with adolescent amygdala–PFC white matter connectivity; and the interaction between childhood violence exposure and social deprivation would be associated with white matter connectivity such that the effects of high violence exposure would be buffered by decreasing social deprivation. In addition, because Goetschius et al. (2019) was conducted on a smaller subsample (N = 141) of the data used in the present study, and utilized a different diffusion data cleaning pipeline, we attempted to reproduce the associations observed between amygdala–PFC white matter connectivity and amygdala activation in the current, full sample (N = 152).

Materials & Methods

These hypotheses, variables, and analyses were preregistered with the Open Science Framework (https://osf.io/spguw) and the data will be available on the NIMH Data Archive (https://nda.nih.gov/edit_collection.html?id=2106). Prior to preregistering these hypotheses, we had examined the diffusion MRI data on the 141 participants (Goetschius et al., 2019). In this analysis, we examined how the probability of amygdala–PFC white matter connectivity predicted amygdala reactivity to threatening faces; however, we had not evaluated any associations between the early environment and diffusion MRI data.

Participants

One hundred eighty-three adolescents (15-17 years) sampled from the Detroit, MI, Toledo, OH, and Chicago, IL sites of the Fragile Families and Childhood Wellbeing Study (FFCWS) were included in the present study (see Table 2.1 for sample demographics and exclusion criteria). The FFCWS is a population-based sample of children born in large US cities,

with an oversample of non-marital births (~3:1) (Reichman et al., 2001). When weighted, the FFCWS represents children born at the turn of the century in American cities of 200,000 or more. When not weighted (as here), given the oversample for non-marital births, the sample represents mostly low-income, urban families. Given the demographics and sample sizes in Detroit, Toledo and Chicago (Hein et al., 2018), a majority of the sample identified as African American. FFCWS families were interviewed at the birth of the focal child, and again when the child was 1, 3, 5, 9, and 15 years of age. The University of Michigan Medical School IRB approved this study (UM IRBMED: HUM00074392). Informed consent was obtained from the parent/legal guardian for both their participation and their teen's participation and informed assent from the adolescent. These data overlap with prior work from our research group: fMRI and dMRI data, but no environmental data (Goetschius et al., 2019; Hein et al., 2018); violence exposure and social deprivation composites, but no MRI (Peckins et al., 2019).

Behavioral Measures

Childhood Violence Exposure and Social Deprivation Composite Scores

Violence exposure and social deprivation were assessed using composite scores calculated using data from the Fragile Families and Child Wellbeing study at ages 3, 5, and 9 years. Both constructs included primary caregiver or mother report of experiences that directly (i.e., child physical and emotional abuse, child physical and emotional neglect) and indirectly (i.e., intimate partner emotional, physical, or sexual violence against mother, intimate partner support for mother, community violence, community support) affect the child. The primary caregiver was primarily a biological parent or family member. One participant's primary caregiver was not a relative. No participants were in the foster care system. We considered violence exposure to exist on a continuum where higher scores represented more violence

exposure, and lower scores represented more safety. We considered social deprivation to exist on a continuum where higher scores (e.g., where the child experienced either more neglect or witnessed less social support for their mother or less neighborhood social cohesion) approximated deprivation and lower scores (e.g., where the child experienced less neglect or witnessed more social support for their mother or more neighborhood social cohesion) approximated social support. Our approach of including experiences with varying levels of proximity to the child across multiple time points allowed us to comprehensively assess the child's cumulative, dimensional exposure to violence and social deprivation across childhood as has been done in previous research (Hein, 2019; McLaughlin & Sheridan, 2016). With this approach, we did not unpack the effect of proximal versus distal experiences, the effect of the developmental timing of exposures; however, those are important future research directions. These composite scores were first utilized in previous work from our group (Hein, 2019). All items at each time point were weighted equally. See Appendix 1 for specific items and the scales that they come from.

To calculate composite scores, the Z scores (zero-centered) for each of the childhood experiences (child abuse, exposure to intimate partner violence, community violence, child neglect, lack of romantic partner support, lack of neighborhood social cohesion) were summed for each of the childhood experiences within a cumulative dimension (violence exposure and social deprivation) (Song et al., 2013) and then divided by the number of childhood experiences within a dimension for each participant, thus maximizing the number of participants and the diversity of the sample by minimizing drop out due to missing data at any given wave. This means that a score of 0 is approximately average for the sample for that dimension. Scores greater than 0 represent higher than average violence or social deprivation and scores below 0

represent low violence or low social deprivation (i.e., social support). We then mean-centered the scores for violence exposure and social deprivation and created an interaction term (Hein, 2019). In our sample, violence exposure and social deprivation were correlated at $r(181) = 0.50$, $t = 7.69$, $p < 0.001$, but the variance inflation factor (VIF) was 1.326 (Figure 2.1, Table 2.2). VIF reflects how much the estimated regression coefficients are increased due to collinear independent variables. Cutoffs are typically between 5–10, therefore, based on the VIF reported here, the multicollinearity of violence exposure and social deprivation was low (Craney & Surles, 2002; Sheather, 2009).

Gender Identification (Faces) fMRI Task

During fMRI data collection, participants completed an event-related emotional faces task in which they were instructed to identify to the gender of emotional faces displaying one of five emotions: fearful, happy, sad, angry, neutral. Details of the task are in the Appendix 1 (and see Goetschius et al., 2019; Hein et al., 2018). Participants who achieved less than 70% accuracy on the Faces Task were excluded (N = 15). Average task accuracy was 94.74%.

Covariates

To address potential confounds, the present analyses adjusted for race/ethnicity, maternal education at birth, and maternal marital status at birth. We controlled for maternal marital status at birth due to the oversampling of non-marital births in the FFCWS study (Reichman et al., 2001). Additionally, we adjusted for adolescent pubertal development, adolescent internalizing psychopathology and adolescent life stress to ensure that observed effects were not driven by these adolescent factors. Adolescent internalizing psychopathology was assessed using a multi-method, multi-informant latent factor score constructed from the following measures: (1) K-SADS (Kaufman et al., 1997) clinician report of past and current symptoms of dysthymia, social

phobia, generalized anxiety disorder, major depression, and phobia and (2) parent and child report on the Mood and Feelings Questionnaire (Angold et al., 1987), Child Depression Inventory (Helsel & Matson, 1984), and the Screen for Child Anxiety Related Disorders (Birmaher et al., 1997) (See Appendix 1 and Hein, 2019 for more detail including the CFA fit statistics). Current life stress was used as a covariate in the present analyses and was measured using the Adolescent Life Events Scale (adapted for Shaw et al., 2003 from Farrell et al., 1998 and Masten et al., 1994). This scale assesses the experience of common adolescent stressful life events in the past year. Descriptive statistics for all covariate variables are in Table 2.2. See Appendix 1 for more information on how covariates were measured. All analyses were done *with and without* covariates.

MR Measures - Adolescence

MR images were acquired using a GE Discovery MR750 3T scanner with an 8-channel head coil located at the UM Functional MRI Laboratory. Head movement was minimized through: (a) instructions to the participant and (b) padding and pillows placed around the head, which are well-tolerated, yet limit motion. These procedures have been outlined in previous work (Goetschius et al., 2019; Hein et al., 2018).

T1-weighted gradient echo images were taken before the functional scans using the same field of view (FOV) and slices as the functional scans (TR = 9.0 seconds, TE = 1.8 seconds, TI = 400 ms, flip angle = 15°, FOV = 22 cm; slice thickness = 3 mm; 256 x 256 matrix; 40 slices). DMRI data were collected using a spin-echo EPI diffusion sequence (scan parameters: TR 7250ms, Minimum TE, 128x128 acquisition matrix, 22 cm FOV, 3 mm thick slices (no gap), 40 slices acquired using an alternating-increasing order, b value = 1000 s/mm^2, 64 non-linear directions, five b=0s/mm^2 T2 images (b0) acquired). Functional MRI (fMRI) T2*-weighted

BOLD images were acquired using a reverse spiral sequence (Glover & Law, 2001) of 40 contiguous axial 3 mm slices (TR = 2000 ms, TE = 30 ms, flip angle = 90°, FOV = 22 cm, voxel size = 3.44mm x 3.44mm x 3mm, sequential ascending acquisition).

Slices were prescribed parallel to the AC-PC line (same locations as structural scans). Images were reconstructed into a 64x64 matrix. Slices were acquired contiguously, which optimized the effectiveness of the movement post-processing algorithms. Images were reconstructed off-line using processing steps to remove distortions caused by magnetic field inhomogeneity and other sources of misalignment to the structural data, which yields excellent coverage of subcortical areas of interest.

dMRI Processing

Diffusion images were converted from DICOM to NIFTI format using MRIcron (dcm2niix – 2MAY2016) for offline analysis using MRtrix (v.3.0.R3) (Veraart et al., 2016) and the FSL (v. 5.0.9) FMRIB's Diffusion Toolbox (FDT) (v. 3.0) (Jenkinson et al., 2012) (see Appendix 1 for more processing details).

DMRI data were then processed using probabilistic tractography in FSL. This involved building a distribution of diffusion parameters at each voxel using bedpost (Hernández et al., 2013) and estimating the probability of amygdala–PFC white matter connectivity for 4 PFC ROIs bilaterally (8 total) using probtrackx (Hernandez-Fernandez et al., 2016) (Appendix 1). Those ROIs were BA10, BA11, BA25, and BA47 and they were selected due to a previous stronger likelihood of amygdala white matter connectivity in our previous work (Goetschius et al., 2019). ROIs, including both seed amygdalae (AAL Atlas) and target PFC regions (TD Brodmann’s Areas) were created from masks in WFU PickAtlas (Maldjian et al., 2003). The maximum-likelihood of amygdala–PFC connectivity was then extracted for each individual from

a group-level peak (6mm sphere around peak point) identified for each ROI (Greening & Mitchell, 2015) (details in Appendix 1). The MNI coordinates (x,y,z) for the peak for each target are as follows: BA10 (left: -30, -4, -14; right: 32, -2, -12), BA11 (left: -30, -4, -14; right: 32, -2, -14), BA25 (left: -16, 0, -14, right: 18, 0, -14), BA47: (left: -30, -4, -14; right: 34, 0, -20).

In the present study, we did not use waypoint or termination masks in the probabilistic tractography analysis. Thus, we cannot guarantee that streamlines did not cross the midline or enter the temporal pole. However, this does not appear to be the case for the measured connections here based on the average streamline images (Supplemental Figure 2.1 in Appendix 1).

The dMRI processing approach used (i.e., from bedpostx through data extraction) was identical to a previously reported analysis; however, the present study's sample size was larger (N=152 with both usable fMRI and dMRI data compared to N=141) because the dMRI data were processed for artifacts using a different, more reliable, and automated method that allowed us to retain more subjects (Andersson et al., 2017; Andersson & Sotiropoulos, 2016; Veraart et al., 2016). Due to the increased sample size and different dMRI cleaning method, we needed to reproduce the associations with amygdala activation seen in Goetschius et al. (2019).

fMRI Processing

First-level statistical analyses for functional activation were performed using the general linear model implemented in SPM12. For each participant, conditions were modeled with the SPM12 canonical hemodynamic response function. Incorrect trials were modeled as a separate condition and were not included in subsequent analysis. A statistical image for each condition contrast in the Faces Task at each voxel was generated. Mean activation was extracted for both the left and right amygdala using MarsBaR (v. 0.44) (Brett et al., 2002) from the contrast image

representing a combination of threat (fear + anger) trials vs. baseline (Goetschius et al., 2019; Hein et al., 2018). ROI masks used in the extraction were created using the left and right amygdala from the AAL Atlas in WFU Pickatlas (Maldjian et al., 2003).

Statistical Analysis

Preregistered Analyses

To determine how childhood exposure to violence and social deprivation at ages 3, 5, and 9 years were associated with amygdala–PFC white matter connectivity, we performed eight multiple regression analyses – one for each amygdala-PFC target pair (bilateral BA10, BA11, BA25, BA47). In each regression analysis, we first ran the analysis without any covariates. Then, we controlled for a list of preregistered covariates, including participant gender (male or female), race (African American, Caucasian, or Other), maternal education at birth, maternal marital status at birth). Additionally, in a separate analysis, we controlled for three variables that we did not pre-register, pubertal status, current life stress, and the internalizing disorders latent factor score (Hein, 2019), in addition to the preregistered covariates, though none of these variables changed the overall effect. We used a Bonferroni-corrected significance threshold based on those eight ROIs ($p<0.05/8$ tests per hemisphere = 0.0063). To interpret significant interactions, simple slope and regions of significance analyses were conducted to determine the nature of the interaction and ensure that the interaction was within our observable data using methods outlined by Preacher et al., (2006).

Our main preregistered analysis plan proposed a structural equation model (SEM) where childhood dimensions of early adversity predicted internalizing psychopathology in a way that was mediated by amygdala–PFC white matter connectivity. We did not continue with this analysis plan when white matter connectivity was not significantly associated with internalizing

psychopathology (Appendix 2). Thus, we proceeded with our secondary analysis plan to examine the pieces of the SEM using multiple regression, including the violence exposure x social deprivation interaction. We did not have adequate statistical power to perform a moderated-mediation model to examine the interaction in a larger SEM framework given the likely small effect size (Preacher et al., 2007).

Non-preregistered Analyses

Due to the use of an automated diffusion MRI data cleaning and artifact detection method which increased sample size, we reproduced the associations between amygdala–PFC white matter connectivity and amygdala activation that were previously reported where amygdala–OFC (right BA47, left BA11) and amygdala–dmPFC (bilateral BA10) white matter connectivity was associated with amygdala reactivity (Goetschius et al., 2019). To do this, we performed eight regressions predicting ipsilateral amygdala activation to threat faces from amygdala–PFC white matter connectivity – one for each amygdala-PFC target pair (bilateral BA10, BA11, BA47, BA25). In these regressions, we used a Bonferroni-corrected significance threshold (p=0.05/8 tests=0.0063).

Results

Probabilistic tractography was used to estimate the white matter connecting the amygdala with all eight PFC targets (bilateral BA10, BA11, BA25, BA47). For a visual representation, see figure 2.2.

Violence Exposure x Social Deprivation predicted right hemisphere amygdala–OFC white matter connectivity

The interaction between violence exposure and social deprivation significantly predicted the probability of right hemisphere amygdala–BA47 (OFC) white matter connectivity (Table 2.3).

This association held when adjusting for our pre-registered covariates (gender, race, maternal education at birth, and maternal marital status at birth) (β=-0.319, p=0.004) and non-preregistered covariates (pubertal status, current life stress, and internalizing psychopathology in addition to the preregistered covariates) (β=-0.317, p=0.005). Contrary to our preregistered hypotheses, there were no main effects of violence exposure or social deprivation. To better understand the interaction in the context of our data, simple slopes and regions of significance are plotted in Figure 2.3. Simple slopes analysis revealed that when social deprivation was 0.78 or greater, violence exposure and probability of white matter were inversely related (β = -0.29, p = 0.048). When social deprivation was 1 standard deviation below the mean, there was no association between violence exposure and amygdala–OFC white matter connectivity (β = 0.02, p = 0.209). Thus, in our data, at relatively high values of social deprivation, violence exposure was related to a lower likelihood of amygdala–OFC connectivity, suggesting that violence exposure had the greatest association with amygdala–OFC white matter connectivity when social deprivation was also high.

Violence Exposure x Social Deprivation predicted right hemisphere amygdala–dmPFC white matter connectivity

The interaction between violence exposure and social deprivation significantly predicted the probability of right hemisphere amygdala–BA10 (dorsomedial prefrontal cortex - dmPFC) white matter connectivity (β=-0.268, p=0.011). This interaction, however, did not remain significant when controlling for the demographic covariates (β=-0.185, p=0.091) (Supplemental Table 2.3 in Appendix 2). There were no main effects of violence exposure or social deprivation on right hemisphere amygdala–BA10 white matter connectivity.

Greater amygdala–OFC and amygdala–dmPFC white matter connectivity was related to attenuated amygdala reactivity

We reproduced results from previous analyses (Goetschius et al., 2019). The probability of amygdala white matter connectivity significantly predicted ipsilateral amygdala activation to threatening (fearful and angry) faces for the four PFC regions where it was previously related (bilateral BA10, left BA11, right BA47), even when adjusting for the specified covariates, using a hemisphere Bonferroni-corrected significance level (0.05/8 = 0.0063) such that increased probability of white matter was associated with decreased amygdala activation (Table 2.4). Additionally, amygdala–PFC white matter connectivity was not related to amygdala reactivity in regions where it had not been related in our previous report (right BA11, bilateral BA25, left BA47) (Supplemental Table 2.10 in Appendix 2).

Null findings

Violence exposure, social deprivation, or their interaction did not significantly predict the likelihood of *left* hemisphere amygdala–BA10 white matter connectivity, *left* hemisphere amygdala–BA47 white matter connectivity, bilateral amygdala–BA11 white matter connectivity, or bilateral amygdala–BA25 white matter connectivity (Supplemental Tables 2.4-2.9 in Appendix 2).

Discussion

Using an open science framework and preregistered hypotheses, the present study examined how two dimensions of adversity - violence exposure and social deprivation - were associated with structural connectivity between the amygdala and OFC in the right hemisphere, a critical circuit for emotion processing and regulation. Whereas, contrary to our hypotheses, there were no main effects of the two dimensions on white matter connectivity, the interaction of

violence exposure and social deprivation at ages 3, 5, and 9 prospectively predicted the degree of right amygdala–OFC white matter connectivity in adolescence. Specifically, the combination of more violence exposure and more social deprivation in childhood prospectively predicted less amygdala–OFC white matter connectivity in adolescence; however, violence exposure was not associated with white matter connectivity when social deprivation was at mean or low levels (i.e., when children were in relatively socially supportive contexts). Thus, social deprivation may exacerbate the effects of childhood violence exposure on the development of white matter connections whereas social support may act as a buffer. This interaction remained even after adjusting for gender, race, pubertal development, current internalizing psychopathology, current life stress, maternal marital status at birth, and maternal education at birth. Importantly, the work was conducted in a well-sampled cohort of adolescents with high rates of poverty and a large proportion of African Americans, groups that are understudied in neuroimaging research (Falk et al., 2013).

As a secondary objective, we reproduced in an expanded, overlapping sample (Goetschius et al., 2019) the finding that increased amygdala–OFC and amygdala–dmPFC white matter connectivity was associated with attenuated amygdala-reactivity to threat faces. This association remained after adjusting for gender, race, pubertal development, current internalizing psychopathology, current life stress, maternal marital status at birth, and maternal education at birth. When considered in conjunction with the violence exposure by social deprivation interaction, these findings suggest that early adversity shapes white matter connections that modulate the amygdala, a structure involved in threat processing (Phelps & LeDoux, 2005).

The association between violence exposure and decreased amygdala–OFC white matter connectivity in the context of social deprivation builds on prior work (for review, see

McLaughlin et al., 2019). Extant dMRI research indicates that child maltreatment or trauma are generally, but not exclusively (Gur et al., 2019), associated with both weaker structural connectivity within the uncinate fasciculus (Govindan et al., 2010; Hanson, Knodt, et al., 2015; Ho et al., 2017) and weaker global structural connectivity, including within the OFC (Puetz et al., 2017). Additionally, consistent with the present findings, fMRI work found that violence exposure is associated with altered amygdala activation (Hein, 2019; McCrory et al., 2011) and amygdala–PFC functional connectivity (Herringa et al., 2013; Kaiser et al., 2018). Further, neural tract tracer research in nonhuman primates revealed that stress affects amygdala–OFC structural connections via increased levels of dopamine (Zikopoulos et al., 2017) and that amygdala–OFC connections serve as a primary inhibitory pathway for amygdala function (Ray & Zald, 2012). Last, research examining the cortisol response to a social stressor in this sample found a similar interaction where the effect violence exposure was exacerbated by high social deprivation (Peckins et al., 2019). Taken together with the increased specificity provided by the current study, childhood violence exposure, when combined with social deprivation, may act as a potent stressor that is associated with decreased white matter in adolescence between the amygdala and the OFC. Expanding on the current DMAP model, our results suggest that the effect of violence exposure (a specific subtype of threat) on fronto-amygdala white matter may depend on the concurrent degree of social deprivation or support.

Extant literature is consistent with the right hemisphere-specific effects of the present study. Amygdala–OFC structural connections are posited to play a role in automatic emotion regulation (Phillips et al., 2008) with right hemisphere connections being more heavily involved in fear extinction learning (Gottfried & Dolan, 2004). Further, in healthy adults, greater right hemisphere amygdala–OFC functional connectivity has been observed in response to

unpredictable threat (Gold et al., 2015), supporting the potential inhibitory role of the structural connections observed here.

In addition to the exacerbating effects of social deprivation and violence exposure, the present findings indicate that low social deprivation (i.e., social support) may exert a "protective-stabilizing" (Proctor, 2006) effect against the negative behavioral sequelae of violence exposure (H. Foster & Brooks-Gunn, 2009; Ozer, 2005). Consistent with the idea of a protective-stabilizing factor, the present study found that social support was associated with a lessening of the negative association between violence exposure and amygdala–OFC connectivity that was observed in the context of social deprivation (i.e., low social support). The present findings suggest that policies aimed at boosting social support for youth in high violence environments may lessen the effect of violence exposure on a primary neural circuit for emotion regulation.

Similar to the interaction in the amygdala–OFC connectivity, we found a violence exposure-social deprivation interaction when predicting right hemisphere amygdala-dmPFC (BA10) connectivity. However, the association was not significant when adjusting for the demographic covariates. BA47, the OFC ROI used, is rostrally bordered by the dmPFC (Petrides & Pandya, 2002), and neighboring cortical regions are often connected (Bullmore & Sporns, 2012). Thus, amygdala-dmPFC tracts may pass through the OFC, explaining the weaker association with the dmPFC.

Importantly, in contrast to our hypotheses, there were no main effects of childhood violence exposure or social deprivation on adolescent amygdala–OFC, amygdala-dmPFC, or amygdala–subgenual cingulate connectivity. Thus, it may not be fruitful to consider dimensions of adversity in isolation and out of context of other salient ecological variables (McLaughlin & Sheridan, 2016). Rather, in order to construct a more complete picture of how early adversity

influences the brain, it is important to measure and model the effects of multiple dimensions that have been established to impact development.

The present study had limitations worth noting. First, due to the population-based sampling methodology used in the FFCWS, youth were not preselected based on their ability or willingness to participate in an MRI study, a common procedure in many neuroimaging studies. Thus, 41 participants of the available sample were ineligible or refused to complete the dMRI scan. Although it is a limitation that our full sample could not participate, the group of excluded participants does not differ from the included participants on demographic factors. A second limitation is that due to demographics of the current sample, our findings may not generalize to more affluent, rural, or other race/ethnic populations. Third, due to changes in the FFCWS questionnaire at year 15, we were unable to control for current life stress using the composite scores we created for ages 3, 5, and 9 years (Hein, 2019). To compensate, we used a life stress scale to control for current stress and found that it did not impact our main findings, suggesting that the effects were unique to childhood, rather than adolescent, adversity. Additionally, the FFCWS study did not collect data between ages 9 and 15, so it was not possible to prospectively account for exposures during this important developmental period. Fifth, human neuroimaging methods precluded us from determining how white matter may influence the direction of signaling between the amygdala and OFC. Consistent with models from non-human primate neural tract tracer research (Ray & Zald, 2012; Zikopoulos et al., 2017), we posit that the OFC inhibits the amygdala; however, the influence may be bidirectional. Last, previous research identified white matter tracts outside of those preregistered in the present study connecting the amygdala and PFC that may be shaped by early adversity (Choi et al., 2012; Huang et al., 2012; Jackowski et al., 2008). Additionally, although the present work used Brodmann's Areas for ROI

selection, previous work has used different anatomical parcellations. Future research examining potential effects of violence exposure and its interaction with social deprivation on additional pathways and using more precise anatomical parcellations would help to better understand how early adversity shapes the brain.

Results from the present study clarify possible directions for future research. Although longitudinal environmental data was a strength of the present study, we only had imaging data at one timepoint. Future research with longitudinal MRI data (Casey et al., 2018) may be able to better examine potential directional relations between dimensional early adversity and the brain by charting trajectories of development. Additionally, future research could characterize possible effects of other dimensions of adversity. We conceptualized violence exposure and social deprivation as composites made up of multiple timepoints in development and sources of exposure to create a cumulative assessment of dimension exposure to violence and social deprivation during childhood. However, it is likely that the proximity of exposure to the child and its developmental timing influence the magnitude of its effect (Dunn et al., 2013). Future research could work to break down the composites for each dimension to determine the importance of source and timing of exposure. Last, the items included in the dimension encompassing social deprivation – social support do not include all potential sources of social support. Future research should work to account for additional sources of social support (i.e., school connectedness) which may influence white matter.

Conclusions

Exposures related to early adversity are complex and can be broken down into dimensions which may affect brain development in different ways. The present study shows, for the first time, that two dimensions of childhood adversity, violence exposure and social

deprivation, interact to predict adolescent white matter connecting right hemisphere amygdala–OFC which is involved in socio-emotional function. High childhood violence exposure together with high social deprivation led to a lower probability of amygdala–OFC white matter in adolescence and, based on the negative correlation between amygdala–OFC white matter connectivity and amygdala reactivity, potentially less OFC regulation of the amygdala to threat. This association was not present with low social deprivation (i.e., social support), potentially implicating social support as a neuroprotective factor.

Table 2.1: Participant exclusions and demographics

Reasons for Exclusions		
Reason	**Number Excluded-Right**	**Number Excluded-Left**
No dMRI Data	41	41
Preprocessing outliers > 5% in diffusion data[1]	1	1
No probabilistic tractography model convergence	5	5
Less than 70% of voxels in PFC masks	1	1
Statistically influential outlier[2]	6	6
Poor fMRI data[3]	31	31

Demographic Characteristics – Included vs. Full Sample Comparison			
	Right Hemisphere Sample (N=183)	**Left Hemisphere Sample (N=183)**	**Full Sample (N=237)**
Age	*M* = 15.85 yrs | *SD* = 0.52 yrs	*M* = 15.85 yrs | *SD* = 0.53 yrs	*M* = 15.88 yrs | *SD* = 0.54 yrs
Puberty	*M* = 3.25 | *SD* = 0.58	*M* = 3.26 | *SD* = 0.59	*M* = 3.24 | *SD* = 0.59
Gender	F = 98 | M = 85	F = 99 | M = 84	F = 125 | M = 112
Race	African American: 132 Caucasian: 26 Other: 25	African American: 133 Caucasian: 26 Other: 24	African American: 170 Caucasian: 34 Other: 33
Annual Income	$4,999 or less: 23 $5,000 to $19,999: 31 $20,000 to $39,999: 54 $40,000 to $69,999: 33 $70,000 or more: 28 Not Report/Missing: 14	$4,999 or less: 22 $5,000 to $19,999: 31 $20,000 to $39,999: 54 $40,000 to $69,999: 33 $70,000 or more: 28 Not Report/Missing: 15	$4,999 or less: 28 $5,000 to $19,999: 41 $20,000 to $39,999: 66 $40,000 to $69,999: 46 $70,000 or more: 35 Not Report/Missing: 21

[1]These outlier slices were detected using the automated diffusion MRI cleaning method from MRtrix (v.3.0.R3). Slices with an average intensity four or more standard deviations lower than predicted by eddy's Gaussian process model were marked as outlier slices and replaced with model predictions.

[2]The same number of participants were excluded in each hemisphere due to being a statistical outlier on their violence exposure and social deprivation composite scores; however, only one of the participants is an outlier in both hemispheres.

[3]These participants are only excluded for the analyses looking at the association between amygdala–PFC white matter connectivity and amygdala activation. This is due to no functional MRI data (N=6), artifacts in the data (N=7), less than 70% accuracy on the Faces task (N=15), or less that 70% of voxels included in the amygdala mask (N=3)

Table 2.2: Descriptive statistics for the main continuous predictor variables and covariates

Predictor	Mean (SD)	Minimum - Maximum
Violence Exposure[1]	0.04 (0.53)	-0.82 – 2.17
Social Deprivation[1]	0.03 (0.50)	-0.76 – 2.67
Internalizing Psychopathology[2]	0.02 (0.42)	-0.60 – 1.29
Current Life Stress[3]	10.13 (5.35)	0 – 25
Maternal Education[4]	2.13 (1.03)	1 – 4

[1] To calculate composite scores, the Z scores (zero-centered) for each of the childhood experiences were summed for each of the childhood experiences within a dimension (violence exposure and social deprivation) and then divided by the number of childhood experiences within a dimension for each participant.

[2] This variable is a multi-method, multi-informant latent factor that is constructed from the following measures: (1) K-SADS clinician report of past and current symptoms of dysthymia, social phobia, generalized anxiety disorder, major depression, and phobia and (2) parent and child report on the Mood and Feelings Questionnaire, Child Depression Inventory, and the Screen for Child Anxiety Related Disorders.

[3] This variable is the sum of all of the items from the Adolescent Life Events Scale (ALES).

[4] This is a self-report categorical variable with the following response options: 1 - less than high school, 2 - high school or equivalent, 3 - some college/technical school, 4 - college or graduate school

Table 2.3: *Stepwise regression predicting right amygdala–BA47 white matter*

These additive models show the base model with only covariates, the R^2 change when adding the non-significant main effects of violence exposure and social deprivation, and then the R^2 change when adding the significant interaction between violence exposure and social deprivation when predicting right amygdala–BA47 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.14	[0.02, 0.25]				
Race_1[2]	0.00	[-0.04, 0.05]	0.02	[-0.19, 0.23]		
Race_2[2]	-0.01	[-0.04, 0.03]	-0.03	[-0.24, 0.18]		
Gender	0.01	[-0.02, 0.04]	0.04	[-0.16, 0.24]		
Pubertal Status	0.02	[-0.01, 0.05]	0.10	[-0.07, 0.26]		
Maternal Education	0.00	[-0.02, 0.03]	0.03	[-0.16, 0.22]		
Maternal Marital Status	-0.00	[-0.00, 0.00]	-0.10	[-0.26, 0.06]		
Current Life Stress	-0.00	[-0.02, 0.01]	-0.07	[-0.23, 0.10]		
Internalizing Psychopathology	-0.01	[-0.05, 0.02]	-0.08	[-0.25, 0.09]		
					R^2 = .022 95% CI[.00,.03]	
(Intercept)	0.14	[0.02, 0.25]				
Violence Exposure	-0.00	[-0.03, 0.02]	-0.01	[-0.19, 0.18]		
Social Deprivation	0.01	[-0.02, 0.03]	0.04	[-0.14, 0.22]		
Race_1[2]	0.00	[-0.04, 0.05]	0.01	[-0.20, 0.23]		
Race_2[2]	-0.01	[-0.04, 0.03]	-0.04	[-0.25, 0.18]		
Gender	0.01	[-0.02, 0.04]	0.04	[-0.16, 0.24]		
Pubertal Status	0.02	[-0.01, 0.05]	0.09	[-0.08, 0.26]		
Maternal Education	0.00	[-0.02, 0.03]	0.03	[-0.16, 0.23]		
Maternal Marital Status	-0.00	[-0.00, 0.00]	-0.10	[-0.26, 0.07]		
Current Life Stress	-0.00	[-0.02, 0.01]	-0.06	[-0.23, 0.11]		
Internalizing Psychopathology	-0.01	[-0.05, 0.02]	-0.08	[-0.25, 0.09]		
					R^2 = .023 95% CI[.00,.01]	ΔR^2 = .001 95% CI[-.01, .01]
(Intercept)	0.14	[0.03, 0.26]				
Violence Exposure	0.01	[-0.02, 0.04]	0.07	[-0.12, 0.25]		
Social Deprivation	0.02	[-0.01, 0.05]	0.13	[-0.06, 0.32]		
Interaction[1]	**-0.04***	**[-0.08, -0.01]**	**-0.26**	**[-0.44, -0.08]**		
Race_1[2]	0.01	[-0.03, 0.06]	0.07	[-0.15, 0.28]		
Race_2[2]	-0.01	[-0.04, 0.03]	-0.05	[-0.26, 0.16]		
Gender	0.01	[-0.02, 0.04]	0.04	[-0.16, 0.24]		
Pubertal Status	0.02	[-0.01, 0.05]	0.09	[-0.08, 0.26]		
Maternal Education	0.00	[-0.02, 0.03]	0.03	[-0.17, 0.22]		

Maternal Marital Status	-0.00	[-0.00, 0.00]	-0.09	[-0.25, 0.07]		
Current Life Stress	-0.00	[-0.02, 0.01]	-0.05	[-0.21, 0.12]		
Internalizing Psychopathology	-0.02	[-0.05, 0.01]	-0.09	[-0.25, 0.08]		
					R^2 = .069	ΔR^2 = .046**
					95% CI[.00,.09]	95% CI[-.01, .11]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
***** Significant predictor using a Bonferroni corrected threshold (p<0.05/8 tests = 0.0063)
** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation
[2]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)

Table 2.4: Regression results from amygdala-prefrontal cortex white matter connectivity predicting ipsilateral amygdala activation to threat faces adjusting for covariates.

