DISSERTATION

EARLY NEIGHBORHOOD DISADVANTAGE AND RISK FOR INTERNALIZING PROBLEMS: UNDERLYING NEUROBIOLOGICAL MECHANISMS

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ABSTRACT

EARLY NEIGHBORHOOD DISADVANTAGE AND RISK FOR INTERNALIZING PROBLEMS: UNDERLYING NEUROBIOLOGICAL MECHANISMS

Early neighborhood-level socioeconomic disadvantage and air pollutant exposure predict an increased risk for mental health difficulties across the lifespan. However, the neurobiological mechanisms underlying these associations remain unclear. In this study, I investigated the associations of early neighborhood-level disadvantage and air pollutant exposure with neural network topology and stress-related outcomes in emerging adulthood. Participants were typically developing 18- to 19-year-olds ($N = 77$) from socioeconomically diverse backgrounds. They reported their home address history and underwent resting-state functional magnetic resonance imaging (rs-fMRI). Geocoding of participants' residential locations was used to compute neighborhood disadvantage and fine particulate matter 2.5 ($PM_{2.5}$) exposure. Global efficiency and clustering in the central executive (CEN), default mode (DMN), and salience networks (SN) were computed. Hair cortisol concentration (HCC) and perceived stress were measured for the full sample. For a subsample ($N = 40$), resting heart rate variability (HRV) was measured in a separate session after the MRI scan. Results showed that greater early neighborhood disadvantage was significantly associated with lower global efficiency in the DMN as well as higher clustering coefficient in the DMN and greater resting respiratory sinus arrhythmia (RSA) at a trend level. Greater early exposure to average $PM_{2.5}$ levels was associated with reduced global efficiency in the DMN. Greater number of days with exposure to high $PM_{2.5}$ levels was associated with reduced clustering coefficient and higher global efficiency in the CEN. Early

$PM_{2.5}$ exposure was not associated with stress physiology or perceived stress. These findings align with the notion that early-life neighborhood conditions may influence the organization of large-scale neural networks known to be associated with mental health. Findings from this study can be used to inform future prevention efforts and policies aimed at reducing health disparities and promoting resilience.

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Introduction

Early exposure to socioeconomic disadvantage is prevalent and increases risk for stress-related mental health difficulties, such as internalizing disorders (e.g., anxiety disorders, depression), across the lifespan (Alegría et al., 2014; Allen et al., 2014; Almeida et al., 2012; Elovainio et al., 2020; Leventhal, 2018; Reiss, 2013; Rudolph et al., 2014; Vine et al., 2012). Socioeconomic disadvantage reflects reduced access to social and economic resources. Although researchers have often focused on socioeconomic factors at the family level (e.g., household income, parental educational attainment), socioeconomic factors at the neighborhood level are also recognized as important. Neighborhood level environmental factors influence children's experiences and have been associated with mental health outcomes across many studies (Alegría et al., 2014; Leventhal, 2018). A more recent literature has focused on the associations between neighborhood disadvantage during childhood and adolescence (hereafter termed *early* neighborhood disadvantage) and neurobiological function, but many questions remain unanswered about these associations.

Early neighborhood disadvantage may alter the development and function of neural circuitry responsible for top-down control over emotional processes (Merz et al., 2024). These changes at the neural level may lead to altered functioning of physiological stress response systems, increasing the likelihood that stressors experienced later in life will amplify negative emotions (Chiang et al., 2015). Yet, the influences of early neighborhood disadvantage on neural function and stress physiology, and how neural function and stress response system function relate to each other, are not well understood. Thus, one main goal of this study was to investigate the associations of neighborhood disadvantage during childhood/adolescence with neural function and physiological and perceived stress in emerging adulthood (see Figure 1).

Neighborhood Disadvantage: Definition and Measurement

Although family socioeconomic status (SES) and neighborhood SES are interrelated, they are distinct constructs. While lower-SES families often reside in lower-SES neighborhoods, and higher-SES families in higher-SES neighborhoods, individual/family-level SES and neighborhood-level SES are only moderately correlated (Moss et al., 2021). Neighborhood disadvantage is associated with increased exposure to multiple forms of adversity (e.g., stressors). On average, families in more disadvantaged neighborhoods are exposed to greater environmental pollutants and toxins and higher crime rates and community violence, while having limited access to high-quality education and health resources (Crowder & Downey, 2010; Evans & Kantrowitz, 2002).

Broad composite measures of neighborhood disadvantage and adversity are commonly employed in research. For example, the Area Deprivation Index (ADI; Barrington et al., 2014; Kind & Buckingham, 2018) is a composite measure that includes a variety of U.S. Census indicators such as poverty prevalence, average adult educational attainment, property values, and household crowding (Kind & Buckingham, 2018). These composites tend to include indicators beyond the main socioeconomic indices (components of SES)—income, education, occupation. Another approach is to measure neighborhood concentrated poverty, which more specifically captures the extent to which economic resource scarcity is clustered within a neighborhood (Brooks-Gunn et al., 2000; Hyde et al., 2020).

Early Neighborhood Disadvantage and Risk for Internalizing Problems

Neighborhood disadvantage during childhood/adolescence has been repeatedly linked with an increased risk for internalizing disorders (Crump et al., 2011; Galea et al., 2007; Hackman et al., 2019; Joshi et al., 2017). *Early* exposure (i.e., during childhood and/or

adolescence) has been theorized and found to explain more variability in these outcomes compared to *later* exposure in adulthood (Evans, 2004; Knudsen, 2004; McManus et al., 2022; Teicher et al., 2022). Exposure to socioeconomic disadvantage during childhood or adolescence (especially early childhood and the peri-pubertal period), when the brain is rapidly developing, is theorized to have enduring effects because neurobiological systems are particularly sensitive to environmental influences during this time (Gee, 2022; Hertzman & Boyce, 2010; Knudsen, 2004). Although prior research has focused primarily on family-level socioeconomic factors, findings indicate overlapping yet distinct associations of family-level and neighborhood-level SES with mental health outcomes (E. Chen & Paterson, 2006; Murtha et al., 2021).

Internalizing Problems and Stress Response Systems

Increased perceived stress and difficulty with emotion regulation (e.g., downregulating negative affect in stressful situations) are transdiagnostic, underlying factors found across internalizing disorders (Aldao et al., 2016). Stress and emotion processing systems depend on two core physiological stress response systems – the hypothalamic-pituitary-adrenal (HPA) axis and autonomic nervous system (ANS) – which are both heavily implicated in internalizing disorders (Badanes et al., 2011; F. R. Chen et al., 2015; El-Sheikh et al., 2008). These stress response systems serve an adaptive purpose, mobilizing bodily resources to respond to a stressor or threat (Evans & Kim, 2007; Gunnar & Quevedo, 2007; Gunnar, 1992).

HPA axis. Stressors are detected and appraised at the neural level (e.g., frontolimbic circuitry), sending signals to modulate the HPA axis stress response (Pruessner et al., 2010). In response to an immediate threat, secretion of corticotropin-releasing hormone from the hypothalamus prompts the pituitary gland to release adrenocorticotropic hormone, which in turn prompts the adrenal glands to release glucocorticoids (e.g., cortisol in humans). An increase in

cortisol then triggers a negative feedback loop involving the hypothalamus and hippocampus, which leads to reduced circulation of cortisol (Sapolsky et al., 2000). Acute HPA axis responses are important and necessary for an adaptive response to a stressor or threat. HPA axis overactivity or underactivity have both been associated with increased risk for internalizing disorders (Koss & Gunnar, 2018; Staufenbiel et al., 2013).

Although cortisol can be measured in various ways (e.g., saliva, serum, urine), hair cortisol concentration (HCC) reflects the accumulation of cortisol over a span of months, providing insight into relatively long-term cortisol output (Stalder et al., 2017). Unlike saliva or serum, which capture cortisol levels at specific moments, HCC is less affected by acute environmental influences and time of day (Stalder & Kirschbaum, 2012).

ANS. The ANS consists of the complementary sympathetic and parasympathetic branches. It acts in concert with the HPA axis. The sympathetic branch of the ANS activates the "fight or flight" response, while the parasympathetic branch facilitates the "rest and digest" response, contributing to the restoration of physiological homeostasis after a stress response (Gibbons, 2019). Altered regulation of the ANS, often characterized by heightened sympathetic activity and/or diminished parasympathetic activity, has been implicated in anxiety disorders and depression (Carney et al., 2005; Friedman, 2007; Vazquez et al., 2016). Specifically, altered ANS regulation is associated with emotion regulation difficulties (Stifter et al., 2011), which increase risk for internalizing disorders.

Heart rate variability (HRV), a reflection of ANS functioning, refers to the measurement of beat-to-beat changes in heart rate. It has been theorized to be an indicator of self-regulatory capacity or top-down emotion regulation (Holzman & Bridgett, 2017). HRV reflects the dynamic balance between sympathetic and parasympathetic activity, with higher HRV indicating greater

flexibility in activating and modulating response to environmental stressors (Berntson et al., 1997).

Respiratory sinus arrhythmia (RSA), specifically, is often interpreted as an index of parasympathetic (vagal) activity, reflecting the body's capacity to flexibly regulate physiological arousal by decreasing activation following stress and restoring homeostasis after environmental demands (Beauchaine et al., 2007; Beauchaine & Thayer, 2015; Thayer et al., 2022). RSA indexes the degree to which heart rate changes relative to respiratory cycles. RSA, and closely related high-frequency HRV (HF-HRV), both reflect vagal cardiac influence.

Lower baseline HRV, indexed by measures such as RSA, is often interpreted as reflecting diminished parasympathetic regulation, which has been linked to poorer emotion regulation capacities and greater difficulties flexibly engaging with social and emotional demands (Porges, 1995, 2001). Studies suggest that lower resting HRV predicts heightened risk for internalizing disorders (Hinnant & El-Sheikh, 2009; Koch et al., 2019; Rottenberg et al., 2007), presumably related to its association with altered emotion regulation (Beauchaine et al., 2007). Conversely, higher baseline HRV has been linked to adaptive emotion regulation, greater resilience, and reduced risk for internalizing symptoms (Beauchaine & Thayer, 2015). In the conceptual model guiding this study (see Figure 1), early neighborhood disadvantage was expected to lead to altered regulation of both the HPA axis and ANS, ultimately increasing risk for internalizing difficulties.

Neighborhood Disadvantage and Multisystemic Stress Physiology

Exposure to early adversity (e.g., early life stress) has long been found to alter regulation of the HPA axis (McEwen, 2007; McEwen & Gianaros, 2010). For example, early life adversity (e.g., family-level poverty, child maltreatment) has been frequently associated with higher or

lower levels of cortisol (Koss & Gunnar, 2018). Yet, few studies have focused on neighborhood-level environmental factors, such as neighborhood disadvantage, in relation to multisystemic stress physiology.

Hair Cortisol

Research on composite measures of neighborhood disadvantage (e.g., ADI) and HCC has yielded mixed results (Gunnar et al., 2022; Ip et al., 2024; Malanchini et al., 2021; Verheyen, Remy, Govarts, et al., 2021; Vliegenthart et al., 2016). More specifically, neighborhood disadvantage has been associated with higher HCC in children in some studies (Gunnar et al., 2022) but with lower HCC in children in other studies (Ip et al., 2024; Vliegenthart et al., 2016). These studies have variably focused on children across a wide age range (Vliegenthart et al., 2016), low-income, Mexican-American adolescents (Ip et al., 2024), and adolescent boys (Verheyen, Remy, Bijnens, et al., 2021). Some studies have also failed to find significant associations between neighborhood disadvantage and HCC in children and adolescents (Malanchini et al., 2021; Vaghri et al., 2013; Verheyen, Remy, Govarts, et al., 2021). Research is needed to further understand these inconsistent results and elucidate how neighborhood disadvantage during childhood/adolescence may influence HCC during emerging adulthood.

