

Associations Between Resting State Functional Connectivity and Trajectories of General Psychopathology in Emerging Adolescents

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ABSTRACT

Background: The general psychopathology “p-factor” captures shared variance across psychiatric symptoms and is associated with alterations in resting state functional connectivity (RSFC). Specifically, aberrant connectivity within and between networks responsible for higher-order cognition and attention has been concurrently related to higher levels of p-factor scores in youth. It remains unclear whether and how RSFC prospectively relates to future psychopathology during early adolescence, a developmental period during which many forms of disorders onset and worsen. **Methods:** Data from 9,344 preadolescents from the Adolescent Brain Cognitive Development (ABCD) study were analyzed to examine the relationship between baseline RSFC and trajectories of general psychopathology in early adolescence. I used longitudinal multilevel modeling to determine whether altered connectivity of the default mode (DMN), frontoparietal (FPN), salience (SN), ventral and dorsal attention (VAN and DAN), and cingulo-opercular (CON) networks were associated with between-person differences and within-person rates of change of p-factor scores over three years. **Results:** Findings indicate that reduced connectivity within-DMN and DAN, and reduced connectivity between DMN-DAN, DMN-CON, and VAN-CON were associated with higher levels of p-factors scores at baseline, and at the one-year, two-year, and three-year follow-ups. VAN-DAN and SN-CON hyperconnectivity and DMN-VAN hypoconnectivity were prospectively associated with steeper within-person quadratic rates of change in p-factor scores over time. **Conclusions:** Results suggest that altered connectivity between networks responsible for self-referential processing, filtering salient information, attention processing, and cognitive control may be a vulnerability for increased transdiagnostic psychopathology, exacerbated by significant developmental changes associated with emerging adolescence.

General Audience Abstract

Rates of co-occurring psychiatric disorders (i.e., comorbid psychopathology) are high during adolescence and are related to many negative outcomes, such as greater future psychiatric diagnoses, psychotropic medication use, and worse functional impairment. Previously, factor analytic studies have identified a general factor of psychopathology, or “p-factor”, that captures shared variance across psychiatric symptoms while accounting for cooccurrence and severity. The p-factor is associated with altered patterns of brain connectivity within and between neural networks responsible for higher-order cognition and attention. However, it remains unclear whether and how alterations in neural network connectivity prospectively relate to future psychopathology during early adolescence, a developmental period during which many forms of disorder onset and worsen. The present study used data from 9,344 preadolescents from the Adolescent Brain Cognitive Development (ABCD) study to examine the longitudinal relationship between altered brain connectivity of the default mode (DMN), frontoparietal (FPN), salience (SN), ventral and dorsal attention (VAN and DAN), and cingulo-opercular (CON) networks at ages 9-10 and p-factor scores three years later (ages 12-13). Results indicate that reduced connectivity within-DMN and DAN, and reduced connectivity between DMN-DAN, DMN-CON, and VAN-CON were associated with higher levels of p-factors scores at baseline, and at the one-year, two-year, and three-year follow-ups. VAN-DAN and SN-CON hyperconnectivity and DMN-VAN hypoconnectivity were prospectively associated with steeper rates of change in p-factor scores over time. These findings suggest that altered connectivity between networks responsible for higher-order cognitions and attention may be a vulnerability for increased psychopathology during emerging adolescence.

Introduction

Current categorical classification systems of psychological disorders fail to fully address the co-occurrence of two or more diagnoses (i.e., comorbid disorders). Rates of psychiatric comorbidity have been reported as high as 40% among adolescents, suggesting that comorbidity is the rule rather than the exception (Merikangas et al., 2010). Additionally, current categorizations of psychiatric disorders contain high levels of symptom heterogeneity (Segal et al., 2024). For example, there are 16,400 possible symptom combinations to meet criteria for major depressive disorder, with 50% of people endorsing unique symptom profiles and only 1.3% of people reporting identical symptoms (Fried & Nesse, 2015). As such, increasing research has highlighted the need to study dimensional characteristics of psychopathology across traditional diagnostic boundaries. For example, the Hierarchical Taxonomy Of Psychopathology (HiTOP) model provides a framework for understanding the high instances of comorbidity between traditional disorders and attempts to reduce heterogeneity in taxonomy by grouping together related symptoms and combining co-occurring disorders into dimensional spectra (Kotov et al., 2017). In line with the HiTOP model, prior factor-analytic studies have identified a general factor of psychopathology, often called the “p-factor”, which captures the shared variance across categorical disorders, accounting for their comorbidity and severity (Caspi et al., 2014; Caspi & Moffitt, 2018). The p-factor has been identified using various assessment modalities (e.g., self-report, parent-report, clinical interview) across the lifespan (Caspi et al., 2014; Clark et al., 2021; Laceulle et al., 2015; Michelini et al., 2019) and has neurobiological and cognitive correlates (Hoy et al., 2023). The p-factor also has demonstrated clinical relevance – higher p-factor scores are associated with greater histories of childhood maltreatment, future psychiatric diagnoses, psychotropic medication use, and worse functional impairment (Caspi et al., 2014; Caspi & Moffitt, 2018; Pettersson et al., 2018). As such, it is critical to study the neurobiological processes underlying the p-factor to identify youth at risk for developing comorbid psychopathology.

Historically, research examining the neurobiological correlates of specific psychiatric disorders have had mixed results, potentially driven in part by the aforementioned heterogeneity in clinical diagnoses, as well as the lack of specificity of neurobiological correlates to distinct forms of disorder (Goodkind et al., 2015; Romer et al., 2020, 2023; Segal et al., 2024). Taking a transdiagnostic, dimensional approach to measuring psychopathology may better map onto neural dysfunctions than case-control studies focused on specific disorder categories (Caspi & Moffitt, 2018; Kotov et al., 2017; Parkes et al., 2021). Resting-state functional connectivity (RSFC) is one method to examine neurocognitive processes underlying psychopathology (Fox & Greicius, 2010; Woodward & Cascio, 2015). Broadly, functional connectivity refers to temporal correlations of neural activation in anatomically distinct but functionally-related regions of the brain, and provides an architecture for understanding how large-scale neural networks organize information and interact with each other (Fox et al., 2005; Glover, 2011; Woodward & Cascio, 2015). When not otherwise engaged in cognitively demanding tasks, functional connectivity during the resting state can be captured by correlating spontaneous changes in blood oxygenation level-dependent (BOLD) signal in the brain. Converging work from structural, functional, behavioral, and lesion studies have identified multiple unique neural networks (Gordon et al., 2016). Of these, three RSFC networks have been identified as central to various forms of psychopathology: the default mode network (DMN); frontoparietal network (FPN); and salience network (SN).