	B	***SEB***	***β***	***t***	***p***
Model: R. Amygdala Activation (Threat) ~ R. Amygdala-BA47 White Matter Connectivity					
RAmy_BA47*	**-2.578**	**0.706**	**-0.290**	**-3.651**	**<0.001**
Internalizing	0.184	0.146	0.115	1.261	0.209
Pubertal Status	0.068	0.120	0.059	0.570	0.569
Gender	0.177	0.150	0.131	1.182	0.239
Race_1[1]	-0.030	0.204	-0.016	-0.149	0.882
Race_2[1]	-0.013	0.160	-0.009	-0.082	0.935
Current Life Stress	0.017	0.011	0.139	1.614	0.110
Maternal Education	-0.083	0.057	-0.124	-1.467	0.145
Maternal Marital Status	0.096	0.146	0.055	0.655	0.514
$F(9, 139) = 2.746$, $p = 0.005$, $R^2 = 0.151$					
Model: R. Amygdala Activation (Threat) ~ R. Amygdala-BA10 White Matter Connectivity					
RAmy_BA10*	**-4.105**	**1.363**	**-0.249**	**-3.013**	**0.003**
Internalizing	0.141	0.147	0.088	0.957	0.340
Pubertal Status	0.048	0.122	0.042	0.396	0.692
Gender	0.154	0.142	0.114	1.015	0.332
Race_1[1]	-0.067	0.207	-0.036	-0.325	0.746
Race_2[1]	0.024	0.164	0.016	0.148	0.883
Current Life Stress	0.020	0.011	0.163	1.881	0.062
Maternal Education	-0.089	0.058	-0.133	-1.542	0.125
Maternal Marital Status	0.156	0.147	0.091	1.061	0.290
$F(9, 139) = 2.238$, $p = 0.023$, $R^2 = 0.127$					
Model: L. Amygdala Activation (Threat) ~ L. Amygdala-BA10 White Matter Connectivity					
LAmy_BA10*	**-12.567**	**3.165**	**-0.322**	**-3.970**	**<0.001**
Internalizing	0.175	0.130	0.122	1.342	0.182
Pubertal Status	0.006	0.104	0.006	0.054	0.958
Gender	0.077	0.131	0.065	0.590	0.556
Race_1[1]	0.072	0.176	0.043	0.406	0.685
Race_2[1]	0.119	0.140	0.091	0.864	0.389
Current Life Stress	0.012	0.009	0.112	1.287	0.200
Maternal Education	-0.071	0.049	-0.120	-1.436	0.153
Maternal Marital Status	0.032	0.128	0.021	0.253	0.801
$F(9, 140) = 2.789$, $p = 0.005$, $R^2 = 0.152$					
Model: L. Amygdala Activation (Threat) ~ L. Amygdala-BA11 White Matter Connectivity					
LAmy_BA11*	**-6.743**	**1.871**	**-0.290**	**-3.604**	**<0.001**
Internalizing	0.129	0.130	0.090	0.991	0.323
Pubertal Status	0.062	0.106	0.062	0.587	0.558
Gender	0.158	0.132	0.132	1.192	0.235
Race_1[1]	0.066	0.178	0.039	0.368	0.713
Race_2[1]	0.103	0.139	0.079	.743	0.458
Current Life Stress	0.017	0.009	0.156	1.813	0.072
Maternal Education	-0.078	0.050	-0.132	-1.556	0.122

Maternal Marital Status	-0.010	0.130	-0.007	-0.80	0.936

$F(9, 140) = 2.463$, $p = 0.012$, $R^2 = 0.137$

* significant at p<0.0063 (Bonferroni corrected significance level for 8 tests)

[1]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)

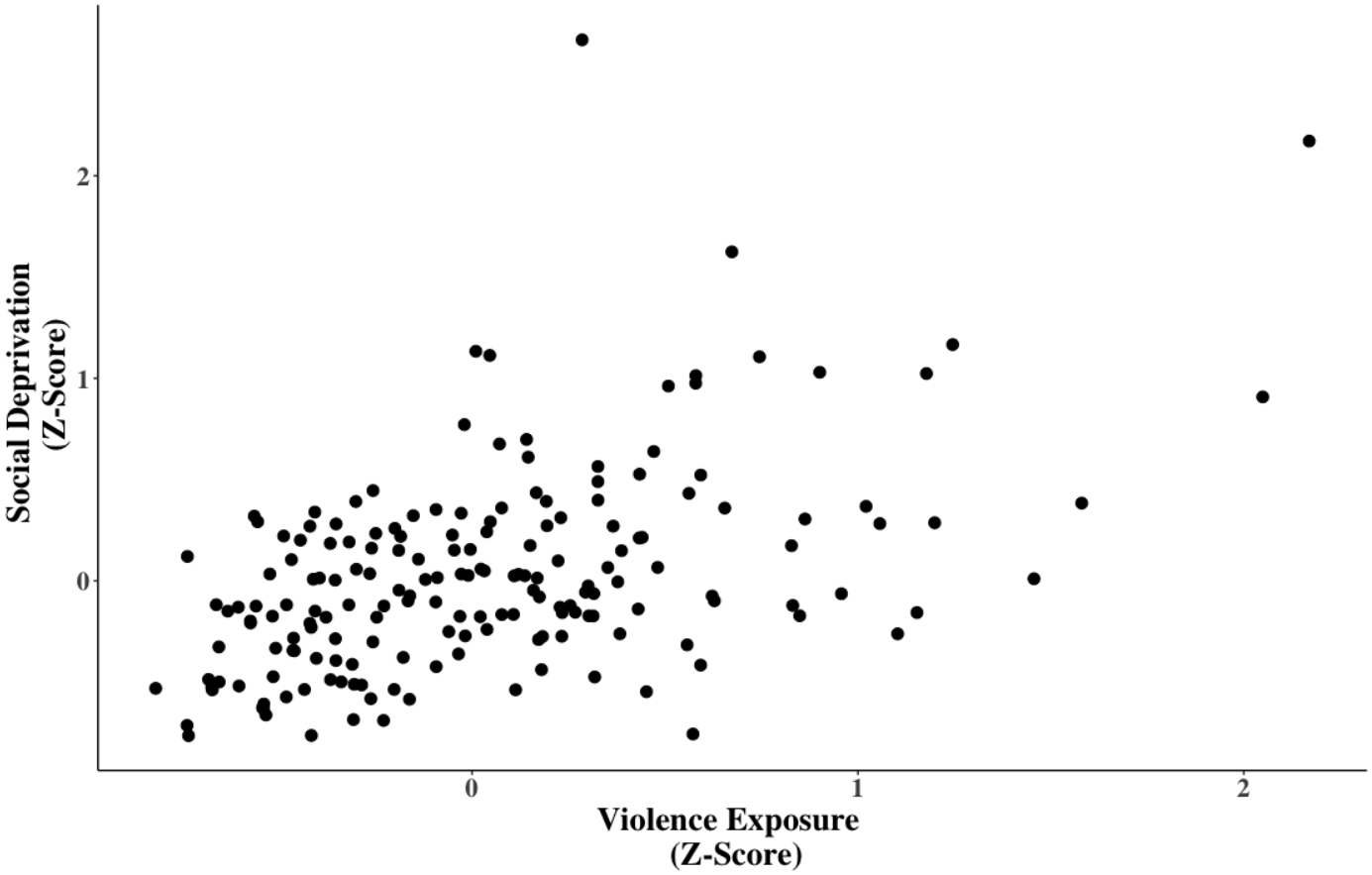


Figure 2.1: Association between violence exposure and social deprivation.

To calculate composite scores, the Z scores (zero-centered) for each of the childhood experiences were summed for each of the childhood experiences within a dimension (violence exposure and social deprivation) and then divided by the number of childhood experiences within a dimension for each participant. In our sample, violence exposure and social deprivation were correlated at $r(181) = 0.50$, $t = 7.69$, $p < 0.001$, but the VIF was 1.326.

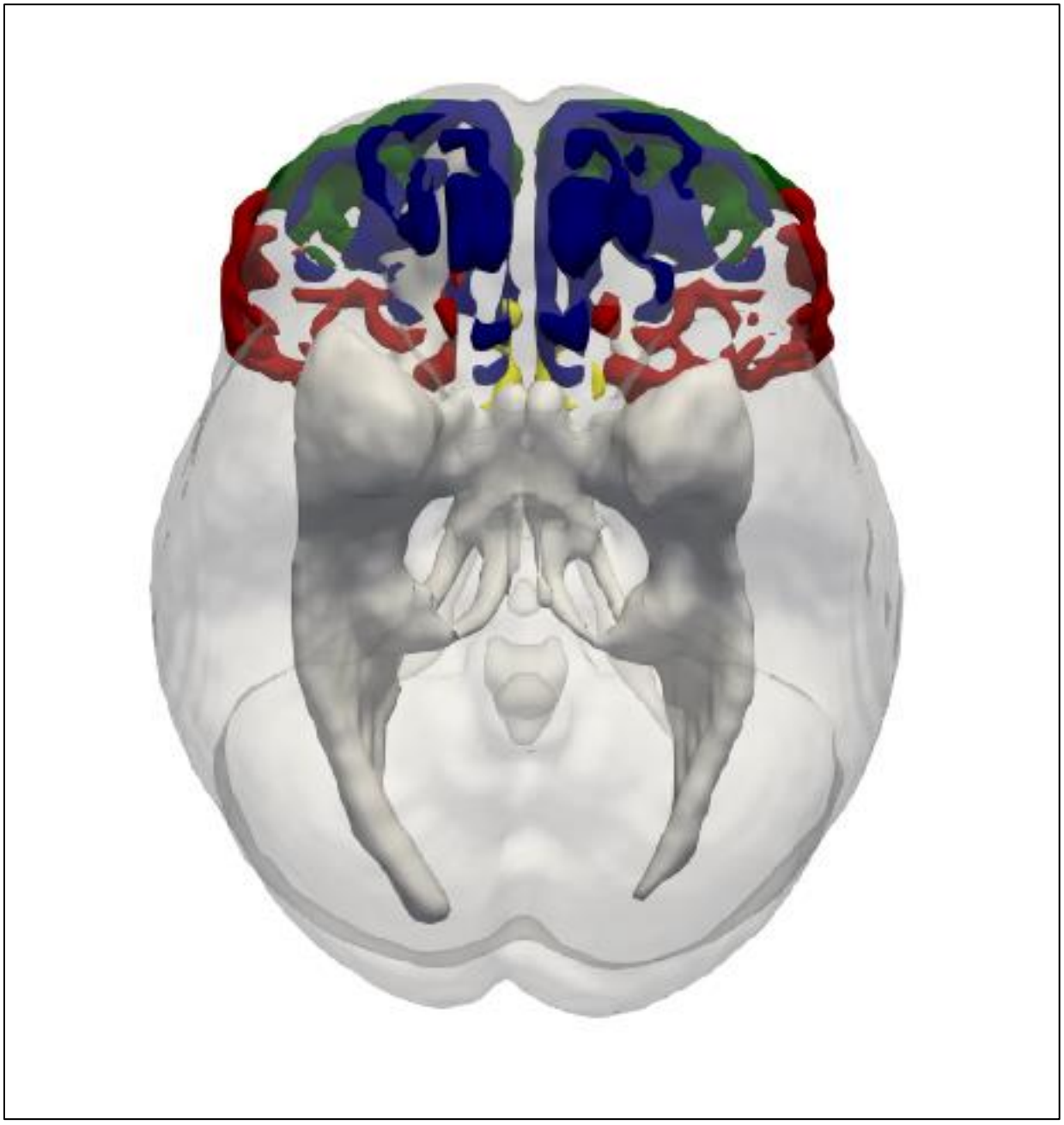


Figure 2.2: White matter tracts from the left and right amygdalae

For illustrative purposes, the Brodmann's Area (BA) masks used as targets are superimposed on the brain in different colors: BA10 (green), BA11 (blue), BA25 (yellow), BA47 (red).

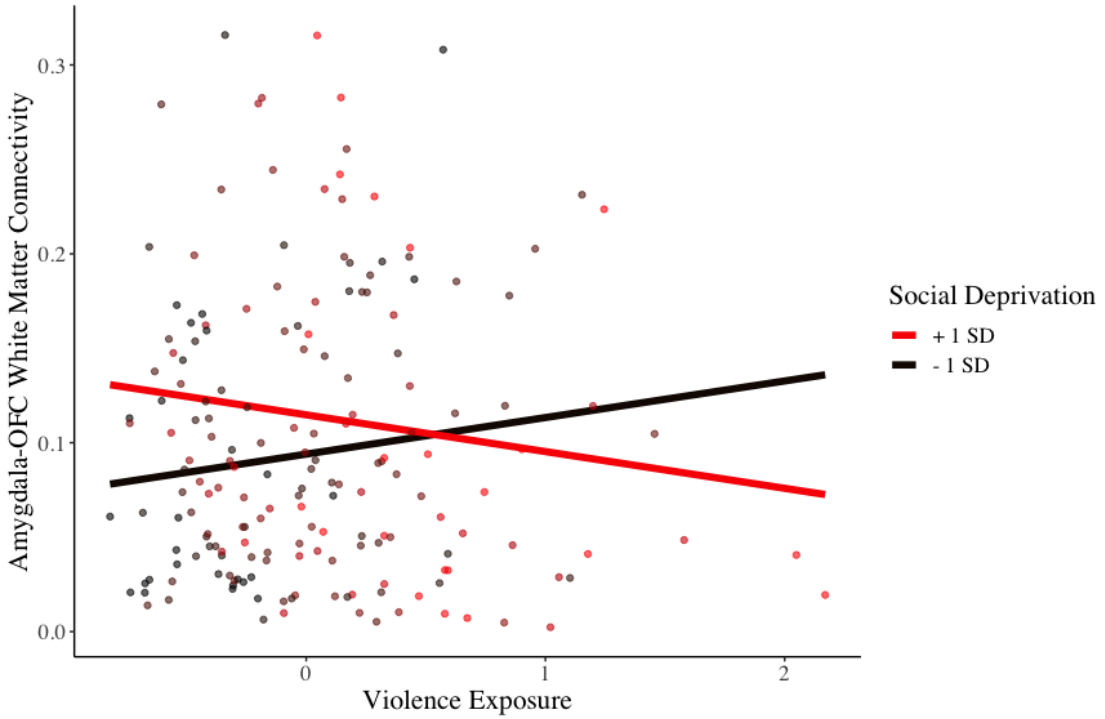


Figure 2.3: Simple slopes plot for VExSD interaction predicting white matter

Plot illustrating the interaction between childhood violence exposure and social deprivation (ages 3, 5, 9) in predicting the probability of white matter connectivity between the amygdala and orbitofrontal cortex (OFC – Brodmann's Area 47) in the right hemisphere (adolescence).

The continuous moderator (social deprivation) has been plotted at a +/- 1 standard deviation (SD) interval. A Johnson-Neyman interval shows that violence exposure and white matter connectivity are significantly, inversely correlated when social deprivation = 0.78 and greater. The range of social deprivation values (zero-centered where 0 is the mean) in the data are [-0.76 2.67]. This figure illustrates that at relatively high values of social deprivation, violence exposure and likelihood of amygdala–OFC connectivity are negatively correlated. Rug plots depict real data points along axes.

Chapter 3[2]: Association of Childhood Violence Exposure with Adolescent Neural Network Density

Early adversity is an unfortunately common and detrimental public health issue. Adverse childhood experiences (ACEs) negatively impact physical and mental health, and effects likely persist into adulthood (Finkelhor et al., 2015; Green et al., 2010; Nusslock & Miller, 2016). Early adverse environments have underlying dimensions, such as violence exposure (e.g., neighborhood violence) and social deprivation (e.g., neglect) (Hein, 2019; McLaughlin et al., 2014), which have distinct neural correlates related to emotion, fear, and reward processing (Goetschius, Hein, Mitchell, et al., 2020a; McLaughlin et al., 2014). For instance, violence exposure and social deprivation are associated with blunted amygdala and ventral striatum reactivity, respectively (Hein, 2019). However, it is unclear how these dimensions affect *neural circuitry*. ACEs (not measured dimensionally) are associated with alterations in resting-state functional connectivity (rsFC) of the salience network (SN; task-positive network, including the anterior insula, involved in identifying and integrating salient input (Uddin, 2017a)) and the default mode network (DMN; task-negative network, including the inferior parietal lobule, linked to internal thought, memory, and social-cognitive processes (Menon, 2011)) (Marusak, Etkin, et al., 2015; Marusak, Martin, et al., 2015; van der Werff et al., 2013), but inferences are limited by relatively small, homogeneous samples focused on few brain regions using retrospective reports of adversity (Falk et al., 2013). Thus, there are significant knowledge gaps

[2] Chapter 3 corresponds to Goetschius et al., 2020 published in *JAMA Network Open*

concerning the ways in which early violence exposure and social deprivation prospectively impact later functioning of neural circuits and how that varies across individuals.

Neural circuits are typically studied using a network framework, with key features including density (i.e., number of connections (Beltz & Gates, 2017)) and node degree (i.e., number of connections involving a specific brain region (Sporns, 2010)) (Power et al., 2010). Across development, network density increases between distal nodes and node degree increases for hub regions, such as the anterior insula (Fair et al., 2009). Understanding how early adversity relates to network density has significant potential for revealing how the environment affects brain development (Sporns, 2010).

These effects are likely to be person-specific because there is considerable variability in neural responses to environmental stress (Marder & Goaillard, 2006), and thus, mean-based analyses may not accurately reflect an individual's circuitry (Molenaar, 2004). Indeed, data from recent neuroimaging projects, such as the Midnight Scan Club, have illustrated that the organization of an individual's rsFC is unique and qualitatively different from the group average (Gordon et al., 2017). Moreover, consistent with behavioral studies of early adversity (Hughes et al., 2017), average estimates of adversity's effects on the brain often have high variances (Gee et al., 2013; Hanson, Hariri, et al., 2015; Hanson, Nacewicz, et al., 2015). This begs the question of whether there is valuable information about individual differences and their etiology – important for eventual prevention and intervention – that is not being conveyed by mean-based conclusions.

In the present study, we examined the association between dimensional indices of childhood exposure and individualized adolescent rsFC networks. We employed a large, longitudinal sample of adolescents with a substantial representation of African American and

low-income participants — who are often underrepresented in neuroimaging research (Falk et al., 2013)– and a person-specific rsFC approach that detects only meaningful connections among brain regions while identifying subgroups of participants that share network features (GIMME) (Gates et al., 2010; Lane et al., 2019). We hypothesized that childhood violence exposure and social deprivation would be associated with person-specific indices of SN and DMN density, respectively. This study was pre-registered (https://osf.io/mrwhn/), and the data will be openly available (https://nda.nih.gov/edit_collection.html?id=2106).

Materials & Methods

Participants were from the FFCWS, a population-based cohort study of children born in large US cities, with an oversample of non-marital births as well as a large proportion of families of color with low resources (Reichman et al., 2001). This study followed STROBE (acronym in Table 3.1) reporting guidelines. In the FFCWS, data were collected from the primary caregiver (94% biological mother) and focal child at their birth and at ages 1, 3, 5, 9, 15 years through in-home visits and phone calls (*Public Data Documentation | Fragile Families and Child Wellbeing Study*, 2019). Data for the violence exposure and social deprivation composites, which have been previously reported (Goetschius, Hein, Mitchell, et al., 2020a; Hein, 2019; Peckins et al., 2019), came from surveys collected at ages 3, 5, and 9. During Wave 6 (when focal child was approximately age 15), 237 teens from Detroit, Toledo, and Chicago participated in a supplementary visit where rsFC data were collected. RSFC data and their association with early adversity have not been previously published. Due to the representative sampling of the FFCWS, youth were not preselected based on their willingness to participate in an MRI study, a common procedure in neuroimaging. This led to missing or incomplete MRI data (N=54), but the included

sample did not statistically differ from the recruited sample (Appendix 3 and Supplemental Table 3.1).

Participants & Procedure

Participants were 175 adolescents aged 15-17 years. Teens and their primary caregiver came to a university lab and completed questionnaires regarding the focal child's current life stress, pubertal development, and other demographic variables. During an MRI scan, eight minutes of data were collected while teens were instructed to remain still and focus on a white fixation cross on a black screen. This study was approved by the University IRB. Participants provided informed consent or assent.

Childhood Violence Exposure and Social Deprivation Measures

Violence exposure and social deprivation were each operationalized by composite z-scores calculated from FFCWS data at ages 3, 5, and 9; thus, 0 is the approximate mean. Both constructs included primary caregiver report of experiences that directly (i.e., physical abuse) and indirectly (i.e., community support) affect the child (Appendix 3). Violence exposure was operationalized as physical or emotional abuse directed at the child, exposure to intimate partner violence, and witnessing or being victimized by community violence. Social deprivation was operationalized as emotional or physical neglect, lack of romantic partner support for the mother, and lack of neighborhood cohesion (Hein, 2019). To reflect a comprehensive assessment of cumulative, dimensional childhood exposure to violence and social deprivation, both constructs included experiences with several levels of proximity to the child (e.g., home, neighborhood) at multiple ages (Hein, 2019; McLaughlin & Sheridan, 2016).

Covariates

To address potential confounds, sensitivity analyses adjusted for gender, race, pubertal development (Carskadon & Acebo, 1993), adolescent life stress (Farrell et al., 1998; Masten et al., 1994; Shaw et al., 2003), maternal education at birth, and maternal marital status at birth (Reichman et al., 2001) (Appendix 3).

Neuroimaging Measures

MR Acquisition. MR data were acquired using a GE Discovery MR750 3T scanner with an 8-channel head coil. Head movement was minimized through instructions to the participant and padding placed around the head. Functional T2*-weighted BOLD images were acquired using a reverse spiral sequence (Glover & Law, 2001) of 40 contiguous axial 3 mm slices (Appendix 3).

Imaging Data Analysis. Preprocessing was primarily conducted in FSL (v.5.0.7) (Beltz et al., 2019; Jenkinson et al., 2012). Structural images were skull-stripped and segmented. Functional images were skull-stripped, spatially smoothed, registered to subject-specific structural and MNI space, and corrected for motion using MCFLIRT (Jenkinson et al., 2002) and ICA-AROMA (Pruim et al., 2015). Nuisance signal from white matter and CSF was removed. Data were high-pass filtered. As an additional precaution against motion-related artifacts, participants with an average relative framewise displacement (FD) greater than 0.5mm (prior to motion preprocessing) were excluded (N=4) (Appendix 3).

Participant-specific time series (235 functional volumes) from seven ROIs per hemisphere (14 total) were extracted. ROIs and their locations were selected using Neurosynth (Yarkoni et al., 2011) and preregistered. Five bilateral ROIs defined the SN: amygdala, insula, dACC, and dlPFC. Three bilateral ROIs defined the DMN: IPL, PCC, and MTG (Table 3.1 for

acronyms). ROIs were 6.5mm spheres around central coordinates (Supplemental Table 3.2 in Appendix 2) linearly adjusted for participant brain volume.

GIMME. Subgrouping GIMME (S-GIMME; v0.5.1) (Gates et al., 2010, 2017; Lane et al., 2019) in R (v3.5.1) was used for rsFC analyses (Supplemental Figure 3.1 in Appendix 3). Beginning with an empty network, S-GIMME fits person-specific unified structural equation models (Gates et al., 2010) in a data-driven manner by using Lagrange multiplier tests (Sörbom, 1989) to add directed connections among ROIs that are contemporaneous (occurring at the same functional volume) or lagged (occurring at the previous volume, including autoregressives), and that apply to the group-level (everyone in the sample), subgroup-level (everyone in a data-derived subsample), or individual-level (unique to an individual). Importantly, S-GIMME is a sparse mapping approach in which only connections that account for a significant amount of variance are added to each participant's network until the model fits the data well according to standard fit indices (RMSEA$\leq$0.05; SRMR$\leq$0.05; CFI$\geq$0.95; NNFI$\geq$0.95) (Brown, 2006; Gates & Molenaar, 2012). During model generation, connections that have become non-significant with the addition of new connections are pruned. S-GIMME uses a community detection algorithm (Walktrap) to detect subgroups of participants and determine their shared connectivity patterns. S-GIMME has been described and validated in large-scale simulations and applied to empirical data (Gates et al., 2014, 2017; Gates & Molenaar, 2012; Price et al., 2017). Network density and node degree of only the contemporaneous connections were extracted from person-specific S-GIMME networks, as lagged connections control for sequential dependencies (e.g., hemodynamic response function) (Gates et al., 2010; S. M. Smith, 2012).

Statistical Analysis

Inferential analyses were completed in R (v3.5.1) and examined whether childhood adversity statistically predicted adolescent neural network features. First, binary logistic regression was used to statistically predict S-GIMME-detected subgroup membership from childhood violence exposure and social deprivation. Second, multiple regression was used to statistically predict *density* (i.e., number of connections) within the SN, within the DMN, and between the SN and DMN from childhood violence exposure and social deprivation. Third, multiple regression was used to statistically predict *node degree* (i.e., number of connections involving a specified node) for each of the 14 ROIs from violence exposure and social deprivation using a Bonferroni-corrected significance threshold ($p<0.004$). In follow-up sensitivity analyses, covariates were added to all regressions to assess the robustness of observed effects.

Results

Analyses included 175 15-17-year-olds (M_{age}=15.88, SD=0.53; 56% female; 73% African American). All person-specific resting state networks fit the data well, according to average indices: RMSEA=.06, SRMR=.05, CFI=.93, NNFI=.96 (individual indices in Appendix 5). Group-level connections were detected within and between the SN and DMN (Figure 3.1a), two subgroups of participants with subgroup-level connections were identified (Figures 3.1b, 3.1c), and all person-specific maps contained individual-level connections ($M_{individual\_connections}$ =11.61, SD=5.32). Final maps revealed that the first subgroup (N=42; Figure 3.1b, 3.1d) was qualitatively homogeneous with 27 subgroup-level connections and few individual-level connections ($M_{individual\_connections}$ =5.60, SD=3.19), and that the second subgroup (N=133; Figure 3.1c, 3.1e) was qualitatively heterogeneous, with 8 subgroup-level connections and many

individual-level connections ($M_{individual\_connections}$=13.50, *SD*=4.36). Possible extreme outliers or overfit models did not impact results (Appendix 4).

Violence Exposure was Associated with Neural Subgroup Membership

Violence exposure was associated with subgroup membership (*b*=1.12, *p*=0.030). With a unit increase in violence exposure, participants were 3.06 times more likely to be classified in the larger, heterogeneous subgroup (Table 3.2). In sensitivity analyses, the odds ratio was 2.54 (Supplemental Table 3.6 in Appendix 4). On average, members of the heterogeneous subgroup experienced higher levels of childhood violence exposure (*M*=0.09, *SD*=0.53) than those in the homogenous subgroup (*M*=-0.15, *SD*=0.43). Social deprivation was not associated with subgroup membership (Table 3.2).

Violence Exposure was Associated with Density Within the SN and between the SN and DMN

Childhood violence exposure was related to reduced density (i.e., sparsity) in the person-specific maps (β=-0.25, *p*=0.005) (Figure 3.2). Specifically, violence exposure was associated with sparsity within the SN (β=-0.26, *p*=0.005) and between the SN and DMN (β=-0.20, *p*=0.023) (Table 3.3A), including in sensitivity analyses (Supplemental Table 3.3 in Appendix 4). Violence exposure was not associated with DMN density, and social deprivation was not related to network metrics (Table 3.3A).

Violence Exposure was Associated with Node Degree for Left IPL and Right Insula

Childhood violence exposure was related to reduced node degree for the right insula (β=-0.29, *p*=0.001) and left IPL (β=-0.26, *p*=0.003) using a Bonferroni-corrected significance threshold (Tables 3.3B and Supplemental Table 3.4 in Appendix 4), including in sensitivity

analyses (Supplemental Table 3.5 in Appendix 4). There were no significant associations with social deprivation.

Discussion

Results from a predominantly under-studied and under-served sample with high rates of poverty revealed that childhood violence exposure, but not social deprivation, was associated with adolescent neural circuitry. Data-driven analyses identified a subset of teens with heterogeneous patterns of connectivity (i.e., few shared and many individual connections) in two key neural networks associated with salience detection, attention, and social-cognitive processes (i.e., the SN and DMN) (Menon, 2011; Uddin, 2017a). This subgroup of teens was exposed to more violence in childhood than the other subgroup, whose patterns of neural connectivity were relatively more homogeneous (i.e., had many connections in common), suggesting that violence exposure may lead to more person-specific alterations in neural circuitry. Beyond subgroups, network density within the SN and between the SN and DMN was sparse for teens with high violence exposure, likely due to few connections involving the right insula and the left IPL. These effects could not be accounted for by social deprivation, in-scanner motion, race, gender, pubertal development, current life stress, or maternal marital status or education at birth. Findings regarding the neural network subgroups are noteworthy because the community detection algorithm within GIMME detected rsFC patterns in the brain from exposures that occurred at least 6 years earlier. Moreover, high childhood violence exposure in the subgroup characterized by neural heterogeneity likely reflects the person-specific effects of early adversity on the brain and suggests that research on the developmental sequelae of ACEs must consider individual differences in neural compensatory responses to stress (Marder & Goaillard, 2006). Although it important to replicate these findings in other samples, S-GIMME has reliably

classified subgroups in empirical data (Gates et al., 2017; Price et al., 2017), and there is evidence from simulations that modeling connections at the subgroup-level, in addition to the group-level, improves the validity and reliability of results (Gates et al., 2017).