Heart-Rate Variability (HRV)

Higher self-reported childhood neighborhood quality (physical conditions, safety, socioeconomic measures) was associated with greater decreases in HRV metrics in response to a social stressor in young women (Lin et al., 2024). In another study, neighborhood-level education and employment were unrelated to HRV, yet housing burden was positively associated with HRV during a social exclusion task (Ugarte et al., 2022). Thus, more research is needed on the associations between neighborhood disadvantage and HRV.

Neighborhood Disadvantage and Neural Network Functional Connectivity

Neighborhood disadvantage during childhood and adolescence may influence brain function in regulatory neural networks that play roles in top-down control over physiological stress response systems (Chiang et al., 2015). These effects on neural function may partially explain associations of early neighborhood disadvantage with altered functioning of physiological stress response systems and difficulty with emotion regulation. Resting-state functional MRI (rs-fMRI) captures neural activation and connectivity in the absence of task-related demands. Resting-state networks (RSNs) are synchronous patterns of brain activity that occur during rest. RSNs correspond to different cognitive functions and provide insights into the brain's intrinsic organization (Grayson & Fair, 2017; Seeley et al., 2007).

The salience network (SN), central executive network (CEN), and default mode network (DMN) are implicated in internalizing disorders (Menon, 2011). The CEN, a fronto-parietal network with primary hubs in the dorsolateral prefrontal cortex (dlPFC) and posterior parietal cortex (Vincent et al., 2008), is predominantly known for its role in executive control processes (Menon, 2011; Seeley et al., 2007). The DMN is activated when the brain is “at rest” and is anchored in the medial PFC, posterior cingulate cortex, and medial temporal cortex (Grayson & Fair, 2017; Greicius et al., 2003; Raichle, 2015). The DMN supports social cognition, including thinking about others’ states of mind, self-referential processing, and introspection (Sherman et al., 2014). The SN plays a key role in integrating sensory, emotional, and cognitive information, serving as an interface between the DMN and CEN to regulate internal mental processes with externally driven cognitive and emotional responses. It has main hubs in the anterior insula and dorsal anterior cingulate (Seeley et al., 2007). Consistent with the Triple Network Model of

Psychopathology (Menon, 2011), altered connectivity within and between these neural networks has been associated with depression and anxiety (Liston et al., 2014; Menon, 2011).

Prior work has demonstrated associations between neighborhood disadvantage and altered functional connectivity across these networks and other circuits (Ip et al., 2022; Rakesh, Seguin, et al., 2021; Rakesh, Zalesky, et al., 2021; Ramphal et al., 2020). Most studies have largely been limited to traditional measures of connectivity strength, such as functional connectivity. However, the human brain operates as a non-random network with a "small-world" architecture, where densely clustered local connections and long-range connections allow for efficient communication between nodes (brain regions) (Latora & Marchiori, 2001; Watts & Strogatz, 1998). Graph theory methods, which are used to measure neural network topology, offer strengths compared to traditional functional connectivity methods. Measures such as global efficiency and clustering coefficient allow for insight into both specialized processing and high-level integration across brain regions within a modular framework. This approach provides a more comprehensive picture of brain network organization by evaluating not only the coactivations of individual connections but the overall structural organization, integration and segregation of networks, and efficiency of information transfer across the network (Bullmore & Sporns, 2009). Thus, examining the topology of large-scale neural networks may provide an indicator of how environmental exposures uniquely influence brain organization.

Graph theory models a network as a graph consisting of nodes representing brain regions and edges representing functional connections between regions. Global efficiency is a measure of the overall efficiency of information transfer across an entire network. Global efficiency at a node (brain region) is defined as the average of inverse-distances between the node and all other nodes in the same graph, or network, with distance defined as the number of connections

between two nodes (Achard & Bullmore, 2007; Latora & Marchiori, 2001). Higher global efficiency suggests a more interconnected brain network for faster and more efficient communication between brain regions. The clustering coefficient quantifies the extent to which nodes in a graph tend to cluster together. It provides insight into the local structure of the network by indicating the degree of interconnectedness, or density of connections, among neighboring nodes (Achard & Bullmore, 2007; Latora & Marchiori, 2001). A higher clustering coefficient suggests stronger local communication and specialized processing within the network.

Few studies have examined associations between neighborhood disadvantage and topological properties of the CEN, DMN, and SN. In one study, children and adolescents (6–17 years of age) in more distressed communities showed lower efficiency and integrity of core SN nodes (Gellci et al., 2019). This study used a broad, composite measure of neighborhood disadvantage that reflected the amount of poverty, low educational attainment, unemployment, and unoccupied housing in the neighborhood. In another study, neighborhood SES, a composite of poverty rates, income, education, employment, family characteristics, and population density, moderated age-related changes in DMN segregation among 8- to 22-year-olds (Tooley et al., 2020a). Youth in high-SES neighborhoods showed increased clustering in the DMN with age, suggesting that greater neighborhood SES associates with stronger DMN maturation (Tooley et al., 2020a). Similarly, neighborhood *poverty* has been shown to moderate age-related changes in brain network organization. Adolescents from high-poverty neighborhoods were found to exhibit delayed network segregation across both the entire brain and in the CEN, DMN, and SN (Michael et al., 2023). Most of these studies, with the exception of Michael et al. (2023), have utilized broad, composite measures of neighborhood-level disadvantage and adversity. Given the

strengths of graph theory methods, additional research is needed that uses these methods to shed light on how neighborhood factors may influence network topology. Additionally, the samples in most previous studies have had wide age ranges; a narrower age range in early adulthood may help elucidate these associations and rule out residual confounding due to age. In this study, I examined the associations of neighborhood disadvantage with global efficiency and clustering coefficient in the SN, CEN, and DMN, as altered topology of these neural networks may partially explain socioeconomic disparities in internalizing disorders.

Neural and Stress Response System Function

At the neurobiological level, stress physiology and brain function are overlapping and closely interconnected, with each signaling to the other and neural function providing top-down control over the HPA axis and ANS (McEwen, 2006). Prior research has suggested that differences in neural structure and function may link adversity exposure with altered functioning of stress response systems (McEwen & Gianaros, 2010). For example, early life adversity has been linked to changes in the structure and function of corticolimbic circuitry crucial to stress reactivity and regulation (Chiang et al., 2015; Demir-Lira et al., 2016; Hanson et al., 2015; Kaiser et al., 2018; Merz et al., 2018). Furthermore, corticolimbic activation has been shown to mediate the association between adversity exposure and cortisol levels and reactivity in adults (Kaiser et al., 2018; Seo et al., 2019). Early life adversity may alter neural function in the CEN, SN, and DMN, leading to altered physiological stress responses in adulthood (Kaiser et al., 2018).

Function of the CEN, SN, and DMN is thought to be associated with the regulation of physiological stress response systems such as the HPA axis and ANS. Foundational models of the central autonomic network (CAN; Benarroch, 1993) described an integrated cortical—

subcortical—brainstem system responsible for coordinating sympathetic and parasympathetic responses. The CEN, SN, and DMN share nodes with the CAN. For example, a meta-analysis of ANS-related neuroimaging studies identified consistent autonomic regulatory activity in the anterior insula, midcingulate cortex, amygdala, posterior cingulate cortex, and ventromedial prefrontal cortex, regions that closely match the hubs of the SN, CEN, and DMN (Beissner et al., 2013). Their findings further demonstrated that task-positive networks (e.g., SN and CEN) may send signals to modulate sympathetic activation, whereas parasympathetic modulation is more strongly linked to DMN regions (Beissner et al., 2013). This overlap suggests that higher-order neural networks may provide cortical input that influences autonomic regulation (Thayer et al., 2012). As such, in the current study, I examined the potentially mediating role of neural network function in linking early neighborhood disadvantage with altered physiological stress response system functioning and perceived stress in emerging adulthood.

Neighborhood Air Pollutant Exposure

Neighborhood disadvantage may exert its effects on neurodevelopment and mental health in part through its associations with the quality of the physical environment including increased exposure to air pollutants (Basner et al., 2011; Beane Freeman et al., 2017; Evans et al., 1998; Hajat et al., 2013; Zare Sakhvidi et al., 2018). Exposure to air pollutants disproportionally impacts socioeconomically disadvantaged communities (Arregi et al., 2024; Clark et al., 2014; Cserbik et al., 2020; Hajat et al., 2013; Ugarte et al., 2022) and is associated with increased depressive and anxiety symptoms (Kioumourtzoglou et al., 2017; M. C. Power et al., 2015; Shin et al., 2018).

Among these pollutants, fine particulate matter 2.5 ($PM_{2.5}$) consists of airborne particles with diameters ≤ 2.5 μm and can be particularly concerning for neurocognitive and mental health

outcomes due to its small aerodynamic size, which allows these particles to penetrate deep into the lungs, enter the bloodstream, and cross the blood-brain-barrier to trigger systemic oxidative stress and neuroinflammation (W. Li et al., 2022). These mechanisms have been described as part of a broader "lung–brain axis," through which respiratory exposures can influence central nervous system functioning (Bajinka et al., 2022). $PM_{2.5}$ is produced by combustion-related sources like vehicle traffic, industrial activity, and heating and cooking (U.S. Environmental Protection Agency, 2025), as well as increasingly frequent and severe wildfire events, which can generate exceptionally high concentrations of $PM_{2.5}$ over large geographic areas (Goss et al., 2020; McClure & Jaffe, 2018). Increased exposure to $PM_{2.5}$ specifically has been associated with increased internalizing symptoms (Bakolis et al., 2021; Buoli et al., 2018; Kioumourtzoglou et al., 2017; M. C. Power et al., 2015; Xie et al., 2023).

Emerging longitudinal evidence also suggests that early life $PM_{2.5}$ exposure may be linked with later emotional difficulties (Mazahir et al., 2025). Higher $PM_{2.5}$ exposure during early childhood is associated with higher emotional problems and externalizing behavior (Ahmed et al., 2022; Tokuda et al., 2023), and higher $PM_{2.5}$ exposure during middle childhood is also linked with later internalizing symptoms in adolescence (Roberts et al., 2019). Although, other studies have found no associations between early $PM_{2.5}$ exposure and later emotional problems (Bradley et al., 2024; Harris et al., 2016). Together, these findings suggest that there is some evidence of sensitive developmental windows during which exposure to $PM_{2.5}$ may shape vulnerability to internalizing psychopathology, but the associations are still unclear.

Many of these studies have focused on chronic $PM_{2.5}$ exposure, defined as long-term average exposure levels across months or years. However, even acute high exposures of $PM_{2.5}$, short-term spikes in $PM_{2.5}$ (e.g., daily levels exceeding regulatory thresholds), have been linked

to increased risk for mental health disorders. For example, greater number of days with $PM_{2.5}$ levels above United States Environmental Protection Agency (EPA) standards was associated with higher internalizing symptoms in children that lasted a year after exposure (Smolker et al., 2024). More research should be conducted on the associations between high acute exposure days and risk for mental health problems.

Air Pollutant Exposure and Stress Physiology

Animal models have found consistent evidence that air pollutants, such as particulate matter (PM), affect the functioning of the HPA axis and ANS, leading to the repeated release of stress hormones and shaping stress response systems (Snow et al., 2018). Only a few studies have addressed whether neighborhood air pollutant exposure is associated with stress physiology in humans as measured by HCC and HRV. In one study, $PM_{2.5}$ exposure was not associated with HCC in adolescence, yet higher levels of other air pollutants (NO_2 and PM_{10}) were significantly associated with higher HCC (Verheyen, Remy, Bijnens, et al., 2021). Chronic exposure to air pollutants $PM_{2.5}$ has been linked to lower HRV in response to a social stressor (Miller et al., 2019; Ugarte et al., 2022) and at rest (Park et al., 2010). Specifically, chronic exposure to $PM_{2.5}$ was associated with a slight decrease in another HRV measure, root-mean square differences of successive normal-to-normal intervals (RMSSD), at rest in adults (Adhikari et al., 2016; Park et al., 2010). Higher $PM_{2.5}$ exposure in adolescents was associated with lower high frequency (HF) HRV in response to a social stress task (Miller et al., 2019) and lower RSA during rest (Ugarte et al., 2022). High acute exposures to $PM_{2.5}$ related to wildfire smoke events have also been associated with a lower HF-HRV and RMSSD compared to a control group (Lankaputhra et al., 2023).