To address why these three networks all are related to multiple psychiatric disorders, Menon (2011) proposed a triple neurocognitive network model of psychopathology, positing that dysfunction in one of these three core networks can impact the other networks, and that this altered pattern of connectivity gives way to psychopathology broadly. In the context of stimulus-driven cognitive and affective information, this theory suggests that the SN and FPN, which are networks needed for higher-order functions, should increase activation, whereas the DMN, which is active during times of self-referential thought (e.g., self-reflection, mind wandering) and when

the brain is “at rest” (Fox et al., 2005; Menon, 2011; Raichle, 2015), should decrease activation in response to external stimuli. Specifically, the SN is responsible for detecting, filtering, and integrating relevant information (Menon, 2011; Menon & D’Esposito, 2022; Seeley, 2019) and is thought to serve as a “switch” between the DMN and FPN, such that when a stimulus is important enough to warrant attention, the SN helps deactivate the DMN and engage the FPN. The FPN is a top-down control network that is activated when engaged in “cold” executive functions such as maintaining and manipulating memory, decision making, and executing goal-directed behaviors (Marek & Dosenbach, 2018; Menon & D’Esposito, 2022; Mezzacappa, 2011). Previous work has identified decreased activation in central nodes of the FPN and misattributions of salience (i.e., finding a benign stimulus to be important, or the inverse) as related to psychopathology (Marek & Dosenbach, 2018; Menon, 2011; Menon & D’Esposito, 2022). This theory posits that failure to properly engage or disengage use of any of these networks may significantly contribute to the onset and maintenance of psychopathology. Specifically, network dysfunction can manifest as (1) altered connectivity within-networks (e.g., increased/reduced co-activation among nodes within a given functional network) or (2) altered connectivity between-networks (e.g., over recruitment of individual nodes outside of a given network, or aberrant patterns of co-activation between entire functional networks). While many studies have examined alterations in RSFC in specific disorder categories, fewer studies have examined how the triple neurocognitive network model can account for comorbidity and severity of psychopathology across diagnostic categories (i.e., the p-factor).

Given that alterations within and between these three networks occur across psychiatric disorders, the triple neurocognitive network model may provide a theoretical framework for understanding functional connectivity abnormalities related to transdiagnostic psychopathology. Results from a meta-analysis identified shared RSFC alterations across eight psychiatric disorders – specifically, increased functional connectivity between the DMN and FPN and reduced

functional connectivity between the FPN and SN were commonly implicated (Sha et al., 2019). Additionally, Sha and colleagues (2019) found that both hyper- and hypoconnectivity between DMN and SN was shared across diagnoses, likely driven by unique nodes within the SN relating differently to DMN nodes. Similarly, another study demonstrated that loss of functional network segregation between DMN and FPN and DMN and SN is associated with increased psychopathology across all dimensions (Xia et al., 2018). Consistent with these results, limited work examining the association between altered RSFC and the p-factor found that reduced within-DMN RSFC and hyperconnectivity between the DMN and FPN was associated concurrently with higher p-factor scores in early adolescents (Karcher et al., 2022; Sripada et al., 2021). There is also emerging evidence that greater general psychopathology is associated with altered connectivity between the three key networks (DMN, FPN, SN) and three additional, related networks involved in bottom-up and top-down attention and cognitive control: the ventral and dorsal attention networks (VAN and DAN) and cingulo-opercular network (CON). Specifically, within-DAN hypoconnectivity and hyperconnectivity between DMN-DAN, DMN-CON, and FPN-VAN was associated with higher p-factor scores in early adolescence (Karcher et al., 2022; Lees et al., 2021; Sripada et al., 2021). Together, these findings broadly indicate that altered connectivity within- and between-networks in and related to the triple neurocognitive network model relates to general psychopathology. However, more work is needed to clarify the association between RSFC and the p-factor and whether this relationship is prospective in nature. Identifying early neurobiological markers that relate to levels and rates of change of psychopathology could help determine who is most at risk for increased psychiatric symptoms during the transition to adolescence, a vulnerability period for the onset of many disorders (Solmi et al., 2022). Previous longitudinal neuroimaging work has found that alterations in brain structure at one timepoint were associated with future p-factor scores in preadolescents (Romer et al., 2023); however, research examining relations between RSFC and the p-factor have been cross-

sectional. Given this, it is currently unclear whether altered RSFC is a risk factor for increases in general psychopathology. Patterns of neural connectivity within and between networks responsible for cognitive control (including DMN, FPN, and SN) evolve during adolescent development – network segregation, integration, and long-distance connection all improve with age in non-clinical samples (Gu et al., 2015; Morgan et al., 2018; Stevens, 2009). It is plausible that failure for networks to properly form connections and appropriately integrate and segregate during adolescence is what drives increases in psychopathology. Identifying whether altered RSFC can predict future change in p-factor scores is the first step to understanding the nature of this relationship. As adolescence is a developmental period when the brain undergoes significant neurodevelopment and many major forms of disorder have their onset (Gu et al., 2015; Morgan et al., 2018; Solmi et al., 2022; Stevens, 2009), studying the prospective relations between RSFC and the p-factor during the transition to adolescence is particularly important for identifying early neural risk markers of general psychopathology.

The present study seeks to clarify whether and how alterations in RSFC within and between three core networks (DMN, FPN, SN) prospectively relate to increases in the p-factor during emerging adolescence. To do this, I employed data from the Adolescent Brain Cognitive Development (ABCD) Study to examine whether baseline RSFC is prospectively related to the trajectory of p-factor scores from baseline to the three-year follow-up (four total timepoints). I used longitudinal multilevel modeling to test whether baseline within- and between-network RSFC of the three core networks relate to between-person differences and within-person rates of change in p-factor scores over three years. As two studies have already identified reduced within-DMN RSFC related concurrently to elevated p-factor scores in the ABCD study preadolescents (Karcher et al., 2022; Sripada et al., 2021), the first aim of the current study seeks to clarify whether reduced within-DMN connectivity prospectively predicts future increases in p-factor scores. Among the three core networks, within-DMN hypoconnectivity is the only within-network alteration that has been

indicated theoretically or in prior analyses. A majority of previous work has implicated between-network alterations as being particularly relevant to general psychopathology, including that increased connectivity between the DMN and FPN was associated concurrently with elevated p-factor scores in the ABCD study preadolescents (Sripada et al., 2021). Thus, the second aim of this study seeks to clarify how altered between-network RSFC among the three core networks prospectively relates to the trajectory of p-factor scores over time. Finally, I conducted an exploratory analysis to determine whether alterations within and between networks involved in attention and cognitive control (DMN, FPN, SN, VAN, DAN, CON) prospectively relate to p-factor scores during this developmental period.

