THESIS

CUMULATIVE EARLY ADVERSITY AND RESTING-STATE NETWORK TOPOLOGY IN 18- AND 19-YEAR-OLDS

Submitted by

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ABSTRACT

CUMULATIVE EARLY ADVERSITY AND RESTING-STATE NETWORK TOPOLOGY IN 18- AND 19-YEAR-OLDS

It is well established that the accumulation of early adversity exposure significantly increases risk for mental health problems across the lifespan. Yet, the neural mechanisms underlying these associations are not well understood. Cumulative early adversity may influence connectivity in large-scale resting-state networks known to be crucial to mental health – namely, the salience (SAL), default mode (DMN), and central executive (CEN) networks. However, little is known about the associations between cumulative early adversity and the topology of these networks in emerging adults or whether these associations vary based on the timing of stressor exposure. Thus, the goal of this study was to use a graph theory approach to examine how cumulative early adversity exposure and the developmental timing of adversity exposure may relate to SAL, DMN, and CEN topology in emerging adults. Participants were typically developing 18- and 19-year-olds ($N = 76$) from socioeconomically diverse backgrounds. They completed questionnaires and a magnetic resonance imaging (MRI) session. Using a well-validated measure—the Stress and Adversity Inventory for Adults (STRAIN) (Slavich & Shields, 2018)—participants reported on whether they experienced 55 different major stressors (e.g., death of a loved one, financial difficulties, car accident, neighborhood danger), and the timing, severity, and duration of endorsed stressors. Using this data, separate scores were computed reflecting the count and severity of stressors in early childhood (0-7 years), adolescence (13-18 years), and within the past year. Resting-state functional topology was computed as the average

clustering coefficient and global efficiency of nodes in the DMN, SAL, and CEN. Multiple linear regression analyses were run to examine how cumulative stressor exposure (stressor count and severity < 18 years), stressor count and severity during early childhood and adolescence, and recent stressor exposure may relate to DMN, SAL, and CEN topology (clustering and efficiency).

Exposure to more severe stressors during early childhood was associated with reduced clustering of the DMN, independent of exposure during other developmental periods (β = -.24, p = .04). Additionally, both recent stressor count (β = .28, p = .02) and severity (β = .24, p = .04) predicted increased SAL clustering and trending lower efficiency. No significant associations were seen for cumulative or adolescent stressor exposure, or for network topology of the CEN.

Findings from this study provide insight into neurodevelopment following early life adversity. Results suggest that the neurodevelopmental impacts of childhood adversity may depend on the timing and severity of exposure, particularly in the DMN and SAL. Focusing on emerging adults allowed for capturing the influences that early life stressors may have as the brain nears maturity. The use of the STRAIN enabled quantifying exposure to a breadth of adversities beyond those that are typically studied in this field (e.g., maltreatment, institutional deprivation), potentially producing more generalizable results. The timing and severity of early adversity are important to consider when investigating effects on neural function and implications for risk for psychopathology.

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INTRODUCTION

Exposure to early life adversity (e.g., abuse, neglect, poverty) is prevalent in the United States (U.S.) and worldwide. It is estimated that 6 in 10 children under five years of age endure psychological or physical aggression on a regular basis (UNICEF, 2024). And, approximately 64% of U.S. adults report at least one type of adverse childhood experience (ACE), while 17% report four or more ACEs (CDC, 2025). Extensive evidence indicates that childhood adversity has long-term consequences for mental health (Juwariah et al., 2022; McGinnis et al., 2022; Riem & Karreman, 2019). Childhood adversity demands substantial psychological, social, and neurobiological adaptation that differs from how predictable and typical upbringings shape children's development (McLaughlin, 2017). Given the heightened plasticity of the developing brain, early life adversity is believed to alter brain development in ways that increase risk for mental health problems that persist over time (Merz et al., 2024; Nelson & Gabard-Durnam, 2020). Numerous studies suggest that early life adversity may alter patterns of neural activity, setting the stage for long-term mental health difficulties (Bick & Nelson, 2016; Hanson et al., 2015). Yet, little is known about how these effects may vary by the developmental timing of adversity exposure. The purpose of this study is to address this gap by examining whether associations between early life stressor exposure and neural function in emerging adults depend on the timing of exposure.

Cumulative Early Adversity and Mental Health Outcomes

The cumulative risk model has been applied to research on early stressor exposure and neurobiological and mental health outcomes (Chad-Friedman et al., 2021; Dufford & Kim, 2017; G. W. Evans et al., 2013; Khundrakpam et al., 2016). In research using a cumulative risk

approach (e.g., studies of ACEs), early adversity exposure is analyzed as a composite of exposure to multiple risk factors, rather than a single risk factor. Multiple, different risk factors (e.g., adversity exposures) are aggregated into one composite score, which reflects the extent of the individual's exposure to adversity (Evans et al., 2013). Examples of types of adversity indicators include poverty, abuse, neglect, parental substance abuse, maternal depression, and peer victimization.

Decades of research have shown that higher cumulative early adversity increases risk for psychiatric difficulties lasting throughout adulthood. These studies have shown that it is the accumulation of adversity exposure rather than exposure to a single risk factor that explains the most variability in mental health outcomes (G. W. Evans et al., 2013; Huang et al., 2021; Lian et al., 2024; Liu et al., 2023; Wiens et al., 2020). For example, exposure to higher cumulative stressors has been associated with increased internalizing symptoms (e.g., anxiety) and conduct, attention, and impulse control problems in children (Bøe et al., 2018; Kunin-Batson et al., 2024; O'Hare et al., 2023; Stein et al., 2022). Similarly, higher cumulative early stressor exposure has been associated with increased substance use, internalizing symptoms, and antisocial behavior in adolescence, into adulthood (X.-Y. Chen et al., 2023; C. B. R. Evans et al., 2019; Hoffmann & Jones, 2022; Jiang et al., 2020; Künzi et al., 2022; Lloyd & Taylor, 2006; McMullin et al., 2021a; Paquola et al., 2017; Pegg et al., 2019; Schilling et al., 2008; Shen, 2021; Turner & Lloyd, 1995). Yet, studies find that the association may also be explained by the severity of adverse experiences, not merely the accumulation of them (Schilling et al., 2008).

Childhood adversity has become an increasingly prevalent topic in the study of neurodevelopment. Yet, there is little consensus on which measures best capture early adversity. Among the most common are measures of early trauma (e.g., child maltreatment), such as the

childhood trauma questionnaire (Bernstein et al., 2011), and the adverse childhood experiences (ACEs) scale (Felitti et al., 1998). The ACEs measure captures interpersonal adversities such as abuse (e.g., physical, emotional, or sexual), neglect (e.g., physical and emotional), and environmental adversities like household dysfunction. While these measures have provided insights into the impacts of early life adversity and ways to measure it, they do have limitations. One limitation is that they are not comprehensive, leaving exposure to other, more common stressors unmeasured.

The Stress and Adversity Inventory (STRAIN) addresses this issue by inquiring on 55 major life stressors, each characterized by interpersonal loss, physical danger, humiliation, entrapment, or role change/disruption (Slavich & Shields, 2018). It includes questions about stressful life circumstances (e.g., housing, financial, or health difficulties) and experiences (e.g., interpersonal/family struggles, life threatening situations, legal issues). Responses to these questions can be aggregated to form a cumulative stressor exposure score, which has been used to examine the influences of stressors on mental health in various populations. Numerous studies have associated cumulative stressor count and severity scores from the STRAIN with anxiety, depression, somatization symptoms, increased alcohol use, anhedonia, risky behavior, impulsivity, food addictive behaviors, and worse subjective physical, emotional, and cognitive well-being (Clay et al., 2023; Klatzkin et al., 2023; McLoughlin et al., 2021; McMullin et al., 2021b; Parra et al., 2023; Senft Miller et al., 2022; Slavich et al., 2019).

Although extensive research has documented the deleterious impact of cumulative early adversity exposure on mental health, less is known about the neural mechanisms underlying these associations. Research has started to uncover how early stressors/adversity may alter brain function and development, but many questions remain unanswered.

Resting-State Functional MRI and Functional Connectivity

Functional magnetic resonance imaging (fMRI) is used to investigate brain function via blood-oxygen-level dependent (BOLD) signal, capturing fluctuations of blood flow in the brain that are thought to reflect neural activity. Functional MRI can be task-based or non-reliant on a task (i.e., resting-state). Resting-state fMRI involves scanning the brain while the subject is idle, without the presence of stimuli or task demands. It can be used to assess baseline functional architecture of the connectome based on BOLD activity during passive rest (Biswal et al., 1995; Biswal & Uddin, 2025). Resting-state fMRI has been widely used to investigate meaningful patterns of spontaneous neural activity, providing important insights into brain function across various populations (Lv et al., 2018). Advantages of resting-state fMRI include its lack of cognitive demand placed on participants and ease of data acquisition in diverse populations (e.g., children and adolescents) (Barkhof et al., 2014; Biswal & Uddin, 2025; Smitha et al., 2017).