Further, acute induced exposure to PM associated with wood smoke showed reduced HRV immediately following exposure compared to a control group (Unosson et al., 2013). Both chronic and high acute exposures may disrupt neurobiological systems that regulate stress and autonomic functioning, and as such, considering both types of exposure are essential for understanding how environmental pollutants influence trajectories of brain development and stress regulation, as each may uniquely and interactively shape vulnerability to emotional and physiological difficulties later in life.

Air Pollutant Exposure and Neural Function

Exposure to outdoor air pollutants has also been associated with reduced connectivity in RSNs such as the DMN and reduced network segregation (Glaubitz et al., 2022; Pérez-Crespo et al., 2022; Pujol et al., 2016). In a large-scale study, chronic neighborhood air pollutant exposure was found to be associated with altered resting-state functional connectivity in children, with different pollutants associating with RSN connectivity in differing directions (Cotter et al., 2023). Specifically, higher $PM_{2.5}$ exposure in childhood was linked to greater inter-network functional connectivity development over time between the DMN and SN and the DMN and CEN, demonstrating a less-mature pattern of network segregation (Cotter et al., 2023)

In a longitudinal study, higher exposures to $PM_{2.5}$ from birth to 3 years were associated with higher within-network resting-state functional connectivity across task-positive and task-negative networks in preadolescence (Pérez-Crespo et al., 2022). There is a need for more research on neighborhood $PM_{2.5}$ exposure during childhood/adolescence and its influences on stress physiology and neural network function during emerging adulthood.

Summary

The aim of this study was to elucidate the neurobiological mechanisms through which neighborhood disadvantage and air pollutant ($PM_{2.5}$) exposure during childhood/adolescence may increase risk for internalizing disorders in early adulthood (Figure 1). I first examined how early neighborhood disadvantage and air pollutant exposure may influence stress physiology (HCC, HRV) and perceived stress in emerging adulthood. I then examined the extent to which altered functional organization of the CEN, DMN, and SN may partially explain these associations. Together, altered function of these neural networks and stress response systems may underlie the association of early neighborhood disadvantage and air pollutant exposure with increased internalizing symptoms in emerging adulthood.

Current Study

The goals of this research were to investigate the associations of early neighborhood disadvantage and $PM_{2.5}$ exposure with (1) physiological and perceived stress and (2) DMN, CEN, and SN functional organization in emerging adults. A third goal was to examine whether DMN, CEN, and SN organization mediates the associations of these neighborhood factors with physiological and perceived stress (see Figure 1). Participants were typically developing 18- to 19-year-olds ($N = 77$), primarily students attending a university in the western U.S. Geographic information systems (GIS) data were used to compute geocoded indicators of Census-derived neighborhood factors based on participants' home addresses during childhood/adolescence. Resting-state fMRI data were acquired and used to compute DMN, CEN, and SN organization and efficiency.

Specifically, I addressed the following main research questions: 1) How do neighborhood disadvantage and $PM_{2.5}$ exposure during childhood/adolescence relate to physiological stress (HCC, resting HRV) and perceived stress in emerging adulthood? 2) How do neighborhood

disadvantage and $PM_{2.5}$ exposure relate to the organization and efficiency of the CEN, DMN, and SN in emerging adulthood? 3) Does CEN, DMN, or SN organization and efficiency mediate associations of early neighborhood disadvantage and $PM_{2.5}$ exposure with physiological and perceived stress in emerging adulthood?

Hypotheses - Neighborhood Factors and Stress-Related Outcomes

1. Higher childhood neighborhood disadvantage will be associated with higher HCC, lower resting HRV, and higher perceived stress in emerging adulthood, independent of family-level socioeconomic context.
2. Higher childhood neighborhood $PM_{2.5}$ exposure will be associated with higher HCC, lower resting HRV, and higher perceived stress in emerging adulthood, independent of neighborhood- and family-level socioeconomic context.

Hypotheses - Neighborhood Factors and Neural Network Efficiency

3. Higher childhood neighborhood disadvantage will be associated with lower efficiency and organization in the CEN, DMN, and SN in emerging adulthood, independent of family-level socioeconomic context.
4. Higher childhood neighborhood $PM_{2.5}$ exposure will be associated with lower efficiency and organization in the CEN, DMN, and SN in emerging adulthood, independent of neighborhood- and family-level socioeconomic context.

Hypotheses - Neural Network Efficiency and Stress-Related Outcomes

5. Lower CEN, SN, and DMN organization and efficiency will be associated with higher HCC, lower HRV, and higher perceived stress in emerging adulthood.
6. CEN, SN, and DMN organization and efficiency will mediate associations of neighborhood disadvantage and $PM_{2.5}$ exposure with stress-related outcomes.

Methods

Participants

Participants in this study were recruited in a medium-sized city in the western U.S. by posting flyers in local neighborhoods and utilizing a university psychology department subject pool. Potential participants completed an online form to determine their eligibility to participate in the study. To be eligible for the study, individuals needed to be 18 or 19 years of age and fluent in English. Exclusionary criteria included a past or current diagnosis of a neurological disorder, autism spectrum disorder, bipolar disorder, schizophrenia, or intellectual disability; a history of head trauma; contraindications for MRI (e.g., claustrophobia, presence of ferromagnetic metal in or on the body); and being the product of a multiple birth (e.g., twin, triplet).

Participants were enrolled to create a sample representative of a full range of socioeconomic backgrounds. To ensure diversity in socioeconomic backgrounds, eligible individuals were invited to participate if their parents' highest level of educational attainment fit into a bin for which the participant quota was not met. Of the 89 participants who completed a first session, 26 participants (29.2%) have parents with a high school diploma or GED as their highest level of education; 13 participants (14.6%) have parents with some college experience (but no degree); 21 participants (23.6%) have parents with a bachelor's degree; and 29 (32.6%) have parents with a master's or doctoral degree

Participants ranged from 18 to 19 years of age (55.1% female). They were 62.3% White, non-Hispanic/Latine, 24.6% Hispanic/Latine, with the remaining participants identifying as Black or African American (7.2%), Asian American (4.3%), American Indian or Alaska Native

(1.4%), or Native Hawaiian or other Pacific Islander (1.4%) (see Table 1 for full sample characteristics).

Procedure

Eligible individuals were invited to participate in the first session during the years of 2022-2026. During this session, they first provided written informed consent. They then completed a set of questionnaires, provided a small hair sample, and were screened for MRI eligibility. Participants who were eligible for the MRI portion of the study were invited to participate in the second session. During the second session, which was intended to be within one month of the first session, participants underwent MRI scanning. Three or more months after their second session, participants were invited to complete a third, follow-up session. During the third session, HRV data was collected. All participants received monetary compensation, and if they were CSU students in introductory psychology courses, they also received class credit. The CSU Institutional Review Board (IRB) approved the protocol for this study. Appendix Figure A1 provides a summary of the procedures for this study, including the timing of each session and data collected in each session.

Home Address Data Collection and Classification

During session 1, each participant provided their primary home address "growing up" (hereafter, called their "childhood" home address). In addition, participants reported their current (permanent) home address and the date they began residing at that location. Participants who completed session 3 also provided a detailed residential history, including full address records with corresponding dates. Based on these data, we tried to determine when each participant lived at their primary childhood home address. Of the 89 participants with usable address data, we were able to determine this with certainty for 67 (75.3%) participants. The primary childhood

home address matched the current address for 43 total participants. Twenty-four participants provided complete home address histories in session 3, allowing us to determine when they lived at their primary childhood home address. We did not know with certainty when the remaining 22 participants (24.7%) lived at their primary childhood home address. Of the 77 participants with usable neuroimaging data, 58 (75.3%) had high-confidence residential histories for the childhood period. Residential history certainty was included as a binary variable (0 = low certainty, 1 = high certainty) and included as a covariate in significant models in follow-up sensitivity analyses. As another robustness check, significant models were re-estimated after removing participants with low certainty.

Definition of the 5-Year Residential Exposure Window

For neighborhood exposure data, we identified an individualized 5-year exposure window for each participant based on their residential history. The end of the exposure window was anchored to the end of when they reported living at their childhood home address, defined as either (a) the final date the participant was known to reside at their childhood home address if before 18 years old or (b) their 18th birthday, whichever occurred first. The exposure window therefore spanned the five calendar years immediately preceding this end date (i.e., from end date – 5 years through the end date). All residential addresses during this period were geocoded and matched to U.S. Census block groups for each calendar year.

Geocoding

Geocoding was used to assign geographical coordinates to childhood addresses for each year as defined above. The methodology for extraction was based loosely on the Adolescent Brain Cognitive Development (ABCD) study protocol (Fan et al., 2021), with the code extensively updated with more appropriate census data and updated software packages. To

calculate neighborhood disadvantage, each address was assigned a geoid which is a unique identifier used to represent a specific geographic area (e.g., census block group) using the 'tigris' package in R (Walker, 2016). The variables used to calculate neighborhood disadvantage were extracted by using a join operation to link the census metrics to each geocoded address for each year.

Neighborhood Disadvantage

Rather than assigning participants to a single fixed American Community Survey (ACS) multi-year release, neighborhood socioeconomic conditions were estimated using year-specific ACS data corresponding to each year of the participant's 5-year exposure window tract (Beaghen & Weidman, 2008; U.S. Census Bureau, 2020). For each calendar year in the window, block-group-level indicators of educational attainment, household income, and poverty rates were extracted for the participant's residential location. These specific metrics were adjusted from previous literature (Malanchini et al., 2021), and narrowed down to more specific socioeconomic metrics.

Annual values were then averaged across the five years to produce participant-level estimates of neighborhood socioeconomic conditions during their individual childhood period. These annual indicators were standardized via z-score calculation and combined to form a composite neighborhood socioeconomic disadvantage score, with higher values reflecting greater socioeconomic disadvantage.

Air Pollutant Exposure

Fine particulate matter 2.5 ($PM_{2.5}$) in the air is measured in micrograms per cubic meter ($\mu g/m^3$) and is often used as one indicator of air pollution levels. To estimate neighborhood exposure to fine particulate matter ($PM_{2.5}$), we utilized publicly available air quality data from

the U.S. Environmental Protection Agency (EPA) and the AirMonitor R package. Each home address was matched to the nearest $PM_{2.5}$ monitor within a 200 km radius for each of the same 5 year period. If no monitors were found within this threshold, the participant was excluded from exposure assignment (n = 1). Further, any participant that was over 50 km from their nearest monitor was excluded in follow up sensitivity analyses (n = 6). Distance from the nearest monitor was also included as a covariate in analyses with $PM_{2.5}$.

Using the RAQSAPI R package (Mccrowey et al., 2023), we then accessed the EPA's Air Quality System (AQS) daily summary data for $PM_{2.5}$ for each year. We then calculated two summary metrics per participant: the average $PM_{2.5}$ level across the year to capture chronic pollutant exposure, and the total number of days with $PM_{2.5}$ concentrations exceeding the US Environmental Protection Agency (US EPA) National Ambient Air Quality Standard for mean 24-h $PM_{2.5}$ exposure of >35 μg/m³ (www.epa.gov) (Smolker et al., 2024) to capture major pollutant exposure events. The average $PM_{2.5}$ levels were then averaged across the 5-year period, and the high exposure days were summed for a total across the 5-year period.

Parental Education

Parental education was used as a measure of family-level socioeconomic context. Participants reported the number of years of education completed by each of their parents. Educational attainment was averaged across parents for each participant. Parental education was used as a covariate in analyses.

