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Early childhood household instability, adolescent structural neural network architecture, and young adulthood depression: A 21-year longitudinal study

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ABSTRACT

Unstable and unpredictable environments are linked to risk for psychopathology, but the underlying neural mechanisms that explain how instability relate to subsequent mental health concerns remain unclear. In particular, few studies have focused on the association between instability and white matter structures despite white matter playing a crucial role for neural development. In a longitudinal sample recruited from a population-based study ($N = 237$), household instability (residential moves, changes in household composition, caregiver transitions in the first 5 years) was examined in association with adolescent structural network organization (network integration, segregation, and robustness of white matter connectomes; $M_{age} = 15.87$) and young adulthood anxiety and depression (six years later). Results indicate that greater instability related to greater global network efficiency, and this association remained after accounting for other types of adversity (e.g., harsh parenting, neglect, food insecurity). Moreover, instability predicted increased depressive symptoms via increased network efficiency even after controlling for previous levels of symptoms. Exploratory analyses showed that structural connectivity involving the left fronto-lateral and temporal regions were most strongly related to instability. Findings suggest that structural network efficiency relating to household instability may be a neural mechanism of risk for later depression and highlight the ways in which instability modulates neural development.

1. Introduction

Childhood adversity is experienced by 40 % of individuals and is linked to close to 30 % of mental health disorders (Kessler et al., 2010; McLaughlin et al., 2012). Early adversity can increase susceptibility for psychopathology later in life through modulation of critical neural systems as demonstrated by both animal (Sánchez et al., 2001) and human studies (Gur et al., 2019; McLaughlin et al., 2019; Nelson and Gabard-Durnam, 2020; Tottenham et al., 2010).

Though theoretical and empirical work has linked several types of adversities to specific developmental and neurobehavioral outcomes, little research has examined the links between unstable environments with human brain development despite growing interest in cross-species translation of neural mechanisms associated with unpredictability (Ellis et al., 2022; Gee, 2021; McLaughlin et al., 2021). Highly variable or stressful environments (e.g., limited bedding or nesting, maternal separation or unpredictable rearing) have primarily been linked to synaptic maturation in rodents (Bath et al., 2016; Guadagno et al., 2020; Ono et al., 2008; Strzelewicz et al., 2019; Walker et al., 2017), and studies focusing on unpredictability and human brain development have only

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recently emerged (Granger et al., 2021).

Instability can create unique challenges as environments that are constantly shifting heighten demands for adaptation and increase the production of stress hormones (Coplan et al., 2001; Martf and Armario, 1997; Muir and Pfister, 1986) that play important roles for neural regions implicated in socioemotional development (Dallman et al., 2004; McEwen, 2008). These effects are especially pronounced during early childhood when rapid neural development is occurring, with long-lasting implications through adolescence and young adulthood (Hensch and Bilimoria, 2012; Luby et al., 2020; Pechtel and Pizzagalli, 2011). For example, unstable environments marked by frequent residential moves (Ziol-Guest and McKenna, 2014) and family instability (Fomby and Osborne, 2017; Mitchell et al., 2015) during early childhood were linked to increased child internalizing and externalizing behaviors. Similarly, environmental unpredictability was associated with increased behavioral problems in adolescence (Belsky et al., 2011), specifically when these changes were experienced during early years of childhood (Doom et al., 2016).

These observed differences in both neural development and mental health outcomes could potentially vary as a function of instability that reflects environmental adaptation. Such inference is consistent with the life history strategy theory (Belsky et al., 2011; Promislow and Harvey, 1990), which posits that early experiences shape one's strategy for survival, leading to certain phenotypic traits that increase the organism's chances of survival and reproduction. These patterns were established in animal models such as squirrels (Dantzer et al., 2013), birds (Martin, 1995), nonhuman primates (Pereira and Fairbanks, 2002), and rodents (Careau et al., 2009), whereby variations in the environment are associated with reproductive behaviors and offspring growth. Such increased growth, however, could come with a potential trade-off cost of shorter lifespans. The "weathering hypothesis" for instance, posits that exposures to adversities could increase allostatic load and accelerate aging (Geronimus, 1992; McEwen, 2007), suggesting that while increased pace of development could be advantageous in the short-term, it could also pose long-term health consequences.

Despite evidence linking unstable early environments, neural development, and mental health, no study to date has examined environmental unpredictability and white matter networks. White matter structures are particularly important markers of neural development as myelination of white matter solidifies neural connections and limits the extent to which the brain remains plastic and sensitive to the environment (Hensch and Bilimoria, 2012). While unpredictable maternal signals have been linked to density of preadolescence white matter corticolimbic structures (Granger et al., 2021), little is known about how unstable environment across early childhood relate to network organization of structural connections. Furthermore, most work examining white matter tractography relating to early adverse experiences and psychopathology have focused on specific major white matter tracts (Granger et al., 2021; Hanson et al., 2013; Hein et al., 2018) or microstructures (Goetschius et al., 2020b; Hardi et al., 2022a), which do not capture the spatial characteristics of structural networks that represent information exchange in the brain (Bullmore and Sporns, 2009; Menon, 2011; Rubinov and Sporns, 2010). Measures of network organization can be accomplished by applying graph analysis to reconstructed white matter fibers streamlines using diffusion magnetic resonance imaging (dMRI) data (Nucifora et al., 2007). Network integration quantified by global efficiency represents how quickly information can travel across the brain through network connections (Bullmore and Sporns, 2009). Network organization can also be characterized by the presence of interconnected nodes (i.e., clusters) that quantify network robustness, which signifies the degree to which structural integrity is retained when the network is perturbed (e.g., removal of any individual node) (Bullmore and Sporns, 2009). Finally, network segregation measured by the modularity index represents the extent to which a network can be subdivided into separate modules (Bullmore and Sporns, 2009). These network organization measures leverage the strength of structural connectivity among brain regions to provide valuable insight into how information is transmitted across the brain via white matter structures.