Resting-state functional connectivity (resting-state FC or rsFC) analyses are used to measure patterns of coactivity between brain regions, providing insight into the brain's dynamic spatiotemporal organization (Biswal & Uddin, 2025). Analytic methods used to examine brain connectivity include seed-based analysis, independent component analysis, and graph theory approaches (Farahani et al., 2019; Hallquist & Hillary, 2018; Smitha et al., 2017). Resting-state FC can be measured between a single pair of different brain regions or in large-scale functional networks.

Intrinsic connectivity networks (ICNs) have been identified that include higher-order association networks such as the default mode network (DMN), salience network (SAL), and central executive network (CEN; Keller et al., 2023; Menon, 2011). These ICNs consist of regions that consistently show temporally synchronous neural activity, often despite the longer

spatial distance between regions. ICNs emerge early in development, existing in a non-random, modular pattern from infancy (Khundrakpam et al., 2016).

Together, the CEN, SAL, and DMN support various functions (e.g., self-regulation and cognitive control) through specialized within-network connectivity and coordinated interactions between networks. Rooted primarily in the ventromedial prefrontal cortex (vmPFC) and posterior cingulate cortex (PCC), the DMN is a task-negative network involved in internally oriented functioning, including self-referential thought, autobiographical memory, internal narrative, and social cognition (Buckner et al., 2008; Fair et al., 2008; Menon, 2023; Menon & D'Esposito, 2022). As such, it shows increased or decreased activity during introspection or task engagement, respectively (Smallwood et al., 2021). Key CEN nodes include the dorsolateral PFC (dlPFC) and posterior parietal cortex (PPC). The CEN is a task-positive network that enables goal-directed behavior via top-down control and is needed for working memory, inhibitory control, and problem-solving (Dosenbach et al., 2007; Keller et al., 2023; Menon, 2011; R. Wang et al., 2021). The SAL is a task-positive network anchored in the dorsal anterior cingulate cortex (dACC) and anterior insula (AI; Menon & D'Esposito, 2022). Crucially, upon detection and filtering of salient (internal or external) information, the SAL modulates activity in both the DMN and CEN by up- or down-regulating their engagement in response to immediate demands (Menon, 2011; Molnar-Szakacs & Uddin, 2022). Altered functional connectivity within and between these neural networks has been theorized and found to underlie various forms of psychopathology, including anxiety disorders and depression (Menon, 2011).

Graph Theory

Graph theoretical analysis is an advanced method used to assess the temporal correlations of activity in ICNs or across a large number of brain regions (Bullmore & Sporns, 2009; J. Wang

et al., 2010). Graph-based analysis models the topological properties of functional networks, which are composed of nodes (brain regions) and edges (connections between regions), elucidating functional integration and segregation across the connectome (Minati et al., 2013). The global clustering and efficiency coefficients are among various metrics that provide insights into neural network topological characteristics (Fair et al., 2007). Clustering coefficient is a measure of local network segregation that reflects the degree to which a node's neighbors are also connected to one another (Tooley et al., 2021). Global efficiency is a measure of network integration, or the efficiency of communication between nodes in a network based on the number of connections it takes to transmit information from one node to another (Farahani et al., 2019). Most studies to date have used conventional rsFC analytic approaches, whereas graph theory methods have been used relatively less frequently despite their methodological advantages.

Cumulative Early Adversity and Resting-State Functional Organization

Numerous studies have demonstrated associations between single types of early adversity (e.g., child maltreatment) and within-network rsFC (a coarse proxy for system segregation) in children and adults, but questions remain about how the accumulation and timing of early adversities may influence rsFC in neural networks. Trends have emerged showing associations between cumulative childhood adversity and differences in rsFC, including in circuitry responsible for higher-order functioning. Greater exposure to ACEs has been associated with reduced rsFC in the DMN, SAL, and CEN (Woodley et al., 2025). Yet, other studies have linked greater cumulative stressor/adversity exposure to *increased* connectivity between main regions for socioemotional processing, such as between the amygdala and the mPFC, anterior insula, and dorsal anterior cingulate, key connections within the SAL (Park et al., 2018; Thomason et al., 2015). Findings show either increased or decreased connectivity in the DMN depending on the

regions assessed (Azarias et al., 2025; Rebello et al., 2018). Severity of childhood trauma has also been associated with rsFC in regions involved in both cognitive and sensorimotor control (Silveira et al., 2020), although there is very little work assessing the CEN specifically. These associations may be attributable to neural adaptation occurring in response to early stressor exposure (Nudelman & Waltz, 2022; Shonkoff et al., 2005).

Although rs-fMRI studies of cumulative early adversity using graph theoretical approaches are few, some have revealed associations between cumulative early adversity and topological properties of the brain. For example, a study of 10-year-olds (from the larger Adolescent Brain Cognitive Development [ABCD] study) found that cumulative adversity was associated with reduced global (whole-connectome) clustering, but not with modularity (a mesoscale measure of segregation)—and that these associations were consistent with all findings from their adversity subscales except physical/sexual abuse (Vedechkina et al., 2024). Subscales of adversity (instability, parental neglect, financial difficulties) were assessed at the network-level and were associated with reduced clustering in networks such as the DMN and SAL (Vedechkina et al., 2024). Another study using the ABCD sample found that total environmental stress exposure (shared variance across various environmental stressors) was associated with lower modularity of networks and higher network efficiency in the DMN in 9- to 10-year-olds (Jeong et al., 2024). These findings suggest altered neural functional organization following greater exposure to stressors/adversity in children. Further work is needed using graph analysis to elucidate the associations between cumulative early adversity and network-level topological features in adulthood.

Research to date has primarily focused on single, extreme forms of early adversity such as trauma (e.g., abuse, neglect, exposure to domestic violence). This approach fails to capture the

consequences of exposure to a range of less extreme, more common stressors, such as relationship problems, financial difficulties, or environmental unpredictability. Few studies have used graph theoretical methods to examine the associations of cumulative early adversity with differences in clustering and efficiency in the DMN, SAL, and CEN in emerging adults.

Adversity Exposure During Sensitive Developmental Periods

Sensitive periods are windows of increased neural plasticity when neural development is particularly malleable to environmental influences (Gee, 2016, 2022; Malave et al., 2022; Meredith, 2015; Michael et al., 2024; Stacy & Van Hooser, 2022). Sensitive periods are thought to correspond to epochs of rapid neural development, which vary to some extent across neural networks. Neural networks underlying sensory systems develop at an earlier age than do the networks underlying association systems. Infancy and early childhood are sensitive periods for maturation of primary sensorimotor and other neural networks (Berardi et al., 2000; Choi, 2018; Gee, 2022; Stacy & Van Hooser, 2022). Adolescence is also considered to be a potential sensitive period, given the continued development and heightened neural plasticity of the PFC and associated higher-order functions during this period (Fuhrmann et al., 2015; Gee, 2022; Schalbetter et al., 2022; Stacy & Van Hooser, 2022). It is hypothesized that the protracted development of association networks during adolescence is accompanied by heightened neural plasticity, as these systems continue to be strengthened and refined toward maturity (Sydnor et al., 2021).

The effects of adversity on the function of these neural systems may depend on the developmental timing of adversity exposure. Research is needed to examine whether the timing of adversity exposure within specific developmental windows matters for adversity effects on neural function. I aim to address this gap by examining whether the count and severity of stressor

exposure at different ages associates with the functional organization of the DMN, CEN, and SAL in emerging adults.

Development of Brain Networks

At birth, ICNs such as the sensorimotor and visual networks are among the first to exhibit coherent activity patterns, reflecting early functional specialization tied to basic sensory and motor processing (Eyre et al., 2021; Fransson et al., 2007; Hoff et al., 2013; Lin et al., 2008). These lower-order networks and functions develop rapidly early in life, reaching relative maturity during early childhood (Huttenlocher & Dabholkar, 1997; Pendl et al., 2017; Sydnor et al., 2021, 2023). Higher-order association networks—such as the DMN, SAL, and CEN—emerge more gradually throughout early childhood, adolescence, and into adulthood, although their foundations originate during gestation (Dennis & Thompson, 2013; Hoff et al., 2013; Song et al., 2024; Sydnor et al., 2021, 2023). Studies of children and adolescents have also shown a positive association between age and segregation, global/local efficiency, and other small-world properties (Song et al., 2024, 2025; Tooley et al., 2022). This may reflect the protracted developmental timeline of long-range functional connectivity, which begins to mature later in the postnatal period (Damaraju et al., 2014; Fair et al., 2007; Khundrakpam et al., 2016).

FC patterns mature in the DMN, CEN, and SAL throughout adolescence. Studies comparing FC across ages have offered compelling insights into the trajectory of connectivity patterns within and between ICNs. For example, during childhood and adolescence, the DMN shows functional connectivity patterns distinct from those observed in adults, before reaching relative stability throughout adulthood (Azarias et al., 2025). Within the DMN, FC between the PCC and mPFC may be the most immature in childhood (Supekar et al., 2010). The DMN—notably its anterior-posterior connections—significantly increases in FC strength and centrality

between ages 7-15 years, suggesting that late childhood/early adolescence may be a crucial time for functional development of the DMN (Sato et al., 2014).