Imaging Data

Image Acquisition

Resting-state fMRI data were acquired using a 3 Tesla (3T) Siemens Skyra scanner with a 64-channel head coil at the CSU Translational Medicine Institute. A resting-state BOLD fMRI

sequence is acquired with the following parameters: voxel size = 2.5 x 2.5 x 2.5 mm; field of view (FoV) = 210 mm; repetition time (TR) = 900 ms; echo time (TE) = 38.0 ms; flip angle = 52°; 60 slices; slice thickness of 2.5 mm; 667 volumes. One run was acquired. Participants were instructed to fixate on a white cross against a black background while remaining motionless during the scan.

Image Preprocessing

A standard preprocessing pipeline was conducted using fMRIPrep 20.2.7 (Esteban et al., 2017, 2019). First, neuroimaging data were converted to the Brain Imaging Data Structure (BIDS) format using HeuDiConv version 0.9.0 (Halchenko et al., 2024) and dcm2niix version v1.0.20240202 (X. Li et al., 2016). T1-weighted (T1w) anatomical images underwent intensity non-uniformity (INU) correction using N4ITK and were subsequently skull-stripped. Tissue segmentation was performed to classify gray matter, white matter, and cerebrospinal fluid (CSF). Each participant's T1w image was then spatially normalized to the MNI152NLin2009cAsymtemplate using ANTs (Advanced Normalization Tools).

For each participant, resting-state fMRI data underwent motion correction. Head motion was estimated and corrected using mcflirt (FSL 5.0.9). Distortion correction was performed using field maps. Functional images underwent coregistration where they were aligned to their corresponding T1w image using boundary-based registration (BBR) and subsequently transformed into MNI152NLin2009cAsym space. To mitigate physiological and motion-related noise in post-processing, confound regressors were extracted using CompCor (Behzadi et al., 2007), motion outliers were identified using a framewise displacement (FD) threshold of 0.5 mm, and ICA-AROMA was applied for additional denoising, reducing motion-related artifacts in functional images (Pruim et al., 2015). To improve the signal-to-noise ratio, gaussian spatial

smoothing was performed with a full-width at half-maximum (FWHM) of 6mm. Seventy-seven of the 79 participants who have been scanned to date have usable rs-fMRI data (see Appendix Figure A2).

Neural Network Organization and Efficiency

First, 13 nodes of the CEN were created via a 100 node parcellation scheme (Schaefer et al., 2018) (see Appendix Table A1). Utilizing the CONN toolbox (Nieto-Castanon, 2020a), a correlation matrix was computed from the time courses of all nodes within the CEN. The correlation matrix was then thresholded to create a binary association matrix, where connections between nodes (edges) were retained if their correlation coefficients exceeded a predefined threshold, set at a recommended level of .15, demonstrating significant functional associations (Nieto-Castanon, 2020b). Graph theory metrics, including measures of network topology (e.g., global efficiency and clustering coefficient) were computed to characterize the organization and efficiency of the CEN. These steps were repeated for the DMN and SN. The 24 nodes of the DMN and 12 nodes of the SN are provided in Appendix Table A1 (Schaefer et al., 2018).

Hair Cortisol Concentration

Hair samples were collected from participants with hair at least 3 cm long. Samples were cut from the back of the head along the midline as close to the scalp as possible. Samples were then stored in aluminum foil at room temperature in dry and dark conditions. For assay, the samples were then sent to our collaborator Dr. Kestutis Bendinskas's lab at the State University of New York at Oswego (SUNY-Oswego). In the lab, the samples were dissolved in an assay diluent and analyzed using commercial cortisol enzyme-linked immunosorbent assay (ELISA). ELISA is a widely used assay method for quantifying cortisol levels in biological samples, providing sensitive and reliable measurements of HCC (Bendinskas et al., 2024; Slominski et al.,

2015). HCC (pg/mg) in the 3 cm of hair closest to the scalp was calculated through this method and utilized in analyses.

Thus far, 70 participants have usable HCC data. During session 1, five participants had hair that was too short for sample collection, and three participants declined to give a sample (see Appendix Figure A3). HCC data for eight participants with values above Q3+1.5*IQR (Q3 = third quartile; IQR = interquartile range) (Bendinskas et al., 2024) was excluded from analyses.

Certain variables (e.g., hair care practices, corticosteroid medication use) may act as confounds in analyses of HCC (Bendinskas et al., 2024; Gray et al., 2018; Merz et al., 2019; Stalder et al., 2017). Thus, participants were asked to report on how frequently they washed their hair, if they used hair dye, and if they took any steroid medications in the 3 months prior to hair sample collection. HCC was not significantly associated with hair washing frequency (p = .70), race/ethnicity (p = .11), or corticosteroid medication use (p = .34). HCC was associated with use of hair dye (p = .037) and contraceptive use (p = .043), so sensitivity analyses would have been conducted if there were significant models.

Heart-Rate Variability (HRV)

Data Acquisition

HRV was measured across resting, stress-induction, and recovery conditions. Data acquisition was performed using a four-channel BioPac MP36AR system, Acqknowledge software, and a three-lead ECG configuration, with signals recorded at a sampling rate of 500 Hz for ECG and 100 Hz for respiratory activity (Mahesh et al., 2022). Disposable electrodes were placed on the participant's torso (below the right the bilateral collarbones and at the bottom of the right ribcage) following site preparation with alcohol swabs and Nuprep gel to ensure low

impedance. A respiratory belt was also secured at the sternum to allow monitoring of breathing patterns and account for respiratory influences on HRV.

The HRV protocol included three conditions: (1) a 10-minute resting baseline, during which participants were instructed to sit still and avoid specific thoughts; (2) a 5-minute mental arithmetic stress task designed to elicit physiological arousal, in which participants performed math problems under evaluative feedback from an experimenter (Kirschbaum et al., 1992); and (3) a 10-minute post-task recovery period. These three conditions allow for the thorough assessment of tonic HRV (baseline) and phasic HRV (changes during stress and recovery) (Laborde et al., 2017). The current study will focus on baseline measures.

Data Cleaning and Processing

ECG data are processed using AcqKnowledge software (version 5.0; Almond, 2017) following standardized preprocessing steps. First, a bandpass filter (0.5–35 Hz) was applied to smooth the HR waveform and enhance signal quality. Human ECG QRS complex boundaries were then located, with only QRS markers retained for HRV computation. Peaks were verified and, if necessary, manually corrected to ensure accurate peak detection. The QRS peak event marks were adjusted to align with the true peak locations, and the heart rate (BPM) was computed, creating a separate channel for further verification. A visual search for anomalies was performed to identify missing QRS peaks, excessive noise, or other irregularities (Laborde et al., 2017). Sections of unusable data (e.g., due to motion artifacts, electrical interference, or poor electrode contact) were excluded from further analysis. If a missing QRS peak was detected, its correct placement was estimated based on surrounding heartbeats. The R-R interval plot was examined to identify potential mislabeling of peaks or outlier intervals.

For HRV computation, time-domain (e.g., RMSSD) and frequency-domain (e.g., RSA) measures were extracted using multi-epoch HRV analysis. Data were segmented into 60-second epochs, and epochs containing artifacts or noise were removed. The final HRV statistics were averaged across the entire recording. RSA values were checked for validity (expected range: 3–9), with problematic epochs flagged for exclusion from analyses.

Higher HRV indicates better stress regulatory capacity, particularly under resting conditions (Beauchaine & Thayer, 2015). RMSSD is a time domain measures that calculates the short-term, beat-to-beat variability, while RSA is a frequency domain measures shows the rhythmic pattern of that variability linked to breathing (Stein et al., 1994). Both indices largely capture vagal tone and are highly correlated, offering complementary variables for analyzing autonomic regulation.

HRV Subsample

There were no significant differences between the HRV subsample and the full sample in parental education, $t(50.96) = .68$, $p = .50$, neighborhood disadvantage rate, $t(48.97) = –.42$, $p = .68$, or average $PM_{2.5}$ exposure, $t(45.96) = –.43$, $p = .67$. These results suggest that participants with HRV data are comparable to the overall sample in parental education, neighborhood socioeconomic conditions, and $PM_{2.5}$ exposure levels.

Perceived Stress

Perceived stress was measured using the Perceived Stress Scale (PSS; Cohen et al., 1983), a widely used instrument for assessing the degree to which situations in a participant's life are subjectively viewed as stressful. The PSS consists of 14 items that ask participants to reflect on their feelings and thoughts over the last month. Participants rated each item on a 5-point Likert scale ranging from 0 (*never*) to 4 (*very often*). Seven items were reverse-scored to account

for positively worded questions. Total scores were computed by summing all item scores, with higher scores indicating greater perceived stress. Possible scores range from 0 to 56. This scale was chosen for its reliability and sensitivity in capturing subjective stressful experiences (S. Cohen, 1988). The PSS demonstrated strong internal consistency in the current sample ($\alpha = .85$).

Statistical Analyses

All analyses were conducted in R (version 4.1.1). Data for each variable was examined to determine whether it was normally distributed (e.g., assessed for skewness). Analyses are described below for each set of hypotheses.

Neighborhood Factors and Neural Network Properties

Multiple linear regression analyses were used to examine associations of neighborhood disadvantage and both metrics of $PM_{2.5}$ exposure (average $PM_{2.5}$ exposure and number of days with $PM_{2.5}$ concentrations >35 $\mu g/m^3$) (independent variables) with CEN, SN, and DMN clustering coefficient and global efficiency (dependent variables). Separate regression models were run for the CEN, SN, and DMN. Covariates included age, sex, lifetime number of residential moves, and parental education. Head motion during the resting-state fMRI scan (mean FD) was also included as a covariate in these analyses because head motion can influence resting-state metrics (Power et al., 2012; Satterthwaite et al., 2012; Van Dijk et al., 2012). Additionally, analyses of neighborhood $PM_{2.5}$ exposure controlled for neighborhood disadvantage and the distance from the EPA monitor. Although sessions 1 and 2 were meant to be within one month of each other, the time between these sessions varied across participants (see Table 1). Thus, time between sessions 1 and 2 was initially included in these regression models as a covariate. However, the time between sessions 1 and 2 did not contribute significantly to the final estimates, so it was removed from the models for parsimony.

Neighborhood Factors and Stress-Related Outcomes

Multiple linear regression analyses were also used to examine associations of neighborhood-level disadvantage and both metrics of $PM_{2.5}$ exposure (independent variables) with HRV, HCC, and perceived stress (dependent variables). Separate regression models were conducted for HRV, HCC, and perceived stress. Covariates included age, sex, parental education, and total lifetime residential moves. Again, analyses of neighborhood $PM_{2.5}$ exposure controlled for neighborhood disadvantage and the distance from the EPA monitor. Analyses of HRV also initially included the time between sessions 1 and 3 as a covariate. Similar to above, time between sessions 1 and 3 was not significant and was removed from the final regression models for parsimony.

Neural Network Properties and Stress-Related Outcomes

Multiple linear regression analyses were used to examine associations of CEN, SN, and DMN clustering coefficient and global efficiency (independent variables) with HRV, HCC, and perceived stress (dependent variables). Separate models were run for each network and each outcome pair. Analyses of HCC, HRV, and perceived stress included age, sex, parental education, and head motion.

Mediation Analyses

To investigate the potential mediating role of neural network efficiency and organization in the association between neighborhood factors and HCC/HRV/perceived stress, subsequent mediation analyses were conducted if the multiple linear regression analyses demonstrated significance of the *a* and *b* paths (see Figure 1). However, these criteria were not met, so no mediation analyses were conducted.

All analyses use a $p < .05$ significance threshold (two-tailed), and trend-level significance was reported when $.05 < p < .10$. Effect sizes were provided for significant results. Specifically, partial eta squared (η_p^2) were presented, with values of .01, .06, and .14 indicating small, medium, and large effects, respectively (J. Cohen, 2013; Richardson, 2011). We did not use multilevel modeling because there is insufficient clustering of participants within neighborhoods to warrant this approach (Raudenbush & Bryk, 2002). In the current sample, all participants were the only individual in their census block (see also Anderson et al., 2014).