Considering the sample as a whole, results also suggest that violence exposure is associated with blunted connectivity within the SN and between the SN and DMN. As expected, the observed reduced SN density in teens with heightened childhood violence exposure differs from typical developmental patterns that show stronger rsFC within SN nodes and increased density of connections with hub regions, such as the anterior insula, as the brain matures (Fair et al., 2009; Menon, 2011). It is difficult, however, to align the present findings with previous work that showed increased SN rsFC in trauma-exposed youth (Marusak, Etkin, et al., 2015; Marusak, Martin, et al., 2015) because those samples were small, used different metrics of connectivity, and had different sample compositions. Moreover, the current sample was likely experiencing chronic adversity and research from animal models of chronic stress propose that, over time, the body's stress response (e.g. HPA-axis reactivity) becomes blunted or habituated to typical stressors (McCarty, 2016). Previous research on HPA-axis reactivity in this sample revealed a blunted cortisol response in teens exposed to heightened childhood violence exposure (M. K. Peckins et al., 2019), and work in other high-risk samples shows blunted activation of the amygdala, a SN node, to threatening stimuli (Gard et al., 2017; Holz et al., 2017). The present study expands this notion to the function of threat detection neural circuits, and future research should examine whether this is compensatory or even adaptive.

Beyond density, childhood violence exposure was associated with reduced node degree of the right anterior insula and left IPL. These results are consistent with the extant literature because the right anterior insula in the SN facilitates shifting between the DMN and central

executive network (Sridharan et al., 2008), which contributes to higher-level executive function (Menon, 2011). Moreover, early life stress has been linked to insular connectivity within the SN (Marusak, Etkin, et al., 2015), DMN (specifically the left IPL, which plays a role in working memory (Philip et al., 2016)), and other neural ROIs (Teicher et al., 2014). These results also illuminate differences in the way that the anterior insula is integrated within and between neural networks in youth exposed to violence in their homes and neighborhoods using longitudinal data from a population-based sample.

This study represents a person-specific approach to the neuroscientific investigation of the sequelae of early adversity. Past research on early adversity and rsFC assumes that the same connectivity patterns characterize all, or a majority of participants, but if this assumption is violated (as is likely the case in studies of diverse populations and biopsychosocial phenomena), then results may not accurately describe any individual(Eavani et al., 2015; Molenaar, 2004). The presence of group- and subgroup-level connections in the current study suggests that there was some consistency in the connections within and between the SN and DMN, aligning with an assumption of homogeneity that is prevalent in rsFC research, but the large number of individual-level connections, especially in adolescents with high early violence exposure, show that there was also notable heterogeneity that required person-specific analyses to accurately reflect rsFC, encouraging future research using person-specific modeling approaches. Interestingly, all significant findings concerned violence exposure, and there were no detected links between social deprivation and rsFC. This set of results could indicate that social deprivation has a less salient influence on patterns of spontaneous neural fluctuations. Some studies have identified links between social deprivation and functional connectivity, but they concerned extreme, non-normative deprivation (i.e., previous institutionalization)(Chugani et al.,

2001; Gee et al., 2013). This deprivation may be qualitatively different from deprivation operationalized in this study, and it may operate through different mechanisms. Additionally, because a hypothesis driven approach to node selection was taken in this study, it is possible that deprivation is associated with rsFC of SN or DMN nodes not measured here, with other networks (e.g., central executive), or in different populations (e.g., with extreme or heightened variability of deprivation). It is also tenable that there are other dimensions of adversity that would have differential associations with rsFC (e.g., those linked to emotionality), which future research should explore. Nonetheless, these findings present evidence for dimensional frameworks of adversity(Hein, 2019; Sheridan & McLaughlin, 2014) because there were *distinct* neural correlates for violence exposure.

Limitations

Based on the demographics of the sample (e.g., 72% African American, born in Midwestern cities), it is not clear how findings will generalize beyond low-income, urban, African American youth; nonetheless, the present work is vital because these populations are often underrepresented in neuroimaging research and underserved by the medical community(Falk et al., 2013). Resting-state fMRI was only collected at a single occasion in adolescence; thus, it is unclear if connectivity patterns reflect stable or changing neural features. Nor is it possible to know the direction of association (e.g., whether neural differences pre-date exposure to adversity). Violence exposure and social deprivation composites were derived from parent-reports. Exposures between the FFCWS collection waves at ages 9 and 15 could not be accounted for in this study. Due to changes in the FFCWS questionnaire at year 15, current adversity could not be controlled using the composite scores created for earlier ages (Hein, 2019). To compensate, a life stress scale was used as a covariate (that did not impact

associations). The ecology of poverty-related adversity is complex, and thus, there are unmeasured variables that may explain these associations or contribute to cascades of risk (e.g., parental psychopathology).

Conclusions

In a prospective, longitudinal study, childhood violence exposure, but not social deprivation, was associated with person-specific differences in how the adolescent brain functions in regions involved in salience detection and higher-level cognitive processes. These differences were potent enough that a data-driven algorithm, blind to child adversity, grouped youth with heightened violence exposure together based on the heterogeneity of their neural networks, suggesting that the impact of violence exposure may have divergent and personalized associations with functional neural architecture. Findings have implications for understanding how dimensions of adversity affect brain development, which can inform future neuroscience-based policy interventions.

Table 3.1: Table of frequently used acronyms

ACE	Adverse Childhood Experience
CFI	Confirmatory Fit Index
dACC	Dorsal Anterior Cingulate Cortex
dlPFC	Dorsolateral Prefrontal Cortex
DMN	Default Mode Network
FFCWS	Fragile Families and Child Wellbeing Study
IPL	Inferior Parietal Lobule
MTG	Medial Temporal Gyrus
NNFI	Non-Normed Fit Index
PCC	Posterior Cingulate Cortex
RMSEA	Root Mean Square Error of Approximation
ROI	Region of Interest
rsFC	Resting State Functional Connectivity
SN	Salience Network
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
S-GIMME	Subgrouping Group Iterative Multiple Model Estimation
SRMR	Standard Root Mean Residual

Table 3.2: Logistic regression results for subgroup membership

Logistic regression results for association between violence exposure and social deprivation and subgroup membership (heterogeneous or homogeneous subgroup) while controlling for motion.

Predictor	*b*	*SE*	*Odds Ratio*	*Odds Ratio* 95% CI [LL, UL]
(Intercept)	0.55	0.32	1.73	[0.90, 3.22]
Violence Exposure*	**1.12**	**0.52**	**3.06**	**[1.17, 8.92]**
Social Deprivation	-0.49	0.46	0.61	[0.25, 1.54]
Motion[1]*	**7.96**	**3.59**	**2860.05**	**[6.33, 8236598]**

***** Significant predictor of subgroup membership

[1]Motion indicated by mean relative framewise displacement

Table 3.3: Regression results for network density and node degree

Regression results for association between dimensional exposure to adversity and (A) network density (i.e., number of connections modeled by network) and (B) node degree (i.e., the sum of the modeled connections involving each node). For space, only significant nodes are reported here. Results for all nodes are in the Appendix 4.

Predictor	***b***	***b* 95% CI [LL, UL]**	***beta***	***beta* 95% CI [LL, UL]**	***r***	**Fit**
Total Density						
(Intercept)	41.88	[40.57, 43.19]				
Violence Exposure*	**-3.08**	**[-5.20, -0.96]**	**-0.25**	**[-0.41, -0.05]**	**-.12**	
Social Deprivation	1.81	[-0.28, 3.91]	0.15	[-0.05, 0.30]	.06	
Motion [1]*	**12.98**	**[3.55, 22.40]**	**0.21**	**[0.06, 0.37]**	**.17***	
						R^2 = .075
						95% CI[.01,.15]
Salience Network Density						
(Intercept)	15.92	[15.47, 16.37]				
Violence Exposure*	**-1.09**	**[-1.82, -0.36]**	**-0.26**	**[-0.43, -0.08]**	**-.11**	
Social Deprivation	0.65	[-0.08, 1.37]	0.15	[-0.02, 0.32]	.07	
Motion [1]*	**5.17**	**[1.91, 8.43]**	**0.24**	**[0.09, 0.39]**	**.20**	
						R^2 = .088
						95% CI[.02,.16]
Density Between Salience & Default Mode Networks						
(Intercept)	16.03	[15.32, 16.74]				
Violence Exposure*	**-1.33**	**[-2.48, -0.19]**	**-0.20**	**[-0.38, -0.03]**	**-.07**	
Social Deprivation	0.76	[-0.37, 1.90]	0.12	[-0.06, 0.29]	.06	
Motion [1]*	**8.58**	**[3.48, 13.67]**	**0.25**	**[0.10, 0.40]**	**.23**	
						R^2 = .079
						95% CI[.01,.15]
Default Mode Network Density						
(Intercept)	9.93	[9.50, 10.36]				
Violence Exposure	-0.66	[-1.35, 0.03]	-0.17	[-0.35, 0.01]	-.13	
Social Deprivation	0.40	[-0.28, 1.09]	0.10	[-0.07, 0.28]	.00	
Motion [1]	-0.77	[-3.85, 2.30]	-0.04	[-0.19, 0.12]	-.06	
						R^2 = .024
						95% CI[.00,.07]
Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	*r*	Fit
Left Inferior Parietal Lobule Degree						
(Intercept)	7.33	[6.98, 7.68]				
Violence Exposure**	**-0.85**	**[-1.41, -0.28]**	**-0.26**	**[-0.44, -0.09]**	**-.17**	
Social Deprivation	0.49	[-0.07, 1.05]	0.15	[-0.02, 0.33]	.03	
Motion [1]	1.16	[-1.35, 3.68]	0.07	[-0.08, 0.22]	.03	
						R^2 = .050

					95% CI[.00,.11]
Right Insula Degree					
(Intercept)	7.72	[7.48, 7.96]			
Violence Exposure**	**-0.65**	**[-1.04, -0.26]**	**-0.29**	**[-0.47, -0.12]**	**-.16**
Social Deprivation	0.42	[0.03, 0.80]	0.19	[0.01, 0.36]	.06
Motion [1]	1.38	[-0.36, 3.12]	0.12	[-0.03, 0.27]	.08
					R^2 = .067
					95% CI[.01,.14]

Note. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

* indicates significant predictor of network density at $p<0.05$.

** indicates significant predictor of node degree at a Bonferroni corrected $p<0.05/14$ nodes ($p<0.004$).

[1]Motion indicated by mean relative framewise displacement prior to motion correction

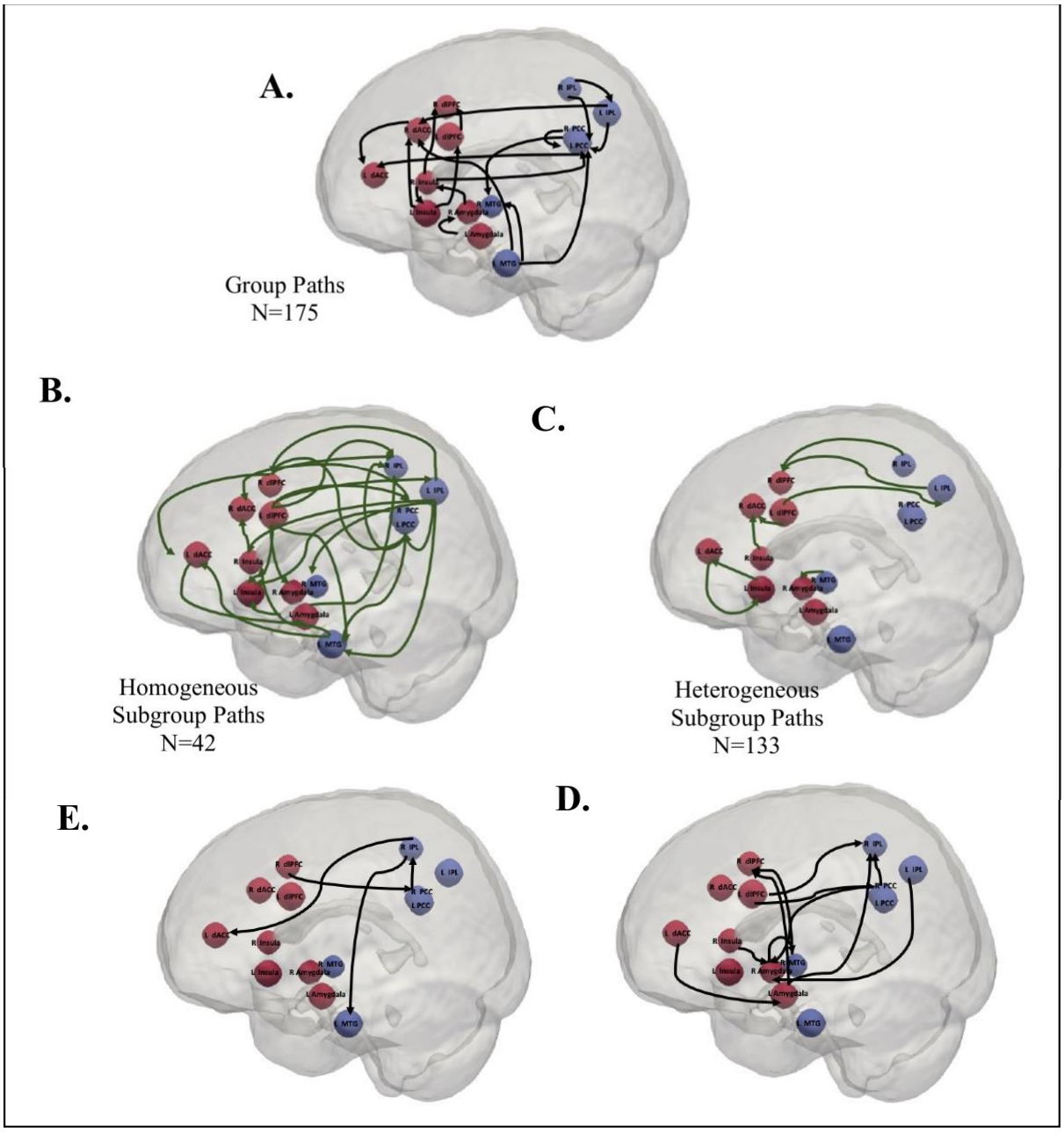


Figure 3.1: S-GIMME connectivity results

A) Connections fit at the group-level (i.e., statistically meaningful for ≥ 75% of sample); B) Subgroup-level connections for first, relatively homogenous (i.e., many shared, common paths), algorithm-detected subgroup (N=42); C) Subgroup-level connections for second, comparatively heterogenous (i.e., few common paths), algorithm-detected subgroup (N=133); D) Individual-level connections for illustrative participant in first subgroup which shows relatively fewer individual level paths; E) Individual-level connections for illustrative participant in second subgroup which shows comparatively more individual level paths. All connections are directed and contemporaneous. Red nodes are part of the salience network. Blue nodes are part of the default mode network.

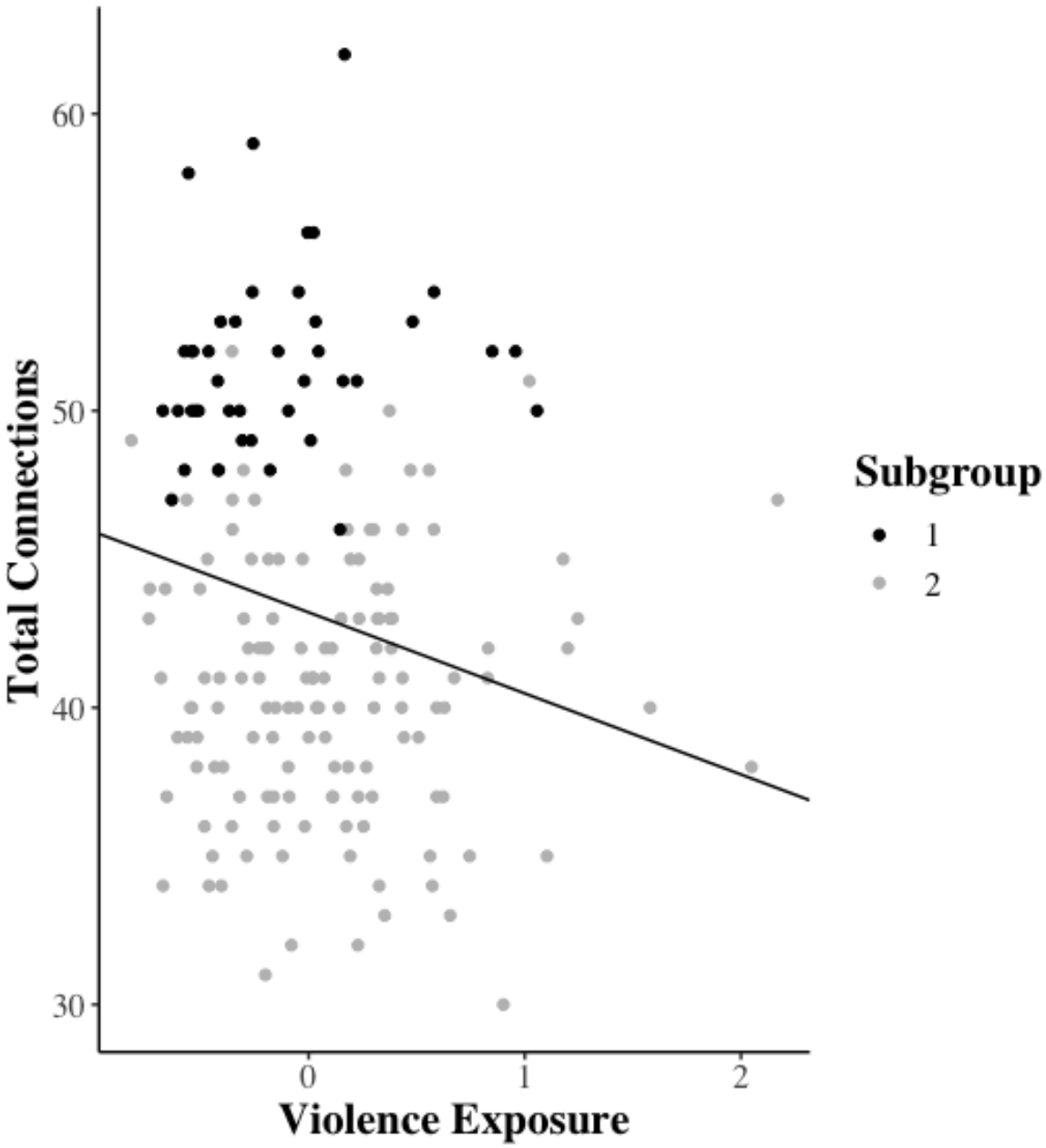


Figure 3.2: Association between violence exposure and network density

Association between childhood violence exposure and reduced network density (i.e., number of connections modeled for each individual).

Chapter 4: School Connectedness as a Protective Factor Against Childhood Exposure to Violence and Social Deprivation: A Longitudinal Study of Adaptive and Maladaptive Outcomes

Recent data from the Center for Disease Control and Prevention (CDC) show that over 60% of adults have experienced at least one adverse childhood experience (ACE) with close to 25% of adults reporting exposure to three or more ACEs (Merrick et al., 2018). On average, exposure to early adversity is associated with an increased risk for poor mental and physical health outcomes across the lifespan (Green et al., 2010; Nusslock & Miller, 2016). However, many children display resilience to early adversity and do not go on to develop poor outcomes (Masten et al., 2004). Multiple environmental factors, including those outside of the home, such as connectedness to school, have been posited to promote resilience (Barber & Olsen, 1997). The present study examined child and adolescent connectedness to school as protective against childhood exposure to early adversity. Consistent with prominent calls in the field (Masten & Cicchetti, 2016), this study is precise in its measure of two dimensions of adversity that index multiple levels of environmental exposure and examines resilience in terms of both the absence of negative outcomes and the presence of positive outcomes.

Though a wealth of literature has examined the effects of either specific adversities (e.g., abuse, harsh parenting) or the cumulative effects of adversity broadly (i.e., cumulative risk research), dimensional models of adversity propose that the complex experiences of early adversity can be broken down into core underlying dimensions that influence neural and behavioral development through both distinct, specific, and more common, global mechanisms

(McLaughlin et al., in press; McLaughlin & Sheridan, 2016). In the dimensional model of adversity and psychopathology (DMAP), one of the first and more prominent dimensional models, adversity is broken down into threat and deprivation (McLaughlin et al., 2014). We have specifically adapted these constructs to examine the dimensional correlates of violence exposure and social deprivation on child and adolescent functioning. Violence exposure, similar to the threat construct in DMAP, includes experiences of abuse, exposure to intimate partner violence in the home, and community violence (Hein et al., 2020). Social deprivation, which differs from the DMAP dimension of deprivation, includes experiences of neglect, a lack of support in the home, and a lack of a sense of cohesion and support in the neighborhood (Hein et al., 2020). These dimensions are conceptually and statistically distinct (Hein et al., 2020; Lambert et al., 2017; McLaughlin et al., 2014; Miller et al., 2018). For example, research has shown that violence exposure is specifically linked to differences in areas of the brain involved in fear-learning and emotion processing, while social deprivation has been related to differences in areas involved in social reward processing (Hein et al., 2020; McLaughlin et al., 2019). However, these dimensions coexist, are likely correlated, and interact with each other to create a unique environment of adversity for each individual (Goetschius, Hein, Mitchell, et al., 2020b; McLaughlin & Sheridan, 2016). Thus, it is important to assess exposure to both dimensions simultaneously.

Early Adversity & Resilience

Equally important to our understanding of how early adversity shapes development is understanding what factors help youth do well even when exposed to adverse environments. Resilience is defined as having positive outcomes in the context of adversity and research on the effects of early adversity has shown that “resilience is common” (Masten, 2001). Protective

factors that promote resilience to adversity can be internal factors, such as positive self-perception, or external factors, such as social support in the home reflected by quality of attachment or parenting (Luthar, Crossman, & Small, 2015; Masten, 2001). In the present study, we examine the protective effects of an extrafamilial source of social support – school connectedness.

Protective processes can differ depending on risk processes and domain of function (National Research Council (US) & Institute of Medicine (US) Board on Children, Youth, and Families, 2001), thus it is necessary to examine resilience effects not only in the context of multiple dimensions of childhood adversity, but also multiple outcomes. As outlined briefly above, evidence supports that dimensions of adversity shape development in ways that are at least partially distinct, but there is a lack of work empirically testing protective processes within this dimensional framework. Of equal importance to modeling the complexity of adversity in the study of resilience is the selection of outcomes. Much of the research on resilience to early adversity has focused on factors that mitigate negative function, such as symptoms of psychopathology; however, it is important to determine whether specific factors promote adaptive function using a strengths-based approach (Luthar et al., 2015; Masten & Cicchetti, 2016). Positive function (Kern et al., 2016), as measured in the present study, indexes qualities such as optimism and perseverance, two qualities that promote positive outcomes (Chen & Miller, 2012). However, successful adaptation can exist simultaneously with maladaptive factors (Luthar et al., 2015). Thus, to gain greater understanding of protective processes, it is important to evaluate factors in terms of both the presence of adaptive and the absence of maladaptive outcomes.

School Connectedness as Hypothesized Protective Factor

Youth spend a majority of their day at school and thus their experiences there play a large role in both socioemotional development and resilience (Masten & Cicchetti, 2016; Roeser, Eccles, & Sameroff, 2000). Connectedness to school, defined as a youth's sense of belonging or closeness with others at their school, has been associated with positive adolescent outcomes including reduced emotional distress, suicidality, violence, and substance use (Bond et al., 2007; Brookmeyer et al., 2006; Kalu et al., 2020; Resnick et al., 1997). This extrafamilial source of social support is posited to promote socioemotional function through the creation of supportive relationships with teachers and peers (Loukas et al., 2006). School connectedness as a buffer is rooted in the risk and resilience framework, which posits that factors that promote developmental competency may also promote resilience to early adversity (Luthar et al., 2000).

The school environment is a salient source of protective factors for a number of reasons. First, school context factors, such as connectedness, have been shown to buffer against the detrimental effects of cumulative, unidimensional adversity constructs as well as specific exposures, such as neighborhood violence and negative family relations (Hardaway et al., 2012; Lensch et al., 2020; Loukas et al., 2010; Markowitz, 2017). Additionally, social support in the school environment may compensate for low social support in other domains, such as in the home (Barber & Olsen, 1997; H. Foster & Brooks-Gunn, 2009). Last, school connectedness is a potentially socially modifiable factor, making it an attractive target for interventions (H. Foster & Brooks-Gunn, 2009; McNeely et al., 2002).

There are number of limitations, however, in previous research examining the protective effects of school connectedness. Most studies have focused solely on either externalizing behaviors, such as conduct disorder (Klika et al., 2013; Loukas et al., 2010), or internalizing behaviors (Lensch et al., 2020; Markowitz, 2017). Given the high comorbidity of internalizing

and externalizing disorders in children (Caron & Rutter, 1991; G. T. Smith et al., 2020), surprisingly little research has examined the protective effects of school connectedness against early adversity when predicting internalizing and externalizing *simultaneously* (Hardaway et al., 2012). There is also a lack of work examining the buffering effects of school connectedness when predicting positive function. In addition to addressing these limitations, the present study adds to this area of research by examining school connectedness as a contributor to resilience in the context of a dimensional model of adversity.

The Present Study

In the present study, we examined whether childhood exposure to two dimensions of early adversity (composite across ages 3, 5, 9), violence exposure and social deprivation, predicted latent variables indexing child (Age 9) and adolescent (Age 15) internalizing and externalizing symptoms (4 separate variables) and adolescent positive function in youth from the Fragile Families and Child Wellbeing Study (FFCWS), a longitudinal birth cohort study. We hypothesized that both violence exposure and social deprivation would predict increased internalizing and externalizing symptoms and decreased positive function. Additionally, we tested whether connectedness to school in either childhood or adolescence was protective against early adversity and whether this differed across dimensions of adversity or by the outcome examined (i.e., internalizing symptoms, externalizing symptoms, or positive functioning) using a latent variable moderation approach. We did not have specific hypotheses regarding whether protective effects of school connectedness would be stronger for one dimension of adversity than another; however, given the multidimensional nature of resilience, we did not assume it would be uniformly protective.

Methods

Sample

In the present study, we used data from the FFCWS, a population-based, longitudinal, birth-cohort study of approximately 5000 children born between 1998 and 2000 and their parents across 20 U.S. cities. The FFCWS oversampled for non-marital births at an approximately 3:1 ratio (Reichman et al., 2001). When weighted, the FFCWS is representative of children born at the turn of the century in American cities of 200,000 or more. When not weighted (as here), given the oversample for non-marital births, the sample represents mostly low-income, urban families. FFCWS families were interviewed at the birth of the focal child, and again when the child was 1, 3, 5, 9, and 15 years of age through a combination of in-person interviews and phone surveys.

Analyses in the present study use data from 3246 families primarily from the mother and primary caregiver interviews at ages 3, 5, 9, and 15 and the focal child interviews at ages 9 and 15. Over the 3 waves where we used data from the primary caregiver survey (ages 3, 5, 9), an average of 96% of respondents were mothers. Therefore, for parsimony, we refer to responses from mothers and primary caregivers as maternal reports. Families were excluded if they were missing data from all included variables from the year 3, 5, 9, and 15 waves (N=1167). Full information maximum likelihood (FIML) estimation was used to account for missing data in participants who were not missing all variables but were missing individual items (Kline, 2015; Muthén & Muthén, 1998). See Table 4.1 for sample demographic information.

Previous work has separately examined dimensional models of adversity (Miller et al., 2020) and school connectedness (Kalu et al., 2020) in the FFCWS sample. However, the present study differs from that research in marked ways and is also the first to study them together. Our dimensions of violence exposure and social deprivation and the method for measuring them are

distinct from previous work. Additionally, the work examining the beneficial effects of school connectedness in the FFCWS sample only used data from girls in the wave at age 15 data, rather than the full sample at both ages 9 and 15 as is done in the current study. Previous work in a subsample of the FFCWS data from our lab has related childhood exposure to violence and social deprivation to the brain using functional and diffusion MRI (Goetschius, Hein, McLanahan, et al., 2020; Goetschius, Hein, Mitchell, et al., 2020b; Hein et al., 2020) and to the stress hormone, cortisol (Peckins et al., 2019). This research used the same composite scores but was done in a subsample of the FFCWS (N=237) that participated in a supplemental study that collected, among other things, neuroimaging and hormone data. Examining the protective effects of school connectedness on positive and negative adolescent function within a dimensional framework of adversity in the FFCWS dataset is a novel contribution to the literature.

Measures

Childhood Violence Exposure and Social Deprivation Composite Scores

Violence exposure and social deprivation were assessed using composite scores calculated using data from the Fragile Families and Child Wellbeing study at ages 3, 5, and 9 years. Both constructs included the maternal report of experiences that directly (i.e., child physical and emotional abuse, child physical and emotional neglect) and indirectly (i.e., intimate partner emotional, physical, or sexual violence, intimate partner support, community violence, community support) affect the child. Our approach of including experiences with varying levels of proximity to the child across multiple time points allowed us to comprehensively assess the child's cumulative, dimensional exposure to violence and social deprivation across childhood as has been done in previous research (Hein et al., 2020; McLaughlin & Sheridan, 2016). These composite scores were first utilized in previous work from our group (Hein et al., 2020).

Childhood Exposure to Violence. Included in this composite was the maternal report of child physical and emotional abuse based on items from the Parent-Child Conflict Tactics Scale (Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Five items were used to assess physical abuse including, "hit him/her on the bottom with a hard object" and "shook him/her" and five items were used to assess emotional abuse including whether the parent/caregiver has "sworn or cursed at," or "called him/her dumb or lazy or some other name like that." Each item was rated on a 7-point Likert scale ranging from "never happened" to "more than 20 times." Maternal report of the child's exposure to or victimization of violence in the neighborhood (S. Zhang & Anderson, 2010) was also included in the composite. This was measured using the maternal report of the child witnessing or being the victim of beating, attacks with a weapon, shootings, and killings (witness only) on a 5-point Likert scale ranging from "never" to "more than 10 times." At age 9, the mother was not asked about whether the child had witnessed killings or if they had been the victim of a shooting, so these items were only included for ages 3 and 5 years. Lastly, we included maternal report of intimate partner violence (IPV) (physical-2 items, emotional-3 items, or sexual-1 item) in the home at each wave (Hunt et al., 2017). Each item was rated on a 3-point Likert scale ranging from "never" to "often." Physical IPV items included "he slapped or kicked you" and "he hit you with his fist or a dangerous object." Emotional IPV items included "he tried to isolate you from family and friends," and "he tried to prevent you from going to work and/or school." The sexual IPV was "he tried to make you have sex or do sexual things you didn't want to do." The child's exposure to IPV against the mother was coded as missing for a given wave if the child did not live with their mother at least 50% of the time.

Childhood Exposure to Social Deprivation. Included in this composite was maternal report of child physical and emotional neglect based on items from the CTS-PC (Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Four items from the CTS-PC were used to assed physical neglect including whether the parent was ever "so drunk or high that you had a problem taking care of your child." One item, whether the parent was "ever so caught up in your own problems that you were not able to show or tell your child that you loved him/her," was used to assess emotional neglect. These items from the CTS-PC were reported on the same 7-point Likert scale as the items in the violence exposure composite. Maternal report of social cohesion in the neighborhood was also included in this composite (reverse coded such that higher scores corresponded to lower cohesion) (Donnelly et al., 2016; Morenoff et al., 2001). This included 4 items, such as "this is a close-knit neighborhood," rated on a 5-point Likert scale ranging from "strongly agree" to "strongly disagree." Lastly, we included maternal report of the level of intimate partner support for each wave using six items, such as "how frequently (the current romantic partner) expresses love and affection (for the mother)," that were rated on a 3-point Likert scale ranging from "never" to "often" (Manuel et al., 2012). This was also reverse coded such that higher scores corresponded to less support. Child exposure to the mother's intimate partner support was coded as missing for a given wave if the child did not live with their mother at least 50% of the time.