Other studies on ICN development have shown similar patterns of network architecture trajectories, with less organized control networks in childhood, and increasingly differentiated, efficient network patterns throughout adolescence and adulthood (Clark et al., 2022; de Bie et al., 2012; Dennis & Thompson, 2013; Keller et al., 2023; Marusak et al., 2017; Pines et al., 2022; Sherman et al., 2014; Song et al., 2024; Supekar et al., 2009; Sydnor et al., 2023; Truelove-Hill et al., 2020). Throughout the expected course of neurodevelopment, neural circuitry is refined based on environmental input, genetics, and the interaction of the two. However, the extent of such refinement depends in part on the developmental timing of childhood experiences.

Current Study

The main goal of this study was to investigate whether and how the associations between cumulative adversity exposure and CEN, SAL, and DMN topological features (e.g., global efficiency) depend on the developmental timing of the adversity exposure in emerging adults (18 to 19 years of age). I hypothesize that higher cumulative early stressor exposure (before age 18) will be associated with lower network segregation (i.e., global clustering) and lower network integration (i.e., global efficiency) of the DMN, CEN, and SAL in emerging adults. Based on sensitive period perspectives, I expect that adversity exposure will have differential associations with network topology depending on its timing. I hypothesize that high early childhood (0-7 years) adversity exposure will predict reduced clustering and efficiency in the SAL, DMN, and CEN, independent of adversity exposure during other developmental periods. I expect that increased exposure to stressors during adolescence (13-18 years) will have the strongest impact on clustering and efficiency in the CEN and SAL, independent of exposure during other

developmental periods. This hypothesis is informed by findings that suggest stress-induced reallocation of neurocognitive resources between the two networks (Hermans et al., 2014; Zhang et al., 2020), which may be amplified by the heightened neuroplasticity characteristic of adolescence.

In sum, a large body of work has focused on the effects of early adversity on the developing brain (Bick & Nelson, 2016; McLaughlin et al., 2014, 2019; Nelson & Gabard-Durnam, 2020). Yet, these studies have most often focused on exposure to single types of extreme, often traumatic events during childhood, with little insight into cumulative risk or the role of adversity exposure during different developmental periods. The effects of cumulative stressor exposure on the neural architecture of resting-state networks in emerging adulthood are not well understood. Findings from this study shed light on how these effects may depend on the timing of early adversity exposure.

METHODS

Participants

Recruitment

Typically developing emerging adults were recruited in a medium-sized city in the western U.S. Participants were recruited by posting and distributing flyers and using a university subject pool. Interested individuals completed an eligibility screener. Eligibility criteria required participants to be 18 or 19 years of age and fluent in English. Exclusionary criteria were as follows: diagnosis of a neurological disorder, autism spectrum disorder, schizophrenia, bipolar disorder, or an intellectual disability; history of a severe head injury; and magnetic resonance imaging (MRI) contraindications (e.g., claustrophobia, ferrous metallic implants).

Enrollment of participants was conducted to ensure socioeconomic diversity, similar to previous studies (Merz et al., 2019, 2020). Participants were enrolled if they fit into a parental education bin (highest in the household) for which the participant quota had not been filled. The bins were as follows: high school graduate or fewer years of education (≤ 12 years of education); some college (13-15 years of education); 4-year college degree (16 years of education); beyond college (> 16 years of education).

Sample Characteristics

The final sample included 76 18- and 19-year-olds (51% female), each having usable behavioral and neuroimaging data. Descriptive statistics for sample characteristics are provided in Table 1.

Table 1. Descriptive statistics for sample characteristics ($N = 76$)

	M	***SD***	***Range***
Age (years)	18.91	0.53	18.02-19.97

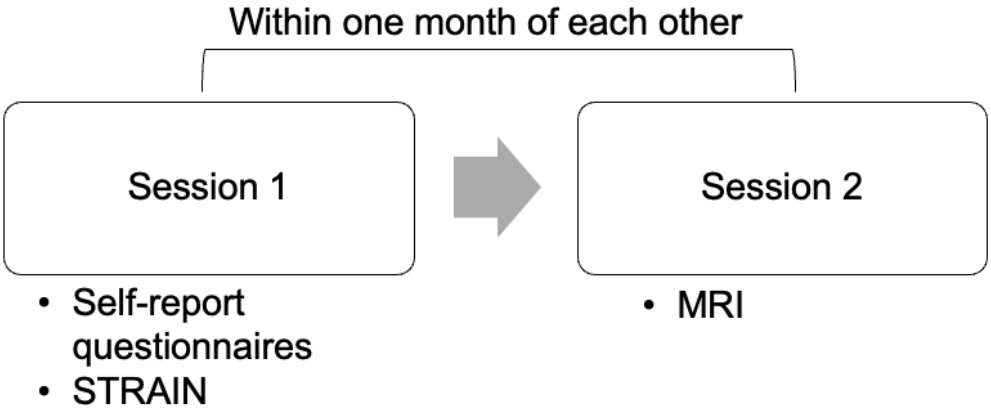
Parental education (years)	14.24	4.12	0.00-22.00
Time between sessions 1 and 2 (days)	19.71	21.89	0-111
	n	**%**	
Sex (female)	39	51.3	--
Race/ethnicity			
White, non-Hispanic/Latine	50	65.8	--
Black or African American	4	5.3	--
Asian American	3	3.9	--
American Indian or Alaska Native	1	1.3	--
Native Hawaiian or other Pacific Islander	1	1.3	--
Hispanic/Latine	17	22.4	--

Note. --, not applicable

Procedure

Written informed consent was provided by all participants during the first of two sessions. Participants then completed a series of questionnaires, including a demographic questionnaire. The Stress and Adversity Inventory for Adults (STRAIN) (Slavich & Shields, 2018) was then administered on a computer. Within one month later, participants returned for a second session, during which they underwent neuroimaging (see Figure 1). During the scanning session, resting-state fMRI was conducted. All study procedures were approved by the CSU Institutional Review Board (IRB).

Figure 1. Overview of data collection procedures



Note. During session 1, participants completed self-report questionnaires and the Stress and Adversity Inventory for Adults (STRAIN). During session 2, participants completed a multimodal magnetic resonance imaging (MRI) scan.

Measures

Early Life Stressor Exposure

The STRAIN (Slavich & Shields, 2018) was used to assess participants' self-reported lifetime exposure to stressors. The STRAIN is an online system that presents the participant with questions about exposure to 55 different major life stressors (www.strainsetup.com) (e.g., car accident, financial difficulties, job loss, death of a loved one, abuse). Example items are 'Was there ever a period of time when you were separated from a parent (or main caregiver) for at least one month before you were 18?' and 'Have you ever been laid off or fired from a full-time job?'. The version of the STRAIN used in this study included a "transition to college" module, which inquires on aspects of this major life change that may be particularly difficult (e.g., separation from loved ones, financial difficulties). Upon endorsing any stressor exposure, participants are asked follow-up questions about the timing, duration, severity, and frequency of the stressor. Participants rate the severity of each stressor on a 5-point scale, ranging from "Very slightly or not at all" (1) to "Extremely" (5). The STRAIN shows strong predictive, concurrent, and discriminant validity, and excellent test-retest reliability (Ahn et al., 2024; Banica et al., 2020; Cazassa et al., 2020; Mayer & Slone, 2024; Murphy et al., 2024; Olvera Alvarez et al., 2019; Slavich & Shields, 2018; Sturmbauer et al., 2019) (see Appendix A for a detailed description of the STRAIN).

Two indices of cumulative early stressor exposure (before 18 years of age) were generated via the STRAIN: (a) total early life stressor *count* and (b) total early life stressor

severity were used in analyses. Composite stressor scores were calculated for two developmental periods theorized to be sensitive periods: early childhood (0-7 years) and adolescence (13-18 years) (Banica et al., 2020; Slavich & Shields, 2018). Additionally, a recent score using stressors occurring within the past year was computed. Two scores (count and severity) for each time frame were calculated using the same values from the initial cumulative score, only separating them by the age at which the stressor occurred. Due to positive skew, cumulative stressor count (skew = 2.3) was log-transformed prior to analyses. Descriptive statistics for cumulative stressor exposure and exposure during each developmental period are included in Table 2.

Neuroimaging

MRI Acquisition

Participants underwent resting-state fMRI for ten minutes and ten seconds while looking at a fixation cross. A 3 Tesla (3T) Siemens Skyra scanner with a 64-channel head radiofrequency coil was used to obtain resting-state fMRI volumes. Volume parameters were a voxel size of 2.5 x 2.5 x 2.5 mm, repetition time (TR) of 900 ms, and a field of view (FoV) read of 210 mm. One sequence of 667 volumes was acquired, the echo time (TE) being 38.0 ms, with 2.5 mm thickness.

MRI Preprocessing

FMRI prep was used to run a standard preprocessing pipeline. Imaging data were first converted to BRAIN Imaging Data Structure (BIDS) format using HeuDiConv version 0.9.0 (Halchenko et al., 2024) and dcm2niix version v1.0.20240202 (Li et al., 2016). The BIDS-converted neuroimaging dataset was then preprocessed in fMRIPrep 20.2.7 (Esteban et al., 2019). Intensity non-uniformity (INU) correction was performed on participants' T1-weighted (T1w) anatomical images using N4ITK, before being skull-stripped. Gray matter, white matter,

and cerebrospinal fluid (CSF) were differentiated using tissue segmentation. Finally, individual T1w images were spatially normalized to the MNI152NLin2009cAsymtemplate using ANTs (Advanced Normalization Tools).