Results

Descriptive Statistics

Descriptive statistics for the main study variables are provided in Table 2, and zero-order correlations between the main study variables are provided in Table 3.

Parental Education, Neighborhood Disadvantage, and Neighborhood $PM_{2.5}$ Exposure

As expected, parental education was inversely associated with neighborhood disadvantage ($\beta = -.31$, $p < .001$, $\eta_p^2 = .14$). Parental education was not associated with number of days with $PM_{2.5}$ exposure > 35 μg/m³ ($p = .68$) or average $PM_{2.5}$ exposure ($p = .88$). Neighborhood disadvantage was positively associated with average $PM_{2.5}$ exposure ($\beta = .35$, $p = .044$, $\eta_p^2 = .05$) and number of days with $PM_{2.5}$ exposure > 35 μg/m³ at a trend level ($\beta = .18$, $p = .091$, $\eta_p^2 = .04$) (Figure 2).

Neighborhood Factors and Stress-Related Outcomes

Neighborhood Disadvantage

Neighborhood disadvantage was positively associated with baseline RSA at a trend level ($\beta = .44$, $p = .054$, $\eta_p^2 = .08$). Neighborhood disadvantage was not associated with baseline RMSSD, HCC, or perceived stress.

Air Pollutant Exposure

Neither average $PM_{2.5}$ exposure or number of days with $PM_{2.5}$ exposure > 35 μg/m³ were associated with HCC, HRV metrics, or perceived stress with or without including neighborhood disadvantage as a covariate.

Neighborhood Factors and Neural Network Properties

Neighborhood Disadvantage

Higher neighborhood disadvantage was associated with lower global efficiency in the DMN (β = -.27, p = .032, η_p^2 = .06) (Figure 3) and higher clustering coefficient in the DMN at a trend level (β = .24, p = .063, η_p^2 = .05). Neighborhood disadvantage was not associated with SN or CEN organization with or without including parental education as a covariate.

Air Pollutant Exposure

Higher number of days with $PM_{2.5}$ exposure > 35 μg/m³ was associated with higher global efficiency in the CEN (β = .39, p < .001, η_p^2 = .19) and reduced clustering coefficient in the CEN (β = -.27, p = .028, η_p^2 = .09) while controlling for neighborhood disadvantage (Figure 4). $PM_{2.5}$ exposure was not associated with SN or DMN properties.

Neural Network Properties and Stress-Related Outcomes

Hair Cortisol Concentration

Network organization and efficiency were not associated with HCC.

Heart-Rate Variability

Higher clustering coefficient in the SN was associated with higher baseline RSA (β = .39, p = .043, η_p^2 = .12), but not baseline RMSSD. Neither organization or efficiency in the DMN and CEN were associated with HRV metrics.

Perceived Stress

Higher clustering coefficient in the DMN was associated with lower perceived stress (β = -.25, p = .040, η_p^2 = .06). Neither organization or efficiency in the CEN and SN were associated with perceived stress.

Sensitivity Analyses

Distance from Air Monitor

The results were re-run after excluding participants whose home address was located more than 50 km from the nearest $PM_{2.5}$ monitor (n = 6) to account for error introduced due to monitor distances. Additionally, analyses were repeated using the continuous distance to the nearest monitor as a covariate to adjust for variability in exposure precision. All results remained consistent.

Address Confidence (Residential History Certainty)

The significant analyses were re-run after additionally controlling for residential history certainty (0 = low certainty, 1 = high certainty) to account for potential misclassification of exposure due to incomplete address histories. All results remained consistent.

Discussion

The results from this study provide insights into associations of neighborhood disadvantage and air pollutant exposure during childhood/adolescence with CEN, DMN, and SN functional organization and stress-related outcomes in emerging adulthood. Neighborhood disadvantage was significantly linked to lower global efficiency in the DMN and marginally higher clustering in the DMN. Greater exposure to high-$PM_{2.5}$-pollution days was linked to higher global efficiency and reduced clustering in the CEN. Neighborhood disadvantage and $PM_{2.5}$ exposure were not significantly associated with stress-related outcomes. Together, these findings suggest that different neighborhood exposures may exert distinct influences on neural

network organization, highlighting both potential adaptive and risk-related patterns. In the following sections, I discuss the full set of results in detail.

Associations Among Neighborhood and Family Factors

Consistent with prior research, neighborhood-level disadvantage was significantly and inversely associated with family-level parental educational attainment, suggesting that socioeconomic disadvantaged families tend to reside in more disadvantaged neighborhoods (Moss et al., 2021). Lower parental education was not associated with higher average $PM_{2.5}$ exposure in this sample, in contrast to prior studies that have reported greater pollutant exposure among lower-SES families (Cserbik et al., 2020; Ugarte et al., 2022).

Higher neighborhood-level disadvantage was associated with higher average $PM_{2.5}$ exposure, consistent with prior literature (Boing et al., 2022; Hajat et al., 2015; Ugarte et al., 2022). Further, neighborhood-level disadvantage was marginally associated with more frequent $PM_{2.5}$ exceedances of the 35 $\mu g/m^3$ threshold suggesting residents of more disadvantaged neighborhoods may experience greater exposure to acute spikes in air pollution, such as those associated with wildfires. This result aligns with previous research that has found that socioeconomically disadvantaged communities face disproportionate exposure to wildfire smoke, a major and growing source of $PM_{2.5}$ pollution across the U.S. (Dennin et al., 2025).

However, high $PM_{2.5}$ exposure was associated with neighborhood disadvantage at a trend level. This may be due, in part, to the fact that wildfire smoke events can affect broad geographic regions regardless of neighborhood socioeconomic status. At the same time, structural factors may shape the disparities in health outcomes associated with these exposures (Reid et al., 2023), as disadvantaged households may have fewer resources such as air filtration systems or climate-controlled housing that mitigate exposure. Therefore, studying $PM_{2.5}$ exposure and its health

implications is important across all communities, while also recognizing that socioeconomic disadvantage may exacerbate vulnerability to the negative health effects of these exposures.

Neighborhood Disadvantage and Neural Network Functional Organization

Higher neighborhood disadvantage was significantly associated with lower global efficiency in the DMN and marginally associated with higher clustering coefficient in the DMN. These findings suggest reduced long-range within-network integration and possibly denser local connections within the DMN. Thus, exposure to greater neighborhood disadvantage during developmental periods (childhood, adolescence) may be linked to re-organization of the DMN in emerging adulthood, with more fragmented long-range connections along with a stronger locally clustered topology. These results align with prior research suggesting that reorganization of large-scale networks can occur in response to environmental adversity (Hermans et al., 2011; Y. Zhang et al., 2020), such as that associated with neighborhood disadvantage. Studies of neighborhood disadvantage and rsFC in the DMN have found reduced connectivity with greater disadvantage (Rakesh, Seguin, et al., 2021; Rakesh, Zalesky, et al., 2021). One study additionally found alterations in connectivity patterns associated with greater neighborhood disadvantage (Ip et al., 2024), providing concurring evidence that reorganization, not just reduction in connectivity, can occur under neighborhood disadvantage. The marginal increase in clustering coefficient observed in this study suggests greater local connectivity within the DMN, which is not consistently reported in previous rsFC studies and may reflect a more complex pattern of network reorganization rather than uniform disruption.

Prior work utilizing graph theory metrics has shown that neighborhood socioeconomic context moderates developmental changes in DMN organization, with youth from more advantaged environments exhibiting greater increases in network segregation (e.g., clustering)

across development (Tooley et al., 2020b). Similarly, Michael et al. (2023) found that youth from higher-poverty neighborhoods exhibit delayed segregation across large-scale networks, including the DMN, suggesting altered or slower maturation of functional network organization. These results are broadly consistent with the present findings in suggesting that neighborhood disadvantage is associated with altered DMN network organization; however, whereas prior work has primarily suggested differences in network segregation and reduced clustering alongside neighborhood disadvantage, the current findings highlight alterations in long-range integration and reflect diminished coordination across distributed DMN regions even in the presence of preserved or increased local connectivity.

The DMN is a task-negative network that supports internally directed cognitive processes including self-monitoring and emotion processing (Menon, 2011; Raichle, 2015). Lower global efficiency may suggest a reduced capacity for long-range information integration across distributed DMN nodes. However, given the broader literature on altered DMN functional connectivity in chronic stress and psychopathology (Soares et al., 2013; Whitfield-Gabrieli & Ford, 2012), the behavioral and long-term implications of this reorganization of the DMN remain unclear. Greater neighborhood disadvantage exposure was associated with reductions in global efficiency *with* marginal increases in local connectivity. This dissociation could suggest that neighborhood disadvantage exerts a compensatory reorganization of DMN topological properties.

Higher neighborhood disadvantage was significantly associated with lower global efficiency in the DMN and marginally associated with higher clustering coefficient in the DMN, independent of parental education, suggesting that neighborhood-level factors contribute uniquely to neural network organization beyond family-level socioeconomic context.

Air Pollutant ($PM_{2.5}$) Exposure and Neural Network Functional Organization

Greater exposure to high air pollutant days (days with high $PM_{2.5}$ exposure) was linked with lower clustering coefficient in the CEN and higher global efficiency in the CEN. These results could suggest less efficient and less integrated functional architecture of the CEN. High $PM_{2.5}$ exposure events, such as those associated with wildfires, may weaken local connectivity among CEN regions that support higher-order cognitive skills and executive control (Reineberg & Banich, 2016). Clustering coefficient is a measure of local segregation and increases with age throughout adolescence and young adulthood as a function of maturation (Wu et al., 2013). Neighborhood disadvantage has been associated with reduced maturation related to clustering and network segregation in other cognitive-related networks (Michael et al., 2023). Reduced clustering in the CEN in the current study may therefore indicate diminished network maturation in the CEN, potentially compromising cognitive control and adaptive regulation.

While reduced clustering in the CEN in the current study may indicate diminished local network specialization, potentially reflecting disrupted maturation of executive control systems, there is also an increase in global efficiency in the CEN associated with exposure to high air pollutant days. The observed increase in global efficiency may reflect a reorganization in which long-range connections are strengthened to offset reduced local cohesion, suggesting a decoupling of integration and segregation processes that are typically coordinated during normative development (Fair et al., 2009; Rubinov & Sporns, 2010). This pattern could represent a less specialized network configuration, even with strong efficiency, that may have implications for cognitive control and emotion regulation.

Given that atypical organization of the CEN has been linked to risk for psychopathology (Menon, 2011), these results may be relevant for understanding affective and behavioral

functioning. Because of the socioeconomic inequities in pollutant exposure (Dennin et al., 2025), these results could point to a multi-level environmental inequality: structural disadvantage increases exposure to pollutants like $PM_{2.5}$, which in turn may lead to neural network re-organization, potentially perpetuating health disparities across generations.

In this study, $PM_{2.5}$ exposure was not associated with either perceived stress or indices of physiological stress. This suggests that $PM_{2.5}$ exposure within the range observed, may not directly influence stress-related outcomes, or that the effects operate through more long-term or indirect pathways. Historical evidence from a major wildfire smoke event suggests that wildfire smoke exposure led to immediate health consequences, but behavioral consequences emerged later in life (Meier et al., 2025). Similarly, the current findings may reflect an early neural change that may signal a latent risk for future affective or cognitive difficulties. Other longitudinal research has suggested that early disruptions in DMN and CEN maturation can predict later internalizing symptoms and poorer executive functioning (Whitfield-Gabrieli et al., 2020), providing evidence that neurobiological changes can occur years before behavioral symptoms. In this sample, the effects of chronic and acute pollutant exposure may be detectable, possibly before clinical change, as evidenced by null findings for stress-related outcomes.