Specific Aims/Hypotheses

AIM 1: Identify the prospective relations between alterations in DMN RSFC and general psychopathology trajectories.

H1: Reduced within-DMN connectivity will be prospectively related to higher levels of and steeper within-person increases in p-factor scores over the course of three years.

AIM 2: Determine RSFC alterations between the three core networks that prospectively relate to general psychopathology trajectories.

H2.1: Increased DMN-FPN connectivity will be prospectively related to higher levels of and steeper within-person increases in p-factor scores over the course of three years.

H2.2: Increased DMN-SN connectivity will be prospectively related to higher levels of and steeper within-person increases in p-factor scores over the course of three years.

H2.3: Reduced FPN-SN connectivity will be prospectively related to higher levels of and steeper within-person increases in p-factor scores over the course of three years.

Methods

Participants

Data from the Adolescent Brain Cognitive Development (ABCD) study were accessed through the National Institute of Mental Health Data Archive (NDA) 5.1 release (DOI: 10.15154/z563-zd24). The ABCD study consists of data from 11,875 preadolescents aged 9-10 at baseline, collected from 21 sites nationwide. Complete recruitment details have been published elsewhere (Garavan et al., 2018). Briefly, participants were excluded from the larger ABCD study for the following reasons: not being fluent in English; having a serious medical, neurological, or mental health disorder (i.e., currently diagnosed schizophrenia, moderate or severe autism spectrum disorder, intellectual disability, or substance use disorder); any contraindications to MRI scanning, including history of traumatic brain injury; pre-term birth (<28 weeks) or low birthweight (<1200g). Each study site obtained approval from their own institutional review board prior to data collection. Participants were excluded from the present study analyses for the following reasons: missing demographic information; complete nonresponse on clinical data; participated at a study site with less than 40 participants; did not pass MRI quality assurance measures according to guidelines from release 5.1 for imaging quality assurance (DOI: [10.15154/z563-zd24](https://doi.org/10.15154/z563-zd24)). A flowchart detailing participant exclusion for the present study is shown in Figure 1. The final number of participants analyzed at each timepoint are as follows: baseline n=9,344; one-year follow-up n=8,840; two-year follow-up n=8,618; three-year follow-up n=8,034.

Procedures

The present study analyses used a subset of neuroimaging and questionnaire data collected during the baseline, one-, two-, and three-year follow-up study waves. Relevant procedures are described below, and descriptions of procedures for the full study can be found elsewhere: (Barch et al., 2018; Casey et al., 2018).

Imaging Acquisition and Processing

A detailed description of full imaging acquisition and processing procedures is available elsewhere (Casey et al., 2018; Hagler et al., 2019). Briefly, a standard scan series for each participant consisted of the following: two structural scans (T1w and T2w), one diffusion MRI series, four resting-state scans, and two functional scans for each of the three tasks. Data from the four resting-state scans was analyzed in the present study. The scan protocol was optimized across sites for use of five different 3T scanners: Siemens Prisma and Prisma Fit, Philips Achieva and Ingenia, and General Electric 750 MR. The fMRI scan parameters were made as similar as possible across manufacturers. Following image acquisition, the ABCD Data Analysis and Informatics Resource Center (DAIRC) inspected the data for protocol adherence and completeness and performed standardized processing procedures, including head motion and B0 distortion corrections. After data collection, the DAIIRC performed centralized pre-processing and minimal analysis of all resting-state data. First, head motion was corrected by registering each frame to the first. Additionally, B0 and gradient nonlinearities distortion corrections were performed. After fMRI preprocessing, data were further processed pre-analysis, including removal of initial frames and time series correlated with motion, and normalization to remove quadratic trends.

Resting State Functional Connectivity (RSFC)

The DAIIRC calculated average time courses using FreeSurfer's automated brain segmentation for each region of interest (ROI) and resampled to align with fMRI data. Functionally defined parcellations (Gordon et al., 2016) were then resampled from atlas-space to individual subject-space. For each of the 13 Gordon networks, within-network RSFC was calculated by averaging together Fisher-transformed pairwise correlations of all ROIs comprising a specific network. Between-network RSFC was calculated by averaging all unique Fisher-transformed pairwise

correlations of all ROIs in the first network with all ROIs in the second network. To further reduce residual effects of head motion, timepoints with a framewise displacement (FD) greater than 0.2mm were excluded from these correlation calculations. Finally, correlation values were averaged across the four resting-state scans (weighted by number of remaining frames after censoring for motion) to calculate within- and between-network RSFC.

Psychopathology

Psychopathology was assessed at each wave using the Child Behavior Checklist (CBCL), a 119-item form measuring child behavior and emotion (Achenbach, 2011). Parents rate their child's emotions/behaviors on a scale of 0 ("Not True [as far as you know]"), 1 ("Somewhat or Sometimes True"), or 2 ("Very True or Often True").

Covariates

Baseline age (in months) and sex assigned at birth for each child was reported by their parent/guardian. Each type of MRI scanner was assigned a dummy coded variable: Prisma, Discovery, Achieva, and Ingenia (Prisma Fit = reference group). Motion in the scanner was captured by mean framewise displacement (FD; measured in millimeters; variable "rsfmri_meanmotion"). Current (past two weeks) medication use also was reported by parents/guardians, which was included as a covariate in sensitivity analyses.

Statistical Analyses

Confirmatory Factor Analysis

Previously, a higher-order factor model of psychopathology using CBCL item-level data was identified in longitudinal ABCD studies (see Romer et al., 2023; Romer & Pizzagalli, 2021 for more details). The model consists of five lower-order factors (internalizing, externalizing,

neurodevelopmental, somatic, and detachment) and the higher-order p-factor, which is consistent with the structure of psychopathology in this age range (Michelini et al., 2019). The model provided an adequate fit to the data at each wave (CFI = .925; RMSEA = .032) and demonstrated longitudinal measurement invariance (Figure 2; see Romer et al., 2023; Romer & Pizzagalli, 2021). Thus, p-factor scores were calculated at each wave for subsequent longitudinal analysis by multiplying each CBCL item by its unstandardized factor loading and then summing the weighted items for each factor.