Resting-state fMRI data were motion corrected using mcflirt, which estimated and corrected for head movement (FSL 5.0.9). Field maps were then used to perform distortion correction. For coregistration, functional images were aligned to corresponding T1w images using boundary-based registration (BBR) before they were transformed to MNI152NLin2009cAsym space. Motion and physiologically related noise were addressed during post-processing by extracting confound regressors using CompCor (Behzadi et al., 2007). Then, motion outliers were classified using a framewise displacement (FD) threshold of 0.5 mm, followed by additional denoising using ICA-AROMA, producing functional images with reduced motion-related artifacts (Pruim et al., 2015). Gaussian spatial smoothing—with a full-width at half-maximum (FWHM) of 6—was used to further improve signal-to-noise ratio.

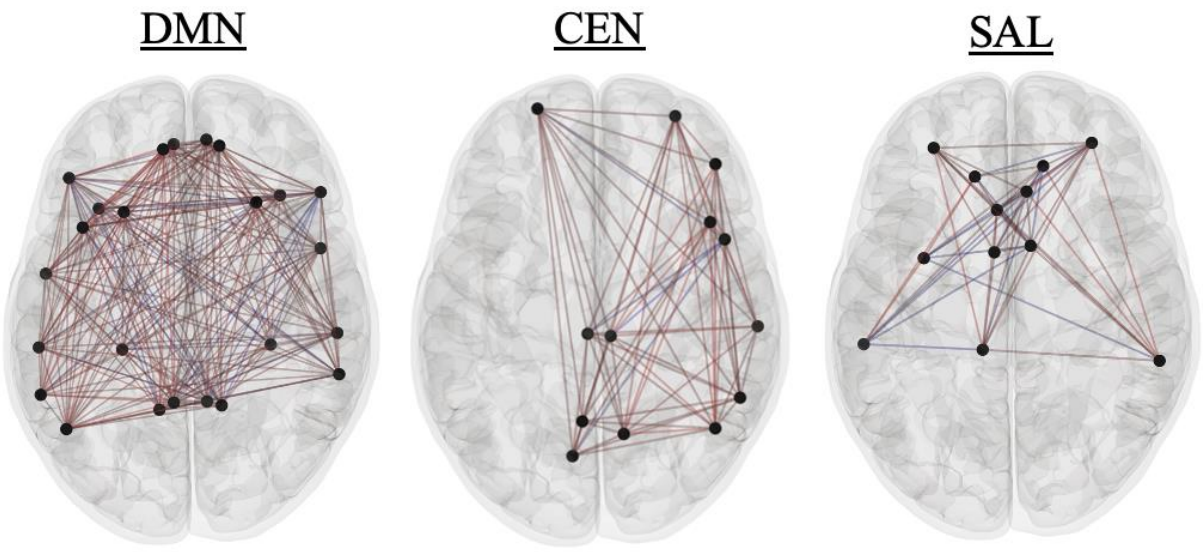
Functional Network Topology

Graph analysis was run using CONN version 21.a (Whitfield-Gabrieli & Nieto-Castanon, 2012). During first-level analyses, ROI-to-ROI connectivity matrices (RRC) are estimated characterizing the patterns of functional connectivity among 49 seeds from the 7-network Schaefer parcellation scheme (Nieto-Castanon, 2020; Schaefer et al., 2018). Functional connectivity strength is represented by Fisher-transformed bivariate correlation coefficients from a weighted general linear model (weighted-GLM; Nieto-Castanon, 2020), defined separately for each pair of seed and target areas, modeling the association between their BOLD signal timeseries. Adjacency matrices were defined using a fixed network-level cost of 0.15 and a two-sided FDR-correction threshold of $p < 0.05$, reducing RRC matrices to an undirected graph

characterizing the strongest connections between ROIs in each network. Global efficiency and clustering coefficients were calculated from these undirected graphs for each network, then extracted from CONN for second-level analyses in R. Table 2 includes descriptive statistics for graph metrics of the DMN, CEN, and SAL. A list of ROIs used for each neural network is provided in Appendix B (see Figure 2 for a spatial map of ROIs). Of the 79 scanned participants, three were excluded from analyses due to corrupted MRI data (n = 1), an incidental finding (n = 1), and incomplete behavioral data (n = 1).

Graph theory methods were used to measure network-level organizational characteristics—specifically, global clustering and efficiency. A local clustering coefficient represents the proportion of connections between a region's neighboring nodes (i.e., the level of clustering within a set of nodes), while the global clustering coefficient is calculated as the mean of the local clustering coefficients for all nodes in a network (Masuda et al., 2018). A higher global clustering coefficient indicates higher inter-connectedness of nodes within a network, suggesting greater network segregation. The global efficiency coefficient is calculated as the inverse of the average shortest path length (i.e., distance/number of edges) between pairs of nodes within a network (Farahani et al., 2019; Khundrakpam et al., 2016; J. Wang et al., 2010).

Figure 2. Default Mode Network, Central Executive Network, and Salience Network nodes.



Note. Axial view of seed regions used for graph-theoretic analysis, drawn from the 7-network Schaefer parcellation scheme (Nieto-Castanon, 2020; Schaefer et al., 2018). See Appendix B for a full list of seeds.

Statistical Analyses

Analyses were conducted in R (version 2024.04.2+764) to examine the associations between early adversity exposure and topology of the DMN, CEN, and SAL in emerging adulthood. First, descriptive statistics and zero-order correlations were examined. Then, multiple linear regression analyses were conducted to examine whether cumulative early adversity exposure (total count and severity of adversity exposure before 18 years of age) was associated with average network efficiency and average clustering coefficient within the DMN, CEN, SAL in twelve separate models. Next, separate regression models were run for the total count and severity of stressors during each time period (early childhood, adolescence, and the past year) as independent variables, and within-network clustering and efficiency coefficients as dependent variables (Banica et al., 2022). Separate regression analyses were run for the DMN, CEN, and

SAL, and each of their average clustering and efficiency coefficients and for the count and severity of stressors, resulting in a total of 16 regression models. Covariates included age, sex, parental education, and mean framewise displacement (FD; head movement during resting-state fMRI scanning).

Although sessions 1 and 2 were meant to be within one month of each other, the time between sessions varied across participants (see Table 1). Thus, time between sessions was initially included in the main regression models as a covariate. Because time between sessions did not significantly contribute to the final estimates, it was removed from the models for parsimony. Effect sizes are provided for significant results. Specifically, partial eta squared (η_p^2) is presented, with values of .01, .06, and .14 indicating small, medium, and large effects, respectively (J. Cohen, 2013; Richardson, 2011). For significant results, sensitivity analyses included controlling for adversity exposure during each of the other developmental periods, then adding recent stressor exposure as an additional covariate. To control for multiple comparisons, false discovery rate (FDR) correction was then applied (Benjamini & Hochberg, 1995).

RESULTS

Descriptive Statistics

Descriptive statistics for primary variables of interest, including stressor and neural network topology measures, are provided in Table 2.

Table 2. Descriptive statistics for variables of main interest (N = 76)

Measure	Item	*M*	*SD*	*Range*
Stressor exposure				
Cumulative (up to age 18)	Total count	10.04	7.55	0-51
	Total severity	25.14	15.90	0-76
Early childhood (ages 0-7)	Total count	3.11	3.27	0-16
	Total severity	5.00	5.30	0-18
Adolescence (ages 13-18)	Total count	10.42	5.79	1-27
	Total severity	26.45	15.23	3-69
Recent (past year)	Total count	3.62	2.52	0-12
	Total severity	12.61	9.55	0-45
Network topology				
DMN	Clustering coefficient	0.53	0.09	0.33-0.75
	Global efficiency	0.36	0.06	0.19-0.45
SAL	Clustering coefficient	0.47	0.22	0.00-0.90
	Global efficiency	0.24	0.04	0.16-0.33
CEN	Clustering coefficient	0.58	0.18	0.04-0.96
	Global efficiency	0.22	0.04	0.15-0.41

Note. DMN = default mode network. CEN = central executive network. SAL = salience network.

Zero-order correlations among primary variables are shown in Appendix C. As expected, stressor count and severity were strongly correlated within each developmental period. Cumulative stressor count and severity were positively and significantly associated with the corresponding measures across developmental periods. Stressor exposure measures between developmental periods were positively correlated, although these relationships varied in strength. Early childhood stressor exposure was weakly correlated with adversity during adolescence (r = .18 - .23), with the exception of the count of early stressors, which showed a small-to-moderate association with recent stressor count (r = 0.31) and severity (r = .29). Recent stressor exposure

was moderately associated with adolescent stressor measures (count: r = .41; severity: r = .43); the only other association was with early childhood stressor count, as reported above.