Beyond examining the direct link between unstable early environment and neural mechanisms relating to mental health, more work is needed to establish evidence for neural specificity linked to different types of childhood experiences. Extant research on childhood adversity has largely focused on distinguishing the effects of emotional and cognitive enrichment that parents are able to provide to children (Brooks-Gunn and Duncan, 1997; McLoyd, 1998); parent-child interaction (e.g., harsh or supportive parenting) (Chang et al., 2003; Gard et al., 2017; Wiggins et al., 2015); or differential dimensions of adversity across threat-related or deprivation exposures (Goetschius et al., 2020a, 2020b; Hein et al., 2020; McLaughlin et al., 2019; Sheridan and McLaughlin, 2014). These efforts have collectively shown that specific types of childhood experiences could have differential impact on children's brain and socioemotional development. In regards to unpredictability, events such as residential moves and caregiver transitions contain elements of stochasticity that may not be present in the experiences of poverty or parental harshness (Belsky et al., 2011; Ellis et al., 2022).

The present investigation sought to examine the neural developmental pathways between unstable environments and white matter structures by testing three aims: (1) examine the association between childhood household instability (i.e., residential moves, household composition, and caregiver transitions during first five years of life) (age 0–5) and adolescent structural neural network architecture (age 15); (2) determine the specificity in childhood adversity by examining the associations between household instability and structural networks are distinct from the association of other types of adversity (i.e., harsh parenting, neglect, poverty) and structural organization; and (3) test indirect effects of household instability through structural networks on prospective anxiety and depressive symptoms during young adulthood (age 21). Additionally, to provide specificity, an exploratory aim of the study was to identify specific regions that may be particularly important by examining the association of early instability and structural connectivity strength of network regions (overall, within-region, between-regions). We hypothesized that greater childhood instability would be related to patterns of more developed structural networks that are more integrated (i.e., increased global efficiency), more robust (i.e., increased transitivity), and less segregated (i.e., decreased modularity) (Hagmann et al., 2010), and that these associations would be distinct from associations with other types of adverse experiences. Furthermore, we hypothesized that instability would predict subsequent symptoms of anxiety and depression via individual differences in white matter organization.

2. Material and methods

2.1. Samples and procedures

Participants were recruited from Fragile Families and Child Wellbeing Study (FFCWS), a population-based sample of 4898 children born in large cities in the United States (population over 200,000) with a 3:1 oversampling for non-marital births (Reichman et al., 2001). Given this sampling strategy (i.e., urban births to unmarried parents), low-income families were disproportionately represented in the FFCWS. These children were followed throughout childhood and when children were 15–17 years old, a cohort of 237 families from midwestern sites (Detroit, MI; Toledo, OH; Chicago, IL) participated in the Study of Adolescent Neural Development (SAND) ($N = 237$; mean age 15.87 years; 52 % females, 76 % Black, median household income $36,195; descriptive statistics on included sample are in Table S2). Six years later (coinciding with the COVID-19 pandemic), participants self-reported their anxiety and depressive symptoms through online and phone interviews. All participants provided informed consent or verbal assent as minors with parents' consent at each wave, and study protocols were approved by the University of Michigan ethics committee (IRB: HUM00167754; HUM00074392).

2.2. Household instability

The early household instability construct was adapted from prior literature examining environmental unpredictability (Belsky et al., 2011; Doom et al., 2016) and applied to the Fragile Families and Child Wellbeing Study longitudinal data (Reichman et al., 2001). The construct of early household instability in the present study includes the extent of residential moves (i.e., number of times family moved from one wave to the next), change in household composition (i.e., difference in the number of individuals living within the home between waves), and change in living situation with caregivers (i.e., mother, father, mother's cohabitating partners, and grandparents moved in and out of the home between waves) during the first five years (between ages 0 and 1, 1 and 3, 3 and 5). Scores across each component were then standardized and summed to create an overall household instability score for each individual. More details on this construct are available in the Supplement.

2.3. Neuroimaging measures

2.3.1. Data acquisition and preprocessing

Magnetic Resonance Imaging (MRI) scans were acquired using 3 T GE Discovery MR750 scanner with 8-channel head coil at the University of Michigan Functional MRI laboratory. Head movement was limited through the use of head paddings and detailed instructions provided to participants. T1-weighted gradient echo images were first captured (TR = 12 ms, TE = 5 ms, TI = 500 ms, flip angle = 15°, field of view = 26 cm, slice thickness=1.44 mm, 256 × 192 matrix, 110 slices). Diffusion MRI (dMRI) data were then acquired using spin-echo EPI diffusion sequence using repetition time of 7250 ms, minimum echo time, 128 × 128 acquisition matrix, FOV = 22 cm, 3 mm no-gap thick slices with 40 slices acquired using alternating-increasing order, b-value of 1000 s/mm^2, 64 non-linear directions. dMRI images were first inspected visually for quality, and slices with an average intensity < 4 standard deviations or more were marked as outliers and replaced with predicted models (Andersson et al., 2016). Participants were excluded if more than 5 % of slides were replaced and images for 10 participants who had most replaced slices were further visually inspected. These data were also utilized and described in previous publications (Calabrese et al., 2022; Goetschius et al., 2019, 2020b; Hardi et al., 2022a; Hein et al., 2018).