Composite Score Calculation. To calculate composite scores, the Z scores for each of the childhood experiences (child abuse, exposure to intimate partner violence, community violence, child neglect, lack of romantic partner support, lack of neighborhood social cohesion) were summed for each of the childhood experiences within a dimension (violence exposure and social deprivation) (Song et al., 2013) and then divided by the number of childhood experiences

within a dimension for each participant, thus maximizing the number of participants and the diversity of the sample by minimizing drop out due to missing data at any given wave. In our sample, violence exposure and social deprivation were correlated at $r = 0.400$ with a variance inflation factor (VIF) of 1.191. VIF reflects how much the estimated regression coefficients are increased due to collinear independent variables. Cutoffs are typically between 5–10, therefore, based on the VIF reported here, the multicollinearity of violence exposure and social deprivation was low (Craney & Surles, 2002; Sheather, 2009).

Internalizing Symptoms

Child Internalizing Symptoms. Child (age 9) internalizing symptoms were measured using maternal report on 28 items from the Child Behavior Checklist (CBCL) (Achenbach & Edelbrock, 1983). These items came from three subscales; "anxious/depressed" (i.e., "Child cries a lot"), "withdrawn/depressed" (i.e., Child enjoys very little), and "somatic complaints" (i.e., "Child has nightmares"). These items were rated on a three-point Likert scale ranging from 1- "not true" to 3- "very true or often true." Four items from these subscales were not included due to very low endorsement (less than 20 responses for a category) which resulted in correlations with other items which exceeded +/-0.985 due to one or more zero cells.

Adolescent Internalizing Symptoms. Adolescent internalizing symptoms in the FFCWS were measured at age 15 using teen-report on five items for depression and five items for anxiety. The items for depression were from the Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) and include statements such as, "I feel I cannot shake off the blues, even with help from my family and my friends." Teens responded with their degree of agreement based on the last four weeks on a four-point Likert scale ranging from 1- "strongly agree" to 4- "strongly disagree." The items for anxiety were adapted from the Brief Symptom

Inventory 18 (BSI-18) (Derogatis & Savitz, 2000) and include statements such as , "I have spells of terror or panic." Teens responded on a four-point Likert scale ranging from 1- "strongly agree" to 4- "strongly disagree." Items corresponding to both depression and anxiety were reverse coded such that higher values represented more internalizing symptoms.

Externalizing Symptoms

Child Externalizing Symptoms. Child (age 9) externalizing symptoms were measured using maternal report on 25 items from the Child Behavior Checklist (CBCL) (Achenbach & Edelbrock, 1983). These items came from two subscales; "rule-breaking behavior" (i.e., "Child lacks guilt") and "aggressive behavior" (i.e., "Child argues a lot"). These items were rated on a three-point Likert scale ranging from 1- "not true" to 3- "very true or often true." Ten items from these subscales were not included due to very low endorsement (less than 20 responses for a category) which resulted in correlations with other items which exceeded +/-0.985 due to one or more zero cells.

Adolescent Externalizing Symptoms. Adolescent externalizing symptoms were measured at age 15 using teen-report of delinquency (6 items), impulsivity (6 items), and substance use (5 items). The items for delinquency were adapted from the National Longitudinal Study of Adolescent Health (Add Health - (Harris, 2013) and included items such as "painted graffiti or signs on private property/public spaces." Items were rated on a four-point Likert scale ranging from 1- "never" to 4- "5 or more times." Seven items from the delinquency scale were not included due to very low endorsement (less than 20 responses for a category) which resulted in correlations with other items which exceeded +/-0.985 due to one or more zero cells. The items for impulsivity were adapted from the dysfunctional impulsivity items on Dickman's Impulsivity scale (Dickman, 1990), and included items such as, "I will often say whatever comes

into my head without thinking first." Items were rated on a four-point Likert scale ranging from 1- "strongly agree" to 4- "strongly disagree." Impulsivity items were reverse coded so that higher scores represented increased impulsivity to match the delinquency scale. The items for substance use were binary variables (yes/no) indexing cigarette use, alcohol use (more than 2 drinks without parents), marijuana, illegal drugs other than marijuana, and taking prescription drugs without a prescription.

Adolescent Positive Function

Adolescent positive function was measured at age 15 using teen-report on 20-items adapted from the EPOCH Measure of Adolescent Wellbeing (Kern et al., 2016). These items make up five subscales representing perseverance (i.e., "I finish whatever I begin"), optimism (i.e., "I am optimistic about my future"), connectedness (i.e., "When something good happens to me, I have people who I like to share the good news with"), happiness (i.e., "I feel happy"), and engagement (i.e., "when I do an activity, I enjoy it so much that I lose track of time"). Teens responded with their degree of agreement to the items based on the last 4 weeks on a four-point Likert scale ranging from 1- "strongly agree" to 4- "strongly disagree." These items were recoded so that higher values represented more positive function.

School Connectedness

School connectedness was measured in the FFCWS via self-report at ages 9 and 15 years based on questions developed for the Panel Study on Income Dynamics Child Development Supplement (PSID-CDS) (*The Panel Study of Income Dynamics Child Development Supplement: User Guide for CDS-III*, 2010). The focal child was asked if they "feel close to people at school," "feel like part of school," are "happy to be at school," and "feel safe at school." At age 9, children responded on a five-point Likert scale with the frequency that they felt the above

questions ranging from “0 - not once in the past month” to “4 - every day.” At age 15, teens responded on a five-point Likert scale with the degree of agreement with the same statements ranging from “0 - strongly agree” to “4 - strongly disagree.” The Age 15 school connectedness items were reverse coded so that higher scores represented more connectedness to be consistent with the Age 9 items.

Covariates

Focal child sex at birth, self-reported race/ethnicity at age 15 (dummy coded – African American, Caucasian, Latinx, Other), and average income-to-needs ratio across the study waves were used as covariates in robustness checks. Self-reported race/ethnicity was coded using a set of 3 dummy coded variables to represent the following groups: African American, Caucasian, Hispanic/Latinx, and Other. The average income-to-needs ratio (referred to as poverty ratio in the FFCWS data) is calculated by taking the average of ($\frac{household\ income}{poverty\ threshold}$) across the six study waves. The poverty threshold accounts for household size and composition as well as inflation; however, it does not differ geographically (U.S. Census Bureau, 2020).

Statistical Analysis

Analyses for the present study were done in a combination of R (v4.0.2) and Mplus (v.8.4-Muthén & Muthén, 1998-2017). Data were cleaned in R and were prepared for analysis in Mplus using the MplusAutomation package (v.0.7-3) (Hallquist & Wiley, 2018). The fit of the measurement and structural models were assessed using accepted fit indices: *RMSEA*, *CFI, TLI*, and *SRMR* (Hu & Bentler, 1999). The X^2 value of these model are reported but were not interpreted since X^2 is likely inflated by the large sample size and thus the significant value cannot be taken as an indicator of poor fit (Schermelleh-engel et al., 2003). To account for the multisite design of the FFCWS, all measurement and structural models were clustered by city at

baseline and estimated using Taylor-series linearization using Type = Complex in Mplus. Cluster effects could not be accounted for in the same way in the moderation models because they required a different analysis type that allows for random slopes and models heterogeneity in the residual variance (Muthén & Muthén, 1998).

Measurement Model

Latent variables were created using item level indicators to measure school connectedness at age 9, school connectedness at age 15, internalizing symptoms at 9, externalizing symptoms at 9, internalizing symptoms at age 15, externalizing symptoms at age 15, and positive function at age 15 (a single latent variable though there are subscales), in Mplus. We used the WLSMV estimator because we had categorical as indicators of the latent factors (Kline, 2015). Items were excluded if they did not have a standard YX loading of at least 0.4 (Kline, 2015). Standardized coefficients (β) are effect size estimates (Kline, 2015) and all factor loadings reported in the present study are standardized.

Main Effects Models

We tested a model that estimated the main effects of childhood violence exposure, childhood social deprivation, and child and adolescent school connectedness on our predicted outcomes. It is tenable that internalizing symptoms, externalizing symptoms, or positive function could influence how connected teens feel to school rather than how it specified in our hypothesized model. Therefore, we also ran a reverse effects model where the outcome variables predicted school connectedness at the same time point (e.g., age 9 internalizing symptoms predicting age 9 school connectedness) and compared model fit and path estimates. All path estimates reported in the present study are standard YX estimates. We used the WLSMV

estimator because we had categorical variables included the latent variables (Kline, 2015). Models controlled for the demographic covariates listed above.

Moderation Models

Two moderation models were run. The first tested whether school connectedness at either age 9 or age 15 moderated the association between childhood violence exposure and internalizing and externalizing symptoms (age 9 and 15) or the association between childhood social deprivation and internalizing and externalizing symptoms. The second tested whether school connectedness at either age 9 or 15 moderated the association between childhood violence exposure and adolescent positive function or the association between childhood social deprivation and adolescent positive function. All moderation models were tested in Mplus using a latent variable moderation approach (Maslowsky et al., 2015). In this approach, interaction terms are created from an observed (i.e., social deprivation composite score) and a latent variable (i.e., school connectedness at age 9) using the XWITH option along with ANALYSIS TYPE=RANDOM which allows for random slopes that model heterogeneity in the residual variance (Muthén & Muthén, 1998). We used the MLR estimator, because the WLSMV estimator cannot be used with TYPE=RANDOM, and the Monte Carlo option for numerical integration with 10000 randomly generated integration points (Muthén & Muthén, 1998). The fit of these moderation models was assessed using a Satorra-Bentler scaled chi-square difference test (TRd) using the log likelihood values for the main effects model verses the interaction model (Satorra & Bentler, 2010). This was done because the traditional global fit indices are not produced in Mplus when the analysis TYPE=RANDOM. Models controlled for demographic covariates.

Simple Slopes Analysis. To interpret significant interactions, simple slope and regions of significance analyses were conducted to determine the nature of the interaction and ensure that the interaction was within our observable data using methods outlined by Preacher et al, (2006). This was done in R using factor scores extracted from Mplus. Factor scores were extracted from measurement models containing all of the latent variables in the model (e.g., school connectedness at age 9, school connectedness at age 15, and adolescent positive function). Interactions were plotted using the *interactions* (v1.1.1) toolbox in R (Long, 2019).

Results

Descriptive statistics for and zero-order correlations between childhood exposure to violence and social deprivation as well as the factor scores representing child and adolescent school connectedness, internalizing and externalizing psychopathology, and adolescent positive function are in Table 4.2.

Measurement Model

The final measurement model using CFA to fit the Age 9 School Connectedness, Age 15 School Connectedness, Age 9 Internalizing Symptoms, Age 15 Internalizing Symptoms, Age 9 Externalizing Symptoms, Age 15 Externalizing Symptoms and Age 15 Positive Function items to their respective factors fit the data well (Table 4.3 - *RMSEA*: 0.010, *RMSEA 95% CI* 0.009-0.010, *CFI* = 0.935, *TLI* = 0.933, *SRMR* = 0.064, $X^2(4928) = 6619.97$). During the process of fitting the CFA model, we discovered that three items from the internalizing subscales of CBCL did not load well with the other items with standard factor loadings below the 0.4 threshold. These items were excluded in the final measurement model. Additionally, we discovered that the engagement subscale of the positive function (EPOCH) scale (4 items) did not load well with the rest of the items from the other four subscales with standard factor loadings well below 0.4. Therefore, we

excluded the items from the engagement subscale from further analyses. All factor loadings in the final measurement model had standard YX estimates of greater than 0.4.

Main Effects Models

The main effects model testing the main effects of childhood violence exposure, childhood social deprivation, and child and adolescent school connectedness on our predicted outcomes fit the data well (*RMSEA*=0.009, 95% CI 0.008-0.010, *CFI* = 0.928, *TLI* = 0.926, *SRMR* = 0.065, X^2(5602) =7094.661 - Figure 4.1). In this model, childhood violence exposure predicted greater internalizing symptoms (age 9: β=0.154, *SE*=0.030, p<0.001; age 15: β=0.061, *SE*=0.018, p=0.001), greater externalizing symptoms (age 9: β=0.256, *SE*=0.021, p<0.001; age 15: β=0.148, *SE*=0.012, p<0.001), and lower positive function at age 15 (β=-0.031, *SE*=0.041, p=0.029). Childhood social deprivation also predicted greater internalizing symptoms (age 9: β=0.212, *SE*=0.023, p<0.001; age 15: β=0.079, *SE*=0.025, p=0.002) and externalizing symptoms at age 9 (β=0.128, *SE*=0.019, p<0.001) but not age 15. Social deprivation also predicted lower positive function at age 15 (β=-0.127, *SE*=0.022, p<0.001). School connectedness at age 9 predicted lower age 9 internalizing (β=-0.141, *SE*=0.021, p<0.001) and externalizing (β=-0.157, *SE*=0.029, p<0.001) symptoms, but did not predict age 15 internalizing symptoms, externalizing symptoms, or positive function. School connectedness at age 15 predicted lower age 15 internalizing (β=-0.371, *SE*=0.016, p<0.001) and externalizing (β=-0.289, *SE*=0.020, p<0.001) symptoms as well as greater adolescent positive function (β=0.567, *SE*=0.020, p<0.001).

The reverse effects model where the outcomes at age 15 (internalizing symptoms, externalizing, symptoms, and positive adolescent function) predicted school connectedness at age 15 and the outcomes at age 9 (internalizing and externalizing symptoms) predicted school connectedness at age 9 fit the data worse than our hypothesized model (*RMSEA*=0.017, 95% CI:

0.016-0.017, *CFI* = 0.751, *TLI* = 0.745, *SRMR* = 0.115, X^2(5611) =10745.500) and the standardized path estimates were lower suggesting that our hypothesized model may be a better fit for the data.

As a check, structural models were run separately for age 9 (*RMSEA*=0.010, 95% CI: 0.009-0.011, *CFI* = 0.950, *TLI* = 0.948, *SRMR* = 0.059, X^2(2808) =3712.128) and for age 15 (*RMSEA*=0.021, 95% CI: 0.020-0.022, *CFI* = 0.897, *TLI* = 0.891, *SRMR* = 0.083, X^2(1336) =3301.428) and path estimates were similar. The only difference was that there was a main effect of age 9 school connectedness (β=0.178, *SE*=0.017, p<0.001) when age 15 school connectedness was not in the model.

Moderation Models

A latent variable moderation model which included the interaction between school connectedness at 15 and early adversity (violence exposure and social deprivation) was initially tested for both for sets of outcomes; however, the moderation paths including school connectedness at age 15 were non-significant. In order to retain a more parsimonious model, those paths were removed in the final model.

Child and Adolescent Internalizing and Externalizing Symptoms as Outcome

This latent variable moderation model showed a significant interaction between social deprivation and child school connectedness (age 9) when predicting child externalizing symptoms (β=0.073, *SE*=0.036, p=0.043 - Figure 4.2). This moderation model fit the data better than the main effects model without interactions based on a Satorro-Bentler Scaled Chi-Square Difference Test (*TRd*=18.765, *df*=8, p=0.016) and better than the moderation model with the school connectedness at age 9 and social deprivation interaction path predicting child externalizing symptoms set to 0 (*TRd*=6.515, *df*=1, p=0.011).

A simple slopes analysis revealed that at all conditional levels of age 9 school connectedness that were tested (+1 standard deviation (SD), mean, -1 SD), social deprivation was positively correlated with externalizing symptoms at age 9 (Figure 4.3). However, when school connectedness was high (+1 SD), the slope of this association was steeper (b=0.162, p<0.001) and the intercept was lower (*intercept*=-0.193) than when school connectedness was at mean (b=0.123, p<0.001, *intercept*=-0.001) or low (b=0.0784, p=0.005, *intercept*=0.196) levels. For ease of interpretation of these intervals, all variables were scaled such that the mean was 0 and SD was 1. An evaluation of Johnson-Neyman intervals showed that the interaction was significant until social deprivation was 2.56 SD above the mean and when school connectedness was greater than -1.55 SD below the mean. This suggests that school connectedness at age 9 was protective against social deprivation but that the protective effects diminished when social deprivation was moderately high (+2.56 SD). Additionally, when school connectedness at age 9 was low (-1.55 SD), it was not protective against social deprivation.

Adolescent Positive Function as Outcome

This latent variable moderation model showed a significant interaction between social deprivation and school connectedness at age 9 when predicting adolescent positive function (β=-0.051, *SE*=0.026, p=0.045), even when accounting for the main effects of school connectedness at age 9, school connectedness at age 15, social deprivation, violence exposure, and the interaction between violence exposure and school connectedness at age 9 (Figure 4.4). This moderation model fit the data better than the main effects model based on a Satorro-Bentler Scaled Chi-Square Difference Test (*TRd*=7.088, *df*=2, p=0.029) and better than the moderation model with the school connectedness at age 9 and social deprivation interaction set to 0 (*TRd*=6.603, *df*=1, p=0.010).

A simple slopes analysis revealed that at all conditional levels of age 9 school connectedness that were tested (+1 SD, mean, -1 SD), social deprivation was negatively correlated with positive adolescent function (Figure 4.5). However, when school connectedness was high (+1 SD), the slope of this association was steeper (b=-0.150, p<0.001) and the intercept was higher (*intercept*=0.209) than when school connectedness was at mean (b=-0.112, p<0.001, *intercept*=-0.001) or low (b=-0.075, p=0.002, *intercept*=-0.210) levels. An evaluation of Johnson-Neyman intervals showed that the interaction was significant until social deprivation was 2.83 SD above the mean and when school connectedness was greater than -1.41 SD below the mean. For ease of interpretation of these intervals, all variables were scaled such that the mean was 0 and SD was 1. Similar to the interaction predicting externalizing symptoms at age 9, school connectedness at age 9 was protective against social deprivation when predicting positive adolescent function; however, it became less protective as social deprivation becomes more extreme. Additionally, when school connectedness at age 9 was very low (-1.41 SD), it was not protective against social deprivation.

Discussion

In the present study, we examined whether school connectedness was protective against childhood exposure to violence and social deprivation based on multiple indices of child and adolescent function in a longitudinal sample of close to 4,000 youth from the Fragile Families and Child Wellbeing Study. We found that both child and adolescent school connectedness were correlated with better concurrent outcomes (i.e., higher positive outcomes, lower negative outcomes), even when accounting for the detrimental effects of violence exposure and social deprivation. Additionally, we found that child school connectedness (age 9) specifically interacted with childhood social deprivation, but not violence exposure, to moderate the

association with child externalizing symptoms and adolescent positive function. An analysis of simple slopes for both interactions suggested that child school connectedness was a *protective but reactive* factor against social deprivation, meaning that school connectedness was protective against social deprivation, but that it became less protective as social deprivation became more extreme (Luthar et al., 2000; Proctor, 2006).

Both child and adolescent school connectedness were promotive of contemporaneous outcomes in all youth, regardless of their childhood exposure to violence or social deprivation. Additionally, school connectedness at age 9 was a *protective but reactive* buffer against social deprivation when predicting age 9 externalizing symptoms and even six years later when predicting positive function. *Protective but reactive* effects confer protection against a risk factor, but the buffering effect diminishes with increasing stress (Luthar et al., 2000). This pattern of results suggests that the protective effect of age 9 school connectedness is reduced over time and with increasing stress, which is consistent with previous work showing a decreasing promotive effect size over time for late adolescent school connectedness (Markowitz, 2017). The residual protective effect of age 9 school connectedness on positive function six years later supports previous work positing that social connections and support at school can compensate for other areas of social deprivation (H. Foster & Brooks-Gunn, 2009) and it extends that work by showing that those connections seem to enhance adolescent self-reports of perseverance, optimism, connectedness, and happiness.

Our results are consistent with the literature highlighting the protective effects of school connectedness (Brookmeyer et al., 2006; Hardaway et al., 2012; Kalu et al., 2020; Markowitz, 2017), but expand on it in four key ways. First, we utilized two multi-context measures of adversity that prospectively indexed a child's exposure to both violence and social deprivation at

varying levels of proximity to the child (i.e., self, home, neighborhood) and at multiple time points (ages 3, 5, 9 years). Second, we included measures of school connectedness and outcomes at two time points (age 9 and 15 years). This allowed us to gain insight into the longitudinal effects of school connectedness because we were able to assess how school connectedness can be correlated with better outcomes at the same timepoint, but also in the future, while still controlling for the effects of contemporaneous school connectedness. Third, we examined the protective effects of school connectedness at an earlier age (9 years) than previous research studies. Last, we operationalized resilience in terms of enhanced positive function in addition to reduced negative function (i.e., internalizing and externalizing symptoms) which supports examining protective processes in terms of multiple domains of function (National Research Council (US) & Institute of Medicine (US) Board on Children, Youth, and Families, 2001).

Results from the present study underscore the important role that the school environment can play for youth who have been exposed to adversity in other areas of their lives. They also underscore the enduring effect of positive social connections. School-aged children and adolescents spend a majority of their day at school (Roeser et al., 2000), and thus, it is critical to help them develop strong social connections at school, especially those who are exposed to violence and social deprivation in their homes and neighborhoods. Previous research has shown that school connectedness is improved through social support and encouragement in school involvement from teachers, school counselors, peers, and parents, as well as through involvement in school-sponsored extracurricular activities (Daly et al., 2010). However, more research is needed to determine effective interventions to promote school connectedness because there are systematic disparities in school connectedness and climate based on race-ethnicity, gender, and socioeconomic status (Liu et al., 2020; Voight et al., 2015). Promoting school

connectedness may be particularly salient for African American boys who experience disparate treatment at school, including disproportionately high levels of suspensions and expulsions (Thomas & Stevenson, 2009) and systematically lower expectations for academic attainment (Wood et al., 2007). Additionally, Latinx youth are the largest ethnic minority in the U.S.; however, the rate of degree attainment in Latinx high school students is systematically lower and has been linked to academic discrimination (Alfaro et al., 2009). These troubling trends highlight the importance of identifying processes that promote factors, such as school connectedness, that improve academic and socio-emotional outcomes in at-risk youth (Liu et al., 2020).

Results from the present study support modeling adversity in terms of their core underlying dimensions which relate to development in both distinct and overlapping ways (McLaughlin & Sheridan, 2016). We found that both violence exposure and social deprivation predicted childhood symptoms of psychopathology. However, violence exposure distinctly predicted adolescent externalizing symptoms and social deprivation, more strongly predicted reduced adolescent positive function. These findings are largely consistent with previous research modeling the effects of adversity using the DMAP framework, including work done with the FFCWS sample, which found that threat (similar to our violence exposure) was associated with both internalizing and externalizing symptoms (Miller et al., 2018, 2020). We are unaware of any prior research examining links between dimensions of adversity and positive function. The predictive effect of social deprivation suggests that there may be a specific mechanism linking the lack of expected social input in the home and neighborhood environment with teen self-reports of low perseverance, optimism, connectedness, and happiness.

Findings related to social deprivation in the present study are complementary but not identical to those found in previous research using the DMAP framework. This may be due to

varying definitions of deprivation. Deprivation constructs have largely encompassed two areas: cognitive, which indexes information about a lack of cognitive enrichment of the child's environment (e.g., age-appropriate toys, books, measures of SES - Lambert et al., 2017; Miller et al., 2018); and social, which indexes information about a lack of expected social input in the child's environment as was done in the present study. Previous work has explicitly or implicitly operationalized deprivation as either cognitive deprivation, social deprivation, or both. Behavioral correlates of these differing definitions suggest that deprivation may be two dimensions rather than one. Deprivation, when indexed by both cognitive and social deprivation, has been linked to increased internalizing and externalizing symptoms (Miller et al., 2020). However, when deprivation is indexed strictly as cognitive deprivation, it is associated selectively with increased externalizing symptoms (Miller et al., 2018), suggesting that perhaps cognitive deprivation is related to a higher risk for externalizing psychopathology and, consistent with our findings, social deprivation may be a greater risk factor for internalizing psychopathology. Future work using dimensional models of adversity should more directly test whether social and cognitive deprivation form two separate dimensions in addition to violence exposure/threat. Additionally, future work comparing the neural correlates of differing definitions of deprivation may provide insight into if there are diverging neural mechanisms underlying cognitive and social deprivation.

We found that social connections at school can promote resilience broadly, but that protective effects also manifest in more specific ways through interaction with social deprivation. This highlights the importance of studying protective factors within a dimensional model of adversity and suggests that school connectedness may work through multiple mechanisms to influence function. A benefit of employing dimensional models of adversity is that it is possible

to test specific hypotheses regarding how variation in core aspects of childhood adversity relate to outcomes with the idea that particular dimensions are likely to impact development through specific neurobiological mechanisms (McLaughlin et al., 2014). By examining protective processes within a dimensional model of adversity, we were able to identify that social connections at school protected against, and may make up for, a lack of expected social input elsewhere (H. Foster & Brooks-Gunn, 2009), while also promoting outcomes overall. Future research should test whether the compensatory effects of school connectedness operate through distinct neurobiological mechanisms compared to the general promotive effects. Additionally, future research should examine if school connectedness is protective against other dimensions of adversity, such as cognitive deprivation or environmental instability (Ellis et al., 2009; Miller et al., 2018), which would provide additional insight into the mechanisms through which school connectedness promotes resilience.

The present study had limitations. First, there was a six-year gap between FFCWS data collection waves at ages 9 and 15. We would be able to better understand these protective associations and how the strength of school connectedness at age 9 changes over time if we had information about the children and their families during this gap. Second, we did not have a comparable index of positive function at age 9 to test whether school connectedness at age 9 is also correlated with positive function. Third, the violence exposure and social deprivation composites were derived from maternal reports. Data regarding childhood adversity from sources outside the home, such as social workers and teachers, would make our composites more comprehensive. Last, the environment of adversity is complex; thus, there are likely unmeasured variables that may influence these associations or contribute to cascades of risk.

Conclusion

The present findings suggest that school connectedness is a robust protective factor against exposure to early adversity in youth from the FFCWS in terms of both positive and negative metrics of child and adolescent function. Social connections at school may compensate for a lack of expected social support and input in the home and neighborhood to help reduce externalizing symptoms and promote positive adaptive function. Consistent with previous research, our results highlight the important role that the school environment can play for youth who have been exposed to adversity in other areas of their lives. Additionally, the interactive effect of school connectedness with social deprivation, but not violence exposure, supports modeling risk and resilience processes using dimensional frameworks to better identify specific groups of youth that may benefit from interventions that boost social connectedness at school.

Table 4.1: Demographic characteristics

Demographic characteristics of the sample.

	Overall (N=3246)
Child's Sex at Birth	
Female	1585 (48.8%)
Male	1661 (51.2%)
Adolescent Race-Ethnicity – Self-report at Age 15	
African American	1592 (49.0%)
Caucasian	587 (18.1%)
Latinx	808 (24.9%)
Other	259 (8.0%)
Average Income-to-Needs Ratio[1] Across All Study Waves	
Mean (SD)	2.11 (2.10)
Median [Min, Max]	1.46 [0.120, 21.2]
Maternal Marital Status at Child's Birth	
Married	785 (24.2%)
Not Married	2443 (75.3%)
Missing	18 (0.6%)
Maternal Education at Child's Birth	
Less than high school	1025 (31.6%)
High school or equivalent	1030 (31.7%)
Some college or technical school	821 (25.3%)
College or graduate school	365 (11.2%)
Missing	5 (0.2%)

[1] Income-to-needs ratio variable is referred to as the poverty ratio in the FFCWS dataset.

Table 4.2: Descriptive statistics and zero-order correlations

Means, standard deviations, and zero-order correlations with confidence intervals of the variables of the interest.

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. Violence Exposure	0.01	0.53								
2. Social Deprivation	0.00	0.53	.40** [.37, .43]							
3. School Connectedness (Age 9)	-0.05	0.77	-.05** [-.09, -.02]	-.06** [-.09, -.02]						
4. School Connectedness (Age 15)	-0.04	0.78	-.14** [-.17, -.10]	-.13** [-.16, -.10]	.16** [.12, .19]					
5. Internalizing Symptoms (Age 9)	0.08	0.82	.20** [.16, .23]	.26** [.22, .29]	-.12** [-.15, -.08]	-.07** [-.11, -.04]				
6. Externalizing Symptoms (Age 9)	0.05	0.88	.34** [.30, .37]	.24** [.21, .28]	-.15** [-.19, -.11]	-.12** [-.16, -.08]	.60** [.58, .62]			
7. Internalizing Symptoms (Age 15)	0.03	0.87	.08** [.05, .12]	.12** [.08, .15]	-.09** [-.13, -.05]	-.31** [-.34, -.28]	.14** [.11, .18]	.13** [.09, .16]		
8. Externalizing Symptoms (Age 15)	0.04	0.91	.19** [.16, .23]	.11** [.08, .15]	-.08** [-.12, -.05]	-.26** [-.29, -.23]	.08** [.05, .12]	.24** [.21, .28]	.42** [.39, .45]	
9. Positive Function (Age 15)	-0.02	0.89	-.04* [-.08, -.01]	-.11** [-.14, -.08]	.13** [.09, .16]	.42** [.39, .45]	-.12** [-.16, -.08]	-.09** [-.12, -.05]	-.50** [-.53, -.48]	-.27** [-.30, -.24]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). ** indicates $p < .01$.

Table 4.3: Measurement model factor loadings

Standard factor loading values for the latent variable measurement model. All factor loadings were significant at p<0.001. Items were excluded if loading <0.4 or if very low endorsement (less than 20 responses per category). Model was clustered by city at birth.