Consistent with the expected integration-segregation tradeoff, there was a strong inverse correlation between clustering coefficient and global efficiency within each network, such that higher global efficiency is associated with lower clustering coefficient. Additionally, global efficiency of the SAL and CEN were anticorrelated (r = -.28), while clustering coefficient of the SAL and DMN were positively correlated (r = .23). There was no correlation between network architecture of the DMN and CEN. Significant correlations were found between early childhood stressor severity and DMN clustering coefficient (r = -.23), and between recent stressor count and SAL clustering coefficient (r = .24).

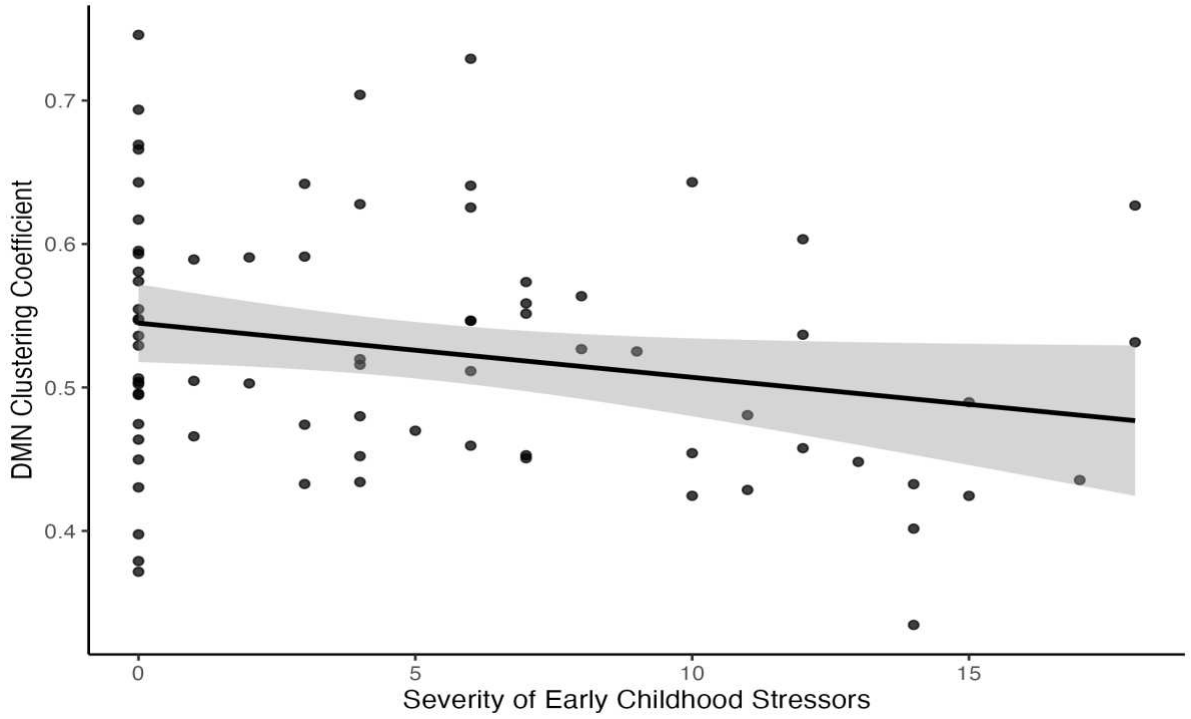
Cumulative Adversity and Network Topology

Count and severity of cumulative stressor exposures were not significantly associated with global efficiency or clustering coefficient in the DMN, SAL, or CEN. Each of these twelve models controlled for age, sex, parental education, and framewise displacement (mean FD; head motion). Tables D.1 and D.2 in Appendix D list β and p values for the cumulative stressor count and severity models, respectively.

Early Childhood Adversity and Network Topology

Higher severity of stressors during early childhood was significantly associated with reduced clustering of the DMN (β = -.24, p = .04, η_p^2 = .05; see Figure 3). There were no other significant associations among early childhood stressor count or severity and the network topology of the DMN, SAL, or CEN. Each model controlled for age, sex, parental education, and mean FD. See Tables D.3 and D.4 of Appendix D for the β and p values corresponding to each of these 12 models.

Figure 3. Relationship between early childhood stressor severity and DMN clustering.



Note. Variables were standardized prior to analysis. β = -.24, p = .04.

Sensitivity analysis

Early childhood stressor severity remained significantly associated with clustering of the DMN when the stressor severity score for adolescence was added as a covariate (β = -.27, p = .03, η_p^2 = .06). This association also remained significant when additionally controlling for the severity of stressors in the past year (β = -.02, p = .03, η_p^2 = .06). However, the association from the original model did not survive FDR correction.

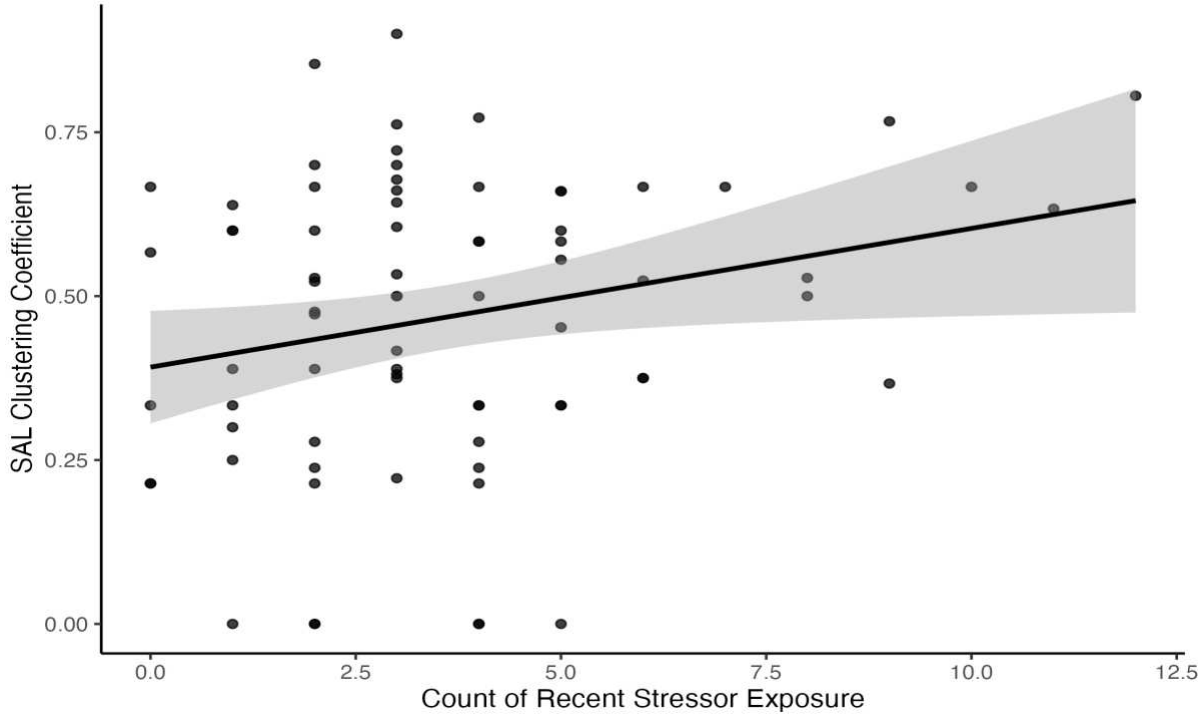
Adversity Exposure in Adolescence and Network Topology

There were no significant associations among the count or severity of stressors during adolescence and the functional organization of the DMN, SAL, and CEN. These 12 analyses, reported in Tables D.7 and D.8 of Appendix D, each controlled for age, sex, parental education, and mean FD.