Wildfire smoke has been linked to accelerated brain aging and heightened risk for neurodegenerative processes later in life (B. Zhang et al., 2023). Chronic or acute exposure to $PM_{2.5}$, particularly the complex and toxic mixture found in wildfire smoke, may exert these effects through neuroinflammatory and oxidative stress pathways that accumulate over time (Fagundes et al., 2015; Schuller & Montrose, 2020). The present pattern of altered CEN organization reflect how exposure-related oxidative stress and neuroinflammation may disrupt more immediate large-scale network connectivity, impairing communication among distributed

cortical hubs critical for cognitive and emotional regulation. However, chronic exposure and large exposures to $PM_{2.5}$ and wildfire smoke during this sensitive period of brain network maturation may lead to enduring changes in neural architecture that represent longitudinal trajectories of risk. These results may suggest early disruptions in network integration and segregation that confer vulnerability to the observed neurodegenerative outcomes later in adulthood or during the aging process.

Neural Network Functional Organization and Stress-Related Outcomes

Greater clustering coefficient (local organization) within the SN was associated with higher resting RSA, suggesting that more cohesive SN processing may support stronger parasympathetic regulation at rest. This aligns with previous work suggesting that there is an integrated cortical—subcortical system that is implicated in coordinating autonomic responses (Beissner et al., 2013). As a network that detects salient stimuli and facilitates switching between large-scale networks (Goulden et al., 2014), the SN may contribute to efficient autonomic regulation through coordinated state shifts. Increased local clustering within the SN may reflect more cohesive local processing, supporting more efficient detection and modulation of environmental demands. However, SN organization was not associated with neighborhood disadvantage or $PM_{2.5}$ exposure. It is possible that other environmental factors at the neighborhood or family level explain variability in SN functional organization among emerging adults.

Neighborhood Disadvantage and Stress-Related Outcomes

Neighborhood disadvantage was not significantly associated with any of the stress-related outcomes. Yet, it was marginally associated with higher baseline RSA. This result could suggest that individuals from more disadvantaged neighborhoods may exhibit greater parasympathetic

regulation and adaptive physiological flexibility (Beauchaine & Thayer, 2015). From an adaptive calibration perspective, heightened baseline vagal response may reflect a context-specific tuning of the stress response system in response to early environmental adversity related to neighborhood disadvantage (Del Giudice et al., 2011). Greater resting RSA among individuals from disadvantaged neighborhoods suggests a physiological adaptation. This adaptation may buffer against the negative effects of chronic stress exposure during early adulthood, though more research is needed to understand whether this association is replicable and if these adaptations occur at the expense of long-term physiological or affective well-being.

Neighborhood disadvantage during childhood/adolescence was not associated with HCC in emerging adults in this study. This result may align with prior studies that have reported null associations between neighborhood disadvantage and HCC (Malanchini et al., 2021; Vaghri et al., 2013; Verheyen, Remy, Govarts, et al., 2021). The absence of an association with HCC could be attributable to multiple factors. For example, it may suggest that the effects of neighborhood disadvantage on HCC vary depending on moderating factors. Evidence also indicates that long-term cortisol hypersecretion is most pronounced when stress exposure is ongoing at the time of assessment (Stalder et al., 2017). As such, HCC could also be more sensitive to proximal or ongoing stressors rather than earlier neighborhood disadvantage.

Higher neighborhood disadvantage was also not associated with perceived stress in this study. This null result could be attributable to multiple factors. For instance, it could reflect adaptive coping mechanisms, resilience, or differing baseline expectations related to stress. Those from more disadvantaged neighborhoods may have developed stress regulation strategies, leading to a diminished perception of stress. In addition, perceived stress was measured using a single self-report questionnaire in this study.

Future Directions

The present findings highlight the importance of investigating adaptive brain organization among youth exposed to socioeconomic disadvantage. The reorganization of DMN efficiency may represent a neural adaptation to context, which could relate to risk or resilience processes. Future research should examine which specific environmental or proximal factors contribute to this adaptive reorganization. For example, supportive caregiving and community resources may facilitate the development of resilient neural and physiological profiles (Olsson et al., 2003; Reich et al., 2010). Identifying specific environmental and protective influences will clarify the reasons behind variability in individual trajectories following early exposure to socioeconomic disadvantage.

Strengths and Limitations

Strengths of this study include its novelty, as many of the associations examined as part of the proposed model (see Figure 1) have not been investigated before. This study addresses a gap in understanding how early neighborhood-level environmental factors influence objective measures of brain function and stress physiology. In addition, applying graph theoretical techniques to fMRI data is a rigorous and advanced analytic approach that allows for a more comprehensive characterization of brain organization compared to traditional functional connectivity analyses. Only a relatively small set of previous studies has collected fMRI and multisystemic stress physiology data at the same time, making the current study a valuable contribution to the literature addressing the associations between neural function and the function of physiological stress response systems. Further, cortisol and other measures of HPA axis function have been studied far more often than HRV or other measures of ANS function. The

sample is also socioeconomically diverse, reducing the impact of restricted range, which often characterizes convenience studies of SES.

There are also limitations to the study to consider when interpreting the results. First, due to the correlational design of the study, causal inferences were not possible. Second, there are challenges with contextualizing the geocoded data. The years when participants lived at their childhood home addresses were variable, which could create challenges in understanding the developmental timing of exposure to environmental adversity. To address this issue, in the third session of the study, participants were asked to provide their entire address history. This data was used to more comprehensively assess neighborhood exposures across different periods of development, but that data was not present for the entire sample. Third, the sample grew up in states across the United States, but no participants have reported addresses within the top 10 of the largest cities in the United States (e.g., New York, Los Angeles, Chicago). There is some data to support differential associations of higher SES neighborhoods experiencing higher pollutant exposure in larger cities (Hajat et al., 2015). Similarly, rurality or urbanicity of participants' residential locations was not explicitly modeled in the analyses. Air pollutant exposure and neighborhood socioeconomic characteristics can differ between rural and urban environments (Kilpatrick et al., 2024). Thus, results may not be generalizable to individuals who grew up in urban and metropolitan hubs. Further research could increase geographic representation by recruiting participants from large metropolitan areas to enhance generalizability. Fourth, wildfire smoke exposure was not directly measured in this study. Although wildfire smoke can substantially elevate ambient $PM_{2.5}$ concentrations during smoke events, $PM_{2.5}$ measurements obtained from air quality monitoring systems represent total particulate matter from multiple sources, meaning that wildfire-specific contributions cannot be

fully isolated (Liu et al., 2016). Fifth, these analyses were conducted during a time when the study was ongoing. Thus, final results may be different when analyses include the full sample. Sixth, although recruited to be socioeconomically diverse, the sample consisted primarily of individuals currently enrolled in a four-year university. Participants in this study were primarily students in their first year of college. Thus, the results may not generalize to other populations. Replication in larger, independent samples is needed to confirm and expand on these results.

Conclusions

This study provides novel evidence linking childhood neighborhood disadvantage and air pollutant exposure to differences in brain network organization and stress physiology in emerging adulthood. Neighborhood disadvantage was associated with reduced efficiency and marginally greater clustering in the DMN, suggesting potential adaptive neural and physiological calibration. Greater exposure to air pollutants ($PM_{2.5}$) was associated with reduced clustering and greater global efficiency of the CEN, which is responsible for executive function skills and goal-directed behavior. Together, these findings highlight that early neighborhood disadvantage and air pollutant exposure may have distinct associations with resting-state network functional organization, potentially reflecting adaptation to context and related to risk for internalizing disorders. Understanding these mechanisms can inform applied strategies to promote resilience in youth exposed to early adversity, including community investments, environmental protections, and equitable access to supportive educational and health resources. Ensuring opportunity-rich, low-pollution environments during childhood is key to supporting children's development, building a foundation for lifelong mental health and well-being.

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Table 1. Descriptive statistics for sample characteristics ($N = 77$)

	M	***SD***	***Range***
Age (years)	18.92	.57	18.02 - 19.97
Parental education (years)	15.43	3.97	6 - 23
Time between sessions 1 and 2 (days)	20.58	21.46	0 - 109
Time between sessions 1 and 3 (days)	811.3	217.49	357 - 1113
	%	***n***	
Sex (female)	55.1	38	--
Race/ethnicity			
White, non-Hispanic/Latine	62.3	43	--
Black or African American, non-Hispanic/Latine	7.2	5	--
Asian American, non-Hispanic/Latine	4.3	2	--
American Indian or Alaska Native, non-Hispanic/Latine	1.4	1	--
Native Hawaiian or other Pacific Islander, non-Hispanic/Latine	1.4	1	--
Hispanic/Latine	24.6	17	--

Note. 77 participants currently have complete and usable home address, rs-fMRI, and perceived stress data.

--, not applicable

Table 2. Descriptive statistics for main study variables

Variable	**N**	**M**	**SD**	**Min**	**Max**
Neighborhood disadvantage	89	0.07	0.08	0.0	0.5
Average $PM_{2.5}$ (μg/m³)	89	7.24	1.38	3.5	10.8
Days > 35 μg/m³ ($PM_{2.5}$)	89	4.94	4.51	0.0	15.0
Hair cortisol concentration (pg/mg)	70	6.95	3.32	2.6	16.6
Baseline RSA (ms²)	40	6.37	0.97	4.2	8.2
Baseline RMSSD (ms)	40	42.02	23.62	13.8	122.5
Perceived stress scale score	90	25.51	6.98	11.0	46.0
CEN clustering coefficient	77	0.58	0.18	0.0	1.0
CEN global efficiency	77	0.22	0.04	0.2	0.4
SN clustering coefficient	77	0.47	0.22	0.0	0.9
SN global efficiency	77	0.24	0.04	0.2	0.3
DMN clustering coefficient	77	0.53	0.09	0.3	0.7
DMN global efficiency	77	0.36	0.06	0.2	0.4

Table includes descriptive statistics for all primary study variables, including sample size (**N**), mean (**M**), standard deviation (**SD**), minimum (**Min**), and maximum (**Max**) values. **$PM_{2.5}$** = particulate matter 2.5 concentration. **RMSSD** = root mean square of successive differences. **RSA** = respiratory sinus arrhythmia. **CEN** = central executive network, **DMN** = default mode network, **SN** = salience network. Sample sizes vary across variables due to missing data across physiological, questionnaire, and neuroimaging modalities.

Table 3. Zero-order correlations for main study variables

Variable	Parental Education	Neighborhood Disadvantage	High $PM_{2.5}$ Days	Average $PM_{2.5}$	HCC	RMSSD	RSA	PSS	CEN GE	CEN CC	DMN GE	DMN CC	SN GE	SN CC
Parental Education	—													
Neighborhood Disadvantage	-0.49***	—												
High $PM_{2.5}$ Days	0.03	0.10	—											
Average $PM_{2.5}$	-0.05	0.23*	0.39***	—										
HCC	0.01	0.06	0.04	-0.08	—									
RMSSD	0.09	0.16	0.00	-0.04	0.48**	—								
RSA	0.20	0.24	0.18	0.10	0.30	0.76***	—							
PSS	-0.13	-0.02	-0.20	0.01	0.10	-0.11	-0.18	—						
CEN GE	0.05	0.02	0.41***	0.21	0.09	-0.10	-0.02	0.10	—					
CEN CC	-0.07	0.08	-0.28*	-0.10	-0.03	0.12	-0.03	-0.12	-0.76***	—				
DMN GE	0.22	-0.20	-0.15	-0.20	0.13	-0.04	0.13	0.16	0.09	-0.10	—			
DMN CC	-0.09	0.24*	0.14	0.15	0.04	0.30	0.15	-0.23*	0.01	0.02	-0.63***	—		
SN GE	-0.05	0.05	0.03	-0.02	-0.04	-0.01	-0.05	-0.04	-0.27*	0.25*	-0.06	-0.15	—	
SN CC	-0.01	0.07	0.10	0.01	0.08	0.00	0.26	-0.04	0.22	-0.13	-0.02	0.22	-0.65***	—

Note. High $PM_{2.5}$ Days = summed number of days with PM2.5 > 35 µg/m³. Average $PM_{2.5}$ = annual average $PM_{2.5}$ concentration (µg/m³). **HCC** = hair cortisol concentration (pg/mg). **RMSSD** = Root Mean Square of Successive Differences (ms) = Respiratory Sinus Arrhythmia (ln[ms²]). **PSS** = Perceived Stress Scale total score. **CEN** = Central Executive Network; **DMN** = Default Mode Network; **SN** = Salience Network. **GE** = Global Efficiency; **CC** = Clustering Coefficient.