Latent Variable	Item	Standard YX Factor Loading
School Connectedness Age 9	K5E1A: Felt part of school	0.684
	K5E1B: Felt close to people at school	0.586
	K5E1C: Happy to be at school	0.753
	K5E1D: Felt safe at school	0.766
School Connectedness Age 15	K6B1A[1]: Feel close to people at school	0.663
	K6B1B[1]: Feel part of school	0.772
	K6B1C[1]: Happy to be at school	0.772
	K6B1D[1]: Feel safe at school	0.637
Internalizing Symptoms Age 9	P5Q3M: Cries a lot	0.644
	P5Q3AB: Fears certain animals/situations/places	0.463
	P5Q3AD: Fears might do something bad	0.628
	P5Q3AF: Feels/complains no one loves him/her	0.745
	P5Q3AH: Feels worthless/inferior	0.820
	P5Q3AR: Nervous movements or twitches	0.643
	P5Q3AV: Too fearful or anxious	0.689
	P5Q3AX: Feels too guilty	0.753
	P5Q3BQ: Self-conscious or easily embarrassed	0.570
	P5Q3CK: Talks about killing self	0.845
	P5Q3DB: Worries	0.533
	P5Q3E: Enjoys verry little	0.475
	P5Q3AO: Would rather be alone than with others	0.593
	P5Q3BK: Refuses to talk	0.641
	P5Q3BO: Secretive, keeps things to self	0.639
	P5Q3CU: Underactive, slow moving, lacks energy	0.651
	P5Q3DA: Withdrawn, doesn't get involved with others	0.713
	P5Q3AS: Has nightmares	0.576
	P5Q3AU: Constipated, doesn't have bowel movements	0.524
	P5Q3AZ: Overtired without good reason	0.754
	P5Q3BB1: Aches or pains without medical cause	0.649
	P5Q3BB2: Headaches without medical cause	0.506
	P5Q3BB5: Rashes/skin problems without medical cause	0.436
	P5Q3BB6: Stomachaches without medical cause	0.610
	P5Q3BB7: Vomiting without medical cause	0.618
Externalizing Symptoms Age 9	P5Q3X: Doesn't feel guilty after misbehaving	0.618
	P5Q3AA: Breaks rules	0.744
	P5Q3AL: Hangs around with others who get in trouble	0.642
	P5Q3AP: Lies or cheats	0.712
	P5Q3BI: Prefers being with older kids	0.421
	P5Q3BZ: Steals at home	0.749
	P5Q3CJ: Swears or uses obscene language	0.768
	P5Q3C: Argues a lot	0.611
	P5Q3O: Cruel, bullies, shows meanness to others	0.774
	P5Q3R: Demands a lot of attention	0.644
	P5Q3S: Destroys own things	0.774
	P5Q3T: Destroys things belonging to others	0.781
	P5Q3U: Disobedient at home	0.726
	P5Q3V: Disobedient at school	0.710
	P5Q3AJ: Gets in many fights	0.765

	P5Q3BC: Physically attacks people	0.832
	P5Q3BN: Screams a lot	0.730
	P5Q3CF: Stubborn, sullen, irritable	0.745
	P5Q3CG: Sudden changes in mood/feelings	0.762
	P5Q3CH: Sulks a lot	0.718
	P5Q3CI: Is suspicious	0.692
	P5Q3CN: Teases a lot	0.678
	P5Q3CO: Temper tantrums or hot temper	0.772
	P5Q3CQ: Threatens people	0.860
	P5Q3CW: Unusually loud	0.655
Internalizing Symptoms Age 15	K6D2AG[1]: Nervous or shaky inside	0.773
	K6D2AI[1]: Feel fearful	0.587
	K6D2D[1]: Spells of terror or panic	0.643
	K6D2J[1]: Feel tense or keyed up	0.563
	K6D2T[1]: Suddenly scared for no reason	0.685
	K6D2AC[1]: Feel depressed	0.878
	K6D2AK[1]: Feel so restless I can't sit still	0.543
	K6D2C[1]: Cannot shake off the blues even with help	0.616
	K6D2N[1]: Feel sad	0.820
	K6D2X[1]: Feel life is not worth living	0.768
Externalizing Symptoms Age 15	K6D2A[1]: Don't spend enough time thinking before act	0.508
	K6D2P[1]: Say/do things without considering consequences	0.675
	K6D2R[1]: Plans don't work because haven't gone over	0.550
	K6D2Z[1]: Make up mind without taking time to consider	0.551
	K6D2AB[1]: Say whatever comes into mind	0.572
	K6D2AJ[1]: Get into trouble because don't think before act	0.753
	K6D61C: Taken something from store without paying	0.902
	K6D61D: Gotten into a serious physical fight	0.644
	K6D61E: Hurt someone badly enough for medical care	0.628
	K6D61K: Stolen something worth less than $50	0.861
	K6D61L: Taken part in group fight	0.583
	K6D61M: Were loud/rowdy/unruly in public place	0.472
	K6D40[1]: Smoked entire cigarette	0.656
	K6D48[1]: Drank alcohol more than twice without parents	0.554
	K6F63[1]: Ever tried marijuana	0.597
	K6F68[1]: Ever tried illegal drugs other than marijuana	0.625
	K6F74[1]: Ever used prescription drugs (not prescribed)	0.681
Positive Function Age 15	K6D2B[1]: Love life	0.755
	K6D2F[1]: Am a cheerful person	0.672
	K6D2G[1]: Have friends that I really care about	0.444
	K6D2I[1]: Keep at my schoolwork until I am done	0.497
	K6D2K[1]: Make plans and stick to them	0.556
	K6D2L[1]: People in my life who really care about me	0.724
	K6D2M[1]: Finish whatever I begin	0.564
	K6D2O[1]: Think good things are going to happen to me	0.568
	K6D2S[1]: Feel happy	0.866
	K6D2V[1]: Am a hard worker	0.592
	K6D2W[1]: Believe that things will work out	0.643
	K6D2Y[1]: Have someone who will be there if I have problem	0.659
	K6D2AA[1]: Have a lot of fun	0.721
	K6D2AE[1]: In uncertain times I expect the best	0.467
	K6D2AF[1]: Have person to share good news with	0.615
	K6D2AH[1]: Optimistic about my future	0.484

1 Item was reverse coded

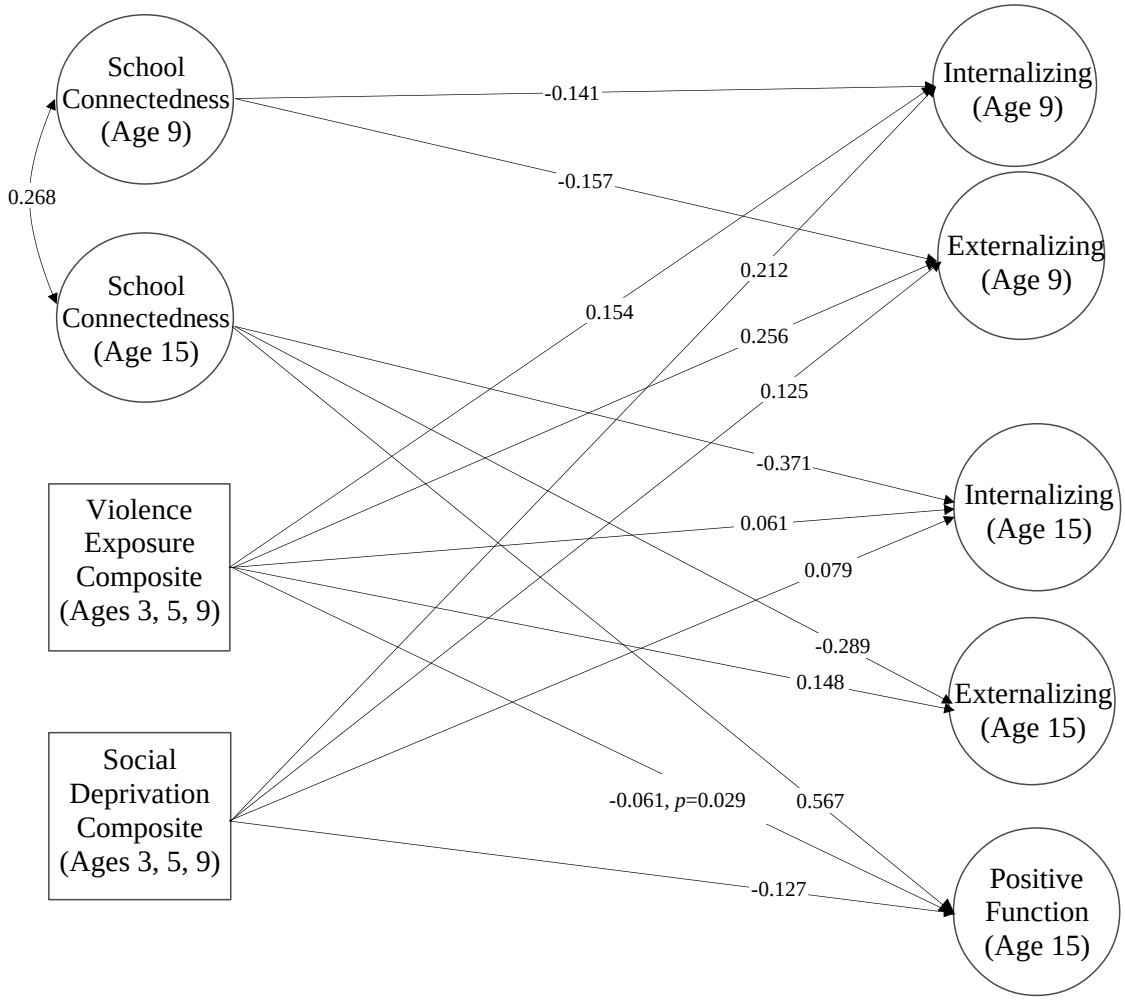


Figure 4.1: Diagram of the main effects model including school connectedness

Model controlled for average income-to-needs ratio, race-ethnicity, and sex and was clustered by city at birth.

Path estimates shown are StandardYX estimates. To make this figure more readable, only paths significant at $p<0.05$ are shown, but all were modeled. P-values of all paths are $p<0.01$ except where reported otherwise. Correlations between all outcome latent variables are not shown to simplify the figure but are modeled and are all significant at $p<0.01$.

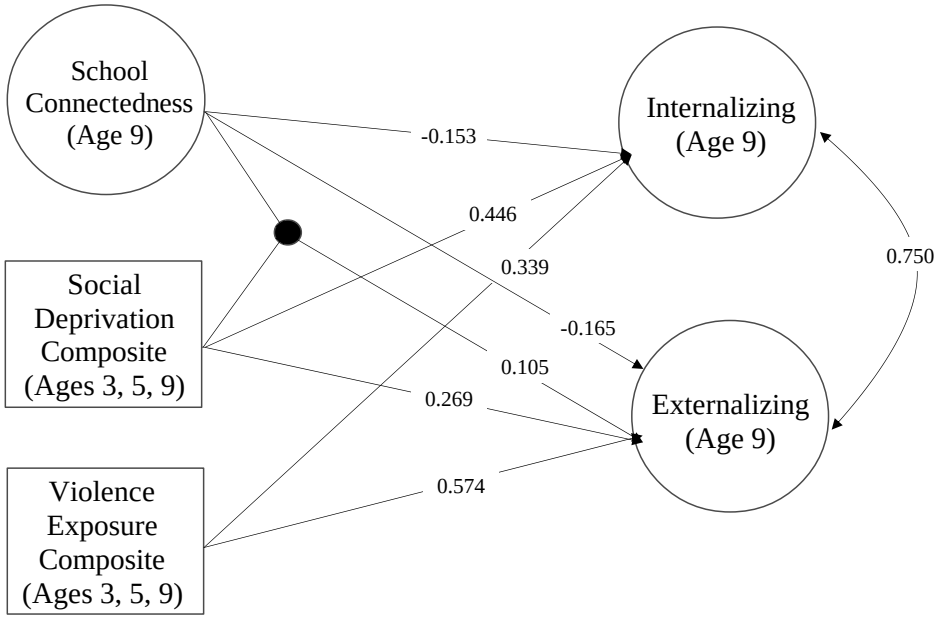


Figure 4.2: Diagram of latent moderation model predicting psychopathology

Diagram of the latent variable moderation model showing that school connectedness at age 9 moderates the association between social deprivation (ages 3, 5, 9) and externalizing symptoms (ages 9). Model controlled for average income-to-needs ratio, race-ethnicity, and sex.

Note: Path estimates shown are StandardYX estimates. To make this figure more readable, only paths significant at $p<0.05$ are shown. All paths, including all 4 interaction paths, are retained in the model even though they are not shown. Including age 15 school connectedness and symptoms of psychopathology does not change the results of this model.

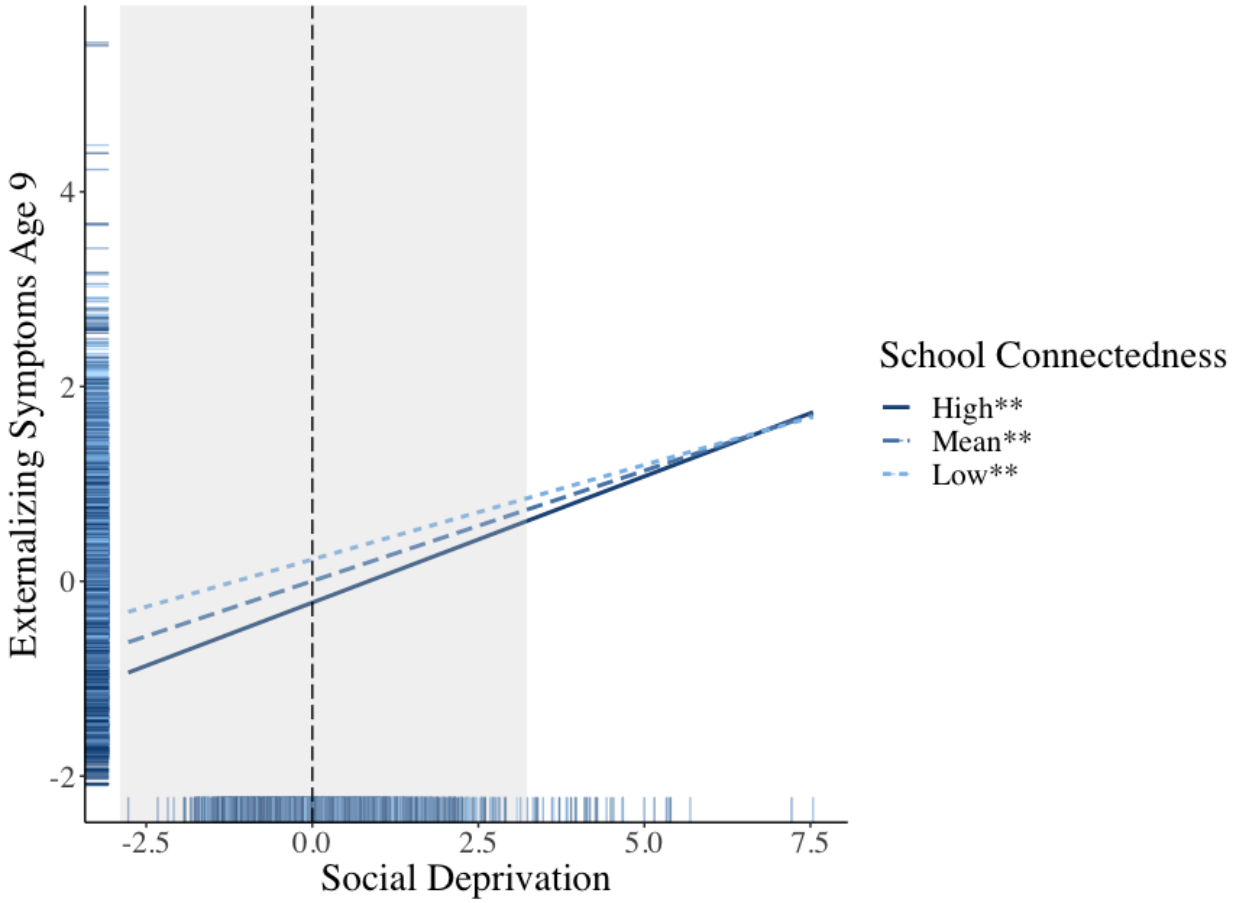


Figure 4.3: Simple slopes plot for SCxSD interaction predicting externalizing symptoms

Plot illustrating the interaction between childhood social deprivation (ages 3, 5, 9) and school connectedness at age 9 in predicting childhood externalizing symptoms (age 9). For ease of interpretation, all variables have been centered and z-scored so that the mean is 0 and the standard deviation (SD) is 1. The dashed line represents mean levels of social deprivation. School connectedness has been plotted at mean and +/- 1 SD. An evaluation of Johnson-Neyman intervals shows that, in this sample, the interaction was significant until social deprivation was very high (+3.22 SD) and when school connectedness was greater than -1.55 SD. This suggests that school connectedness at age 9 also had a *protective but reactive* association with social deprivation when predicting externalizing symptoms at age 9, meaning that school connectedness was protective against social deprivation but that the protective effects diminished when social deprivation was extreme. Additionally, when school connectedness at age 9 was low (-1.55 SD), it was not protective against social deprivation. The range of school connectedness at age 9 in this sample was [-3.31, 1.59] and the range of social deprivation values was [-2.77, 7.54].

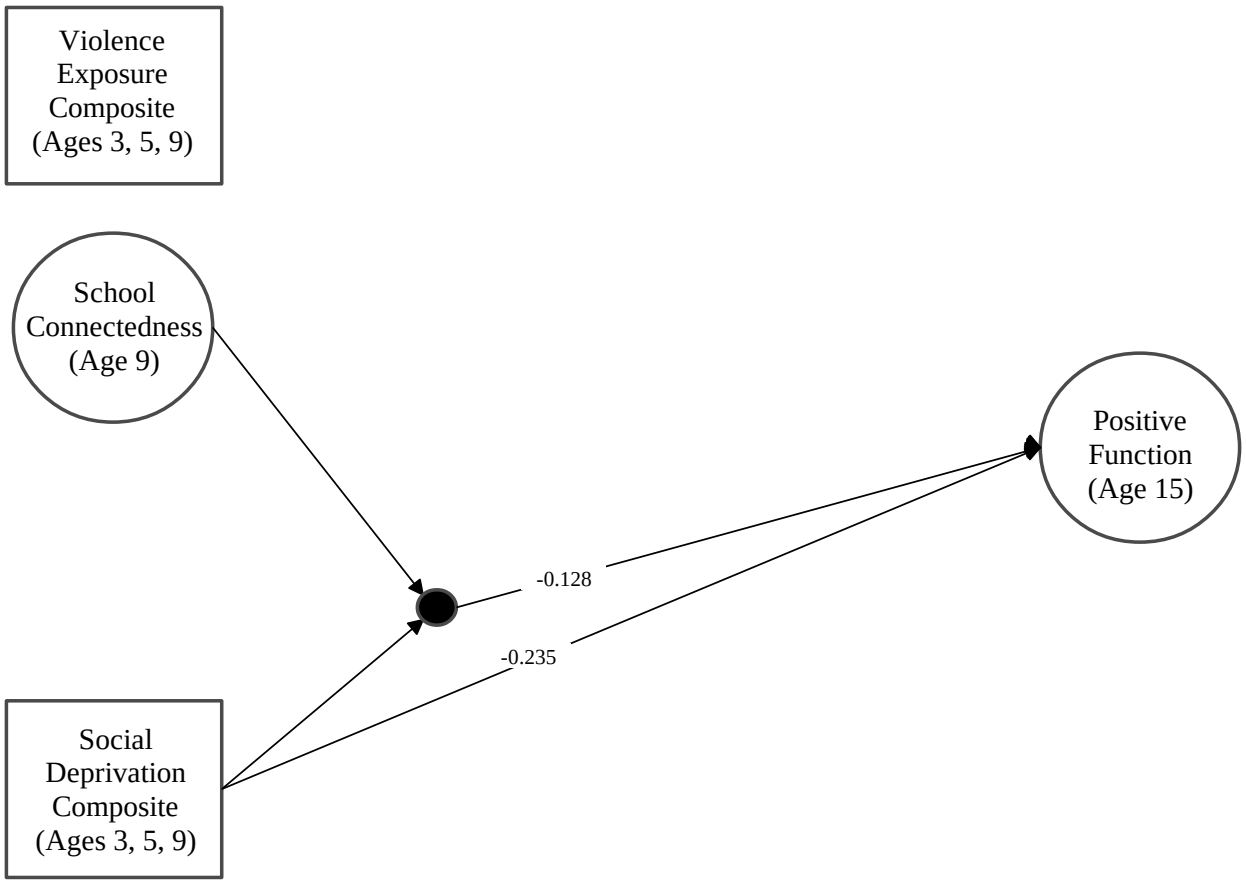


Figure 4.4: Diagram of latent moderation model predicting positive function

Diagram of the latent variable moderation model showing that school connectedness at age 9 moderates the association between social deprivation (ages 3, 5, 9) and positive adolescent function (age 15). Model controlled for average income-to-needs ratio, race-ethnicity, and sex.

Note: Path estimates shown are StandardYX estimates. To make this figure more readable, only paths significant at $p<0.05$ are shown. All paths, including all both interaction paths, are retained in the model even though they are not shown. Including age 15 school connectedness does not change the results of this model.

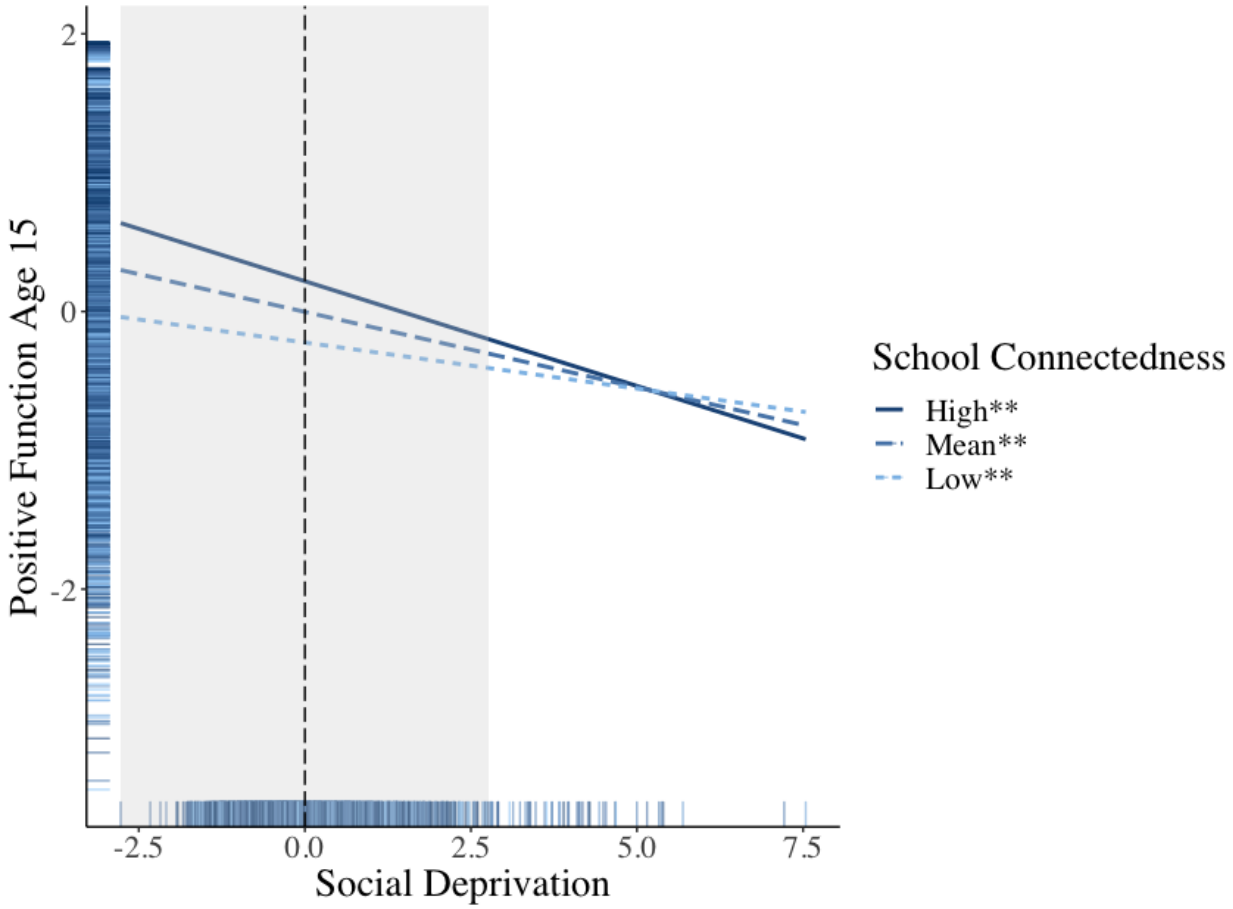


Figure 4.5: Simple slopes plot for SCxSD interaction predicting positive function

Plot illustrating the interaction between childhood social deprivation (ages 3, 5, 9) and school connectedness at age 9 in predicting adolescent positive function (age 15). For ease of interpretation, all variables have been centered and z-scored so that the mean is 0 and the standard deviation (SD) is 1. The dashed line represents mean levels of social deprivation. School connectedness has been plotted at mean and +/- 1 SD. An evaluation of Johnson-Neyman intervals shows that, in this sample, the interaction was significant until social deprivation was very high (+2.77) and when school connectedness was greater than -1.27. This suggests that school connectedness at age 9 had a *protective but reactive* association with social deprivation when predicting positive adolescent function, meaning that school connectedness was protective against social deprivation but that the protective effects diminished when social deprivation was extreme. Additionally, when school connectedness at age 9 was low (-1.27 SD), it was not protective against social deprivation. The range of school connectedness at age 9 in this sample was [-3.26, 1.46] and the range of social deprivation values was [-2.77, 7.54].

Chapter 5: General Discussion

Early adversity is a potent and unfortunately common public health concern that increases the risk of negative physical and mental health outcomes during childhood, and this risk persists throughout the lifespan (Green et al., 2010). Research has shown that experiences of adversity can be distilled down to core underlying dimensions that have at least partially distinct effects on neural and behavioral development (McLaughlin et al., 2014; Miller et al., 2018). This dissertation examined how two such dimensions, violence exposure and social deprivation, related to alterations in neural architecture and function, as well as behavioral outcomes. Importantly, many children exposed to early adversity do not experience negative outcomes (Masten, 2001). However, there is a lack of work empirically testing protective processes within this dimensional framework. Thus, a second objective of this dissertation was to examine how protective factors buffered against adversity and whether this differed across dimensions of adversity or by the outcome examined. This research has implications for understanding how dimensions of adversity affect the brain and behavior during development and what factors can be protective, which can inform future neuroscience-informed policy interventions.

Summary

Study 1. Dimensions of adversity may differentially shape emotion-based neural circuitry, such as the white matter linking the amygdala with regions of the PFC (Etkin et al., 2015; McLaughlin et al., 2014; Swartz et al., 2014). Using a preregistered analysis plan, this chapter showed that childhood violence exposure and social deprivation interacted to predict the probability of adolescent right hemisphere amygdala–OFC white matter connectivity. High

violence exposure with high social deprivation related to less amygdala–OFC white matter connectivity. Violence exposure was not associated with white matter connectivity when social deprivation was at mean or low levels (i.e., relatively socially supportive contexts). Therefore, social deprivation may exacerbate the effects of childhood violence exposure on the development of white matter connections involved in emotion processing and regulation. Conversely, social support may buffer against them.

Study 2. Dimensional exposure to early adversity may also differentially shape functional networks in the brain, such as the salience and default mode networks (Marusak, Etkin, et al., 2015; McLaughlin et al., 2019). Observed effects of adversity on functional networks are likely to be person specific because there is considerable variability in neural responses to environmental stress (Marder & Goaillard, 2006). Chapter 3 used a person-specific approach to modeling resting-state functional networks (GIMME) and a pre-registered analysis plan to show that childhood violence exposure, but not social deprivation, was associated with reduced adolescent resting-state density of the salience and default mode networks in the SAND sample. A data-driven algorithm, blinded to childhood adversity, identified youth with heightened violence exposure based on resting-state connectivity patterns. Childhood violence exposure appears to be associated with adolescent functional connectivity heterogeneity, which may reflect person-specific neural plasticity and should be considered when attempting to understand the impacts of early adversity on the brain.

Study 3. School connectedness, a construct indexing supportive school relationships, has been posited to promote resilience (Barber & Olsen, 1997; Hardaway et al., 2012). Consistent with prominent calls in the field (Masten & Cicchetti, 2016), this study was precise in its measure of two dimensions of adversity which index multiple levels of environmental exposure

and it examines resilience in terms of both the absence of negative outcomes and the presence of positive function. Results from this chapter showed that child and adolescent school connectedness were promotive of concurrent outcomes, even when accounting for the detrimental effects of violence exposure and social deprivation. Additionally, child school connectedness had a *protective but reactive* association (Luthar et al., 2000) with social deprivation, but not violence exposure, when predicting child externalizing symptoms and adolescent positive function. These findings highlight the important role that the school environment can play for youth who have been exposed to adversity in other areas of their lives. Additionally, the interactive effect of school connectedness with social deprivation, but not violence exposure, supports modeling risk and resilience processes using dimensional frameworks to better identify specific groups of youth that may benefit from interventions that boost social connectedness at school in future research.

Considerations

The research discussed in this dissertation provides evidence of dimensional effects of risk and resilience on neural and behavioral indices of socio-emotional function, but also highlights considerations for future research.

Timing Effects. All three studies in this dissertation examined the cumulative effects of childhood exposure to violence and social deprivation on developmental outcomes. However, future work should determine how the timing of exposure impacts its effect on development. Evidence for experience-driven plasticity suggests that plasticity driven by environmental experience is influenced by the timing, quality, and intensity of those experiences (Gottlieb, 2007; Kolb & Gibb, 2014; McLaughlin et al., in press). Indeed, previous work has found evidence for differential effects of neighborhood adversity on adolescent and early adult

corticolimbic function (Gard et al., 2021). This research found specific effects of neighborhood disadvantage in early childhood and adolescence on amygdala reactivity and PFC reactivity, respectively, suggesting that there may be sensitive periods or periods of heightened neuroplasticity that differ across the brain and lead to more specific effects (Hyde et al., 2020).

More work is needed to determine whether there are specific developing timing effects of violence exposure and social deprivation on the neural and behavioral correlates of socioemotional function measured in this discussion. Progress towards that end can be made with the existing SAND data. However, given that the first timepoint of neuroimaging data collected in this sample was in adolescence (discussed in more detail below) and that there is a gap in information about the child's environmental exposures from approximately ages 9 to 15 years, conclusions about timing effects may still be limited. Data from the longitudinal Adolescent Behavioral and Cognitive Development® (ABCD) study (Casey et al., 2018), may be able to address how exposure to adversity shapes neural development in adolescence since there would baseline information about neural structure and function from childhood (Age 9-10). ABCD data would still be limited; however, in drawing conclusions about neural and behavioral correlates of early childhood adversity since the first study wave does not occur until preadolescence and thus all reports of early adversity would be retrospective. Additionally, there are qualitative differences between the samples in the SAND/FFCWS and ABCD studies that may hinder direct comparisons (sample considerations discussed in more detail below). Future studies should be designed to prospectively assess how developmental timing impacts the effects of adversity using dimensional frameworks in samples with adequate representation of marginalized populations (i.e., low-income families or families of color) who likely experience more adversity (Falk et al., 2013; Hyde et al., 2020).

Causal Effects. Studies 1 and 2 found evidence of longitudinal associations between dimensions of adversity and neural connectivity; however, given that there is only a single timepoint of imaging data in the SAND study, it is not possible to determine causal effects of the environment on the brain or vice versa. Additionally, the brain is plastic, and measures of neural structure and function are not static, particularly during childhood, adolescence, and early adulthood when there are times of explosive neural development (Giedd et al., 1999; Richmond et al., 2016). Therefore, we do not know how the patterns of associations observed in studies 1 and 2 differed earlier in development or how they will continue to change as the teens transition to early adulthood and beyond. Of further interest would be change over time in these patterns of association and how longitudinal trajectories of neural structure and function are shaped by adversity and how that corresponds to socioemotional function (Hyde, 2015; Wiggins & Monk, 2013).