Longitudinal Multilevel Modeling

To determine whether alterations in baseline RSFC within and between networks in the triple neurocognitive network model prospectively related to higher levels of and steeper within-person increases in p-factor scores over time, I conducted two-level growth models to test associations between baseline RSFC and p-factor scores over three years. Level 1 accounted for the within-subject trajectory of p-factor scores and Level 2 accounted for between-subject differences in p-factor scores. A random subject-specific intercept and random slope for time were also included. The time variable was recoded as a number from 0 (baseline/wave 1) to 3 (three-year follow-up/wave 4), for a total of four timepoints.

Analyses were conducted using R version 4.5.0 ([http:// www.r-project.org/](http://www.r-project.org/)) using the lme4 package. First, I compared the fit of the unconditional linear and nonlinear growth models to determine the best-fitting functional form of the trajectory of p-factor scores over time. Second, I examined whether within- and between-network RSFC among the three core networks (DMN, FPN, SN) were associated with between-person differences (intercept) and within-person rates of change (slope) of p-factor scores over wave. Baseline within- and between-network RSFC measures were included as time-invariant covariates (TICs). Baseline age, sex assigned at birth, mean FD, and dummy-coded MRI scanner model were included as Level 2 TICs of no interest.

Additionally, to control for nesting within study site and family, random intercepts for site and family unit were included in the models.

If analyses indicated alterations in RSFC relate to between-person levels (intercept) but not within-person trajectories (slope) of p-factor scores over time, follow-up analyses were run to determine whether these associations persist over time (e.g., if altered RSFC at baseline relates to higher p-factor scores at baseline, one-year follow-up, two-year follow-up, and three-year follow-up). Specifically, I used the `glht` function from the `multcomp` package in R (Hothorn et al., 2008) to examine whether altered RSFC at baseline relates to higher p-factor scores at baseline, one-year follow-up, two-year follow-up, and three-year follow-up (i.e., remain stable over time).

Exploratory analyses

As planned exploratory analyses, I also examined within- and between-network RSFC of additional relevant attentional and cognitive control networks at baseline as prospective predictors of p-factor trajectories. Specifically, I investigated whether altered within-network connectivity for the remaining two key networks (FPN and SN) and within- and between-network RSFC among the three core networks (DMN, FPN, SN) and related cognitive control networks (VAN, DAN, CON) are prospectively associated with future p-factor scores, for a total of 17 additional exploratory growth models.

Sensitivity analyses

The optimal amount of post-processing motion correction in RSFC analysis is still under debate. Excessive head motion during resting-state scans can disproportionately influence results and lead to spurious findings, particularly in non-adult samples, as in-scanner motion decreases with age (Power et al., 2015). One method for handling excessive head motion is to simply exclude any data that rises above a certain threshold (e.g., mean FD >0.2mm). However, higher motion

in the scanner is correlated with numerous relevant clinical and demographic factors (Cosgrove et al., 2022), which raises concern that removing high-motion participants may reduce variability on the outcome of interest (i.e., the p-factor). A study comparing baseline demographic data from the ABCD study between “low noise” and “high noise” (mean FD>0.15mm) groups found that the low noise group consisted of significantly more females, non-Hispanic whites, and parents with graduate degrees/income >\$100,000. Additionally, the low noise group was significantly older, had fewer mental and physical health problems, and had better executive functioning compared to the high noise group (Cosgrove et al., 2022). Given this, removing high-motion participants from initial analyses runs the risk of artificially reducing the diversity of the sample. As such, all initial analyses were conducted following basic quality assurance guidance for motion correction without additional exclusions for motion.

I conducted three planned sensitivity tests. First, to address the concern of motion disproportionately influencing results, I removed all participants with greater than 0.2mm mean FD and re-tested all significant findings. Second, to control for the potential effect of psychotropic medication use on brain function and psychopathology, I included medication as a covariate. Third, I re-ran all models excluding all participants with completely missing clinical follow-up data.

Results

Descriptive Statistics

A full summary of descriptive statistics and missingness is included in Table 1 for all variables. There were 9,344 participants with baseline data included in the present study. Of included participants, 1,601 had missing CBCL data at one or more of the follow-up waves. Of those participants with missing follow-up data, 270 participants only had baseline data (i.e., missing data at all three follow-up waves). I tested for baseline differences between participants with any missing follow-up data (n=1,601) versus those with complete follow-up data (n=7,743).

Participants with no missing follow-up data had significantly lower mean FD, lower baseline p-factor scores, lower DMN-DAN, SN-DAN, and VAN-DAN connectivity and higher CON-CON connectivity compared to those with missing data. Given that participants without any missing data had significantly lower p-factor scores compared to participants with missing data, sensitivity analyses were conducted by removing the 270 participants who did not have any CBCL follow-up data.

Unconditional Linear and Non-linear Growth Models

Likelihood ratio testing was employed to compare the fit of linear and non-linear (quadratic) unconditional growth models of the trajectory of p-factor scores. I found that the quadratic model provided a better fit to the data than the linear model ($X^2(4)=36.353$, $p<0.001$), which was then employed in all subsequent conditional growth models. Random effects from the unconditional quadratic model indicated that there were significant intraindividual (i.e., within-person) and interindividual (i.e., between-person) variability in the linear and quadratic intercepts and slopes (Table 2). There was more between-person variability in the intercepts compared to within-person variability in the slopes, and minimal variability in the slopes of p-factor scores over wave. Fixed effects indicated that p-factor scores showed a u-shaped trajectory, initially decreasing from baseline to wave 3 (ages 9-12) and then increasing from waves 3 to 4 (ages 12-13), with an overall increasing quadratic trend over time ($\beta=0.083$, $p<0.001$). The intra-subject correlation for p-factor scores was 0.781.

Within- and Between-Network RSFC Relations with P-Factor Trajectories

Associations between RSFC networks and the intercepts and slopes of p-factor scores over wave are shown in Table 3 and Figure 3. Results indicated that lower within-DMN connectivity at

baseline was significantly associated with higher baseline levels (intercepts) of p-factor scores; however, lower within-DMN connectivity was not related to within-person change (quadratic slope) in p-factor scores over time. Thus, H1 was partially supported in the present study – hypoconnectivity within the DMN was related to higher concurrent levels of p-factor scores but did not prospectively relate to trajectories of p-factor scores over time. None of the other hypothesized (H2.1, H2.2, H2.3) alterations between RSFC networks (DMN-FPN, DMN-SN, FPN-SN) were associated with either between-person levels of or within-person trajectories of p-factor scores over time.