Recent Adversity Exposure and Network Topology

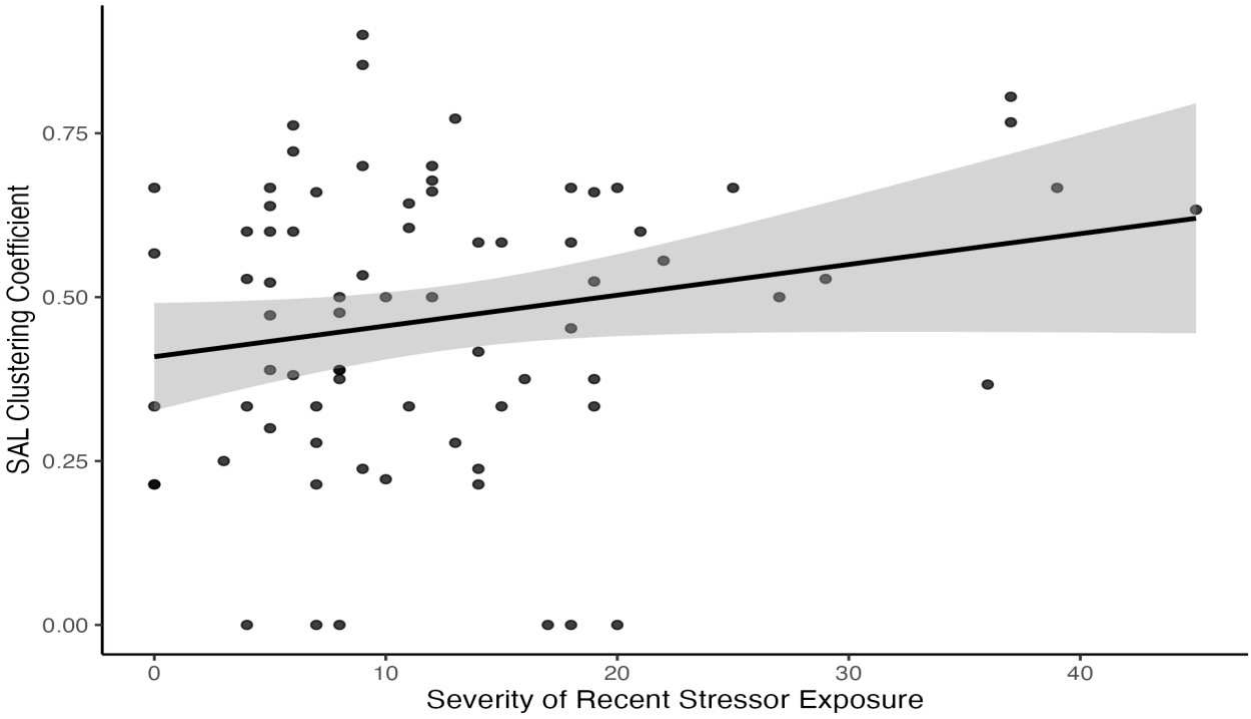
Higher stressor count over the past year was significantly associated with a higher clustering coefficient (β = .28, p = .02, η_p^2 = .06; see Figure 4) and marginally lower global efficiency (β = -.23, p = .06, η_p^2 = .03) in the SAL. Similarly, higher severity of recent stressors was significantly associated with higher clustering coefficient (β = .24, p = .04, η_p^2 = .04; see Figure 5) and trending lower global efficiency (β = -.21, p = .08, η_p^2 = .03) of the SAL. Adversity exposure in the past year was not associated with organization of the DMN or CEN. Each model controlled for age, sex, parental education, and mean FD. The full results of these 12 analyses are reported in Tables D.9 and D.10 of Appendix D.

Figure 4. Relationship between recent stressor count and SAL clustering.



Note. Variables were standardized prior to analysis. β = .28, p = .02.

Figure 5. Relationship between recent stressor severity and SAL clustering.



Note. Variables were standardized prior to analysis. $\beta = .24$, $p = .04$.

Sensitivity Analysis

These associations were similar after adding the stressor scores corresponding to early childhood and adolescence. Specifically, recent stressor count remained positively associated with SAL clustering coefficient ($\beta = .33$, $p = .03$, $\eta_p^2 = .05$) and marginally associated with reduced global efficiency ($\beta = -.25$, $p = .08$, $\eta_p^2 = .02$). Similarly, the severity of stressors over the past year remained significantly associated with increased SAL clustering coefficient ($\beta = .01$, $p = .03$, $\eta_p^2 = .04$), while the p-value for global efficiency decreased to a significant level ($\beta = -.001$, $p = .03$, $\eta_p^2 = .03$). However, none of the associations survived FDR correction.

DISCUSSION

As children and adolescents develop, they are continuously influenced by their environmental inputs, interpersonal interactions, and daily activities, which work dynamically to teach them about the world as their neural circuitry adapts to effectively navigate different contexts. Neural function may adapt in distinct ways depending on whether a child/adolescent is enduring adversity (e.g., abuse, neglect, family conflict, poverty) or thriving in a supportive, enriching environment (Merz et al., 2024). In the current study, I expanded on the literature by examining the associations between childhood adversity and the functional organization of the DMN, CEN, and SAL in emerging adulthood. Findings indicated that the associations between early stressor exposure and neural network organization may depend on the timing of exposure, especially for the DMN and the SAL.

Cumulative Stressor Exposure

Neither the total amount nor severity of stressors before the age of 18 associated with resting-state network topological characteristics. This is not consistent with prior studies of children (ages 9-10) which have linked cumulative stress to altered DMN and SAL efficiency and clustering, suggesting that the cumulative impacts of stress may not extend into later development (Jeong et al., 2024; Vedechkina et al., 2024). However, no other studies to my knowledge have evaluated these relationships in emerging adults, limiting the extent to which direct comparisons can be made. Alternatively, the absence of associations may be due to more dynamic components of adversity and network organization. A cumulative model assumes there are additive effects, while neglecting to account for the nuances of stressor exposure, such as the type or domain, timing and chronicity of the experience, individual response or perception of the

stressor, or resiliency factors. Or, the lack of effects may instead be related to other methodological factors. Adversity exposure may dynamically affect higher-order network reconfiguration in ways that are missed by using a whole-network approach. For example, differences may be present at a different scale than what was examined in this study, such as within or between smaller network clusters or between different networks. Additionally, effects of adversity may be obscured by heterogeneity in developmental trajectories, which may only stabilize after early adulthood when neurodevelopmental plasticity is reduced.

Developmental Timing of Stressors

When developmental timing was considered, results were found for the SAL and DMN. Early childhood (ages 0-7 years) stressor exposure was associated with decreased clustering, but not efficiency, of the DMN in emerging adulthood. Those who experienced more severe stressors in early childhood showed less locally organized topological patterns in the DMN, despite preservation of overall network integration. Between childhood and adolescence, neural networks exhibit increasing integration alongside refinement of network segregation (Fair et al., 2009; Michael et al., 2023). Thus, reduced clustering in early adulthood (as a function of higher early childhood adversity) may indicate altered maturation of DMN architecture, or reduced reductions in local specialization of the DMN in adulthood, potentially due to early disruptions in its developmental scaffolding.

Previous studies have found similar patterns, with higher exposure to adversity predicting lower clustering and higher efficiency of the DMN—however, these studies only looked at outcomes during late childhood (Jeong et al., 2024; Vedechkina et al., 2024). To our knowledge, this is the first study to examine these effects using graph theory measures in a sample of young adults. This finding suggests that the influence of early-life stressors on DMN architecture may

extend to long-term neurodevelopmental alterations. However, early childhood stressor exposure was not associated with global efficiency of the DMN, contrary to what was observed in a late childhood sample (Jeong et al., 2024). This could reflect a compensatory mechanism where network integration increases from its low late-childhood baseline in order to meet the increasing cognitive and socioemotional demands through adolescence. However, this interpretation should be taken cautiously given differences in sample and methodology of the current study and the comparison study, as well as other methodological factors that could account for the null findings.

The DMN supports internally oriented cognition, such as emotional and social evaluation, self-reflection, memory retrieval and integration, and mind-wandering. As such, normative development of the DMN is a key predictor of emotional and cognitive functioning (Azarias et al., 2025). Early childhood adversity has been consistently associated with differences in DMN organization (Giordano et al., 2026), a network implicated in anxiety and depressive disorders (Menon, 2011), making it a candidate neural mechanism linking childhood adversity to long-term mental health outcomes (Chahal et al., 2022; Hardi et al., 2024; Teicher et al., 2016).

SAL topology was found to differ as a function of recent stressor exposure in emerging adults. Associations were found for both the count and severity of recent stressors, and clustering and efficiency of the SAL, although the associations only reached significance for network clustering. Specifically, emerging adults with higher recent stressor exposure showed increased clustering and lower efficiency of the SAL. The SAL supports detection and filtering of behaviorally relevant stimuli (internal or external) and coordinates transitions between internal (e.g., DMN) and external (e.g., CEN) processing systems depending on stimulus demands

(Menon & D'Esposito, 2022). The SAL is frequently implicated in psychopathology, particularly anxiety and related disorders (Menon, 2011). Increased clustering paired with reduced efficiency of a network is indicative of a highly modular, segregated organization. In other words, network functioning relies more on short-range connections and neglects to leverage cross-network integration. The imbalance of efficiency and clustering in the SAL suggests some level of reconfiguration following recent stressor exposure—reconfiguration which may be short- or long-term (T. Chen et al., 2016). Without the inclusion of behavioral metrics, it is difficult to say whether this imbalance is maladaptive or supportive to participants' circumstances. However, lasting changes may prove harmful given that alterations in SAL organization have been repeatedly associated with cognitive and affective difficulties (Schimmelpfennig et al., 2023).

The hypotheses regarding stressor exposure during adolescence (ages 13-18) were not supported by this study. This was unexpected given the continued development of association network circuitry that occurs throughout adolescence. High levels of adversity during development are known to require neural adaptations in order to meet the demands of the environment. These null findings could reflect normative development regardless of levels of stressor exposure, perhaps due to high levels of neuroplasticity and increasing cognitive and emotional skills during these developmental periods (Sydnor et al., 2021). Combined with the early childhood stressor finding and knowledge of the protracted development of higher-order networks, these null findings could suggest that typical development of higher-order networks depends on the integrity of their early developmental scaffolding.