Links between Brain and Behavior. Surprisingly absent from studies 1 and 2 were links between neural correlates of adversity and behavioral outcomes, specifically psychopathology. This could be for a number of reasons which should be the focus of additional research. First, the adversity-related differences in white matter and resting-state function connectivity could be reflective of adaptation and could promote resilience (Lipina & Evers, 2017; Varnum & Kitayama, 2017). Evidence for this potential explanation comes from study 1 where the effects of violence exposure were conditional upon social deprivation. Only in teens with high violence exposure *and* high social deprivation, a relatively small portion of the sample, was there an association with white matter connectivity suggesting that low social deprivation, or relative social support, could be protective. Results from study 2 showing that violence exposure was related to more individual heterogeneity in resting-state functional connectivity also provide

evidence of potential neural adaptation to adversity. This individual heterogeneity may reflect a tuning of neural circuits to the environment that promote stability of network function in the context of violence and threat in the environment (Marder & Goaillard, 2006; Varnum & Kitayama, 2017). Additionally, although we did not examine neural correlates in study 3, the theory that the adversity-related differences in brain structure and function may be adaptive could be tested in future research by determining whether they relate to the measure of positive function and wellbeing used in study 3 (EPOCH - Kern et al., 2016) An important consideration related to resilience and adaptation as an explanation; however, is that protective processes can differ by risk process and by domain of function (National Research Council (US) & Institute of Medicine (US) Board on Children, Youth, and Families, 2001). Therefore, it is also tenable that, although the adversity-related neural correlates we observed did not relate to internalizing psychopathology, they may relate to differences in other domains of function.

A second possible explanation for the lack of association between the observed neural correlates of adversity and psychopathology is the way in which psychopathology was measured in this dissertation. Similar to adversity, psychopathology can be considered to be dimensional, rather than categorical, with two dimensions being internalizing and externalizing psychopathology (Hyde, 2015). Hypotheses for studies 1 and 2 focused only on how neural correlates of adversity were associated with internalizing psychopathology (i.e., depression and anxiety). However, given the high comorbidity of internalizing and externalizing psychopathology in youth (Caron & Rutter, 1991; G. T. Smith et al., 2020; Wiggins et al., 2015), perhaps study 1 and study 2 should have tested for associations with both dimensions of psychopathology simultaneously as was done in Study 3. Alternatively, perhaps a better way to measure broad maladaptive function associated with psychopathology would be to use “p-

factor," which is hypothesized to be an overall latent risk for distress and overall symptomatology across both internalizing and externalizing psychopathology (Hyde, 2015; G. T. Smith et al., 2020). It is tenable that the adversity-related neural correlates would be related to higher order associations indexed by a transdiagnostic "p-factor" (Hyde, 2015). An important direction for future research using dimensional models of adversity would be to test whether dimensions of adversity better predict specific dimensions of psychopathology or if exposure to adversity more generally increases risk for psychopathology using "p-factor."

Similarly, a third potential explanation for why we did not observe strong links between the brain and behavior in this dissertation may be that the dimensional framework generally, or the dimensions that we used specifically, do not correspond to neural correlates that relate to behavior. Critics of dimensional models of adversity posit that the distinctions made between dimensions are social in nature and thus are not likely to have distinct, meaningful effects on the brain and subsequent psychopathology (K. E. Smith & Pollak, 2020). Although this is a possibility, evidence does exist linking neural correlates within dimensional models of adversity to behavior (e.g., Sheridan et al., 2017). Additionally, systematic reviews and meta-analyses have found evidence for the specific effects of dimensional exposure to threat or deprivation on biological metrics (e.g., brain structure and function, cellular aging) which have corresponded to differences in behavior, including indices of psychopathology (Colich et al., 2020; McLaughlin et al., 2019). With that in mind, it is tenable that either the dimensions or neural correlates we studied were not meaningfully linked with behavior, but that other dimensions or other indices of neural structure or function would be.

A last and less optimistic alternative would be that the associations between neural correlates of adversity and psychopathology will develop later as this sample transitions to early

adulthood. Life course theory suggests that key transitions, such as the transition to adulthood, are times of stress and individual life trajectories can be shaped by supportive relationships and by social capital that provides resources in the transition to post-secondary education or the labor force (Elder & Shanahan, 2006; McLoyd et al., 2015). Social resources are especially important for socioeconomically disadvantaged or marginalized youth for whom they may be limited by systematic mechanisms of inequality (McLoyd et al., 2015). Interestingly, rates of psychopathology in the SAND teens were approximately equal to or below national averages (Table 5.1); however, the rates for the SAND parents/guardians (92% biological mothers) were generally higher than the national average, particularly for depressive disorders and PTSD (Kessler et al., 2005; Merikangas et al., 2010). With the transition to adulthood, we may see heterotypic continuity in the link between adversity-related neural correlates and psychopathology outcomes where the same process, or neural correlate, may relate to different outcomes at different developmental stages (Angold et al., 1999; Hyde, 2015). The neural correlates observed in study 1 and study 2 may be adaptive in the short term, or in the teen's current context (Hyde et al., 2020; Varnum & Kitayama, 2017), but it will important to continue to follow these teens to determine if that remains the case as they transition to adulthood.

Generalizability. A strength of the SAND subsample and the larger FFCWS sample is that it includes substantial representation of marginalized populations, specifically African American youth and their families. For example, the SAND subsample is approximately 70% African American, which makes it largely different from other large neuroimaging samples (though see Brody et al., 2017; Shaw et al., 2012 for other examples). However, this may impact how the results compare to the extant literature and highlights the limits of generalizability of results based on sample composition. Previous work in the SAND sample has found adversity-

related neural correlates that do not entirely match existing trends in the literature (e.g., threat relates to attenuated rather than heightened amygdala reactivity (Hein et al., 2020; Hein & Monk, 2017). Similarly, study 2 in this dissertation found that violence exposure was related to reduced salience network density, which is not directly in line with results of resting-state functional connectivity studies showing adversity-related increases in salience network connectivity (e.g., Marusak, Etkin, et al., 2015). This may be due to meaningful differences in sample demographics or systematic differences in the experience of adversity (e.g., chronic vs. acute). For example, research in other high-risk samples found similar blunting of amygdala reactivity (Gard et al., 2017; Holz et al., 2017) and work examining the HPA-axis response to stress has found that experiences of chronic stress, or adversity, relate to blunting of the stress response (McCarty, 2016). Additionally, the representation of different racial, ethnic, and gender identities as well as the systematic differences in their experiences contribute to the complex associations between context, biology, and behavior and it is important to consider how these factors may impact how research findings are interpreted and may generalize (Hyde et al., 2020). Last, discussions regarding generalizability of results are not complete without considering the importance of thoughtful, representative sampling in the first place. In order to be able to more fully understand complex associations between adversity, the brain, and behavior, the research questions must be asked in well-powered studies where there is substantial representation of the communities to which we want the results to generalize (Davis-Kean & Jager, 2017; Falk et al., 2013; Hyde et al., 2020).

Integrative Themes

There are three integrative themes that guided the research reported in this dissertation which merit discussion that center around how to model risk, resilience, and their neural

correlates. A fourth integrative theme that guided this dissertation is the importance of open science to support the transparency and reproducibility of psychological research.

Dimensional Models of Adversity and Resilience. All three data chapters in this dissertation provide evidence and support for breaking adversity down into core, underlying dimensions that have at least partially distinct effects on brain and behavioral development (McLaughlin et al., in press, 2014). Findings from studies 1 and 2 isolate white matter and functional neural correlates of violence exposure. In contrast, although social deprivation moderated the association between violence exposure and the brain in study 1, it did not seem to be related to the neural indices we examined in distinct ways. Additionally, study 3 provided initial support for extending dimensional models to include protective processes that may have both global and more specific buffering effects. In each of these three studies, modeling risk and resilience within a dimensional framework yielded specific results which help to identify mechanisms explaining how risk factors influence developmental outcomes with the goal of ultimately improving both theory and evidence-based interventions (Hyde, 2015; McLaughlin et al., in press).

Importance of Neural Connectivity. A central tenet of this dissertation was modeling the brain using methods that index connectivity because the brain is a complex system of interacting regions (Pessoa, 2018; Rubinov & Sporns, 2010). The use of connectivity or network models, such as those in studies 1 and 2, help to provide a more comprehensive picture of how adversity gets under the skin which may not be picked up when examining individual ROIs, including how the brain may compensate or adapt to environmental adversity (Blair & Raver, 2012; Varnum & Kitayama, 2017). Additionally, this dissertation took a multimodal neuroimaging approach to modeling neural connectivity by measuring both white matter and resting-state functional

connectivity; two methods which provide complementary but distinct insights into the brain (Honey et al., 2007; Rubinov & Sporns, 2010). White matter connectivity (study 1) is posited to index longer-term exposures since axonal myelination is a process that occurs gradually across development (de Prado Bert et al., 2018; Mackey et al., 2012). In contrast, resting-state functional connectivity (study 2), may be more sensitive to short-term, concurrent exposures in addition to picking up on patterns of experience-dependent plasticity that reflect a history of co-activation (Gabard-Durnam et al., 2016; Guerra-Carrillo et al., 2014). Of note, research has not systemically tested if there are differential links between the timing of environmental exposures and diffusion and resting-state functional MRI. However, this is an interesting avenue for future longitudinal neuroimaging research. The understanding of associations between dimensional exposure to adversity and the structure and function of neural circuits gained from this research will hopefully help build and improve existing theoretical models and ultimately help create neuroscience-informed policies and interventions (Farah, 2018).

Heterogeneous Individual Response to the Environment. Results from this dissertation, as well as the discussion of the first two integrative themes, provide evidence suggesting that individuals are unique and their interaction with the environment results in nuanced and probabilistic associations between adversity, the brain, and behavior (Cicchetti & Blender, 2004; Hyde et al., 2020; Wiggins & Monk, 2013). In study 2, we see increased heterogeneity of functional connectivity in teens with more childhood exposure to violence. This may be because individuals do not respond to environmental stressors in uniform ways, likely because people do not compensate for stress in uniform ways (Marder & Goaillard, 2006). This concept is thoroughly discussed in theoretical models of developmental psychopathology which emphasize that risk and protective factors operate within complex, multi-level systems

(Bronfenbrenner & Morris, 2007; Cicchetti & Toth, 2009; Hyde, 2015; Sameroff, 2010). There are multiple, interacting, mechanistic pathways that connect environmental adversity with individual brain and behavioral outcomes (Hankin, 2012; Hyde et al., 2020). This is most clearly evident in studies of resilience where individuals with similar risk factors end up with different outcomes (multifinality - Cicchetti & Rogosch, 1996). In these studies, such as study 1 and study 3, we see conditional and indirect effects which are more likely than large main effects to explain how exposure to adversity gets under the skin and affects the individual (Hyde, 2015). Consistent with that idea is that the extant literature has not identified strong, main effects of environmental adversity in either brain or behavioral outcomes, but rather effects that are much more heterogeneous and variable (Hughes et al., 2017; McLaughlin et al., 2019). Dimensional models of adversity and resilience attempt to account for the heterogeneity of the environment and models of neural connectivity work to better characterize the heterogeneous, dynamic system that is the brain. However, a final, and critically important, set of factors that contribute to these nuanced associations is the systematic differences in unmeasured influences resulting from systems of oppression that marginalized communities experience (Chetty et al., 2020; Lipina & Evers, 2017; Rivas-Drake et al., 2014).

The presence of nuanced and probabilistic associations between adversity, the brain, and behavior highlights the need to incorporate statistical methods that are capable of picking up the dynamic interactions between context, biology, and behavior within an individual (Beltz et al., 2016; Howard & Hoffman, 2018; Molenaar, 2004). Moving forward, research in this domain must balance the parsimony provided by variable-centered models with the specificity provided by person-centered and person-specific approaches to build theoretical models that are representative of the individual but have the potential to be generalized. Methods that bridge

nomothetic and idiographic inferences, such as GIMME (used in study 2), can help identify associations that are more generalizable (e.g., to the full sample or to subgroups) in addition to those that are largely unique to the individual (Beltz & Gates, 2017; Gates et al., 2014; Gates & Molenaar, 2012). Person-centered analysis approaches, such as latent profile analysis and data-driven cluster analysis, can also help identify subgroups within a sample and give insight into how associations differ within and between groups (Howard & Hoffman, 2018). It is likely a combination of variable-centered, person-centered, and person-specific analyses is necessary to develop and test models that explain how environmental adversity and individual neural and behavioral outcomes are associated across development.

Importance of Open Science. One last integrative theme that has guided the work reported in this dissertation has been the importance of open science in psychological research. Studies 1 and 2 in this dissertation has preregistered aims, hypotheses, variables, and analytic plans (Study 1: https://osf.io/spguw; Study 2: https://osf.io/mrwhn). While the final analyses did end up being slightly different from the preregistered plan, especially for Study 1, preregistering these studies helped to increase transparency about what was specifically hypothesized and what was exploratory (Nosek et al., 2018; Simmons et al., in press). Preregistration is not always the best fit for some research questions or methods. For example, it is difficult, though admittedly not impossible, to preregister all of the iterative potential pathways in structural equation models that may arise depending on how models fit the data (e.g., study 3). However, in those and all cases, open science practices can and still should be incorporated which promote transparent, reproducible research practices. For example, the code for the statistical analyses done for all three studies reported in this dissertation are publicly available (https://github.com/lgoetschius) as are the neuroimaging analysis pipelines used to process the white matter

(https://github.com/lgoetschius/ProbabilisticTractography_On_Flux) and resting-state functional (Beltz et al., 2019) connectivity data. Future work should incorporate open-science practices through publicly available code as well as pre-registration or registered reports where appropriate.

Future Directions & Conclusions

The research in this dissertation presents evidence for how two dimensions of childhood adversity, violence exposure and social deprivation, predict differences in adolescent neural structure and function, as well as how protective factors, such as school connectedness, buffers against these dimensions. However, it also points to a few avenues of future research that have not already been discussed. One necessary direction for future research is to strengthen the measurement of dimensions of adversity. Similar to dimensional models of psychopathology (Caron & Rutter, 1991; Hyde, 2015), dimensional models of adversity are only as strong as the measures used to operationalize and quantify them (McLaughlin et al., in press). Currently, much of the research using dimensional models of adversity, this dissertation included, have used measures of environmental adversity that are not designed to isolate specific dimensions, but rather are pieced together from separate measures in existing datasets that measure specific exposures (e.g., child abuse, child neglect, community violence, etc.) (McLaughlin et al., in press). Future work should utilize principles from psychometric theory to build questionnaires that are designed to measure dimensions of adversity and should also work to include more objective measures in addition to data from multiple sources within and outside of the families we are working with (e.g., social workers, teachers). A second, related direction for future research is the consideration of additional dimensions. Results from study 3 and a discussion of how they fit into the larger literature suggest that social and cognitive deprivation may be two

separate dimensions. Future work should directly test this hypothesis using both behavioral and neuroimaging data to determine if they have distinct effects. Additionally, recent theoretical work has posited that unpredictability or instability of the environment may be another dimension of adversity (Ellis et al., 2009; McLaughlin et al., in press). Future work should directly test this as well and determine its unique effect in models that contain the other dimensions.

A third important direction for future research will be to account for the influence of genetic and epigenetic factors. Previous work suggests that genetic and epigenetic factors contribute to the nuanced and probabilistic associations between adversity, the brain, and behavior through conditional and indirect effects (Dunn et al., 2019; Gard et al., 2017; Holz et al., 2016; Hyde, 2015). Additionally, a recent meta-analysis robustly linked the dimension of threat, but not deprivation, to accelerated cellular aging using data about telomere length and epigenetic clocks designed to quantify aging using markers of DNA methylation (Colich et al., 2020). This meta-analysis had limitations, including that it used post-hoc estimates of threat and deprivation for the included studies and that it collapsed across all indices of cellular aging rather than examining distinct effects of adversity on epigenetic clock estimates and telomere length. This second limitation is particularly problematic due to research that has found low correlations between telomere length and metrics from different epigenetic clocks, suggesting that they may work through distinct biological processes (Belsky et al., 2018). However, the results of this meta-analysis, as well as other work linking genomics with environmental adversity and the brain, provide strong evidence for the need to consider and account for links between environmental adversity and measures of genomics within dimensional models of adversity.

Overall, the goal of this dissertation has been to help understand how early adversity may shape neural and behavioral development and what may help buffer against it. Throughout this

work, my goal has been to keep the families who participated in the SAND and FFCWS studies in mind, especially when framing research questions and interpreting results. It has been important to always remember that I am analyzing data about exposure to adversity and stress from real people and to be sensitive to that when working on this important topic. Further, I hope I have communicated that, although we should do everything we can to prevent people from living in high-risk environments and experiencing adversity, exposure to it does not lead to a "broken brain" that condemns people to poor outcomes (Hyde et al., 2020; Varnum & Kitayama, 2017). Rather, I hope it brings attention to the societal inequalities that perpetuate the cycle of poverty and keep families living in high-risk environments (Hyde et al., 2020; Lipina & Evers, 2017; Oliver et al., 2006; Roberts & Rizzo, 2020; Rothstein, 2017). Additionally, I hope that the work on the promotive and protective effects of school connectedness underscores the importance of providing schools with the resources and support necessary to create environments where all students feel connected. It is my ultimate goal that the research reported in this dissertation can eventually help the people from the statistics I reported at the very beginning of this dissertation. The approximately 58% percent of adolescents who experience at least one ACE, the 1 in 4 children who experience some form of child maltreatment, and the approximately 40% are exposed to violence in the home or neighborhood (Finkelhor et al., 2015; McLaughlin, Greif Green, et al., 2012; Sacks & Murphy, 2018). We still have a long way to go.

Table 5.1: Rates of teen and parental psychopathology in SAND

In this table, rates from the SAND teens and their mothers are reported alongside national rates.

Adolescent Psychopathology[1]		
Diagnosis*	**SAND Rate (Count)**	**National Rate[2]**
ADHD	16% (39)	12.8%
Conduct Disorder	8% (19)	6.8%
Generalized Anxiety Disorder	3% (6)	2.3%
Major Depressive Disorder or Dysthymia	16% (39)	11.9%
Oppositional Defiant Disorder	8% (20)	12.6%
Post-Traumatic Stress Disorder	3% (7)	5.2%
Social Phobia	8% (18)	9.1%
Specific Phobia	5% (11)	19.5%
Substance Use Disorder	1% (3)	6.4%
Maternal[3] Psychopathology[4]		
Diagnosis*	**SAND Rate (Count)**	**National Rate[5]**
Generalized Anxiety Disorder	4% (9)	7.1%
Major Depressive Disorder	38% (91)	20.2%
Obsessive Compulsive Disorder	3% (7)	3.1%
Panic Disorder	8% (19)	6.2%
Persistent Depressive Disorder	13% (30)	3.1%
Post-Traumatic Stress Disorder	14% (33)	9.7%
Social Phobia	10% (23)	13.0%
Specific Phobia	15% (35)	15.8%
Substance Use Disorder	<1% (2)	7.5%

Notes:
[1] Diagnosed using K-SADS-PL (Kaufman et al., 1997)
[2] National rates for teens come from the lifetime rates reported by 13-18-years-olds on the NCS-A (Merikangas et al., 2010)
[3] 92% of parents or guardians at SAND visit were mothers, so we called this maternal psychopathology for parsimony
[4] Diagnosed using SCID for DSM5 (First, 2015)
[5] National rates for moms come from estimates of lifetime prevalence in women reported in the NCS-R (Kessler et al., 2005)
* Includes past and current diagnoses and "not otherwise specific" diagnoses

Appendix 1: Chapter 2 Supplemental Methods

Violence Exposure and Social Deprivation Composites

Childhood Exposure to Violence. Included in this composite was the primary caregiver's report of child physical and emotional abuse based on items from the Parent-Child Conflict Tactics Scale (Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Five items were used to assess physical abuse including, "hit him/her on the bottom with a hard object" and "shook him/her" and five items were used to assess emotional abuse including whether the parent/caregiver has "sworn or cursed at," or "called him/her dumb or lazy or some other name like that." Each item was rated on a 7-point Likert scale ranging from "never happened" to "more than 20 times." The primary caregiver's report of the child's exposure to or victimization of violence in the neighborhood (Zhang & Anderson, 2010) was also included in the composite. This was measured using the primary caregiver's report of the child witnessing or being the victim of beating, attacks with a weapon, shootings, and killings (witness only) on a 5-point Likert scale ranging from "never" to "more than 10 times." At age 9, the primary caregiver was not asked about whether the child had witnessed killings or it they had been the victim of a shooting, so these items were only included for ages 3 and 5 years. Lastly, the child's mother reported on intimate partner violence (IPV) (physical-2 items, emotional-3 items, or sexual-1 item) in the home at each wave (Hunt et al., 2017). Each item was rated on a 3-point Likert scale ranging from "never" to "often." Physical IPV items included "he slapped or kicked you" and "he hit you with his fist or a dangerous object." Emotional IPV items included "he tried to isolate you from family and friends," and "he

tried to prevent you from going to work and/or school." The sexual IPV was "he tried to make you have sex or do sexual things you didn't want to do." The child's exposure to IPV against the mother was coded as missing for a given wave if the child did not live with their mother at least 50% of the time.

Childhood Exposure to Social Deprivation. Included in this composite was the primary caregiver's report of child physical and emotional neglect based on items from the CTS-PC (Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Four items from the CTS-PC were used to assed physical neglect including whether the parent was ever "so drunk or high that you had a problem taking care of your child." One item, whether the parent was "ever so caught up in your own problems that you were not able to show or tell your child that you loved him/her," was used to assess emotional neglect. These items from the CTS-PC were reported on the same 7-point Likert scale as the items in the violence exposure composite. The primary caregiver's report of social cohesion in the neighborhood was also included in this composite (reverse coded such that higher scores corresponded to lower cohesion) (Donnelly et al., 2016; Morenoff et al., 2001). This included 4 items, such as "this is a close-knit neighborhood," rated on a 5-point Likert scale ranging from "strongly agree" to "strongly disagree." Lastly, the child's mother reported on the level of intimate partner support for each wave using six items, such as "how frequently (the current romantic partner) expresses love and affection (for the mother)," that were rated on a 3-point Likert scale ranging from "never" to "often" (Manuel et al., 2012). This was also reverse coded such that higher scores corresponded to less support. Child exposure to the mother's intimate partner support was coded as missing for a given wave if the child did not live with their mother at least 50% of the time.

Covariates

Adolescent/Current Internalizing Disorders. To ensure that any observed effects were not driven by internalizing symptoms, a multi-method, multi-informant latent factor that indexed internalizing symptoms was used a covariate. It was constructed from the following measures: (1) K-SADS (Kaufman et al., 1997) clinician report of past and current symptoms of dysthymia, social phobia, generalized anxiety disorder, major depression, and phobia and (2) parent and child report on the Mood and Feelings Questionnaire (Angold et al., 1987), Child Depression Inventory (Helsel & Matson, 1984), and the Screen for Child Anxiety Related Disorders (Birmaher et al., 1997). A CFA of this model in the data had acceptable fit (Hein, 2019), with a χ^2 of 38.558 (p = 0.011), a CFI of 0.936, a TLI of 0.862, and a RMSEA of 0.059 (Hu & Bentler, 1999).

To determine if amygdala–PFC white matter connectivity was a mediator between dimensions of childhood adversity and internalizing psychopathology, we calculated the correlations between white matter connectivity and internalizing psychopathology for each amygdala-PFC target pair. There were no significant associations (Supplemental Table 2.1); therefore, we determined that an analysis of indirect effects would not be appropriate – it was then that internalizing psychopathology was added as a covariate in all analyses.

Supplemental Table 2.1

Correlation between internalizing psychopathology and the probability of amygdala white matter connectivity with four Brodmann's Areas (BAs)(bilaterally) in the PFC.

Region	*r*	*t*	*p*
Left BA10	0.117	1.584	0.115
Left BA11	0.022	0.298	0.766
Left BA25	0.070	0.937	0.350
Left BA47	0.044	0.589	0.557
Right BA10	0.022	0.299	0.765
Right BA11	0.000	0.005	0.996
Right BA25	0.083	1.115	0.267
Right BA47	0.050	0.670	0.504

Adolescent/Current Life Stress. Current life stress was used as a covariate in the present analyses and was measured using the Adolescent Life Events Scale (adapted for Shaw et al., 2003 from Farrell et al., 1998 and Masten et al., 1994). This scale assesses the experience of common adolescent stressful life events in the past year.

Race. Race was used a covariate in all analyses and was a set of two dummy-coded variables based on three race categories reported by the teen on the Multigroup Ethnic Identity Measure (MEIM - (Roberts et al., 1999)): African American (Hispanic and Non-Hispanic), White/Caucasian, and Other. Other included Hispanic, Asian, multi-racial participants, Native American, and Unknown/Not Reported (see Table 2.1). If teen report was not available (n = 12), then parent report was used.

Pubertal Development. Self-report of pubertal status was used as a covariate in the present analyses and was collected using the child report of the pubertal development scale (Petersen et al., 1988) which asks both boys and girls about growth spurt in height, pubic hair, and skin change; facial hair growth and voice change in boys only; and breast development and menarche in girls only (see Table 2.1). When adolescent report pubertal data was not available (N=6), parent report was used. Pubertal development scores reported by parents were not significantly different from those reported by the adolescent (t (5.76) =-0.93, p=0.39).

Maternal Covariates at Birth. We used two maternal covariates at birth– self-report of maternal marital status at birth (yes/no) and of maternal education at birth (1 - less than high school, 2 - high school or equivalent, 3 - some college/technical school, 4 - college or graduate school).

Functional MRI Task – Gender Identification (Faces Task)

During fMRI data collection, participants completed an implicit emotion task in which they were instructed to attend to the gender of emotional faces from the NimStim set (Tottenham et al., 2009) and respond to the gender of the face (Swartz et al., 2014). A trial consisted of a 500 ms fixation cross followed by a face presented for 250 ms. A black screen then appeared for 1500 ms, during which participants indicated the gender of the face by pressing a button (thumb for male, index finger for female). Total trial duration was 2250 ms with an inter-trial interval that was jittered and ranged from 2000 to 6000 ms at intervals of 2000 ms. There were 100 total trials with 20 trials of each of the following emotions: happy, sad, angry, fearful, neutral. There were equal numbers of males and female faces and an equal number of faces from individuals identified as White/Caucasian-American and Black/African American.

Diffusion MRI Processing

Raw diffusion MRI data were denoised using MRtrix. Denoised data were then corrected for motion, eddy-current, and signal dropout using dwipreproc in MRtrix, which utilizes eddy from FSL (v.5.0.11)(Andersson & Sotiropoulos, 2016). Intra-volume motion was corrected using slice-to-volume correction. Mean and standard deviation of the movement parameters as well as their correlations with violence exposure and social deprivation can be found in Supplemental Table 2.2. All correlations between environmental variables and movement parameters were not statistically significant. Slices with an average intensity four or more standard deviations lower than predicted by eddy's Gaussian process model were marked as outlier slices and replaced with model predictions (Andersson et al., 2016). Participants with more than 5% of slices replaced were excluded (N=1). Across included participants, 0.00% to 4.35% of slices were replaced (median 0.43%). Mean and standard deviation of the percentage of outlier slices as well as the correlation between percentage of outlier slices and our environmental variables can be found in

Supplemental Table 2.2. The ten images with the most slices replaced were visually inspected to ensure that no abnormal artifacts were introduced during preprocessing. As an additional quality check, we calculated the percentage of non-zero voxels for each seed and target mask (using fslstats) and excluded participants (N=1) who did not have data for at least 70% of the voxels.

Supplemental Table 2.2

Means, standard deviations, and correlations with confidence intervals between the environmental measures (Violence Exposure and Social Deprivation) and the movement metrics from the diffusion MRI data.

Variable	*M*	*SD*	Violence Exposure	Social Deprivation
Violence Exposure	0.04	0.53		
Social Deprivation	0.03	0.50	.50** [.38, .60]	
Outliers Percentage	0.61%	0.65%	.13 [-.02, .27]	.07 [-.07, .22]
X Rotation (degrees)	0.00	0.37	-.02 [-.16, .13]	-.05 [-.20, .09]
Y Rotation (degrees)	0.00	0.14	.05 [-.10, .19]	-.01 [-.16, .13]
Z Rotation (degrees)	-0.11	0.17	-.00 [-.15, .14]	.10 [-.05, .24]
X Translation (mm)	-0.02	0.16	-.06 [-.20, .09]	.05 [-.10, .19]
Y Translation (mm)	-0.01	0.13	-.02 [-.16, .13]	-.04 [-.18, .11]
Z Translation (mm)	-0.01	0.30	.02 [-.12, .17]	.09 [-.06, .23]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of

population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

Following preprocessing, bedpostx (bedpostx_gpu: (Hernández et al., 2013) was performed using the standard settings (number of fibers modeled per voxel = 2, multiplicative factor weight = 1, burn in = 1000) to build up a distribution of diffusion parameters using Markov Chain Monte Carlo sampling at each voxel (Behrens et al., 2007). The diffusion data were then registered using FLIRT (Jenkinson et al., 2002; Jenkinson & Smith, 2001) to allow for the linear transformation between diffusion, standard, and structural space.

FSL's probtrackx2 (nsamples per voxel = 5000; nsteps per sample = 2000; step length = 0.5mm; curvature threshold = 0.2; fibthresh = 0.01; distthresh = 0.1) (probtrackX2_gpu: (Hernandez-Fernandez et al., 2016) was used to estimate the probability of white matter connectivity between the prefrontal cortex and the amygdala (Behrens et al., 2007; Behrens, Johansen-Berg, et al., 2003; Behrens, Woolrich, et al., 2003; Eickhoff et al., 2010; Johansen-Berg et al., 2004). In this analysis, the amygdala, defined using masks created using WFU Pick Atlas (v 3.0.5b) (Maldjian et al., 2003), was the seed region and eight Brodmann's Areas (BA) were specified as the target regions. Those ROIs were BA10, BA11, BA25, and BA47 and they were selected due to a previous stronger likelihood of amygdala white matter connectivity in our previous work (Goetschius et al., 2019). Individual masks were made for the included regions for each hemisphere using WFU Pick Atlas. Eight separate probabilistic tractography analyses were run in each participant's native diffusion space for the anatomical regions in each hemisphere. Only ipsilateral connections between the amygdala and PFC regions were targeted in this analysis because neural tracer studies in non-human primates suggest that first order amygdala connections are primarily ipsilateral (Ghashghaei et al., 2007).

Following the individual probabilistic tractography analyses, the resulting amygdala images representing the probability of white matter connectivity with the specified PFC targets were transformed to MNI space for further analysis. Prior to the group-level analysis, each of the individual amygdala images were divided by the total number of samples per voxel (5000) which scaled the probability value at each voxel to a range between 0 and 1. Based on the group-level analysis outlined in Greening and Mitchell (Greening & Mitchell, 2015), an average amygdala image was created for each amygdala-PFC target pair using fslmaths (FMRIB, Oxford, UK) and the peak voxel in that image was identified. This peak voxel represents the maximum probability of white matter connectivity between the amygdala and the specified PFC region. This peak, selected at the group level, was used for all participants. The MNI coordinates (x,y,z) for the peak for each target are as follows: BA10 (left: -30, -4, -14; right: 32, -2, -12), BA11 (left: -30, -4, -14; right: 32, -2, -14), BA25 (left: -16, 0, -14, right: 18, 0, -14), BA47: (left: -30, -4, -14; right: 34, 0, -20). A 6mm sphere mask was then created centered around the peak voxel. Once each of the amygdala-target peak voxel masks were created, the average probability of connectivity for each mask was extracted at the individual participant level. This extracted value represents the maximum likelihood estimate of probability for each of the 8 amygdala-target pairs (Goetschius et al., 2019).