The planned exploratory analyses examining within- and between-network connectivity among the core networks (DMN, FPN, SN) and three additional related cognitive control and attentional networks (VAN, DAN, CON) indicated several significant findings. Lower within-DAN connectivity and higher between DMN-DAN, DMN-CON, and VAN-CON connectivity at baseline were significantly associated with higher initial levels (intercepts) of but not rates of change (quadratic slopes) in p-factor scores after FDR correction. Follow-up tests were conducted for all RSFC alterations that were associated with between-person levels but not within-person trajectories of p-factor scores (i.e., DMN-DMN, DAN-DAN, DMN-DAN, DMN-CON, and VAN-CON), which demonstrated that these associations remained significant at the one-year, two-year, and three-year follow-ups (Table 4).

Additionally, lower DMN-VAN and greater SN-CON and VAN-DAN connectivity were associated with higher initial levels (intercepts) of and steeper rates of change (quadratic slopes) in p-factor scores over wave. To illustrate these interactions, simple slopes were estimated at the mean and +/- 1 standard deviation from the mean using the emmeans package (Lenth & Piaskowski, 2025) (Figure 4). Preadolescents with lower baseline DMN-VAN connectivity had higher initial p-factor scores at baseline and showed the steepest u-shaped trajectory of p-factor scores ($\beta=-0.010$, 95% CI [-0.010, -0.004]), followed by youth with average ($\beta=-0.009$, 95% CI [-0.011, -0.006]) and

higher ($\beta=-0.007$, 95% CI [-0.013, -0.008]) DMN-VAN connectivity (all $p<0.001$). Preadolescents with higher baseline SN-CON and VAN-DAN connectivity had higher initial p-factor scores and showed the steepest u-shaped trajectory of p-factor scores (SN-CON: $\beta=-0.009$, 95% CI [-0.012, -0.006]; VAN-DAN: $\beta=-0.011$, 95% CI [-0.013, -0.008]), followed by youth with average (both networks: $\beta=-0.009$, 95% CI [-0.011, -0.006]) and lower (SN-CON: $\beta=-0.008$, 95% CI [-0.011, -0.005]; VAN-DAN: $\beta=-0.006$, 95% CI [-0.009, -0.004]) connectivity (all $p<0.001$). Sensitivity analyses revealed these associations remained significant after excluding participants with mean FD>0.2mm, controlling for medication use, and excluding participants who did not have any CBCL follow-up data (Table 5).

Discussion

The present study utilized data from the ABCD study to examine the prospective relationship between altered resting state functional connectivity and the trajectory of transdiagnostic psychopathology in emerging adolescents. Results indicated that p-factor scores fit best as a nonlinear function, as p-factor scores followed a u-shaped trajectory over time, such that general psychopathology decreased from baseline to the two-year follow-up and then increased from the two-year to three-year follow-ups. Largely consistent with prior research, I found that lower within-DMN and within-DAN connectivity and greater DMN-CON, DMN-DAN, and VAN-CON connectivity were associated with higher between-person levels of p-factor scores. Further, I demonstrated that these relationships persisted over time and that they remained significant while controlling for sex, age, scanner, study site, head motion, and medication use. Additionally, unlike prior studies, I investigated the prospective relationship between RSFC and p-factor scores and found that hypoconnectivity between DMN-VAN and hyperconnectivity between VAN-DAN and SN-CON at baseline was both concurrently associated with higher levels of p-factor scores and prospectively associated with steeper decreases in p-factor scores from baseline to the two-year

follow-up and steeper increases from the two-year to three-year follow-ups. Together, this evidence broadly suggests that early altered connectivity between networks responsible for self-referential thought, reorienting and guiding attention, and filtering salient stimuli are related to future trajectories of general psychopathology during this critical developmental period.

The present findings add to a growing body of evidence that implicate alterations in higher-order cognitive processes as a mechanism underlying psychopathology broadly. In line with prior research that demonstrates functional and structural alterations in cognitive and attentional regions of the brain (Elliott et al., 2018), I found alterations among five out of the six functional networks typically implicated in higher-order cognition and cognitive control (i.e., DMN, SN, VAN, DAN, and CON). These findings both replicate and expand upon previous cross-sectional findings between RSFC and the p-factor in the ABCD study (Karcher et al., 2022; Lees et al., 2021; Sripada et al., 2021) and other adolescent samples (Xia et al., 2018). Replicating prior work, this study found a significant association between lower within-DMN and within-DAN connectivity and higher DMN-CON and DMN-DAN connectivity with higher between-person levels of p-factor scores. Expanding on prior work, results demonstrated that these associations remained significant throughout the transition to adolescence. Additionally, this study is the first to identify lower baseline DMN-VAN connectivity and greater SN-CON and VAN-DAN connectivity as prospectively related to future trajectories of general psychopathology. Previous studies found that DMN-VAN hypoconnectivity and VAN-DAN hyperconnectivity were associated with higher between-person levels of p-factor scores (Karcher et al., 2022; Sripada et al., 2021), which this study replicates and extends to demonstrate that baseline alterations in VAN-DAN and DMN-VAN connectivity prospectively relate to steeper within-person rates of change in p-factor scores over three years.

Many of the current findings related to increased general psychopathology implicated aberrant connectivity of the DMN, the network active when the brain is “at rest” and responsible for self-

referential thought. This is broadly consistent with Menon's (2011) triple neurocognitive network model and additional work demonstrating that DMN alterations are associated with a large variety of psychiatric disorders (Fox et al., 2005; Raichle, 2015). Menon (2011) theorized that alterations in DMN connectivity influence FPN and SN connectivity to give rise to psychopathology. The findings here both support and extend this theory by suggesting that alterations not only between the core theorized networks (DMN, FPN, SN), but also between three additional related networks responsible for reorientation of attention, top-down orientation of attention, and maintaining attention (VAN, DAN, and CON, respectively) may be important RSFC mechanisms underlying general psychopathology. Indeed, I found that altered connectivity within the DMN and between the DMN and VAN, DAN, and CON were related to increased general psychopathology concurrently and over time. I also found that increased connectivity between VAN-DAN and SN-CON and decreased DMN-VAN connectivity prospectively related to p-factor scores.