In the present investigation, preprocessed dMRI data were then processed using the MRtrix pipeline to estimate structural connectivity (Tournier et al., 2012). MRtrix utilizes a novel tensor-fitting method called the Constrained Spherical Deconvolution (CSD) (Farquharson et al., 2013; Tournier et al., 2004, 2007) that outperforms diffusion tractography imaging (DTI) or other conventional deterministic methods, especially in crossing fibers regions (Tournier et al., 2012). This improved method of tracking white matter fibers is further strengthened by the addition of Anatomical Constrained Tractography algorithm, which takes into account other biological tissues (e.g., cerebral spinal fluid) during estimation to restrict fiber tracking only to anatomically plausible fibers (Smith et al., 2012). These advancements in methodology improve the ability to estimate white matter fibers more precisely using dMRI data. Ten million streamlines were generated using probabilistic tractography, which were then combined with nodes from the anatomical AAL2 atlas (Rolls et al., 2015) to create a 94 × 94 individualized connectome matrix representing the number of streamlines or structural connectivity (i.e., edges) between each brain regions (i.e., nodes). Additionally, to examine regional specificity, these nodes were subdivided into 8 brain regions based on AAL2 anatomical parcellation (Rolls et al., 2015): frontal lateral, frontal medial, orbitofrontal, temporal, limbic, subcortical, parietal, and occipital (Fig. 1). Details on specific nodes and coordinates are in Table S5 and more specific details on steps of MRtrix pipeline are available in the Supplement.

2.3.2. Graph analysis

The resulting white matter connectomes were subsequently processed as weighted, undirected, and unthresholded (Civier et al., 2019) graphs using the Brain Connectivity Toolbox (Rubinov and Sporns, 2010) in MATLAB to generate three weighted global metrics of network architecture: global efficiency (Latora and Marchiori, 2001; Onnela et al., 2005; Wang et al., 2017); transitivity (Opsahl and Panzarasa, 2009); and modularity (Newman, 2006; Reichardt and Bornholdt, 2006). Additionally, a localized metric of nodal strength (i.e., strengths of edge connections attached to individual nodes) (Sporns, 2002) was computed and within-region strength (i.e., strength of connections that are within the same region) and between-region strength (i.e., strength of connections between one region and other regions) using custom codes in R. For a weighted graph, global efficiency is the inverse of path with greatest structural connectivity within the network. Thus, highly efficient networks typically contain strong network connections that facilitate faster information transfer within the network (Bullmore and Sporns, 2009). As a measure of network clustering, transitivity was utilized in this study to reduce bias of identifying clusters in a weighted graph (Opsahl and Panzarasa, 2009). Transitivity is a localized measure of efficiency, and captures the presence of triangles in the network. A greater transitivity score indicates a greater number of triangles and often signifies the robustness of information transfer when individual nodes are removed on a local level (Bullmore and Sporns, 2009). Modularity is a measure of network segregation and quantifies the extent to which the network can be subdivided into separate modules (Bullmore and Sporns, 2009); greater modularity score indicates a more segregated network. These metrics were computed for each person

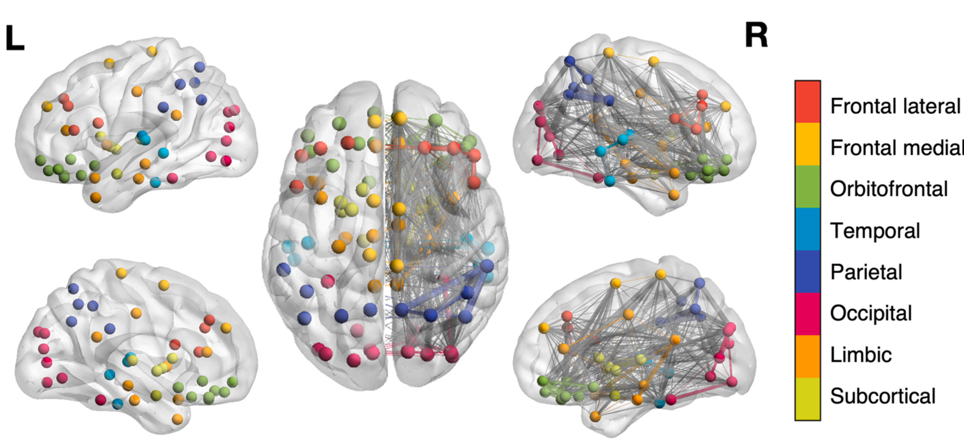

Fig. 1. Structural networks subdivided into 8 subregions. Structural nodes and edges for one subject. Structural networks were subdivided into 8 regions based on the AAL2 anatomical parcellation (Rolls et al., 2015). Details on each node and coordinates are available in Supplemental. Within-region connections are depicted in the same color as the subregion, while between-regions connections are shown in grey. Thickness of edges (i.e., connections) differ based on edge weight (i.e., structural connectivity strength). Edges shown in 25 % sparsity for visualization purposes only and figures were created using BrainNet Viewer (Xia et al., 2013).

based on individual structural connectomes and extracted for further analysis. More information on their computation is in the Supplement.

2.4. Anxiety and depressive symptoms

Anxiety and depressive symptoms during young adulthood (age 21) were self-reported using the 21-item Beck Anxiety Inventory (BAI) (Beck et al., 1988) and 20-item Beck Depression Inventory (BDI) (Beck et al., 1996). The scales showed good internal reliability (Cronbach's $\alpha = 0.95$ for BAI; Cronbach's $\alpha = 0.91$ for BDI). These data were collected during the early peak of the COVID-19 pandemic (between April 30th, 2020 and June 26th, 2021).