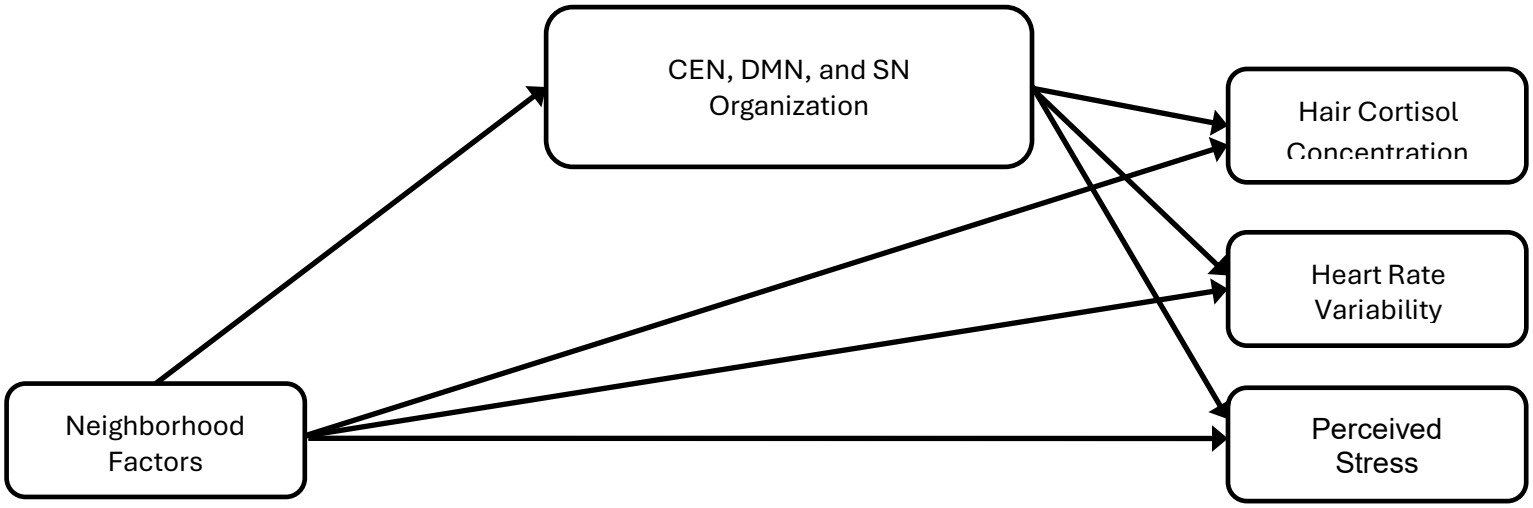
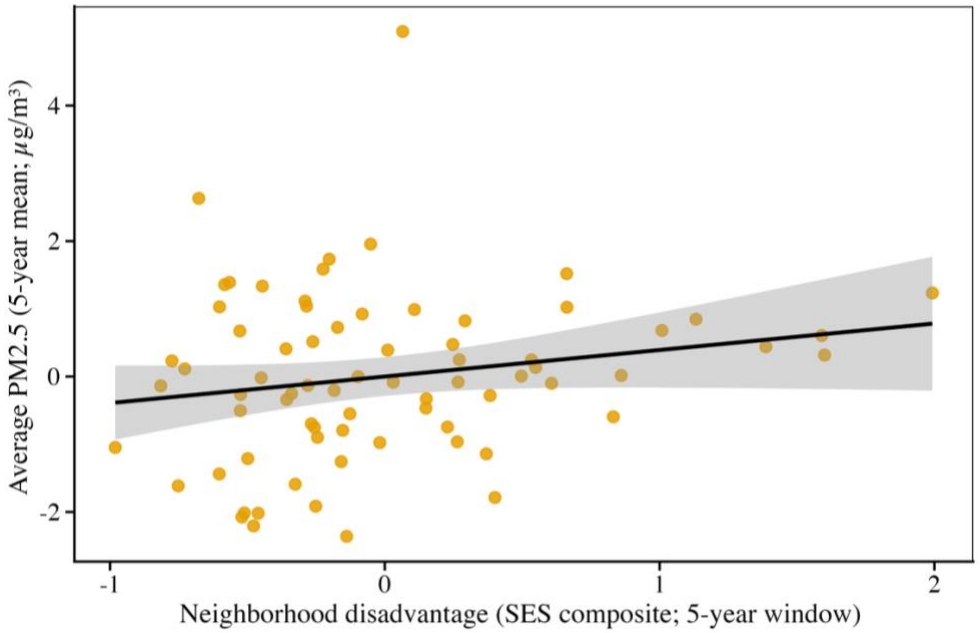


Figure 1. Conceptual Model: This model illustrates the hypothesized associations between neighborhood factors during childhood/adolescence, central executive network (CEN), salience network (SN), and default mode network (DMN) functional organization, and stress-related outcomes in early adulthood. Neighborhood factors are expected to influence multisystemic stress physiology (hair cortisol concentration, heart rate variability) and perceived stress. Indirect effects are also expected. Specifically, neighborhood factors are expected to influence CEN, SN, and DMN functional organization, leading to altered stress-related functioning.

a)



b)

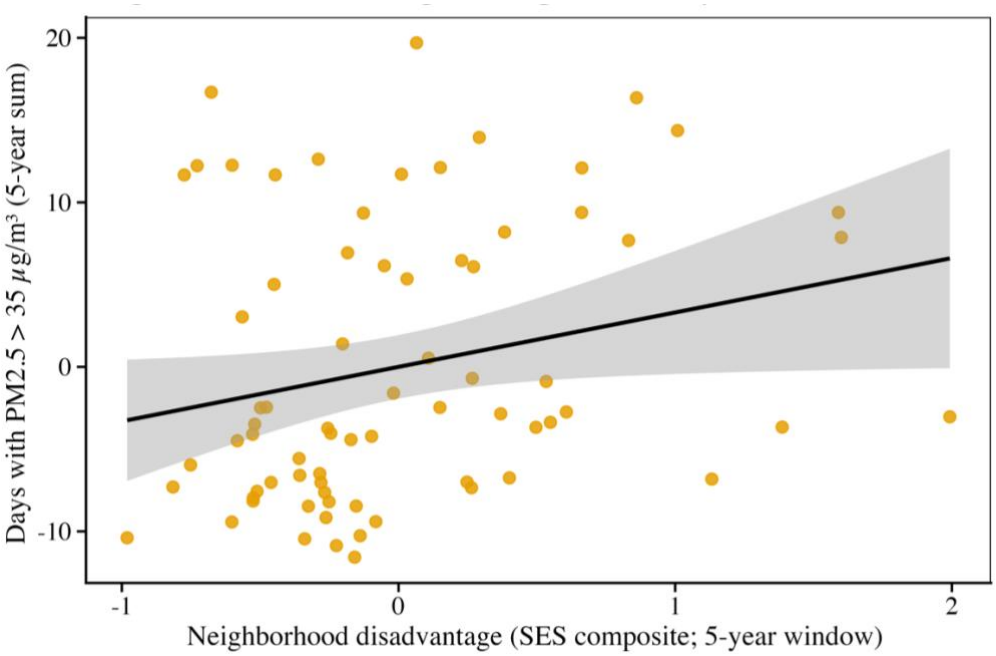


Figure 2. Associations between neighborhood disadvantage and $PM_{2.5}$ exposure (covariate-adjusted partial regression plots). Association between neighborhood socioeconomic disadvantage and **(a)** average $PM_{2.5}$ concentration and **(b)** the number of days with $PM_{2.5}$ concentrations exceeding 35 µg/m³. Both partial regression plots are linear models adjusting for distance to the nearest air quality monitor and number of lifetime residential moves. Points represent covariate-adjusted residuals for individual participants; solid lines indicate fitted regression slopes, and shaded bands denote 95% confidence intervals.

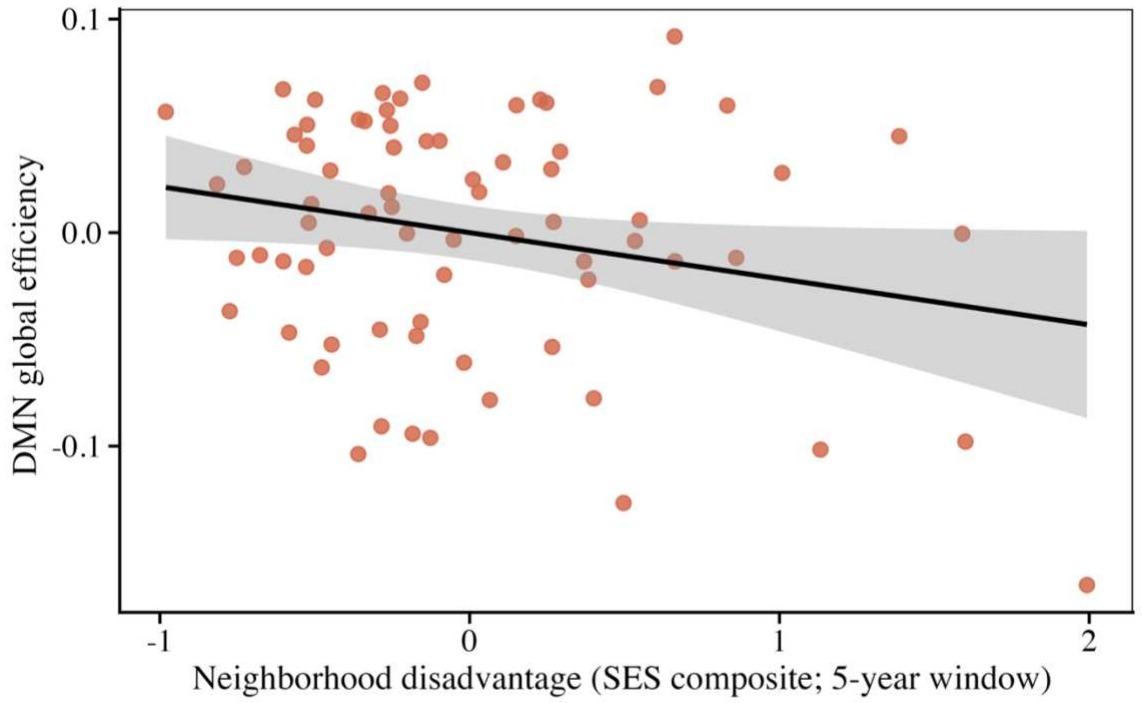
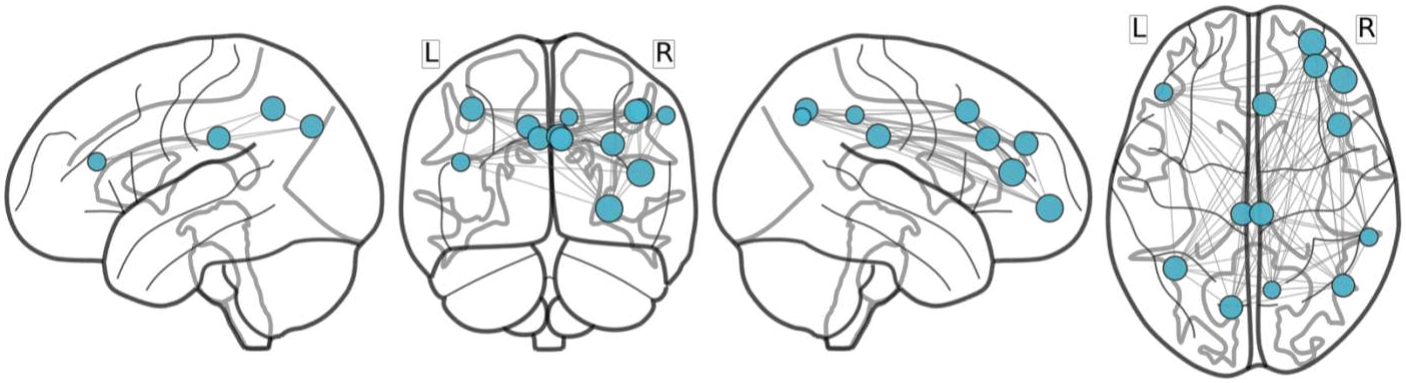