Alterations between the three core networks themselves (i.e., DMN-FPN, DMN-SN, and FPN-SN) were not associated with psychopathology during emerging adolescence. Given that all six networks examined in this study are involved in cognitive control and become increasingly specialized during adolescence (Menon & D'Esposito, 2022; Morgan et al., 2018; Stevens, 2009), it is plausible that early lack of differentiation between these functional networks may drive increases in general psychopathology. In typical development, greater integration of nodes within the DMN, greater segregation between higher-order cognitive networks, and increased connectivity between the DMN and higher-order cognitive networks are positively associated with age and executive functioning skills during adolescence (Gu et al., 2015; Morgan et al., 2018; Satterthwaite et al., 2013; Sherman et al., 2014; Stevens, 2009). In this study, I found that reduced within-DMN connectivity (i.e., poorer integration), increased VAN-DAN, VAN-CON and SN-CON connectivity (i.e., poorer segregation), and reduced DMN-VAN connectivity were associated with higher levels of psychopathology. This may suggest that atypical connectivity among these

networks is an early neurocognitive marker of greater psychopathology and a potential risk factor for greater future psychopathology during early adolescence. Additionally, Sripada and colleagues (2021) demonstrated that higher rates of transdiagnostic psychopathology served primarily as an “attenuating factor”, such that higher psychopathology made neurotypically positive network connections less positive and neurotypically negative network connections less negative. Taken together, one broad hypothesis is that early failure for these functional networks to properly form connections and appropriately integrate and segregate during the transition to adolescence is what drives increases in general psychopathology.

Consistent with previous findings in the ABCD study which found CBCL internalizing and externalizing scores decreased over time (Barch et al., 2021), the current study demonstrated that p-factor scores followed a significant u-shaped developmental trajectory during the transition to adolescence. Specifically, p-factor scores decreased from baseline (ages 9-10) to the two-year follow-up (ages 11-12) before increasing during the transition to adolescence at the three-year follow-up (ages 12-13). It is possible that this reflects a typical trajectory of psychopathology, as many forms of psychopathology do indeed decrease during this developmental period – anxiety and impulse control difficulties tend to peak and begin to decrease during early adolescence before mood, thought, and substance use disorders onset and worsen during adolescence and early adulthood (American Psychiatric Association, 2022; Kessler et al., 2005; McGrath et al., 2020; Solmi et al., 2022). The observed relationship between altered DMN-VAN, SN-CON, and VAN-DAN connectivity and the u-shaped trajectory of psychopathology during emerging adolescence may reflect an early marker of increased vulnerability to transdiagnostic psychopathology during this developmental period.

Notably, the observed effect sizes between RSFC and p-factor scores were small, especially the associations between baseline RSFC and future trajectories of p-factor scores. There may be several reasons for these smaller effect sizes. First, there is evidence that effect sizes in the ABCD

study may be smaller than typically expected overall, with a first quartile value of .03 and average observed effect size of .05 (Owens et al., 2021), which a majority of the intercept effects observed in this study fell between. Additionally, the present study utilized data from a relatively short period of time during the very beginning of adolescence. Given that psychopathology tends to worsen with age and many forms of psychopathology onset during adolescence (Kessler et al., 2005; McGrath et al., 2020; Solmi et al., 2022), it is possible that the influence of aberrant RSFC on general psychopathology will become stronger over time. The three-year follow-up has data from ages 12-13, just as p-factor scores begin to increase. As psychopathology presumably begins to demonstrate greater change and variability at later timepoints, the ability to predict how RSFC relates to future p-factor scores may strengthen. Further, supported by results from the unconditional quadratic model of p-factor scores over wave, there was minimal variability in p-factor scores in the present study. This limited variability in p-factor score trajectories reduced the amount of variability that alterations in functional connectivity can predict, which may also contribute to the small effect sizes.

There are several limitations to note in the present study. First, participants with no missing data had significant differences on important variables compared to participants with missing p-factor follow-up scores (approximately 17% of the sample had missing follow-up data). However, to ensure differences between groups did not influence results, significant models were re-run after removing participants with completely missing follow-up data and all results remained significant. Second, this final timepoint examined in this study is the ABCD three-year follow-up when participants were between the ages of 12 to 13. Rates of psychopathology are still generally low at this age as many forms of internalizing psychopathology do not reach their peak age of onset until a little later, between 14 and 16 years old (Solmi et al., 2022). Given this, in the present study, levels of psychopathology were only just beginning to increase at the final timepoint, and the observed quadratic pattern of p-factor scores was just beginning to emerge. It is important to

examine the relationship between RSFC and trajectories of psychopathology using future study waves to clarify whether the observed associations exist in middle to late adolescence as psychopathology increases. In addition to this, p-factor scores in the present study were calculated based on parent-reported symptoms, which may introduce reporter bias. Future waves of the ABCD study will include youth self-report on symptoms of psychopathology and should be examined. Finally, although results from this study broadly support the hypothesis that failure of functional networks to appropriately integrate and segregate during early adolescence may contribute to transdiagnostic psychopathology, I only examined RSFC at baseline. To determine whether these observed early alterations in connectivity represent atypical development in network integration and segregation, it is an important future direction to investigate how patterns of RSFC change over time and relate to trajectories of psychopathology.

The present study was the first to examine whether alterations in RSFC prospectively related to between-person differences and within-person trajectories of p-factor scores in early adolescence. For the first time, I demonstrated that hypoconnectivity between DMN-VAN and hyperconnectivity between VAN-DAN and SN-CON was associated with higher levels of p-factor scores and prospectively associated with steeper quadratic changes in p-factor scores over three years. Additionally, this study expanded upon previous work with findings indicating that within-DMN and within-DAN hypoconnectivity and hyperconnectivity between the DMN-CON, DMN-DAN, and VAN-CON were associated with higher between-person levels of p-factor scores over three years. Taken together, these results suggest that altered functional connectivity within- and between-networks responsible for cognitive control and attention may serve as a risk factor for increased transdiagnostic psychopathology during emerging adolescence. This study provides a base for future research to examine whether patterns of reduced network integration and segregation among these networks over time is a neurobiological vulnerability for greater rates of comorbidity and severity in psychiatric disorders during middle and late adolescence.