2.5. Covariates

The following covariates were included to adjust for demographical characteristics: gender (male, female); ethnoracial identity (Black, white, Hispanic/LatinX, other), a social construct included to control for differences in exposure to structural racism and other discrimination; birth city (Detroit, Toledo, Chicago, other), to account for any sampling differences by geographical location; pubertal development (parent and self-reported at age 15), to account for neural developmental stemming from puberty; economic hardship (parent-reported at ages 1, 3, 5), to account for differences in socioeconomic circumstances. The following variables were also included to examine the specificity of instability in predicting neurobehavioral outcomes versus other adverse experiences during early childhood: harsh parenting (standardized sum score of parent-reported physical violence and psychological aggression subscales in the Conflict Tactics Scale at ages 3 and 5, and parent-reported spanking at age 1); neglect (sum score of parent-reported neglect in the Conflict Tactics Scale at ages 3, 5); food insecurity (parent-reported at ages 3, 5). In addition, to ensure that anxiety and depressive symptoms during young adulthood were not confounded by earlier levels of symptoms, pre-pandemic levels of symptoms (scores measuring anxiety and depression using self-reported Screen for Anxiety Related Disorders (Birmaher et al., 1997) and Mood and Feelings Questionnaire (Angold et al., 1995) at age 17 (Cronbach's $\alpha = 0.95$ and Cronbach's $\alpha = 0.96$ respectively) were also accounted for during sensitivity analyses. More details on these constructs and sensitivity analyses are available in Supplement.

2.6. Statistical analyses

All statistical analyses were conducted using R Statistical Software v4.2.1 and MPlus v8 (Muthén and Muthén, 2017). For the first aim, zero-order correlations between early childhood instability and structural network properties (i.e., global efficiency, transitivity, modularity) were examined. Distribution of each variable was examined, and models were tested without extreme or influential outliers as robustness checks. For the second aim, a path model was tested using MPlus v8 with early instability, other types of early adversity, and demographic covariates (gender, puberty, ethnoracial identity, birth city, economic hardship) predicting structural network metrics (global efficiency, transitivity, modularity). For the third aim, indirect effects models predicting anxiety and depressive symptoms during young adulthood were separately tested in association with instability, other adverse experiences, and demographic covariates. Pre-pandemic symptoms were subsequently also added in the mediation model as sensitivity analyses (see Supplement for additional information on robustness checks and sensitivity analyses). Finally, for the fourth exploratory aim, zero-order correlations were tested between childhood instability and structural connections of each lateral subregion. False Discovery Rate (FDR) (Benjamini and Hochberg, 1995) was applied to correct for multiple comparisons across 8 structural subregions. Path models were assessed using standard fit indices (Hu and Bentler, 1999) and were utilized to account for shared variance across multiple types of adverse experiences while retaining the full number of participants in the sample. Full Information Maximum Likelihood estimation with robust standard errors (MLR) was used to account for missing data and to deviations in normality of variables and residuals for the path models tested using Mplus. For the models examining zero-order correlations between instability and network metrics or subregion-specific structural connections, subjects with missing household instability data were listwise deleted ($N = 161$). There were no demographical differences across the full sample ($N = 237$) and subsample with missing instability data ($N = 161$) (Table S4), and results do not differ when path models were tested using either sample (see Supplement).

3. Results

3.1. Early instability was related to greater adolescent structural networks efficiency

Greater instability during early childhood was related to greater global efficiency in structural networks ($b^* = 0.180$, $p = .028$; Fig. 2). Instability was not related to clustering or modularity (transitivity: $b^* = 0.143$, $p = .149$; modularity: $b^* = -0.062$, $p = .432$) (Fig. 2). The association between instability and global efficiency remained after adjusting for covariates ($b^* = 0.164$, $p = .042$) (see Table S3 for zero-order correlations of all variables).

3.2. Associations between early childhood instability and adolescent structural network efficiency remained after accounting for other types of early adverse experiences

The association between early instability and global efficiency remained after controlling for harsh parenting, neglect, and food insecurity (b^* [SE] $= 0.183$ [0.077], $p = .017$) (Fig. 3). Beyond instability, increased harsh parenting also related to increased transitivity (b^* [SE] $= 0.312$ [0.142], $p = .029$). The path model showed excellent model fit (CFI = 0.986, TLI = 0.968, RMSEA = 0.036, SRMR = 0.042) and included controls for all demographic characteristics (gender, ethnoracial identity, puberty, birth city, economic hardship; see Fig. S1 for full path model).

3.3. Greater structural network efficiency in adolescence was related to increased depressive symptoms in young adulthood. Household instability predicted depressive symptoms via network efficiency