Identifying Statistical Outliers

Statistical outliers were excluded from multiple regression analyses based on the standardized residuals with a cutoff based on Cook's Distance. This was done separately for each hemisphere in order to retain data from more participants (N=6 in the right hemisphere and N=6 in the left hemisphere) using diagnostic plots, including a Q-Q Norm plot, created using the stats

package (https://www.rdocumentation.org/packages/stats/versions/3.5.1) in R (v.3.5.1). Only one of the six outliers was present in both hemispheres.

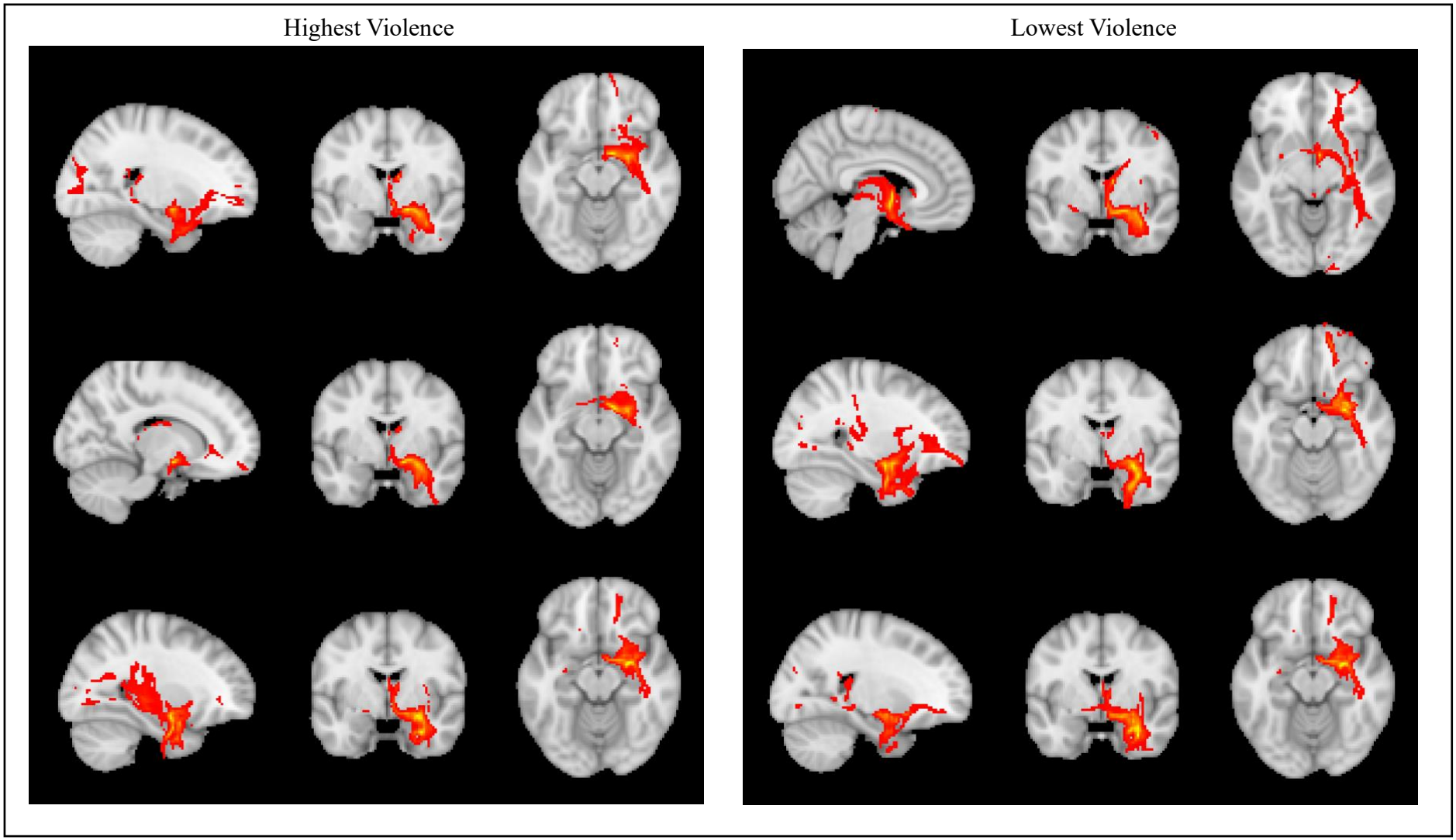
The final sample was slightly different in the right vs. left hemisphere. See Table 2.1 for a comparison of sample demographics in the included versus full samples. The included sample for both the left and right hemispheres did not differ statistically from the full sample on age (Left Hemisphere: $t(396.31) = 0.30$, p=0.765; Right Hemisphere: $t(398.04) = 0.32$, p=0.750), puberty (Left Hemisphere: $t(392.68) = -0.33$, p=0.739; Right Hemisphere: $t(394.16) = -0.15$, p=0.882), gender (Left Hemisphere: : $X^2(1) = 0.020$, p = 0.887; Right Hemisphere: $X^2(1) = 0.000$, p = 0.987), race (Left Hemisphere: $X^2(2) = 0.044$, p = 0.978; Right Hemisphere: $X^2(2) = 0.000$, p = 0.999), or annual income (Left Hemisphere: $X^2(5) = 0.155$, p = 0.999; Right Hemisphere: $X^2(5) = 0.002$, p = 0.999).

Checks for Statistical Assumptions

To confirm that our data conforms to the assumptions required for linear regression, we performed multiple checks of the model residuals. Only the checks for the regression model predicting right amygdala–BA47 white matter connectivity from violence exposure, social deprivation, and violence exposure x social deprivation (including covariates) are reported here since those were the main results from the present study; however, checks were done for all regression models. First, we confirmed that the mean of the model residuals was approximately equivalent to zero ($M_{residuauls} = 0.00000000000000005935$, $t(182) = 0.0000$), $p = 1$). Next, we ran a Durbin-Watson test to confirm that our residuals were not autocorrelated ($DW = 1.917$, $p = 0.265$). Then, we confirmed that our X variable of interest (violence exposure x social deprivation) was not correlated with the model residuals, $t(181) = 0.000$, $p = 1$. Additionally, we tested for multicollinearity in our X variables by calculating the variance inflation factor (VIF).

The maximum VIF for all X variables (including covariates) was 2.03 (one of the two dummy-coded variable for race/ethnicity) with the VIF for a majority of variables being below 2, a relatively conservative cutoff (Sheather, 2009). Based on these checks, we concluded that our data conformed to the assumptions of linear regression.

Supplemental Figure 2.1*: Individual streamlines of participants with the highest and lowest composite scores. Images are taken from the peak voxel per participant. Images thresholded at 1000 streamlines.*



Appendix 2: Chapter 2 Supplemental Results

Supplemental Table 2.3

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict right amygdala–BA10 white matter connectivity when accounting for covariates.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.06	[-0.00, 0.12]				
Race_1[2]	-0.00	[-0.03, 0.02]	-0.04	[-0.25, 0.16]		
Race_2[2]	0.02*	[0.00, 0.04]	0.21	[0.00, 0.41]		
Gender	0.00	[-0.02, 0.02]	0.00	[-0.20, 0.20]		
Internalizing Psychopathology	0.00	[-0.01, 0.02]	0.05	[-0.11, 0.21]		
Pubertal Status	-0.00	[-0.01, 0.01]	-0.00	[-0.19, 0.18]		
Current Life Stress	-0.00	[-0.00, 0.00]	-0.06	[-0.21, 0.10]		
Maternal Education	-0.01	[-0.01, 0.00]	-0.14	[-0.30, 0.03]		
Maternal Marital Status	-0.00	[-0.02, 0.01]	-0.04	[-0.20, 0.12]		
					R^2 = .073 95% CI[.00,.12]	
(Intercept)	0.06	[-0.00, 0.12]				
Violence Exposure	-0.00	[-0.02, 0.01]	-0.02	[-0.20, 0.16]		
Social Deprivation	-0.00	[-0.02, 0.01]	-0.04	[-0.22, 0.14]		
Race_1[2]	-0.00	[-0.03, 0.02]	-0.03	[-0.24, 0.17]		
Race_2[2]	0.02*	[0.00, 0.04]	0.22	[0.01, 0.43]		

Gender	-0.00	[-0.02, 0.02]	-0.00	[-0.20, 0.19]		
Internalizing Psychopathology	0.01	[-0.01, 0.02]	0.05	[-0.11, 0.22]		
Pubertal Status	-0.00	[-0.01, 0.01]	-0.01	[-0.20, 0.18]		
Current Life Stress	-0.00	[-0.00, 0.00]	-0.06	[-0.22, 0.10]		
Maternal Education	-0.01	[-0.01, 0.00]	-0.15	[-0.31, 0.02]		
Maternal Marital Status	-0.00	[-0.02, 0.01]	-0.04	[-0.20, 0.13]		
					R^2 = .076 95% CI[.00,.11]	ΔR^2 = .002 95% CI[-.01, .02]
(Intercept)	0.06	[0.00, 0.12]				
Violence Exposure	0.00	[-0.01, 0.02]	0.02	[-0.16, 0.21]		
Social Deprivation	0.00	[-0.01, 0.02]	0.02	[-0.17, 0.20]		
Interaction[1]	-0.01	[-0.03, 0.00]	-0.15	[-0.33, 0.02]		
Race_1[2]	-0.00	[-0.03, 0.02]	-0.00	[-0.21, 0.21]		
Race_2[2]	0.02*	[0.00, 0.04]	0.21	[0.00, 0.42]		
Gender	-0.00	[-0.02, 0.02]	-0.00	[-0.20, 0.19]		
Internalizing Psychopathology	0.01	[-0.01, 0.02]	0.05	[-0.11, 0.22]		
Pubertal Status	-0.00	[-0.01, 0.01]	-0.02	[-0.21, 0.17]		
Current Life Stress	-0.00	[-0.00, 0.00]	-0.06	[-0.22, 0.10]		
Maternal Education	-0.01	[-0.01, 0.00]	-0.14	[-0.30, 0.03]		
Maternal Marital Status	-0.00	[-0.02, 0.01]	-0.04	[-0.21, 0.12]		
					R^2 = .092 95% CI[.00,.12]	ΔR^2 = .016 95% CI[-.02, .05]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively. * indicates $p < .05$. ** indicates $p < .01$.

[1]Interaction between Violence Exposure/Victimization and Social Deprivation

[2]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)

Supplemental Table 2.4

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict left amygdala—BA10 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.01**	[0.01, 0.01]				
Violence Exposure	-0.00	[-0.01, 0.00]	-0.02	[-0.20, 0.15]		
Social Deprivation	-0.00	[-0.01, 0.01]	-0.01	[-0.18, 0.16]		
					R^2 = .001 95% CI[.00,.01]	
(Intercept)	0.01**	[0.01, 0.01]				
Violence Exposure	0.00	[-0.01, 0.01]	0.00	[-0.17, 0.18]		
Social Deprivation	0.00	[-0.00, 0.01]	0.03	[-0.15, 0.21]		
Interaction[1]	-0.00	[-0.01, 0.00]	-0.12	[-0.29, 0.05]		
					R^2 = .012 95% CI[.00,.05]	ΔR^2 = .011 95% CI[-.02, .04]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.5

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict left amygdala—BA11 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.02**	[0.02, 0.02]				
Violence Exposure	-0.00	[-0.01, 0.01]	-0.06	[-0.23, 0.11]		
Social Deprivation	-0.00	[-0.01, 0.01]	-0.00	[-0.17, 0.17]		
					R^2 = .004 95% CI[.00,.03]	
(Intercept)	0.02**	[0.02, 0.02]				
Violence Exposure	-0.00	[-0.01, 0.01]	-0.04	[-0.22, 0.14]		
Social Deprivation	0.00	[-0.01, 0.01]	0.03	[-0.15, 0.21]		
Interaction[1]	-0.01	[-0.01, 0.00]	-0.09	[-0.26, 0.08]		
					R^2 = .010 95% CI[.00,.04]	ΔR^2 = .006 95% CI[-.02, .03]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.6

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict left amygdala—BA25 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.08**	[0.08, 0.09]				
Violence Exposure	0.00	[-0.02, 0.02]	0.01	[-0.16, 0.19]		
Social Deprivation	0.00	[-0.02, 0.02]	0.00	[-0.17, 0.17]		
					R^2 = .000 95% CI[.00,1.00]	
(Intercept)	0.08**	[0.08, 0.09]				
Violence Exposure	0.00	[-0.02, 0.02]	0.02	[-0.16, 0.20]		
Social Deprivation	0.00	[-0.02, 0.03]	0.01	[-0.18, 0.19]		
Interaction[1]	-0.00	[-0.03, 0.02]	-0.02	[-0.19, 0.15]		
					R^2 = .001 95% CI[.00,1.00]	ΔR^2 = .000 95% CI[-.01, .01]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

* indicates $p < .05$. ** indicates $p < .01$.

[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.7
Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict left amygdala—BA47 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.02**	[0.02, 0.03]				
Violence Exposure	-0.00	[-0.01, 0.00]	-0.09	[-0.26, 0.08]		
Social Deprivation	0.00	[-0.00, 0.01]	0.09	[-0.09, 0.26]		
					R^2 = .008 95% CI[.00,.04]	
(Intercept)	0.02**	[0.02, 0.03]				
Violence Exposure	-0.00	[-0.01, 0.00]	-0.09	[-0.26, 0.09]		
Social Deprivation	0.01	[-0.00, 0.01]	0.10	[-0.09, 0.28]		
Interaction[1]	-0.00	[-0.01, 0.01]	-0.03	[-0.19, 0.14]		
					R^2 = .008 95% CI[.00,.04]	ΔR^2 = .001 95% CI[-.01, .01]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.8

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict right amygdala—BA11 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.08**	[0.07, 0.09]				
Violence Exposure	-0.02	[-0.04, 0.01]	-0.12	[-0.29, 0.05]		
Social Deprivation	0.00	[-0.02, 0.02]	0.01	[-0.16, 0.18]		
					R^2 = .013 95% CI[.00,.05]	
(Intercept)	0.08**	[0.07, 0.09]				
Violence Exposure	-0.01	[-0.04, 0.01]	-0.10	[-0.27, 0.07]		
Social Deprivation	0.01	[-0.02, 0.03]	0.04	[-0.14, 0.22]		
Interaction[1]	-0.01	[-0.04, 0.02]	-0.07	[-0.24, 0.10]		
					R^2 = .017 95% CI[.00,.06]	ΔR^2 = .004 95% CI[-.01, .02]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.9

Stepwise regression results showing that violence exposure, social deprivation, and their interaction do not predict right amygdala—BA25 white matter connectivity.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	Fit	Difference
(Intercept)	0.09**	[0.08, 0.10]				
Violence Exposure	-0.01	[-0.03, 0.01]	-0.08	[-0.25, 0.09]		
Social Deprivation	-0.00	[-0.03, 0.02]	-0.01	[-0.18, 0.16]		
					R^2 = .007 95% CI[.00,.04]	
(Intercept)	0.09**	[0.07, 0.10]				
Violence Exposure	-0.01	[-0.04, 0.01]	-0.09	[-0.26, 0.08]		
Social Deprivation	-0.00	[-0.03, 0.02]	-0.03	[-0.21, 0.15]		
Interaction[1]	0.01	[-0.02, 0.04]	0.04	[-0.13, 0.22]		
					R^2 = .009 95% CI[.00,.04]	ΔR^2 = .001 95% CI[-.01, .01]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.
[1]Interaction between Violence Exposure/Victimization and Social Deprivation

Supplemental Table 2.10

Amygdala-prefrontal cortex white matter connectivity does NOT predict ipsilateral amygdala activation to threat faces

	B	***SEB***	***β***	***t***	***p***
Model: L. Amygdala Activation (Threat) ~ L. Amygdala-BA25 White Matter Connectivity					
L. Amygdala – BA25	-0.210	0.750	-0.023	-0.280	0.780
	$F(1, 150) = 0.079$ p = 0.780, $R^2 = 0.000$				
Model: L. Amygdala Activation (Threat) ~ L. Amygdala-BA47 White Matter Connectivity					
L. Amygdala – BA47	-3.935	1.820	-0.174	-2.162	0.032
	$F(1, 150) = 4.674$, p = 0.032, $R^2 = 0.030$				
Model: R. Amygdala Activation (Threat) ~ R. Amygdala-BA11 White Matter Connectivity					
R. Amygdala – BA11	-1.027	0.765	-0.109	-1.342	0.182
	$F(1, 150) = 1.802$, p = 0.182, $R^2 = 0.012$				
Model: R. Amygdala Activation (Threat) ~ R. Amygdala-BA25 White Matter Connectivity					
R. Amygdala – BA25	-1.012	0.766	-0.107	-1.321	0.188
	$F(1, 150) = 1.746$, p = 0.188, $R^2 = 0.012$				

Note: No models were significant at p<0.0063 (Bonferroni-corrected significance level for 8 tests)

Appendix 3: Chapter 3 Supplemental Methods

Supplemental Participant Information

Five hundred and six families from Detroit, Toledo, and Chicago area who had participated in the FFCWS were contacted. Of the 506 FFCWS families contacted, 237 families participated in SAND data collection, and 183 of those families had teens who were eligible and completed the neuroimaging tasks. 54 teens either were not eligible (i.e., braces, etc.) to complete the neuroimaging portion of the study (*N*=28) or did not complete the full protocol (*N*=26).

Families that agreed to participate in SAND data collection did not differ from families that refused or were unreachable on annual household income at the six waves of the FFCWS (*p*s = .11 - .84). However, nonparticipation was associated with mother reports of community violence exposure when adolescents were ages 3, 5, and 9 ($\chi 2 = 6.72$, $df = 1$, $p < .05$, $V = .12$). Sixty-three percent of all mothers from families that participated in SAND endorsed at least one form of community violence when adolescents were ages 3, 5, or 9 compared to 51.10% of families that did not participate. Within the Detroit, Toledo, and Chicago subsamples, mothers' self-report of race/ethnicity at the time of the child's birth did not differ between mothers who did and did not participate in SAND (*p*s = .13-.49). However, mothers' self-report of race/ethnicity at the time of the child's birth differed between the SAND sample and the larger FFCWS ($\chi 2 = 86.32$, $df = 3$, $p < .01$, $V = .13$). The majority of mothers who participated in SAND data collection identified as Black/African American (75%), and also included mothers identifying as White/Caucasian (16%), Hispanic (6%), and other (3%). In the FFCWS, 46% of

mothers identified as Black/African, 21% identified as White/Caucasian, 29% identified as Hispanic, and 4% as other.

Of the 183 SAND teens with resting-state MRI data, three participants were excluded due to artifacts in the functional or structural MRI data, four participants had excessive motion (as defined by average relative framewise displacement greater than 0.5mm), and one person had signal dropout in the areas of the brain included in the present analysis. The sample included for the neuroimaging analyses did not differ from the full SAND sample (*ps* 0.670 - 0.997) (Supplemental Table 3.1). This information has also been reported in other work from our group (Hein, 2019; Peckins et al., 2019).

MRI Acquisition

Structural MRI Acquisition. T1-weighted gradient echo images were taken before the functional scans using the same field of view (FOV) and slices as the functional scans (TR = 12ms, TE = 5ms, TI = 500ms, flip angle = 15°, FOV = 26cm; slice thickness = 1.4mm; 256 x 192 matrix; 110 slices, voxel size = 1mm x 1mm x 1mm). These methods are described in previous research using this sample (Goetschius et al., 2019).

Resting State MRI Acquisition. Slices were prescribed parallel to the AC-PC line (same locations as structural scans). Images were reconstructed into a 64x64 matrix (TR=2000 ms, TE=30 ms, flip angle=90°, FOV=22 cm, voxel size=3.44mm x 3.44mm x 3mm, bottom-up interleaved). Slices were acquired contiguously, which optimized the effectiveness of the movement post-processing algorithms. Images were reconstructed off-line using processing steps to remove distortions caused by magnetic field inhomogeneity and other sources of misalignment to the structural data, which yields excellent coverage of subcortical areas of interest. These

methods are identical to those described in previous task-based fMRI research using this sample (Goetschius et al., 2019).

Imaging Data Analysis

MRI Preprocessing. Anatomical images were homogeneity-corrected using SPM2, then skull-stripped (f=0.25) using the Brain Extraction Tool (BET) in FSL (version 5.0.7) (Jenkinson et al., 2012; S. M. Smith, 2002). The functional imaging data then had the following preprocessing steps applied: removal of large temporal spikes in k-space data (> 2 std dev), field map correction and image reconstruction using custom code in MATLAB; noise from cardiac and respiratory motion were removed using RETROICOR, and slice-timing correction using SPM8 (Wellcome Department of Cognitive Neurology, London, UK; http://www.fil.ion.ucl.ac.uk). Additionally, the first 10 volumes of functional data were removed to ensure the stability of signal intensity. Lastly, the structural images were segmented into gray matter, white matter, and CSF using FSL's FAST(Y. Zhang et al., 2001).

Following these initial preprocessing steps, the resting state functional data underwent further preprocessing using FEAT (FMRI Expert Analysis Tool) Version 6.00, part of FSL (FMRIB's Software Library, www.fmrib.ox.ac.uk/fsl). Registration to high resolution structural and/or standard space images was carried out using FLIRT(Jenkinson et al., 2002; Jenkinson & Smith, 2001). The following pre-statistics processing was applied: motion correction using MCFLIRT(Jenkinson et al., 2002); non-brain removal for the functional images using BET(S. M. Smith, 2002); spatial smoothing using a Gaussian kernel of FWHM 6.0mm; grand-mean intensity normalization of the entire 4D dataset by a single multiplicative factor. ICA-AROMA(Pruim et al., 2015) was used to remove motion-related artifacts in the data. Nuisance

signal derived from the white matter and cerebrospinal fluid (CSF) were regressed out of the data and then the data was high-pass filtered to remove signal below 0.01Hz.

Violence Exposure and Social Deprivation Composite Scores

The procedure creating these composite scores were first utilized in previous work from our lab (Hein, 2019) and has been used previously in research on cortisol reactivity in this sample(Peckins et al., 2019). Data for these composite scores come from primary caregiver report on survey measures at 3, 5, 9 years.

Childhood Exposure to Violence. Included in this composite was the primary caregiver's report of child physical and emotional abuse based on items from the Parent-Child Conflict Tactics Scale(Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Five items were used to assess physical abuse including, "hit him/her on the bottom with a hard object" and "shook him/her" and five items were used to assess emotional abuse including whether the parent/caregiver has "sworn or cursed at," or "called him/her dumb or lazy or some other name like that." Each item was rated on a 7-point Likert scale ranging from "never happened" to "more than 20 times." The primary caregiver's report of the child's exposure to or victimization of violence in the neighborhood(S. Zhang & Anderson, 2010) was also included in the composite. This was measured using the primary caregiver's report of the child witnessing or being the victim of beating, attacks with a weapon, shootings, and killings (witness only) on a 5-point Likert scale ranging from "never" to "more than 10 times." At age 9, the primary caregiver was not asked about whether the child had witnessed killings or it they had been the victim of a shooting, so these items were only included for ages 3 and 5 years. Lastly, the child's mother reported on intimate partner violence (IPV) (physical-2 items, emotional-3 items, or sexual-1 item) in the home at each wave (Hunt et al.,

2017). Each item was rated on a 3-point Likert scale ranging from "never" to "often." Physical IPV items included "he slapped or kicked you" and "he hit you with his fist or a dangerous object." Emotional IPV items included "he tried to isolate you from family and friends," and "he tried to prevent you from going to work and/or school." The sexual IPV was "he tried to make you have sex or do sexual things you didn't want to do." The child's exposure to IPV against the mother was coded as missing for a given wave if the child did not live with their mother at least 50% of the time. We considered violence exposure to exist on a continuum in which high scores represent violence exposure and low scores represent safety.

Childhood Exposure to Social Deprivation. Included in this composite was the primary caregiver's report of child physical and emotional neglect based on items from the CTS-PC(Straus et al., 1998) that have been used in previous research (Font & Berger, 2015; Hunt et al., 2017). Four items from the CTS-PC were used to assed physical neglect including whether the parent was ever "so drunk or high that you had a problem taking care of your child." One item, whether the parent was "ever so caught up in your own problems that you were not able to show or tell your child that you loved him/her," was used to assess emotional neglect. These items from the CTS-PC were reported on the same 7-point Likert scale as the items in the violence exposure composite. The primary caregiver's report of social cohesion in the neighborhood was also included in this composite (reverse coded such that higher scores corresponded to lower cohesion). The items were selected based on previous research in the FFCWS linking neighborhood cohesion and adolescent mental health (Donnelly et al., 2016) and were adapted from previous neighborhood research (Morenoff et al., 2001). This included 4 items, such as "this is a close-knit neighborhood," rated on a 5-point Likert scale ranging from "strongly agree" to "strongly disagree." Lastly, the child's mother reported on the level of

intimate partner support for each wave using six items, such as "how frequently (the current romantic partner) expresses love and affection (for the mother)," that were rated on a 3-point Likert scale ranging from "never" to "often" (Manuel et al., 2012). This was also reverse coded such that high scores represent low support. Child exposure to the mother's intimate partner support was coded as missing for a given wave if the child did not live with their mother at least 50% of the time. We considered social deprivation to exist on a continuum in which high scores (e.g., the child experienced either high neglect or witnessed low social support for their mother or low neighborhood social cohesion) represent deprivation and low scores (e.g., the child experienced little neglect or witnessed high social support for their mother or high neighborhood social cohesion) represent social support.

Composite Score Calculation. To calculate composite scores, the Z scores for each of the childhood experiences (i.e., child abuse, exposure to intimate partner violence, community violence, child neglect, lack of romantic partner support, lack of neighborhood social cohesion) were summed for each of the childhood experiences within a dimension (i.e., violence exposure and social deprivation) (Song et al., 2013) and then divided by the number of childhood experiences within a dimension for each participant, thus maximizing the number of participants and the diversity of the sample by minimizing drop out due to missing data at any given wave. This procedure has been previously described (Hein, 2019).

In an exploratory attempt to characterize the sample, we examined the abuse and neglect subscales of the Conflict Tactics Scale (CTS). We found that averaged across ages 3, 5, and 9 years, the focal children experienced greater than 3 of the 10 abuse categories (M=3.67, SD=1.61, range: 0-10) and less than 1 of the 5 neglect categories (M=0.19, SD=0.37, range: 0-4) in the CTS scale.

Covariates

With the exception of mean framewise displacement, all covariates have been previously described (Goetschius et al., 2019; Hein, 2019; Peckins et al., 2019).

Current Life Stress. Current life stress was measured using the Adolescent Life Events Scale (adapted(Shaw et al., 2003) from (Farrell et al., 1998; Masten et al., 1994)). This scale assesses the experience of common adolescent stressful life events in the past year.

Race. Race was operationalized as a set of two dummy-coded variables based on three race categories reported by the teen: African American (Hispanic and Non-Hispanic), White/Caucasian, and Other. Other included Hispanic, Asian, multi-racial participants, Native American, and Unknown/Not Reported (Supplemental Table 3.1). If teen report was not available ($N = 12$), then parent report was used.

Pubertal Development. Self-report of pubertal status was assessed using child report of the Pubertal Development Scale (Petersen et al., 1988) that measures growth spurt in height, public hair, and skin change in boys and girls; facial hair growth and voice change in boys only; and breast development and menarche in girls only (Supplemental Table 3.1). When adolescent report was not available (N=8), parent report was used. Pubertal development scores reported by parents were not significantly different from those reported by the adolescent (t(9.04)=-0.74, p=0.48).

Maternal Covariates at Birth. Maternal self-report of marital status at birth (yes/no) and education at birth (1 - less than high school, 2 - high school or equivalent, 3 - some college/technical school, 4 - college or graduate school) was assessed.

Framewise Displacement. Framewise displacement (FD) is a metric that evaluates motion in the scanner and is the average of rotation and translation parameter differences(Power et al., 2012). FD was quantified using fsl_motion_outliers (FSL v.5.0.7).

Sensitivity Analyses

Extreme Outliers. Predictor variables (i.e., violence exposure and social deprivation) were checked for extreme outliers, which were considered to be individuals with a value less than Q1 - 2.2*Interquartile Range (IQR) or greater than Q3 + 2.2*IQR (Hoaglin & Iglewicz, 1987). Four individuals were considered to be extreme outliers. Analyses were run with and without these participants, and inferences did not change. Thus, the participants were not excluded in order to retain as much data as possible.

Protecting Against Model Overfitting. To protect against model overfitting, the psi matrices for each individual were examined – these matrices summarize the variance that was not explained for each network node for each participant. Participants were flagged (*N*=26 or 14.8%) if they had a psi value greater than 1. To ensure that these participants were not driving effects, all analyses were run with and without participants with high psi values, and inferences did not change. Thus, the participants were not excluded in order to retain as much data as possible.

Supplemental Table 3.1

Participant Demographics

Included vs. Full Sample Comparison			
	Included Sample (N=175)	**Full Sample (N=237)**	**Statistically Different?**
Age	*M* = 15.88 yrs | *SD* = 0.53	*M* = 15.88 yrs | *SD* = 0.54	No. $t(379.27) = 0.10$, $p=0.918$
Puberty	*M* = 3.25 | *SD* = 0.58	*M* = 3.24 | *SD* = 0.59	No. $t(377.67) = 0.30$, $p=0.770$
Gender	F = 98 | M = 77	F = 125 | M = 112	No. $\chi^2(1) = 0.18$, $p = 0.670$
Race	African American: 127 Caucasian: 26 Other: 22	African American: 170 Caucasian: 34 Other: 33	No. $\chi^2(2) = 0.40$, $p = 0.820$
Annual Income	$4,999 or less: 22 $5,000 to $19,999: 29 $20,000 to $39,999: 50 $40,000 to $69,999: 33 $70,000 or more: 28 Not Report/Missing: 13	$4,999 or less: 28 $5,000 to $19,999: 41 $20,000 to $39,999: 66 $40,000 to $69,999: 46 $70,000 or more: 35 Not Report/Missing: 21	No. $\chi^2(5) = 0.34$, $p = 0.997$

Supplemental Table 3.2

MNI coordinates for ROIs.

Region	MNI Coordinates	Network
Left Hemisphere		
Insula	-34 20 -4	Salience
Amygdala	-24 -6 -16	Salience
Dorsal Anterior Cingulate Cortex	0 46 6	Salience
Dorsolateral Prefrontal Cortex	-46 12 34	Salience
Inferior Parietal Lobule	-42 -52 48	Default Mode
Posterior Cingulate Cortex	-4 52 48	Default Mode
Medial Temporal Gyrus	-62 -26 -18	Default Mode
Right Hemisphere		
Insula	36 20 -4	Salience
Amygdala	24 -2 -16	Salience
Dorsal Anterior Cingulate Cortex	4 26 28	Salience
Dorsolateral Prefrontal Cortex	52 12 34	Salience
Inferior Parietal Lobule	46 -52 48	Default Mode
Posterior Cingulate Cortex	8 -52 28	Default Mode
Medial Temporal Gyrus	58 -16 20	Default Mode

Note: ROIs were selected based on their inclusion in either the Salience or Default Mode networks according to the NeuroSynth database (search terms were "Salience Network" and "Default Mode," respectively). Additionally, the MNI coordinates for all ROIs were preregistered with the Open Science Framework.