References

- Achenbach, T. M. (2011). Child Behavior Checklist. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Eds.), *Encyclopedia of Clinical Neuropsychology* (pp. 546–552). Springer. https://doi.org/10.1007/978-0-387-79948-3_1529
- American Psychiatric Association. (2022). Diagnostic and statistical manual of mental disorders (5th ed., text rev.). <https://doi.org/10.1176/appi.books.9780890425787>
- Barch, D. M., Albaugh, M. D., Avenevoli, S., Chang, L., Clark, D. B., Glantz, M. D., Hudziak, J. J., Jernigan, T. L., Tapert, S. F., Yurgelun-Todd, D., Alia-Klein, N., Potter, A. S., Paulus, M. P., Prouty, D., Zucker, R. A., & Sher, K. J. (2018). Demographic, physical and mental health assessments in the adolescent brain and cognitive development study: Rationale and description. *Developmental Cognitive Neuroscience, 32*, 55–66. <https://doi.org/10.1016/j.dcn.2017.10.010>
- Casey, B. J., Cannonier, T., Conley, M. I., Cohen, A. O., Barch, D. M., Heitzeg, M. M., Soules, M. E., Teslovich, T., Dellarco, D. V., Garavan, H., Orr, C. A., Wager, T. D., Banich, M. T., Speer, N. K., Sutherland, M. T., Riedel, M. C., Dick, A. S., Bjork, J. M., Thomas, K. M., ... ABCD Imaging Acquisition Workgroup. (2018). The Adolescent Brain Cognitive Development (ABCD) study: Imaging acquisition across 21 sites. *Developmental Cognitive Neuroscience, 32*, 43–54. <https://doi.org/10.1016/j.dcn.2018.03.001>
- Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T. E. (2014). The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders? *Clinical Psychological Science, 2*(2), 119–137. <https://doi.org/10.1177/2167702613497473>
- Caspi, A., & Moffitt, T. E. (2018). All for One and One for All: Mental Disorders in One Dimension. *American Journal of Psychiatry, 175*(9), 831–844. <https://doi.org/10.1176/appi.ajp.2018.17121383>

- Clark, D. A., Hicks, B. M., Angstadt, M., Rutherford, S., Taxali, A., Hyde, L., Weigard, A. S., Heitzeg, M. M., & Sripada, C. (2021). The General Factor of Psychopathology in the Adolescent Brain Cognitive Development (ABCD) Study: A Comparison of Alternative Modeling Approaches. *Clinical Psychological Science, 9*(2), 169–182. <https://doi.org/10.1177/2167702620959317>
- Cosgrove, K. T., McDermott, T. J., White, E. J., Mosconi, M. W., Thompson, W. K., Paulus, M. P., Cardenas-Iniguez, C., & Aupperle, R. L. (2022). Limits to the generalizability of resting-state functional magnetic resonance imaging studies of youth: An examination of ABCD Study® baseline data. *Brain Imaging and Behavior, 16*(4), 1919–1925. <https://doi.org/10.1007/s11682-022-00665-2>
- Elliott, M. L., Romer, A., Knodt, A. R., & Hariri, A. R. (2018). A Connectome-wide Functional Signature of Transdiagnostic Risk for Mental Illness. *Biological Psychiatry, 84*(6), 452–459. <https://doi.org/10.1016/j.biopsych.2018.03.012>
- Fox, M. D., & Greicius, M. (2010). Clinical applications of resting state functional connectivity. *Frontiers in Systems Neuroscience, 4*. <https://doi.org/10.3389/fnsys.2010.00019>
- Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle, M. E. (2005). The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences, 102*(27), 9673–9678. <https://doi.org/10.1073/pnas.0504136102>
- Fried, E. I., & Nesse, R. M. (2015). Depression is not a consistent syndrome: An investigation of unique symptom patterns in the STAR*D study. *Journal of Affective Disorders, 172*, 96–102. <https://doi.org/10.1016/j.jad.2014.10.010>
- Garavan, H., Bartsch, H., Conway, K., Decastro, A., Goldstein, R. Z., Heeringa, S., Jernigan, T., Potter, A., Thompson, W., & Zahs, D. (2018). Recruiting the ABCD sample: Design considerations and procedures. *Developmental Cognitive Neuroscience, 32*, 16–22. <https://doi.org/10.1016/j.dcn.2018.04.004>

- Glover, G. H. (2011). Overview of Functional Magnetic Resonance Imaging. *Neurosurgery Clinics of North America*, 22(2), 133–139. <https://doi.org/10.1016/j.nec.2010.11.001>
- Goodkind, M., Eickhoff, S. B., Oathes, D. J., Jiang, Y., Chang, A., Jones-Hagata, L. B., Ortega, B. N., Zaiko, Y. V., Roach, E. L., Korgaonkar, M. S., Grieve, S. M., Galatzer-Levy, I., Fox, P. T., & Etkin, A. (2015). Identification of a common neurobiological substrate for mental illness. *JAMA Psychiatry*, 72(4), 305–315. <https://doi.org/10.1001/jamapsychiatry.2014.2206>
- Gordon, E. M., Laumann, T. O., Adeyemo, B., Huckins, J. F., Kelley, W. M., & Petersen, S. E. (2016). Generation and Evaluation of a Cortical Area Parcellation from Resting-State Correlations. *Cerebral Cortex (New York, NY)*, 26(1), 288–303. <https://doi.org/10.1093/cercor/bhu239>
- Gu, S., Satterthwaite, T. D., Medaglia, J. D., Yang, M., Gur, R. E., Gur, R. C., & Bassett, D. S. (2015). Emergence of system roles in normative neurodevelopment. *Proceedings of the National Academy of Sciences of the United States of America*, 112(44), 13681. <https://doi.org/10.1073/pnas.1502829112>
- Hagler, D. J., Hatton, Sean N., Cornejo, M. D., Makowski, C., Fair, D. A., Dick, A. S., Sutherland, M. T., Casey, B. J., Barch, D. M., Harms, M. P., Watts, R., Bjork, J. M., Garavan, H. P., Hilmer, L., Pung, C. J., Sicat, C. S., Kuperman, J., Bartsch, H., Xue, F., ... Dale, A. M. (2019). Image processing and analysis methods for the Adolescent Brain Cognitive Development Study. *NeuroImage*, 202, 116091. <https://doi.org/10.1016/j.neuroimage.2019.116091>
- Hothorn, T., Bretz, F., & Westfall, P. (2008). Simultaneous inference in general parametric models. *Biometrical Journal. Biometrische Zeitschrift*, 50(3), 346–363. <https://doi.org/10.1002/bimj.200810425>
- Hoy, N., Lynch, S. J., Waszczuk, M. A., Reppermund, S., & Mewton, L. (2023). Transdiagnostic biomarkers of mental illness across the lifespan: A systematic review examining the genetic and neural correlates of latent transdiagnostic dimensions of psychopathology in the general

population. *Neuroscience & Biobehavioral Reviews*, 155, 105431.

<https://doi.org/10.1016/j.neubiorev.2023.105431>

Karcher, N. R., Michelini, G., Kotov, R., & Barch, D. M. (2022). *Associations Between Resting State Functional Connectivity and a Hierarchical Dimensional Structure of Psychopathology in Middle Childhood*.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, Severity, and Comorbidity of Twelve-month DSM-IV Disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry*, 62(6), 617–627.