Greater adolescent global efficiency was related to increased depressive symptoms during young adulthood (b^* [SE] $= 0.523$ [0.168], $p = .002$). Further, instability predicted depressive symptoms later in adulthood via global efficiency during adolescence (specific indirect effect via global efficiency b^* [SE] $= 0.100$ [0.049], $p = .042$) (Fig. 4). There were no indirect effects via transitivity (b^* [SE] $= -0.041$ [0.031], $p = .190$) or modularity (b^* [SE] $= -0.007$ [0.011], $p = .510$). The model showed excellent fit to the data (CFI = 0.987, TLI = 0.957, RMSEA = 0.035, SRMR = 0.039; see Fig. S2 for full path model). Additionally, results remained after sensitivity analyses including previous levels of depressive symptoms (instability related to global efficiency b^* [SE] $= 0.179$ [0.077], $p = .021$; efficiency related to depression b^* [SE] $= 0.582$ [0.156], $p < .001$; specific indirect effect via efficiency b^* [SE] $= 0.104$ [0.052], $p = .043$; model fit: CFI = 0.988, TLI=0.947, RMSEA=0.035, SRMR = 0.037). There were no significant direct or indirect effects between instability, structural networks, and anxiety symptoms (model fit: CFI = 0.986, TLI = 0.955, RMSEA = 0.036, SRMR = 0.040; association between anxiety and global efficiency b^* [SE] $= 0.201$ [0.156], $p = .196$; indirect association through global efficiency b^* [SE] $= 0.038$ [0.032], $p = .245$). Furthermore, beyond instability, harsh parenting was associated with transitivity (b^* [SE] $= 0.317$ [0.142], $p = .025$), but transitivity was not related to symptomatology, nor did transitivity indirectly explain the

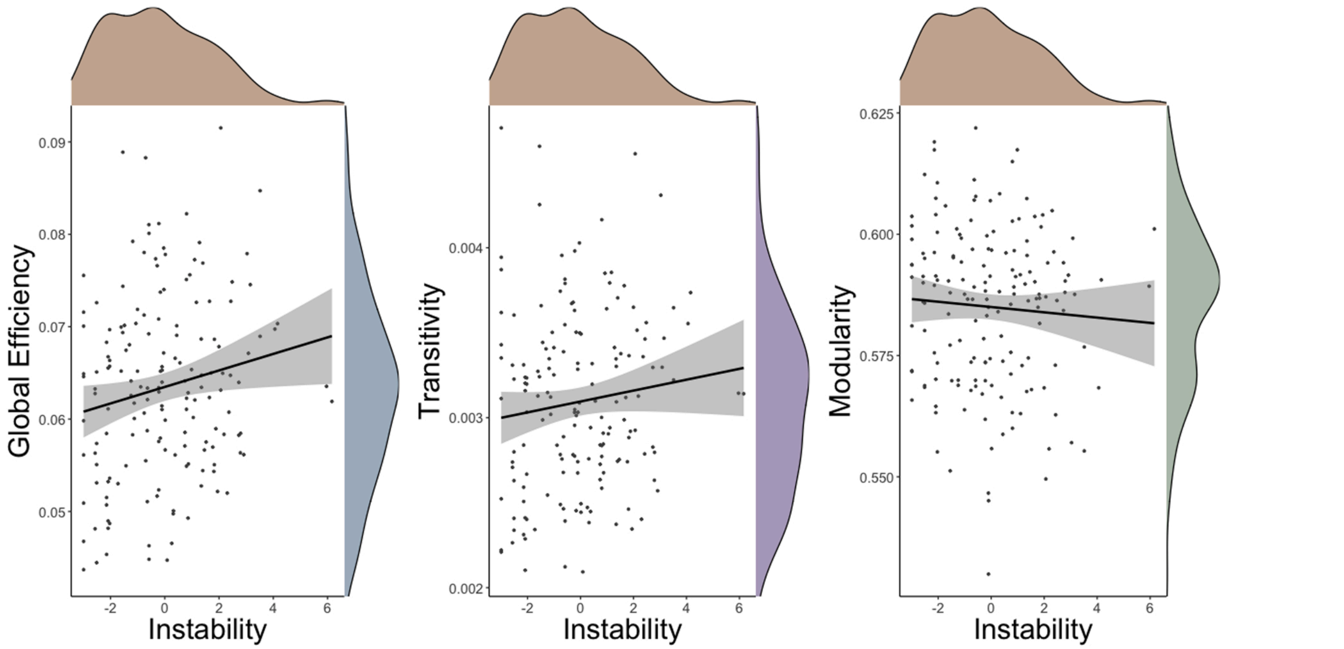

Fig. 2. Associations between household instability and white matter structural networks. Zero-order correlations between instability and structural network properties. From left to right: greater instability was related to greater structural network efficiency ($b^* = 0.173$, $p = .028$), but not transitivity ($b^* = 0.143$, $p = .149$) or modularity ($b^* = -0.062$, $p = .432$). Distributions for each variable are shown in brown (instability), blue (global efficiency), purple (clustering), and green (modularity). Outliers ($n = 2$) were omitted for ease of visualization; results were consistent with or without inclusion of outliers. Household instability was represented by standardized scores.

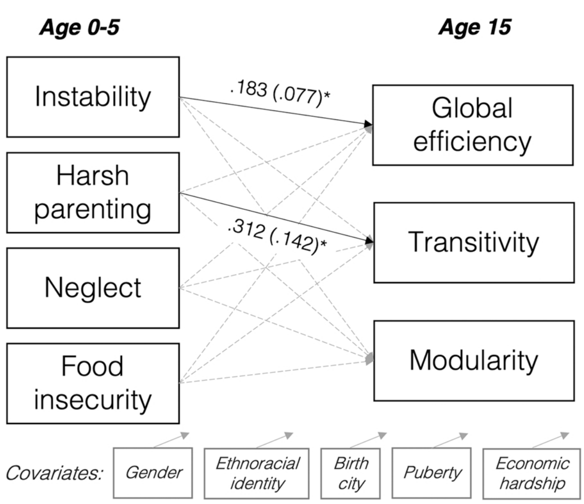

Fig. 3. Path model testing associations among early instability, other types of childhood adversity, and adolescent structural networks. Associations between instability and global efficiency remained (b^* [SE] = 0.183 [0.077], $p = .017$) even after adjusting for other types of early adversity (i.e., harsh parenting, neglect, food insecurity). Additionally, harsh parenting was also associated with greater transitivity (b^* [SE] = 0.312 [0.142], $p = .029$). Model was adjusted for demographic covariates (gender, ethnoracial identity, birth city, puberty, economic hardship) and had excellent fit (CFI = 0.986; TLI = 0.968; RMSEA = 0.036; SRMR = 0.042). Standardized coefficients are shown, and dotted path lines indicate non-significant estimated paths.