Implications

Findings of this study suggest that early adversity may be linked to long-term developmental differences in the organization of the DMN, which is key to cognitive and

affective functioning. Given evidence that the developmental organization of the DMN relies in part on its early developmental roots (Azarias et al., 2025), these findings may support developmental models suggesting that early stress may contribute to long-term alterations in the organization of the DMN. Importantly, the specificity of these associations to early childhood stressor exposure, rather than adolescent or recent stress, may reflect heightened susceptibility of DMN development during earlier sensitive periods. Although these findings do not directly inform causal mechanisms or intervention strategies, they may have implications for the timing of prevention and therapeutic efforts. Given the DMN's role in the development of psychopathology, the results may support the idea that interventions aimed at reducing stress load or promoting protective environments (e.g., through family- or community-based interventions) early in life may influence neurodevelopmental trajectories before the emergence of later psychopathology. Prior work demonstrating buffering effects of positive caregiving environments in childhood (but less so in adolescence) on neural outcomes following early stress further emphasize this possibility (Gee, 2016).

Additionally, because DMN alterations have been implicated across several forms of psychopathology with substantial inter-individual variability in treatment efficacy, these findings may help identify a potential neural pathway through which early adversity contributes to later mental health risk. More broadly, given the heterogeneous environmental and neurobiological underpinnings of mental health outcomes, early childhood adversity may represent an important and underexamined factor in the relationship between the DMN and psychopathology, with possible relevance for understanding treatment response among individuals with histories of childhood adversity (Teicher et al., 2016). Emerging evidence also suggests that changes in DMN connectivity patterns following certain psychotherapeutic (e.g., cognitive-behavioral,

exposure, and mindfulness-based) and pharmacological interventions contribute to improvements in symptoms (Cui et al., 2021; Fahmy et al., 2019; Haipt et al., 2024; Posner et al., 2013; Rubia et al., 2019; Teicher et al., 2016; Yokoyama et al., 2018). Although the present findings do not establish the DMN as a causal treatment target, they may help identify one neural system through which early adversity becomes biologically embedded and potentially relevant to later intervention research. However, given that the present study did not incorporate mental health measures, findings should not be interpreted as direct evidence linking DMN alterations to adversity-related psychopathology or treatment response.

The association between recent exposure to stressors and salience network architecture in early adults may reflect a period of more flexible, state-dependent adaptation to current environmental demands. Given the SAL's role in detecting, filtering, and responding to emotionally and behaviorally relevant stimuli (Menon & Uddin, 2010), these findings may suggest that salience-related neural organization remains particularly responsive to environmental demands into early adulthood. This raises the possibility that interventions targeting stress reactivity, affective regulation, and cognitive control during this developmental period may be particularly supportive in shaping salience-based network dynamics, adaptive responses to ongoing adversity, and potentially reducing risk for maladaptive stress-related outcomes. Further research is needed to clarify whether SAL alterations associated with recent stress reflect normative state-dependent neural adaptations or aberrant patterns of salience processing that may confer vulnerability to later stress-related psychopathology (or both).

Strengths and Limitations

This study has several key strengths that enhance the interpretability of its findings. A comprehensive measure of adversity exposure was used, which improves the generalizability of

findings compared to studies exclusively examining severe forms or singular types of adversity. The use of a novel, robust measure of functional network architecture allowed for a more nuanced understanding of the network-level differences associated with early life adversity, and how they may compare to normative developmental patterns. The triple network model adds to this dynamic understanding, given that the CEN, SAL, and DMN have crucial and distinct, yet interconnected roles in cognition and mental health. The study is also strengthened by the use of a socioeconomically diverse sample, allowing for better representation across backgrounds. Finally, this study is among the first to examine these associations in a sample of emerging adults, adding novel insights regarding the neurodevelopmental impacts of early adversity as association networks near maturity.

While this study has many strengths, it also has limitations that warrant consideration. First, due to the correlational, cross-sectional design, inferences about causality or directionality cannot be made. Second, retrospective, self-report measures of stressor exposure were used. However, grouping stressors according to age of exposure allowed for greater developmental specificity and more comparable variability within each developmental window. It is possible that response biases (e.g., recall or self-report bias) may have influenced reported stressor exposure and consequently, the observed associations. Finally, the sample primarily consisted of healthy emerging adults who were college students. Thus, findings from this study may not generalize to the broader population of emerging adults. Replication in larger, more diverse samples of emerging adults, including those not attending college, will be important to making conclusions based on this study's findings.

Future Directions

Future analyses could include the use of sub-scales of stressor exposures (both cumulative and developmentally specific) to examine whether effects are driven by particular types or characteristics of stressors. This would be particularly useful for comparing developmental timing effects, given the differences in age-appropriateness of stressful experiences. Additionally, examining these associations using a between-network design would allow for a more comprehensive understanding of the influences of cumulative adversity on functional network architecture. Finally, incorporating an outcome measure of current mental health symptoms, behavior, or cognitive functioning would add valuable insights into the implications of this work.

Conclusion

Using a novel framework for understanding stressor/adversity exposure and functional network organization in emerging adults, this research expands on our understanding of the critical role of childhood experiences in shaping neural architecture. The current study used a well-validated, comprehensive self-report measure of stressful life experiences, allowing for the analysis of cumulative adversity as opposed to the more common approach of focusing on single types of severe adversity. The impacts of cumulative early stressor exposure on the topological characteristics of higher-order networks (i.e., the DMN, SAL, and CEN), which are crucial to mental health, are poorly understood. Findings from this study suggest that early exposure to adversity may be associated with DMN functional organization in emerging adulthood. Additionally, recent stressor exposure was significantly associated with SAL architecture, which could reflect acute adaptations in response to challenging environmental demands. This study provides insight into how stressor exposure during specific developmental periods relates to

network architecture in emerging adults. Understanding the effects of early adversity on neural network function is key to elucidating the mechanisms underlying risk for psychopathology and subsequently informing the design of more effective intervention strategies.

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APPENDIX A

Adult Stress and Adversity Inventory

The Adult Stress and Adversity Inventory (STRAIN) inquires on 55 major life stressors, each fitting into one of two types, 12 primary life domains, and five core characteristics (Slavich & Shields, 2018). Stressor types include acute events and chronic difficulties. The 12 life domains are housing, education, work, treatment/health, marital/partner, childhood/early adversity, reproduction, financial, legal/crime, other relationships, death, life threatening situations, accidents, and possessions. Lastly, core characteristics include interpersonal loss, physical danger, humiliation, entrapment, or role change/disruption. For example, the question "Have you ever had a divorce or other breakup of a serious relationship?" inquires on an acute stressor in the marital/partner domain, with the core characteristic of interpersonal loss. The question "Have you ever been laid off or fired from a full-time job?" is an example of an acute stressor in the work domain, with the core characteristic of role change/disruption. A cumulative stressor score is calculated, in addition to sub-scores for each stressor type, domain, and core characteristic.

Initial questions for each stressor require simple yes/no answers. Upon each "yes" response, survey flow initiates several follow-up questions regarding the severity (how stressful or threatening it was on a 5-point scale, 5 being the highest), timing (when it happened; 0-3 months, 3-6 months, 6-12 months, 1-2 years, 2-5 years, or 5+ years ago), age of occurrence, and the duration (months, years) for chronic difficulties or frequency for acute events. Using responses to these questions, additional scores can be calculated, including the total count and

severity of stressors across the lifetime, before the age of 18, within the last year, and within each age/age range, as done for this study.

APPENDIX B

Within-Network Nodes

Network	Cortical Area (component name)	Schaefer Node Label	MNI Centroid Coordinates		
			x	y	z
DMN	Temporal 1	LH_Temp_1	-56	-4	-20
	Temporal 2	LH_Temp_2	-58	-32	-2
	Parietal 1	LH_Par_1	-58	-50	12
	Parietal 2	LH_Par_2	-48	-64	36
	Prefrontal Cortex 1	LH_PFC_1	-34	22	-10
	Prefrontal Cortex 2	LH_PFC_2	-46	34	-4
	Prefrontal Cortex 3	LH_PFC_3	-6	46	0
	Prefrontal Cortex 4	LH_PFC_4	-24	60	-2
	Prefrontal Cortex 5	LH_PFC_5	-8	48	42
	Prefrontal Cortex 6	LH_PFC_6	-40	14	48
	Prefrontal Cortex 7	LH_PFC_7	-26	20	52
	Precuneus posterior cingulate cortex 1	LH_pCunPCC_1	-12	-56	12
	Precuneus posterior cingulate cortex 2	LH_pCunPCC_2	-6	-52	34
	Parietal 1	RH_Par_1	54	-50	30
	Temporal 1	RH_Temp_1	62	-24	-18
	Temporal 2	RH_Temp_2	50	6	-18
	Temporal 3	RH_Temp_3	58	-26	-2
	Ventral prefrontal cortex 1	RH_PFCv_1	36	26	-16
	Ventral prefrontal cortex 2	RH_PFCv_2	50	28	0
	Dorsal prefrontal cortex medial prefrontal cortex 1	RH_PFCdPFCm_1	6	48	0
	Dorsal prefrontal cortex medial prefrontal cortex 2	RH_PFCdPFCm_2	12	50	40
	Dorsal prefrontal cortex medial prefrontal cortex 3	RH_PFCdPFCm_3	26	24	50
	Precuneus posterior cingulate cortex 1	RH_pCunPCC_1	12	-54	14
	Precuneus posterior cingulate cortex 2	RH_pCunPCC_2	6	-52	30
SAL	Parietal operculum 1	LH_ParOper_1	-58	-38	30
	Frontal operculum insula 1	LH_FrOperIns_1	-42	-2	-6
	Frontal operculum insula 2	LH_FrOperIns_2	-38	12	6
	Lateral prefrontal cortex 1	LH_PFCl_1	-30	44	30
	Medial 1	LH_Med_1	-6	20	34
	Medial 2	LH_Med_2	-12	-34	46