Figure 3. Regression plot showing the adjusted association between childhood neighborhood disadvantage and default node network (DMN) global efficiency. Models were adjusted for sex, age, head motion (mean framewise displacement [FD]), and parental education. Higher neighborhood disadvantage was associated with lower DMN global efficiency after accounting for covariates.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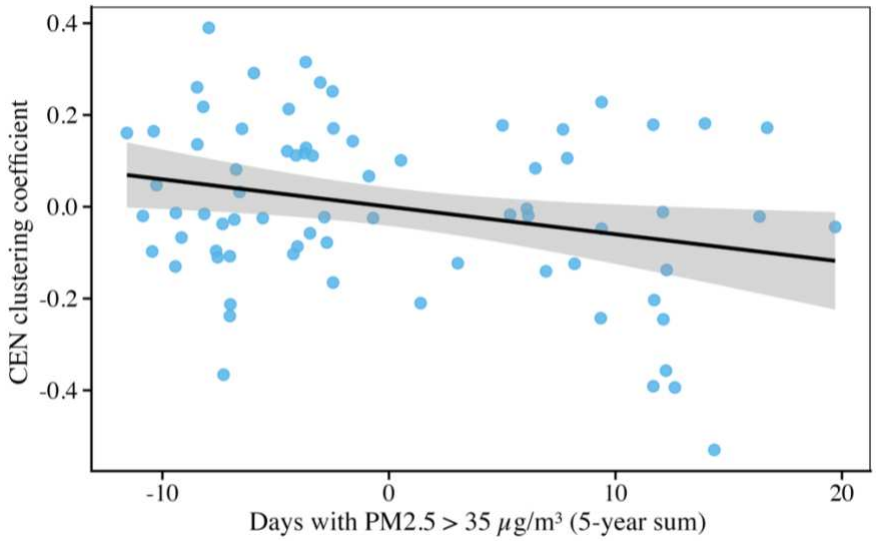
a)


CEN clustering coefficient
0.4
0.2
0.0
-0.2
-0.4
-10
0
10
20
Days with PM2.5 > 35 μg/m³ (5-year sum)

b)

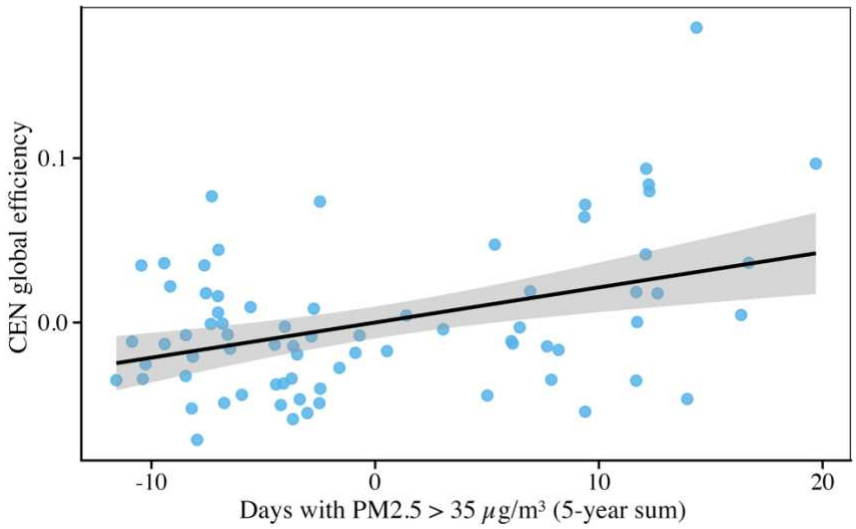

CEN global efficiency
0.1
0.0
-10
0
10
20
Days with PM2.5 > 35 μg/m³ (5-year sum)

Figure 4. Regression plots showing the adjusted association between days with high $PM_{2.5}$ exposure and central executive network (CEN) global efficiency and clustering coefficient. Higher number of days with high $PM_{2.5}$ exposure was associated with **(a)** lower CEN clustering coefficient and **(b)** higher CEN global efficiency after accounting for covariates. Brain renderings depict CEN (blue) defined from the Schaefer (2018) atlas, with node sizes weighted by the average global efficiency of each node in the network.

APPENDIX

Additional HRV Data Considerations

Certain variables (e.g., substance use, medication use, sleep duration) may act as confounds in analyses of HRV (Laborde et al., 2017). Participants reported on substance use, prescription medication use, and sleep duration. To account for these potential influences, we examined associations between HRV indices and these potentially confounding factors in HRV analyses. Substance use ($p = .75$), medication use ($p = .50$), and sleep duration ($p = .34$) were not significantly associated with baseline RMSSD or RSA and were not included in further analyses.

To complement physiological data, participants completed the State-Trait Anxiety Inventory – State scale (STAI-S; Spielberger et al., 1983) immediately before and after the stress task to capture self-reported changes in momentary anxiety. The STAI-S is a brief, validated measure of state anxiety that is widely used in psychophysiological studies examining stress reactivity, including work linking acute stress induction with concurrent changes in HRV (Immanuel et al., 2023; Tarbell et al., 2017; Yamane et al., 2021). Collecting STAI-S scores alongside HRV indices allows for the integration of subjective and physiological responses, providing a more comprehensive assessment of stress regulation. STAI-S scores were not significantly associated with baseline RMSSD ($p = .26$) or RSA ($p = .20$).

Table A1. Nodes of the CEN, DMN, and SN used to compute network properties

CEN (13 nodes)
Left and right parietal cortex (3 nodes)
Left and right lateral prefrontal cortex (5 nodes)
Left and right precuneus (2 nodes)
Left and right cingulate cortex (2 nodes)
Right medial posterior prefrontal cortex (1 node)
DMN (24 nodes)
Left and right temporal cortex (5 nodes)
Left and right parietal cortex (3 nodes)
Left prefrontal cortex (7 nodes)
Right ventral prefrontal cortex (2 nodes)
Right dorsal/medial prefrontal cortex (3 nodes)
Left and right precuneus/posterior cingulate cortex (4 nodes)
SN (12 nodes)
Left parietal operculum (1 node)
Left frontal operculum/insula (2 nodes)
Right frontal operculum/insula (1 node)
Left lateral prefrontal cortex (1 node)
Left medial regions (3 nodes)
Right medial regions (2 nodes)
Right temporal-occipital-parietal regions (2 nodes)

Note. The nodes of each network were based on Schaefer et al. (2018). Abbreviations: DMN, default mode network; SN, salience network; CEN, central executive network.

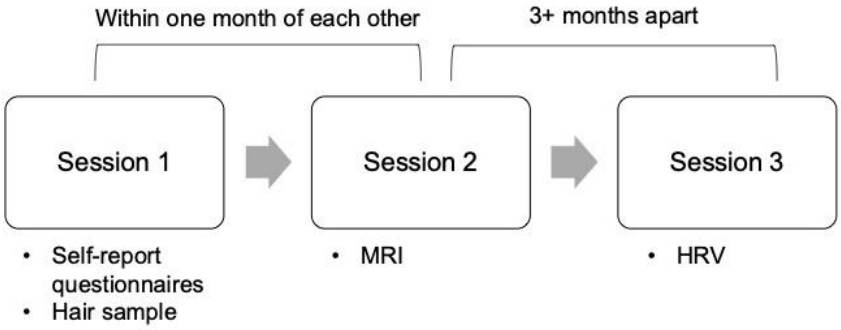


Figure A1. Overview of study procedures showing the study timeline, including three data collection sessions. During session 1, participants completed a set of questionnaires and provided a hair sample. During session 2, participants complete a magnetic resonance imaging (MRI) scanning session. Sessions 1 and 2 were intended to occur within one month of each other. Session 3, which focuses on heart-rate variability (HRV) measurement, took place at least three months after session 2.

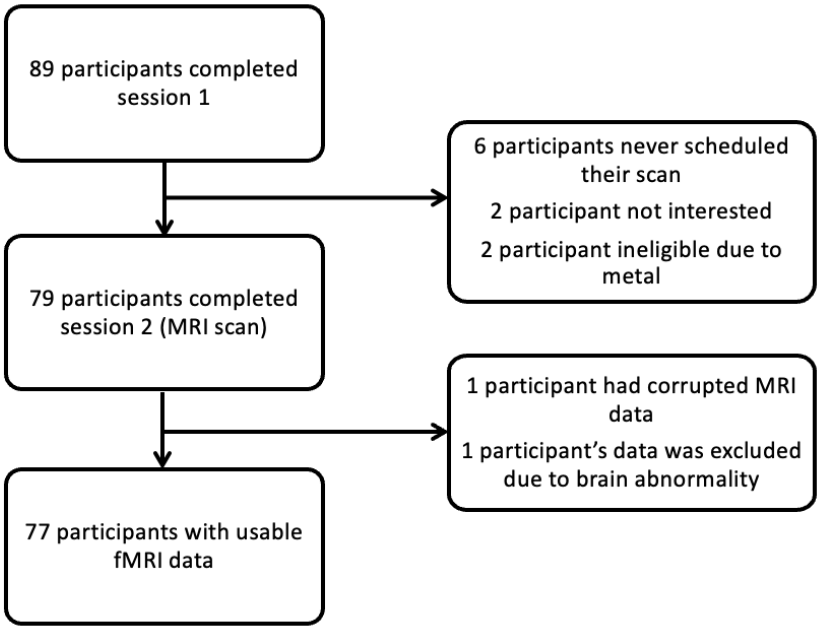


Figure A2. Flowchart illustrating current availability of resting-state functional magnetic resonance imaging (rs-fMRI) for analyses. Reasons for missing data and current sample sizes for rs-fMRI data are highlighted.

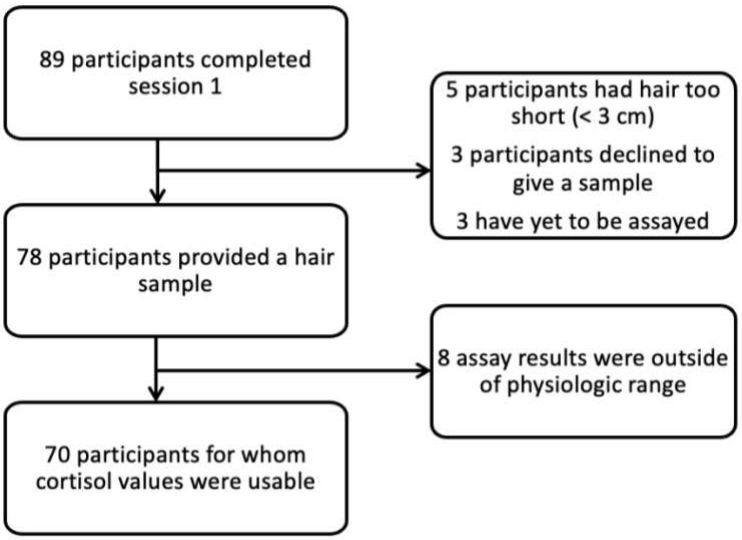


Figure A3. Flowchart illustrating current availability of hair cortisol concentration (HCC) data for analyses. Reasons for missing data and current sample sizes for these HCC data are highlighted.