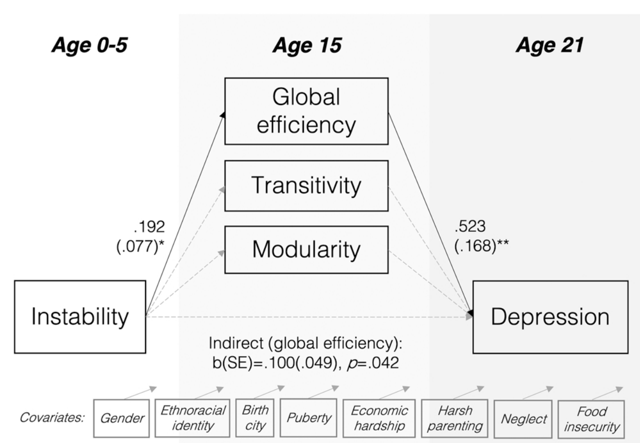

Fig. 4. Early household instability indirectly related to depression at young adulthood via adolescent structural network efficiency. Childhood instability was related to greater structural network efficiency ($b^*[SE] = 0.192$ [0.077], $p = .013$), which in turn related to greater depressive symptoms at young adulthood ($b^*[SE] = 0.523$ [0.168], $p = .002$). Global efficiency indirectly explains the association between instability and depression ($b^*[SE] = 0.100$ [0.049], $p = .042$). Model had excellent fit (CFI = 0.987; TLI = 0.957; RMSEA = 0.035; SRMR = 0.039) and was adjusted for all covariates (gender, ethnoracial identity, birth city, puberty, economic hardship, harsh parenting, neglect, food insecurity). Standardized coefficients are shown, and dotted path lines indicate non-significant estimated paths.

association between harsh parenting and depression (b^* [SE] = $-$ 0.101 [0.067], $p = .131$).

3.4. Structural connectivity of the fronto-lateral and temporal regions was associated with early instability

Exploratory analyses on to identify regional specificity found that early instability was most strongly related to structural connectivity strength of the left frontolateral nodes ($b^* = 0.23$, $q = 0.029$) (Fig. 5). Furthermore, early instability was related to between-region strength connectivity (i.e., connectivity among left frontolateral nodes and all other regions) ($b^* = 0.22$, $q = 0.037$), as well as between-region connectivity among left temporal nodes and all other regions ($b^* = 0.20$, $q = 0.037$) (Fig. 5). All results from subregional connectivity analyses