	Medial 3	LH_Med_3	-6	4	62
	Temporal occipital parietal 1	RH_TempOccPar_1	58	-42	12
	Temporal occipital parietal 2	RH_TempOccPar_2	60	-26	28
	Frontal operculum insula 1	RH_FrOperIns_1	40	8	2
	Medial 1	RH_Med_1	12	-32	46
	Medial 2	RH_Med_2	8	6	52
CEN	Parietal 1	LH_Par_1	-38	-52	46
	Lateral prefrontal cortex 1	LH_PFCl_1	-44	32	20
	Precuneus 1	LH_pCun_1	-10	-74	38
	Cingulate 1	LH_Cing_1	-4	-26	34
	Parietal 1	RH_Par_1	58	-38	44
	Parietal 2	RH_Par_2	46	-62	46
	Lateral prefrontal cortex 1	RH_PFCl_1	30	58	-2
	Lateral prefrontal cortex 2	RH_PFCl_2	46	38	16
	Lateral prefrontal cortex 3	RH_PFCl_3	32	46	30
	Lateral prefrontal cortex 4	RH_PFCl_4	44	16	44
	Cingulate 1	RH_Cing_1	6	-28	34
	Medial posterior prefrontal cortex 1	RH_PFCmp_1	6	28	30
	Precuneus 1	RH_pCun_1	10	-66	42

Note. Regions of interest are selected using default parcellation in CONN, which is based on the Schaefer parcellation scheme (Schaefer et al., 2018). Abbreviations: DMN, default mode network; SAL, salience network; CEN, central executive network; LH, left hemisphere; RH, right hemisphere.

APPENDIX C

Zero-order correlations

Variable	1	2	3	4	5	6	7
1. Cumulative EA Count	--						
2. Cumulative EA Severity	.86**	--					
3. Early Childhood Stressor Count	.55**	.49**	--				
4. Early Childhood Stressor Severity	.48**	.57**	.81**	--			
5. Adolescent Stressor Count	.48**	.56**	.19	.20	--		
6. Adolescent Stressor Severity	.37**	.56**	.18	.23	.90**	--	
7. Recent Stressor Count	.21	.24*	.31**	.19	.41**	.37**	--
8. Recent Stressor Severity	.19	.28*	.29*	.21	.42**	.43**	.96**
9. SAL *C*	.06	.10	.13	.12	.08	.02	.24*
10. CEN *C*	.17	.15	.08	.08	-.02	-.07	-.06
11. DMN *C*	-.00	-.06	-.14	-.23*	.14	.07	.14
12. SAL E_{glob}	-.07	-.10	-.12	.00	-.08	-.00	-.18
13. CEN E_{glob}	-.09	-.07	-.01	-.01	-.01	.02	.12
14. DMN E_{glob}	.01	.00	.12	.11	-.15	-.13	-.05

	8	9	10	11	12	13	14
9. SAL *C*	.20	--					
10. CEN *C*	-.11	-.12	--				
11. DMN *C*	.07	.23*	.02	--			
12. SAL E_{glob}	-.16	-.66**	.23*	-.16	--		

13. CEN E_{glob}	.14	.21	-.76**	.01	-.28*	--	
14. DMN E_{glob}	-.01	-.02	-.09	-.63**	-.07	.08	--

Note. -- indicates the diagonal (variable correlated with itself). * $p < .05$. ** $p < .01$. *C* = clustering coefficient. DMN = default mode network. CEN = central executive network. EA = early adversity. E_{glob} = global efficiency. SAL = salience network.

APPENDIX D

Table D.1. Associations between cumulative stressor count and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Cumulative stressor count	.01	.41	.01	.92		.07	.56	-.16	.20		.11	.42	.02	.85
Age	-.02	.94	-.10	.67		-.20	.40	.18	.46		.02	.93	.04	.87
Sex	-.12	.62	.04	.88		-.01	.97	.15	.54		.01	.97	-.13	.58
Parental education	-.01	.50	.06	.07		.01	.81	-.02	.47		-.01	.74	.01	.71
Mean FD	-2.1	.13	-.59	.66		-.00	.99	-.65	.63		.46	.74	-1.1	.43

Note. FD, framewise displacement.

Table D.2. Associations between cumulative stressor severity and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Cumulative stressor severity	-.04	.75	.08	.54		.11	.38	-.11	.39		.13	.31	-.03	.82
Age	-.01	.96	-.11	.64		-.21	.38	.18	.44		.01	.96	.04	.85
Sex	-.14	.57	.03	.89		.01	.92	.18	.47		-.01	.97	-.14	.57
Parental education	-.03	.34	.06	.05		.01	.77	-.02	.56		-.01	.76	.01	.78
Mean FD	-1.7	.22	-.78	.57		-.13	.93	-.77	.58		.37	.79	-.95	.49

Note. FD, framewise displacement

Table D.3. Associations between early childhood stressor count and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Early childhood stressor count	-.16	.20	.18	.14		.13	.27	-.12	.33		.07	.58	.01	.95

Age	-.02	.94	-.10	.67		-.20	.40	.17	.47		.03	.92	.04	.86
Sex	-.17	.48	.07	.77		.01	.98	.15	.54		.01	.98	-.14	.57
Parental education	-.03	.25	.06	.03		.01	.76	-.02	.56		-.01	.66	.01	.73
Mean FD	-1.64	.21	-.76	.56		.04	.97	-.94	.48		.66	.62	-1.04	.44

Note. FD, framewise displacement

Table D.4. Associations between early childhood stressor severity and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Early childhood stressor severity	-.24	.04	.17	.16		.13	.30	.01	.96		.06	.64	.01	.93
Age	-.01	.96	-.10	.65		-.20	.40	.17	.47		.02	.92	.04	.86
Sex	-.14	.56	.03	.89		-.02	.93	.18	.48		-.01	.98	-.14	.56
Parental education	-.04	.19	.06	.03		.01	.78	-.01	.70		-.01	.65	.01	.73
Mean FD	-1.54	.24	-.76	.56		.05	.97	-1.08	.42		.67	.62	-1.05	.44

Note. FD, framewise displacement

Table D.5. Associations between adolescent stressor count and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Adolescent stressor count	.14	.25	-.15	.24		.05	.69	-.06	.64		-.04	.75	.02	.89
Age	.07	.77	-.19	.44		-.17	.50	.14	.58		.00	1.0	.05	.84
Sex	-.16	.52	.05	.83		-.03	.91	.18	.46		-.00	.99	-.15	.56
Parental education	-.02	.43	.05	.08		.00	.89	-.01	.65		-.02	.56	.01	.72
Mean FD	-1.83	.17	-.55	.67		.19	.89	-1.07	.42		.07	.58	-1.04	.44

Note. FD, framewise displacement

Table D.6. Associations between adolescent stressor severity and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Adolescent stressor severity	.07	.54	-.13	.30		-.01	.94	.02	.86		-.08	.51	.04	.73
Age	.02	.92	.17	.48		-.21	.41	.18	.46		-.02	.93	.06	.80
Sex	-.15	.54	.05	.83		-.02	.93	.17	.49		.00	.99	-.15	.55
Parental education	-.03	.38	.05	.07		.00	.93	-.01	.70		-.02	.55	.01	.72
Mean FD	-1.80	.17	-.58	.66		.19	.89	-1.07	.42		.72	.59	-1.03	.44

Note. FD, framewise displacement

Table D.7. Associations between recent stressor count and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Recent stressor count	.13	.28	-.04	.76		.28	.02	-.23	.06		-.06	.62	.12	.34
Age	-.06	.80	-.09	.71		-.29	.21	.25	.29		.04	.86	.00	.99
Sex	-.15	.54	.04	.88		-.04	.88	.19	.44		-.00	.99	-.15	.55
Parental education	-.02	.41	-.60	.07		.01	.76	-.02	.57		-.02	.55	.01	.67
Mean FD	-1.68	.20	.05	.65		.49	.71	-1.31	.32		.67	.62	-.91	.49

Note. FD, framewise displacement

Table D.8. Associations between recent stressor severity and DMN, SAL, and CEN topology

	DMN					SAL					CEN			
	Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency			Clustering coefficient		Global efficiency	
	β	p	β	p		β	p	β	p		β	p	β	p
Recent stressor severity	.06	.62	.01	.95		.24	.04	-.21	.08		-.12	.34	.14	.24
Age	-.04	.88	-.10	.66		-.28	.24	.24	.31		.06	.80	-.00	.99
Sex	-.15	.53	.03	.89		-.08	.76	.22	.36		.02	.94	-.17	.48

Parental education	-.03	.38	-.55	.06		.01	.83	-.01	.61		-.02	.54	.01	.68
Mean FD	-1.74	.19	.06	.68		.49	.72	-1.33	.31		.59	.67	-.86	.52

Note. FD, framewise displacement