Supplemental Figure 3.1: Flowchart of the S-GIMME analytical steps

Appendix 4: Chapter 3 Supplemental Results

Supplemental Table 3.3

Regression results for network density.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	*r*	Fit
Total Density						
(Intercept)	47.20	[38.30, 56.11]				
Violence Exposure*	**-2.81**	**[-5.01, -0.60]**	**-0.23**	**[-0.41, -0.05]**	**-.13**	
Social Deprivation	1.51	[-0.63, 3.65]	0.12	[-0.05, 0.30]	.06	
Motion [1]*	**13.24**	**[3.52, 22.96]**	**0.21**	**[0.06, 0.37]**	**.17**	
Gender	-2.22	[-4.65, 0.21]	-0.18	[-0.37, 0.02]	-.08	
Race_1[2]	0.90	[-2.17, 3.98]	0.06	[-0.15, 0.28]	-.08	
Race_2[2]	3.16	[-0.61, 6.94]	0.18	[-0.03, 0.39]	.16	
Pubertal Development	-1.37	[-3.44, 0.69]	-0.13	[-0.32, 0.06]	-.03	
Current Life Stress[3]	-0.08	[-0.24, 0.09]	-0.07	[-0.22, 0.08]	-.07	
Maternal Education[4]	-0.08	[-1.10, 0.93]	-0.01	[-0.18, 0.15]	.03	
Maternal Marital Status[4]	-0.29	[-2.80, 2.23]	-0.02	[-0.18, 0.15]	-.08	
						R^2 = .122
						95% CI[.00,.17]
Salience Network Density						
(Intercept)	17.37	[14.22, 20.52]				
Violence Exposure*	**-1.01**	**[-1.79, -0.23]**	**-0.24**	**[-0.42, -0.05]**	**-.12**	
Social Deprivation	0.59	[-0.17, 1.34]	0.14	[-0.04, 0.32]	.06	
Motion [1]*	**4.97**	**[1.54, 8.41]**	**0.23**	**[0.07, 0.39]**	**.20**	
Gender	-0.33	[-1.19, 0.53]	-0.07	[-0.27, 0.12]	-.01	
Race_1[2]	0.24	[-0.85, 1.33]	0.05	[-0.17, 0.26]	-.06	
Race_2[2]	0.78	[-0.56, 2.11]	0.12	[-0.09, 0.34]	.12	
Pubertal Development	-0.37	[-1.10, 0.36]	-0.10	[-0.29, 0.09]	-.07	
Current Life Stress[3]	-0.00	[-0.06, 0.06]	-0.01	[-0.16, 0.14]	-.03	
Maternal Education[4]	-0.05	[-0.40, 0.31]	-0.02	[-0.18, 0.14]	.02	
Maternal Marital Status[4]	-0.31	[-1.19, 0.58]	-0.06	[-0.22, 0.11]	-.09	
						R^2 = .106

					95% CI[.00,.15]
Density Between Salience & Default Mode Networks					
(Intercept)	18.74	[13.95, 23.53]			
Violence Exposure*	**-1.33**	**[-2.51, -0.14]**	**-0.20**	**[-0.39, -0.02]**	**-.09**
Social Deprivation	0.65	[-0.50, 1.80]	0.10	[-0.08, 0.28]	.06
Motion [1]*	**8.55**	**[3.33, 13.78]**	**0.26**	**[0.10, 0.41]**	**.22**
Gender	-1.07	[-2.37, 0.24]	-0.16	[-0.35, 0.04]	-.04
Race_1[2]	0.28	[-1.37, 1.94]	0.04	[-0.18, 0.25]	-.07
Race_2[2]	1.47	[-0.56, 3.51]	0.15	[-0.06, 0.37]	.14
Pubertal Development	-0.79	[-1.90, 0.33]	-0.13	[-0.32, 0.06]	-.07
Current Life Stress[3]	-0.05	[-0.14, 0.04]	-0.08	[-0.23, 0.07]	-.09
Maternal Education[4]	-0.01	[-0.56, 0.54]	-0.00	[-0.17, 0.16]	.01
Maternal Marital Status[4]	0.40	[-0.95, 1.75]	0.05	[-0.12, 0.21]	-.01
					R^2 = .122
					95% CI[.00,.17]
Default Mode Network Density					
(Intercept)	11.09	[8.18, 14.01]			
Violence Exposure	-0.47	[-1.19, 0.25]	-0.12	[-0.31, 0.06]	-.12
Social Deprivation	0.27	[-0.43, 0.97]	0.07	[-0.11, 0.25]	.01
Motion [1]	-0.29	[-3.46, 2.89]	-0.01	[-0.17, 0.15]	-.06
Gender*	-0.82	[-1.62, -0.03]	-0.20	[-0.40, -0.01]	-.19
Race_1[2]	0.38	[-0.62, 1.39]	0.08	[-0.14, 0.30]	-.07
Race_2[2]	0.91	[-0.32, 2.15]	0.16	[-0.06, 0.38]	.15
Pubertal Development	-0.22	[-0.89, 0.46]	-0.06	[-0.26, 0.13]	.09
Current Life Stress[3]	-0.02	[-0.08, 0.03]	-0.06	[-0.22, 0.09]	-.05
Maternal Education[4]	-0.03	[-0.36, 0.31]	-0.01	[-0.18, 0.15]	.05
Maternal Marital Status[4]	-0.38	[-1.20, 0.44]	-0.08	[-0.25, 0.09]	-.13
					R^2 = .079
					95% CI[.00,.11]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

* indicates significant predictor of network density

[1]Motion is measured using mean relative framewise displacement

[2]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)

[3]Current life stress is measured using the Adolescent Life Events Scale

[4]Maternal variable at the child's birth

Supplemental Table 3.4

Node degree for ROIs that were not significantly associated with violence exposure or social deprivation using the Bonferroni-corrected significance threshold.

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	*r*	Fit
Left Amygdala Degree						
(Intercept)	2.87	[2.55, 3.20]				
Violence Exposure	-0.60	[-1.13, -0.07]	-0.19	[-0.36, -0.02]	-.04	
Social Deprivation	0.30	[-0.23, 0.82]	0.09	[-0.07, 0.26]	.07	
Motion [1]	6.04	[3.69, 8.40]	0.37	[0.23, 0.52]	.34	
						R^2 = .143
						95% CI[.05,.23]
Left Dorsal Anterior Cingulate Cortex Degree						
(Intercept)	6.47	[6.15, 6.79]				
Violence Exposure	-0.35	[-0.87, 0.17]	-0.12	[-0.30, 0.06]	-.08	
Social Deprivation	0.18	[-0.34, 0.69]	0.06	[-0.12, 0.24]	.01	
Motion [1]	0.61	[-1.70, 2.92]	0.04	[-0.11, 0.20]	.02	
						R^2 = .011
						95% CI[.00,.04]
Left Dorsolateral Prefrontal Cortex Degree						
(Intercept)	6.96	[6.53, 7.40]				
Violence Exposure	-0.62	[-1.33, 0.08]	-0.16	[-0.34, 0.02]	-.12	
Social Deprivation	0.34	[-0.36, 1.04]	0.09	[-0.09, 0.26]	-.00	
Motion [1]	-0.57	[-3.71, 2.56]	-0.03	[-0.18, 0.13]	-.05	
						R^2 = .020
						95% CI[.00,.06]
Left Anterior Insula Degree						
(Intercept)	6.02	[5.76, 6.28]				
Violence Exposure	-0.50	[-0.93, -0.08]	-0.21	[-0.39, -0.03]	-.12	
Social Deprivation	0.26	[-0.17, 0.68]	0.11	[-0.07, 0.28]	.02	
Motion [1]	1.38	[-0.52, 3.27]	0.11	[-0.04, 0.27]	.08	
						R^2 = .037
						95% CI[.00,.09]
Left Medial Temporal Gyrus Degree						
(Intercept)	6.03	[5.62, 6.45]				
Violence Exposure	-0.55	[-1.21, 0.12]	-0.15	[-0.32, 0.03]	-.06	
Social Deprivation	0.65	[-0.01, 1.31]	0.17	[-0.00, 0.35]	.09	
Motion [1]	-0.68	[-3.65, 2.29]	-0.04	[-0.19, 0.12]	-.04	
						R^2 = .026
						95% CI[.00,.07]
Left Posterior Cingulate Cortex Degree						
(Intercept)	8.40	[8.03, 8.76]				
Violence Exposure	-0.61	[-1.21, -0.01]	-0.18	[-0.36, -0.00]	-.11	
Social Deprivation	0.35	[-0.24, 0.94]	0.11	[-0.07, 0.28]	.02	
Motion [1]	0.78	[-1.87, 3.44]	0.05	[-0.11, 0.20]	.02	
						R^2 = .024

						95% CI[.00,.07]
Right Amygdala Degree						
(Intercept)	4.05	[3.80, 4.29]				
Violence Exposure	0.00	[-0.40, 0.40]	0.00	[-0.16, 0.16]	.13	
Social Deprivation	0.05	[-0.34, 0.45]	0.02	[-0.14, 0.18]	.11	
Motion [1]	5.38	[3.60, 7.16]	0.43	[0.29, 0.57]	.43	
						R^2 = .188
						95% CI[.08,.28]
Right Dorsal Anterior Cingulate Cortex Degree						
(Intercept)	7.39	[7.13, 7.64]				
Violence Exposure	-0.40	[-0.81, 0.01]	-0.17	[-0.35, 0.00]	-.08	
Social Deprivation	0.12	[-0.28, 0.53]	0.05	[-0.12, 0.23]	.02	
Motion [1]	3.01	[1.19, 4.83]	0.25	[0.10, 0.40]	.22	
						R^2 = .069
						95% CI[.00,.14]
Right Dorsolateral Prefrontal Cortex Degree						
(Intercept)	6.38	[6.08, 6.68]				
Violence Exposure	-0.39	[-0.87, 0.09]	-0.14	[-0.32, 0.03]	-.03	
Social Deprivation	0.40	[-0.08, 0.88]	0.15	[-0.03, 0.32]	.10	
Motion [1]	1.71	[-0.45, 3.86]	0.12	[-0.03, 0.28]	.12	
						R^2 = .033
						95% CI[.00,.09]
Right Inferior Parietal Lobule Degree						
(Intercept)	5.45	[5.15, 5.75]				
Violence Exposure	-0.49	[-0.97, 0.00]	-0.18	[-0.36, 0.00]	-.16	
Social Deprivation	0.06	[-0.42, 0.54]	0.02	[-0.15, 0.20]	-.07	
Motion [1]	0.43	[-1.74, 2.59]	0.03	[-0.12, 0.19]	-.01	
						R^2 = .026
						95% CI[.00,.08]
Right Medial Temporal Gyrus Degree						
(Intercept)	4.69	[4.42, 4.96]				
Violence Exposure	0.14	[-0.30, 0.57]	0.05	[-0.12, 0.22]	.18	
Social Deprivation	0.26	[-0.17, 0.69]	0.10	[-0.07, 0.27]	.19	
Motion [1]	3.46	[1.53, 5.39]	0.27	[0.12, 0.41]	.30	
						R^2 = .11
						95% CI[.03,.19]
Right Posterior Cingulate Cortex Degree						
(Intercept)	3.99	[3.67, 4.30]				
Violence Exposure	-0.29	[-0.80, 0.22]	-0.10	[-0.28, 0.08]	-.12	
Social Deprivation	-0.25	[-0.76, 0.25]	-0.09	[-0.26, 0.09]	-.11	
Motion [1]	1.88	[-0.39, 4.15]	0.13	[-0.03, 0.28]	.08	
						R^2 = .032
						95% CI[.00,.09]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

*Significant at a Bonferroni-corrected threshold of p = 0.004 (p=0.05/14 tests)

[1]Motion is measured using mean relative framewise displacement

Supplemental Table 3.5

Node density adjusted for covariates

Predictor	*b*	*b* 95% CI [LL, UL]	*beta*	*beta* 95% CI [LL, UL]	*r*	Fit
Left Inferior Parietal Lobule						
(Intercept)	9.14	[6.79, 11.49]				
Violence Exposure*	**-0.75**	**[-1.33, -0.17]**	**-0.23**	**[-0.42, -0.05]**	**-.17**	
Social Deprivation	0.36	[-0.20, 0.93]	0.11	[-0.06, 0.29]	.03	
Motion [1]	1.27	[-1.29, 3.84]	0.08	[-0.08, 0.24]	.03	
Gender	-0.04	[-0.09, 0.00]	-0.15	[-0.30, 0.01]	-.13	
Race_1[2]	-0.37	[-0.91, 0.18]	-0.13	[-0.32, 0.06]	-.09	
Race_2[2]	-0.71	[-1.36, -0.07]	-0.21	[-0.41, -0.02]	.17	
Pubertal Development	0.30	[-0.51, 1.11]	0.08	[-0.14, 0.29]	.03	
Current Life Stress[3]	0.85	[-0.15, 1.84]	0.18	[-0.03, 0.39]	-.14	
Maternal Education[4]	-0.09	[-0.35, 0.18]	-0.05	[-0.22, 0.11]	.00	
Maternal Marital Status[4]	-0.09	[-0.75, 0.57]	-0.02	[-0.19, 0.14]	-.07	
						R^2 = .117
						95% CI[.00,.16]
Right Insula						
(Intercept)	8.44	[6.83, 10.06]				
Violence Exposure*	**-0.64**	**[-1.04, -0.24]**	**-0.28**	**[-0.46, -0.11]**	**-.17**	
Social Deprivation	0.4	[0.06, 0.84]	0.20	[0.03, 0.37]	.06	
Motion [1]	1.28	[-0.48, 3.04]	0.11	[-0.04, 0.27]	.08	
Gender	-0.00	[-0.03, 0.03]	-0.02	[-0.17, 0.13]	-.05	
Race_1[2]	-0.36	[-0.73, 0.02]	-0.18	[-0.36, 0.01]	-.10	
Race_2[2]	-0.26	[-0.70, 0.19]	-0.11	[-0.30, 0.08]	-.01	
Pubertal Development	0.01	[-0.54, 0.57]	0.01	[-0.21, 0.22]	-.14	
Current Life Stress[3]	0.58	[-0.11, 1.27]	0.18	[-0.03, 0.39]	.21	
Maternal Education[4]	0.17	[-0.01, 0.36]	0.15	[-0.01, 0.31]	.17	
Maternal Marital Status[4]	0.15	[-0.30, 0.61]	0.05	[-0.11, 0.22]	-.07	
						R^2 = .149
						95% CI[.02,.20]

Note. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *beta* indicates the standardized regression weights. *r* represents the zero-order correlation. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

*Significant predictor of node degree adjusting for covariates.

[1]Motion is measured using mean relative framewise displacement

[2]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)

[3]Current life stress is measured using the Adolescent Life Events Scale

[4]Maternal variable at the child's birth

Supplemental Table 3.6

Logistic regression results adjusted for covariates

Predictor	*b*	*SE*	*Odds Ratio*	*Odds Ratio* 95% CI [LL, UL]
(Intercept)	-0.20	1.82	0.82	[0.02, 32.70]
Violence Exposure	**0.93**	**0.54**	**2.54**	**[0.92, 7.87]**
Social Deprivation	-0.38	0.47	0.68	[0.27, 1.77]
Motion [1]	**7.32**	**3.54**	**1516.93**	**[3.58, 4284250.00]**
Gender	0.57	0.50	1.77	[0.67, 4.77]
Race_1[2]	-0.58	0.71	0.56	[0.11, 2.02]
Race_2[2]	-1.13	0.80	0.32	[0.06, 1.46]
Pubertal Development	0.14	0.41	1.16	[0.50, 2.59]
Current Life Stress[3]	0.05	0.04	1.06	[0.98, 1.14]
Maternal Education[4]	0.07	0.20	1.07	[0.72, 1.61]
Maternal Marital Status[4]	0.05	0.49	1.05	[0.39, 2.72]

[1]Motion is measured using mean relative framewise displacement
[2]Dummy coded variables represented 3 category race variable (African American, Caucasian, Other)
[3]Current life stress is measured using the Adolescent Life Events Scale
[4]Maternal variable at the child's birth

Appendix 5: Chapter 3 Supplemental Data

Table 3.7
Supplemental Data

Model fit for each individual participant. Connections were not added to individual models when 2 out of the 4 fit indices met the following thresholds: RMSEA ≤0.05; SRMR ≤0.05; CFI ≥0.95; NNFI ≥0.95.

	χ^2	df	npar	RMSEA	SRMR	NNFI	CFI
1	337.464	215	219	0.0493	0.0498	0.9468	0.9698
2	460.4917	232	202	0.0649	0.0434	0.919	0.9503
3	442.6814	227	207	0.0637	0.0493	0.9176	0.9505
4	369.3483	230	204	0.0509	0.0497	0.9395	0.9632
5	356.2464	224	210	0.0502	0.0499	0.9424	0.9658
6	422.89	230	204	0.0599	0.0472	0.9367	0.9615
7	400.1407	228	206	0.0568	0.0469	0.9183	0.9507
8	395.4893	229	205	0.0557	0.0493	0.9342	0.9602
9	472.9349	228	206	0.0678	0.0485	0.9261	0.9554
10	349.8288	222	212	0.0496	0.0511	0.9436	0.9668
11	446.6057	229	205	0.0637	0.0492	0.9183	0.9505
12	505.731	218	216	0.0751	0.0491	0.9157	0.9514
13	417.9443	221	213	0.0617	0.0485	0.9263	0.9569
14	451.117	227	207	0.065	0.049	0.9287	0.9572
15	405.1967	229	205	0.0573	0.0486	0.9292	0.9571
16	377.3698	228	206	0.0529	0.0486	0.952	0.971

17	375.7425	200	234	0.0613	0.0485	0.9211	0.9583
18	418.1664	219	215	0.0623	0.0482	0.9139	0.9501
19	362.0274	218	216	0.0531	0.0485	0.9154	0.9512
20	489.3867	233	201	0.0686	0.0484	0.9203	0.9509
21	355.5109	230	204	0.0483	0.0547	0.9471	0.9678
22	381.252	231	203	0.0527	0.0492	0.9311	0.9579
23	359.1018	209	225	0.0554	0.0499	0.9229	0.9574
24	366.8721	232	202	0.0498	0.0526	0.9266	0.9549
25	422.4284	226	208	0.0609	0.0466	0.9175	0.9507
26	482.5072	234	200	0.0674	0.0461	0.9201	0.9505
27	357.8082	220	214	0.0517	0.0498	0.935	0.9621
28	441.1115	227	207	0.0635	0.0455	0.9184	0.951
29	415.5675	213	221	0.0638	0.0496	0.9249	0.9577
30	435.3762	225	209	0.0632	0.0469	0.9164	0.9502
31	459.8657	216	218	0.0695	0.049	0.9279	0.9588
32	404.2475	215	219	0.0613	0.0476	0.9241	0.9569
33	363.5178	222	212	0.0522	0.0446	0.9168	0.9511
34	394.4465	217	217	0.0591	0.0498	0.9381	0.9645
35	512.738	221	213	0.0751	0.0461	0.9164	0.9511
36	419.0826	227	207	0.0601	0.0496	0.9189	0.9513
37	391.933	225	209	0.0563	0.0438	0.9204	0.9526
38	355.2865	226	208	0.0494	0.053	0.9259	0.9557
39	431.6301	221	213	0.0638	0.0487	0.9246	0.9559
40	363.5577	232	202	0.0492	0.0502	0.9342	0.9596
41	418.9789	216	218	0.0634	0.0494	0.9146	0.9512
42	370.4016	221	213	0.0537	0.0496	0.9307	0.9595
43	357.6225	219	215	0.052	0.0497	0.9424	0.9666
44	364.269	218	216	0.0535	0.05	0.9169	0.9521
45	404.6379	213	221	0.062	0.048	0.9374	0.9647
46	481.5645	215	219	0.0728	0.0443	0.9148	0.9515
47	360.3157	225	209	0.0507	0.0493	0.9179	0.9511
48	435.7415	217	217	0.0656	0.045	0.9164	0.952
49	387.1817	225	209	0.0555	0.0459	0.9177	0.951

50	428.6833	229	205	0.061	0.0489	0.9352	0.9608
51	414.8962	233	201	0.0578	0.0424	0.9191	0.9501
52	395.9013	212	222	0.0609	0.0447	0.9125	0.9509
53	532.6246	221	213	0.0776	0.047	0.9148	0.9502
54	416.5936	221	213	0.0615	0.0434	0.9152	0.9504
55	434.5957	208	226	0.0682	0.0497	0.9131	0.9522
56	418.6393	236	198	0.0575	0.0485	0.9256	0.9535
57	351.4741	222	212	0.0499	0.0499	0.9385	0.9639
58	398.2812	236	198	0.0542	0.0476	0.9511	0.9695
59	355.4745	228	206	0.0489	0.0483	0.922	0.953
60	471.3257	231	203	0.0667	0.0487	0.9262	0.9549
61	343.2778	206	228	0.0534	0.0495	0.9509	0.9732
62	431.6664	210	224	0.0672	0.0408	0.915	0.9528
63	432.934	225	209	0.0628	0.0465	0.9186	0.9516
64	341.8362	219	215	0.049	0.0512	0.9526	0.9726
65	395.5717	226	208	0.0566	0.0472	0.9167	0.9502
66	445.6803	225	209	0.0647	0.05	0.9164	0.9502
67	481.0292	219	215	0.0715	0.0441	0.9148	0.9506
68	388.5093	217	217	0.0581	0.0445	0.9165	0.9521
69	385.9336	210	224	0.0598	0.0411	0.9142	0.9524
70	422.1714	227	207	0.0606	0.0487	0.9353	0.9612
71	372.7023	236	198	0.0498	0.0488	0.9441	0.9651
72	435.3817	223	211	0.0638	0.0498	0.9187	0.952
73	368.3488	216	218	0.0549	0.0491	0.9404	0.9659
74	379.8688	207	227	0.0597	0.0478	0.9274	0.9602
75	460.2895	214	220	0.0701	0.0496	0.9288	0.9597
76	524.4858	217	217	0.0778	0.0436	0.9139	0.9506
77	356.5818	230	204	0.0485	0.057	0.9345	0.9602
78	428.9118	232	202	0.0602	0.0498	0.9278	0.9557
79	424.6102	226	208	0.0613	0.0497	0.9229	0.9539
80	404.0907	216	218	0.061	0.0439	0.9171	0.9526
81	408.21	214	220	0.0623	0.0496	0.9218	0.9557
82	337.0235	216	218	0.0489	0.052	0.9491	0.9709

83	416.4031	218	216	0.0624	0.0437	0.9179	0.9527
84	473.7268	228	206	0.0679	0.0442	0.919	0.9511
85	388.1109	233	201	0.0533	0.0494	0.9487	0.9684
86	378.0401	221	213	0.0551	0.0492	0.9245	0.9559
87	472.1856	218	216	0.0706	0.0499	0.9155	0.9512
88	417.637	233	201	0.0582	0.0498	0.9384	0.962
89	383.6733	230	204	0.0534	0.0481	0.9293	0.957
90	397.4964	222	212	0.0581	0.0476	0.9174	0.9515
91	358.8544	229	205	0.0492	0.0538	0.9407	0.9641
92	515.0297	237	197	0.0708	0.0479	0.9228	0.9516
93	339.9077	222	212	0.0476	0.0624	0.9418	0.9658
94	408.5504	209	225	0.0639	0.041	0.9114	0.951
95	549.3469	226	208	0.0782	0.0464	0.9165	0.9501
96	376.8911	225	209	0.0537	0.0492	0.9351	0.9614
97	440.472	221	213	0.0651	0.0471	0.9172	0.9516
98	347.1012	222	212	0.0491	0.0489	0.948	0.9695
99	381.2555	224	210	0.0548	0.048	0.9167	0.9506
100	441.3668	230	204	0.0627	0.0499	0.925	0.9543
101	407.0288	221	213	0.06	0.0495	0.9222	0.9545
102	376.2155	216	218	0.0563	0.0464	0.9131	0.9503
103	366.2102	223	211	0.0524	0.0497	0.9416	0.9656
104	381.601	233	201	0.0522	0.0478	0.9242	0.9533
105	457.7367	235	199	0.0636	0.048	0.9206	0.9506
106	423.4817	214	220	0.0647	0.0492	0.9321	0.9616
107	433.4452	227	207	0.0623	0.0468	0.92	0.952
108	449.1592	228	206	0.0644	0.0496	0.9176	0.9503
109	477.1445	227	207	0.0686	0.0496	0.9192	0.9515
110	412.928	223	211	0.0603	0.0486	0.9183	0.9518
111	395.8876	234	200	0.0544	0.0491	0.9222	0.9519
112	509.8378	202	232	0.0807	0.0494	0.9094	0.9516
113	379.6696	224	210	0.0545	0.0485	0.9436	0.9666
114	393.9832	229	205	0.0555	0.0488	0.9277	0.9562
115	420.1125	231	203	0.0591	0.0487	0.9283	0.9562

116	461.147	216	218	0.0696	0.0436	0.9167	0.9524
117	435.54	218	216	0.0653	0.047	0.9172	0.9522
118	400.423	221	213	0.0589	0.0478	0.9294	0.9587
119	405.4778	214	220	0.0618	0.0485	0.9135	0.951
120	435.3515	229	205	0.0621	0.0492	0.9258	0.9551
121	467.4572	219	215	0.0696	0.0495	0.9162	0.9515
122	451.823	226	208	0.0653	0.0457	0.9193	0.9517
123	444.5608	213	221	0.0682	0.0471	0.9164	0.9529
124	360.8171	216	218	0.0535	0.0495	0.9209	0.9548
125	428.2471	220	214	0.0636	0.044	0.9173	0.9519
126	371.5123	213	221	0.0564	0.0456	0.928	0.9594
127	374.1864	232	202	0.0512	0.0453	0.9215	0.9518
128	410.7942	212	222	0.0633	0.0487	0.9291	0.9602
129	480.7249	220	214	0.0712	0.048	0.9154	0.9507
130	408.7257	224	210	0.0594	0.0437	0.9166	0.9506
131	473.8235	209	225	0.0736	0.0416	0.9114	0.951
132	344.6724	219	215	0.0495	0.0532	0.9407	0.9656
133	430.6263	230	204	0.0611	0.0464	0.9211	0.952
134	432.7567	231	203	0.0611	0.0487	0.9241	0.9536
135	511.666	240	194	0.0696	0.0456	0.9231	0.9512
136	435.3927	222	212	0.0641	0.0482	0.9176	0.9516
137	360.0409	216	218	0.0534	0.0489	0.9322	0.9613
138	390.6012	230	204	0.0546	0.0444	0.9307	0.9578
139	411.2425	233	201	0.0572	0.0482	0.9352	0.9601
140	441.3396	221	213	0.0653	0.049	0.9171	0.9515
141	423.1329	221	213	0.0625	0.0455	0.9182	0.9522
142	484.8995	218	216	0.0723	0.0422	0.9205	0.9542
143	424.9599	220	214	0.0631	0.0485	0.9283	0.9583
144	437.5468	233	201	0.0613	0.0492	0.9285	0.9559
145	395.4005	228	206	0.056	0.0491	0.9282	0.9567
146	430.6302	229	205	0.0613	0.0475	0.9205	0.9518
147	505.7797	230	204	0.0716	0.0463	0.9206	0.9517
148	380.6049	215	219	0.0574	0.0444	0.9155	0.9519

149	481.3911	226	208	0.0695	0.0499	0.9295	0.9579
150	361.4001	220	214	0.0524	0.0465	0.9516	0.9718
151	356.5355	227	207	0.0494	0.0513	0.9247	0.9548
152	395.1945	227	207	0.0563	0.0483	0.9302	0.9581
153	420.3603	223	211	0.0615	0.0499	0.9183	0.9518
154	395.4	226	208	0.0566	0.0472	0.9178	0.9508
155	422.053	233	201	0.0589	0.05	0.9216	0.9517
156	361.1325	224	210	0.0511	0.0497	0.9329	0.9602
157	443.7209	233	201	0.0622	0.0493	0.9204	0.9509
158	419.7464	215	219	0.0638	0.0496	0.9331	0.962
159	422.6659	226	208	0.061	0.0489	0.917	0.9504
160	431.8286	228	206	0.0618	0.047	0.9181	0.9506
161	456.3976	235	199	0.0635	0.0475	0.921	0.9509
162	348.7459	222	212	0.0494	0.0498	0.9431	0.9666
163	384.086	226	208	0.0547	0.0487	0.9244	0.9548
164	450.2674	222	212	0.0663	0.0499	0.928	0.9577
165	333.3395	216	218	0.0482	0.0516	0.932	0.9612
166	418.5673	229	205	0.0595	0.0492	0.9204	0.9518
167	398.5382	218	216	0.0595	0.0486	0.9348	0.9624
168	353.7505	225	209	0.0495	0.0509	0.9235	0.9545
169	415.5257	230	204	0.0587	0.0484	0.9317	0.9584
170	402.5641	223	211	0.0587	0.0462	0.9209	0.9534
171	384.9787	229	205	0.054	0.0478	0.939	0.963
172	403.9139	215	219	0.0613	0.0494	0.9161	0.9523
173	374.1356	226	208	0.0529	0.0491	0.9294	0.9578
174	390.1415	216	218	0.0587	0.0488	0.935	0.9628
175	434.5492	215	219	0.0661	0.048	0.9153	0.9518

Appendix 6: Data Processing and Analysis Code

Study 1

Information about and the scripts for the processing of the diffusion MRI data for this study are publicly available on GitHub (https://github.com/lgoetschius/ProbabilisticTractography_On_Flux). The data cleaning and statistical analysis code, including the graph visualizations, for this study are also publicly available on GitHub (https://github.com/lgoetschius/VE_SD_AmygdalaProbtrack).

Visualizations of the white matter data were created using FSL (v.5.0.9), ITK-SNAP (v.3.6.0), and ParaView (v.5.8.0) based on a tutorial in Madan (2015). Figure 2.2 was created by overlaying 3D masks of the target regions (bilateral Brodmann's Areas (BA) 10, 11, 25, 47) and a binarized and thresholded (threshold=1000 streamlines) mask of the averaged FDT_path image for all participants representing the probabilistic tractography streamlines for the left and right amygdala seed regions. Visualizations of the individual white mater streamlines (Supplemental Figure 2.1) was created using FSLeyes (0.31.2) (McCarthy, 2019) from the thresholded (threshold = 1000 streamlines) fdt_paths image with the right amygdala as the seed region for the individual participants with the highest and lowest violence exposure composite scores.

Study 2

Information about and the scripts for the processing of the resting state fMRI data for this study are publicly available on GitHub (https://github.com/lgoetschius/GIMME_Preprocessing) (Beltz et al., 2019). The data cleaning and statistical analysis code, including the graph

visualizations, for this study are also publicly available on GitHub (https://github.com/lgoetschius/VE_SD_GIMME_Analysis).

Visualizations of the group, subgroup, and example individual connectivity maps (Figure 3.1) were created using FSL (v.6.0.3), ITK-SNAP (v.3.6.0), and ParaView (v.5.8.0) based on a tutorial in Madan (2015). Figure 3.1 was created by overlaying 3D masks of the sphere masks for each node central coordinate (Supplemental Table 3.2). Arrows representing the group, subgroup, and example individual level paths were then added manually in Microsoft PowerPoint based on the output from the S-GIMME algorithm.

Study 3

The data cleaning, statistical analysis, and graph visualization code done using Mplus and R for this study are publicly available on GitHub (https://github.com/lgoetschius/VE_SD_SchoolConnectedness). Diagrams of the structural equation models (Figures 4.1, 4.2, 4.4) were created using the online Cacoo software (Nulab Inc.) based on the statistical output from Mplus.

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