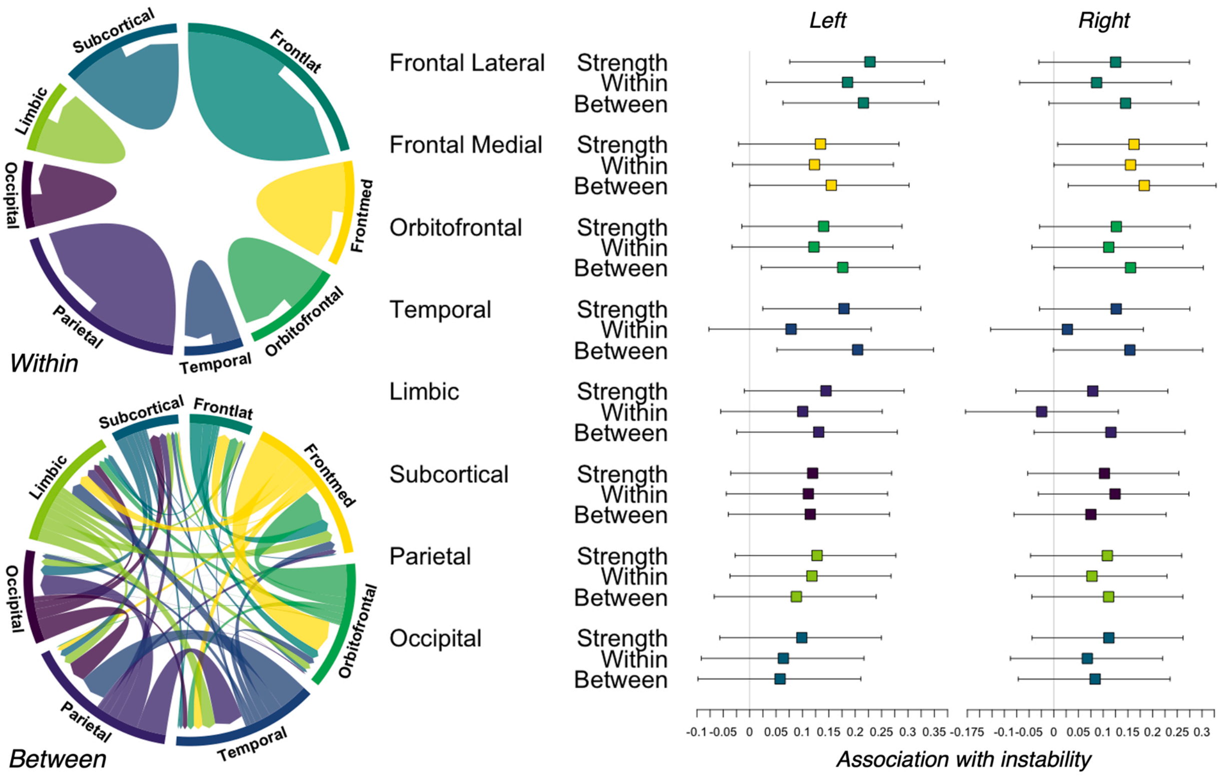

Fig. 5. Associations between regional structural connectivity and early instability.
LEFT: Circular plots illustrating within-region (i.e., connections between nodes within each region) and between-regions (i.e., connections between nodes of each region with all other regions) structural connectivity of one individual in the sample.
RIGHT: Instability was particularly associated with the overall strength of structural paths connected to the left frontal lateral nodes ($b^* = 0.23$, $q = 0.029$). Additionally, instability was related to connections between left frontal lateral nodes and other regions ($b^* = 0.19$, $q = 0.037$), as well as connections between left temporal nodes and other regions ($b^* = 0.20$, $q = 0.037$). Each square box denotes standardized estimate of the association between instability and each subregion connectivity metrics, and whiskers indicate confidence intervals.

are available in Table S6.

4. Discussion

Using a longitudinal design across 21 years, childhood household instability during the first five years of childhood was associated with greater structural network efficiency in adolescence, which in turn predicted to greater depressive symptoms in young adulthood. Furthermore, structural network efficiency indirectly explained the association between instability and depression, and these associations remained even after accounting for other types of adversity (i.e., harsh parenting, neglect, food insecurity) and earlier symptoms of depression. Moreover, exploratory findings showed that associations with structural connectivity were strongest within the left frontolateral subregion and between temporal and other subregions were associated with greater instability. These results suggest that instability during early childhood was related to greater structural network efficiency, particularly in regions important for regulation and cognition, and that these associations may have consequences for mental health later in life. These findings demonstrate the potential long-term neurobehavioral consequences of exposure to early instability and highlight a model in which early instability may lead to greater risk for psychopathology via modulation of structural white matter networks.

Increased attention has been placed recently to identify the differential influence of unpredictability and instability for child development (Ellis et al., 2022; Gee, 2021; McLaughlin et al., 2021). Our findings here are one of the first to establish associations between household instability and white matter network organization in relation to mental health. While previous work has focused on unpredictability within the caregiving environment through observations of mother-child dyads at a more granular timescale (Granger et al., 2021), the present results provide complementary evidence by examining neural mechanisms associated with unstable environment across several years. While parent-child interaction is critical for understanding how caregiving environments impact brain development, a multi-year snapshot of the child's environment could capture the potential accumulation of stress resulting from unstable environment. The interplay between these constructs would be an intriguing avenue for further inquiry as instability at large timescales is likely to be correlated with instability at smaller timescales. For instance, frequent residential moves can lead to an array of experiences such as changes in schools or routines that create instability on a daily basis. Furthermore, instability caused by relocations or fathers moving in and out of the home could also have downstream effects on child development through caregiving practices, given that these events could also function as a potential source of stress for the mothers.

Greater levels of instability during early childhood in the present investigation was associated with network organization that facilitates faster information flow in the brain (i.e., greater global efficiency)—a pattern commonly observed with brain maturation (Chen et al., 2013; Hagmann et al., 2010; Richmond et al., 2016; Vértes and Bullmore, 2015). It is possible that this increased efficiency in structural network reflects an adaptive response to household instability, though with potential long-term implications for mental health. Such inference is consistent with theoretical models such as life history theories (Belsky et al., 2011; Promislow and Harvey, 1990) and the stress acceleration hypothesis (Callaghan and Tottenham, 2016), which posit that less predictable early environments or increased exposure to early life stress accelerates biological maturation to increase organism survival, though potentially at later costs. Relatedly, evidence on the "weathering hypothesis" (Geronimus, 1992; McEwen, 2007) have also demonstrated how adverse environments are associated with increased allostatic load, more rapid aging, and health inequalities, especially among Black mothers. For humans, the prolonged juvenile period may be developmentally advantageous as it allows for continued sensitivity to the environment. Thus, a reduced window of plasticity due to early maturation of neural structures may limit opportunities for subsequent enrichment, potentially increasing susceptibility to psychopathology. From a network theoretical perspective, increased efficiency relating to greater risk for mental health may reflect a greater topological cost of maintaining a more densely structured networks (Bullmore and Sporns, 2012). While greater network density could improve communication efficiency, greater allostatic load and vulnerability to environmental stressors could occur if such increase in wiring cost were attained prematurely and constrain future development and adaption to later contexts.

Despite these inferences, conclusive evidence for accelerated neural development relating to instability requires multiple timepoints of neuroimaging data, which is not possible in present investigation. Even though many studies have found efficiency of structural networks to increase with age (Chen et al., 2013; Hagmann et al., 2010; Richmond et al., 2016; Vértes and Bullmore, 2015), there are evidence to show that global efficiency of streamline-based structural connectivity decreases (Koenis et al., 2015) or stabilizes during adolescence (Koenis et al., 2018). It is important to note however, that current developmental trajectories of structural network organization are predominantly based on samples that are markedly distinct from present investigation. This emphasizes the critical need for developmental trajectories of brain development using more diverse and representative samples (Falk et al., 2013; Garcini et al., 2022; Ricard et al., 2022) to allow for a more definitive understanding of how white matter structures change across time.

The associations between household instability with structural networks was observed after adjusting for other types of early adverse experiences, suggesting that instability could have unique neural correlates for mental health. Additionally, whereas instability had the strongest association with global efficiency, harsh parenting during early childhood was related to greater network clustering. Furthermore, although harsh parenting was related to more clustered networks, transitivity was not related to symptomatology. These results suggest that instability is qualitatively different from exposure to harsh parenting and that different types of early adverse experiences may contribute to the development of different elements of network organization that are potentially implicated in psychopathology. These findings are also in line with the integrated model of childhood adversity (Ellis et al., 2022; McLaughlin et al., 2021) that proposed distinct neural mechanisms through which different types of adverse experiences (i.e., threat, deprivation, unpredictability) could lead to psychopathology. Further research is warranted to identify the common facets between the different types of adversity (e.g., household instability and harsh parenting), and whether the intersections of multiple types of adverse experiences may contribute to more unique neural mechanisms relating to psychopathology or, conversely, whether an environment with differential combinations of adverse experiences can contribute to similarities in neural correlates (i.e., multifinality and equifinality; Cicchetti and Rogosch, 1996).

While the present study does not examine the directional nature of household changes (e.g., whether the child moved into a better or worse neighborhood; or whether a grandparent's move into the home produced beneficial caregiving support or introduced novel stress for the child), these findings suggest that changes in either direction were linked to the organization of white matter networks. This is consistent with evidence showing that adjustment to both positive and negative life changes can produce stress with consequences for health (Brown and McGill, 1989). However, future investigations on the destination of residential moves or children's perception of these changes are warranted, in addition to their developmental timing effects. For instance, Moving to Opportunity (Chetty et al., 2016), a randomized control trial in which families were given the opportunity to move to a lower-poverty area or remain in their homes, found that relocating to a more economically affluent neighborhood produced more beneficial developmental outcomes for youth who moved during early, but not later, childhood. These findings suggest that the impact of household instability on development could be age or developmental stage specific.

In the present investigation, we found associations between efficiency with depression during COVID-19, a period marked with heightened stress. The pandemic has presented stressors such as social isolation, financial adversity, and uncertainty that increase risk for anxiety and depression, especially for young adults (Lee et al., 2020). Thus, it is possible that these elevated symptoms of depression indicate a potential susceptibility to stress. There were also no associations observed between network metrics and depression at age 17 (before the pandemic), suggesting that these effects could be unique to the circumstance of heightened stress during COVID, or could indicate that these brain-depression findings only emerge later in development when rates of depression continue to increase. Interestingly, we found no association between network connectivity and anxiety symptoms, echoing the potentially differential neural mechanisms relating to anxiety and depression during this period (Hardi et al., 2022b), thus indicating a future direction to further tease apart the distinct etiology preceding these mental health disorders.

Results on subregional structural connectivity in the current study suggests that early instability was particularly related to structural connectivity of the frontolateral and temporal regions. Specifically, the average strength of edges within the frontolateral regions (i.e., within-region connectivity strength), as well as connectivity between the frontolateral and temporal nodes with other brain regions (i.e., between-region connectivity strength) were related to greater early instability. These regions play an important role in higher-order processing and are sensitive to rearing environment in rodents (Greenough et al., 1973). More work is thus needed to further examine the significance of structural connectivity of these regions in relation to early environment and mental processes implicated in psychopathology.

Though the current study had several strengths including a 21-year longitudinal study with a well sampled and underrepresented sample, the combination of early measures of adversity with cutting-edge metrics of white matter organization, and the ability to control for multiple other types of adversity, there are a few limitations. First, longitudinal data during early childhood were collected in intervals of one to two years and no data were collected at ages 2 and 4, thus, additional changes occurring within the household (specifically, parental transitions) during that period may not be accounted for. Nevertheless, the present study captured changes across four waves within a period of five years for all individuals. Second, no information regarding experience of other types of adverse experiences (i.e., neglect, food insecurity) were collected prior to age 1; however, these experiences are unlikely to vastly differ across time, and the present investigation showed consistent results when accounting for a multitude of factors across multiple timepoints (i.e., ages 3 and 5). Third, while events such as residential moves and caregiver transitions could be unpredictable and have been deemed as such in previous works, we were not able to determine whether these events are truly unpredictable for the child in present investigation (i.e., regular moves may be interpreted as predicted by a child). Thus, more research is needed to establish whether household changes experienced by the child and family as those measured here are unpredictable. Fourth, the present investigation focused on a narrow age range (ages 0–5) as data were collected at longer and unequal intervals in subsequent waves. This limited our ability to capture developmental timing-specific effects of instability during later childhood or preadolescence; thus more research is needed to test the developmental timing hypothesis of instability using other longitudinal samples in the future. Lastly, no data on externalizing behaviors were collected at age 21.

5. Conclusion

Household instability during childhood is a known risk factor for subsequent mental health problems, but less is known about its neurodevelopmental correlates. Here, using a network analysis approach with diffusion tractography of white matter, we found that early childhood instability related to greater structural network efficiency in adolescence and these network differences were linked to greater depressive symptoms in a lower-income, well-sampled cohort of young adults who are underrepresented in neuroimaging research (Falk et al., 2013). These findings suggest that early childhood household instability was related to greater efficiency in neural networks, which, in turn increased susceptibility for mental health problems in young adulthood. At the same time, these associations may also reflect neural adaptation that is protective against increased instability early in life, thus it is possible that there may be benefits of increased efficiency in structural networks that were not examined in this study. Taken together, these results could inform interventions that promote household stability during early childhood to benefit long-term development of child mental health and well-being.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2023.101253.

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