<https://doi.org/10.1001/archpsyc.62.6.617>

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K., ... Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, 126(4), 454–477.

<https://doi.org/10.1037/abn0000258>

Laceulle, O. M., Vollebergh, W. A. M., & Ormel, J. (2015). The Structure of Psychopathology in Adolescence: Replication of a General Psychopathology Factor in the TRAILS Study. *Clinical Psychological Science*, 3(6), 850–860. <https://doi.org/10.1177/2167702614560750>

Lees, B., Squeglia, L. M., McTeague, L. M., Forbes, M. K., Krueger, R. F., Sunderland, M., Baillie, A. J., Koch, F., Teesson, M., & Mewton, L. (2021). Altered Neurocognitive Functional Connectivity and Activation Patterns Underlie Psychopathology in Preadolescence. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 6(4), 387–398.

<https://doi.org/10.1016/j.bpsc.2020.09.007>

Marek, S., & Dosenbach, N. U. F. (2018). The frontoparietal network: Function, electrophysiology, and importance of individual precision mapping. *Dialogues in Clinical Neuroscience*, 20(2), 133–140. <https://doi.org/10.31887/DCNS.2018.20.2/smarek>

- McGrath, J. J., Lim, C. C. W., Plana-Ripoll, O., Holtz, Y., Agerbo, E., Momen, N. C., Mortensen, P. B., Pedersen, C. B., Abdulmalik, J., Aguilar-Gaxiola, S., Al-Hamzawi, A., Alonso, J., Bromet, E. J., Bruffaerts, R., Bunting, B., de Almeida, J. M. C., de Girolamo, G., De Vries, Y. A., Florescu, S., ... de Jonge, P. (2020). Comorbidity within mental disorders: A comprehensive analysis based on 145 990 survey respondents from 27 countries. *Epidemiology and Psychiatric Sciences*, 29, e153. <https://doi.org/10.1017/S2045796020000633>
- Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences*, 15(10), 483–506. <https://doi.org/10.1016/j.tics.2011.08.003>
- Menon, V., & D'Esposito, M. (2022). The role of PFC networks in cognitive control and executive function. *Neuropsychopharmacology*, 47(1), 90–103. <https://doi.org/10.1038/s41386-021-01152-w>
- Merikangas, K. R., He, J.-P., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., Benjet, C., Georgiades, K., & Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication--Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(10), 980–989. <https://doi.org/10.1016/j.jaac.2010.05.017>
- Mezzacappa, E. (2011). Executive Function. In *Encyclopedia of Adolescence* (pp. 142–150). Elsevier. <https://doi.org/10.1016/B978-0-12-373951-3.00016-8>
- Micheline, G., Barch, D. M., Tian, Y., Watson, D., Klein, D. N., & Kotov, R. (2019). Delineating and validating higher-order dimensions of psychopathology in the Adolescent Brain Cognitive Development (ABCD) study. *Translational Psychiatry*, 9(1), 1–15. <https://doi.org/10.1038/s41398-019-0593-4>
- Morgan, S. E., White, S. R., Bullmore, E. T., & Vértes, P. E. (2018). A Network Neuroscience Approach to Typical and Atypical Brain Development. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 3(9), 754–766. <https://doi.org/10.1016/j.bpsc.2018.03.003>

- Owens, M. M., Potter, A., Hyatt, C. S., Albaugh, M., Thompson, W. K., Jernigan, T., Yuan, D., Hahn, S., Allgaier, N., & Garavan, H. (2021). Recalibrating expectations about effect size: A multi-method survey of effect sizes in the ABCD study. *PLOS ONE*, *16*(9), e0257535.
<https://doi.org/10.1371/journal.pone.0257535>
- Parkes, L., Moore, T. M., Calkins, M. E., Cook, P. A., Cieslak, M., Roalf, D. R., Wolf, D. H., Gur, R. C., Gur, R. E., Satterthwaite, T. D., & Bassett, D. S. (2021). Transdiagnostic dimensions of psychopathology explain individuals' unique deviations from normative neurodevelopment in brain structure. *Translational Psychiatry*, *11*(1), 1–13. <https://doi.org/10.1038/s41398-021-01342-6>
- Pettersson, E., Lahey, B. B., Larsson, H., & Lichtenstein, P. (2018). Criterion Validity and Utility of the General Factor of Psychopathology in Childhood: Predictive Associations With Independently Measured Severe Adverse Mental Health Outcomes in Adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *57*(6), 372–383.
<https://doi.org/10.1016/j.jaac.2017.12.016>
- Power, J. D., Schlaggar, B. L., & Petersen, S. E. (2015). Recent progress and outstanding issues in motion correction in resting state fMRI. *NeuroImage*, *105*, 536–551.
<https://doi.org/10.1016/j.neuroimage.2014.10.044>
- Raichle, M. E. (2015). The Brain's Default Mode Network. *Annual Review of Neuroscience*, *38*(1), 433–447. <https://doi.org/10.1146/annurev-neuro-071013-014030>
- Romer, A. L., Elliott, M. L., Knodt, A. R., Sison, M. L., Ireland, D., Houts, R., Ramrakha, S., Poulton, R., Keenan, R., Melzer, T. R., Moffitt, T. E., Caspi, A., & Hariri, A. R. (2020). Pervasively Thinner Neocortex as a Transdiagnostic Feature of General Psychopathology. *American Journal of Psychiatry*. <https://doi.org/10.1176/appi.ajp.2020.19090934>
- Romer, A. L., & Pizzagalli, D. A. (2021). Is executive dysfunction a risk marker or consequence of psychopathology? A test of executive function as a prospective predictor and outcome of

- general psychopathology in the adolescent brain cognitive development study®. *Developmental Cognitive Neuroscience*, 51, 100994. <https://doi.org/10.1016/j.dcn.2021.100994>
- Romer, A. L., Ren, B., & Pizzagalli, D. A. (2023). Brain Structure Relations With Psychopathology Trajectories in the ABCD Study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 62(8), 895–907. <https://doi.org/10.1016/j.jaac.2023.02.002>
- Satterthwaite, T. D., Wolf, D. H., Erus, G., Ruparel, K., Elliott, M. A., Gennatas, E. D., Hopson, R., Jackson, C., Prabhakaran, K., Bilker, W. B., Calkins, M. E., Loughead, J., Smith, A., Roalf, D. R., Hakonarson, H., Verma, R., Davatzikos, C., Gur, R. C., & Gur, R. E. (2013). Functional Maturation of the Executive System during Adolescence. *Journal of Neuroscience*, 33(41), 16249–16261. <https://doi.org/10.1523/JNEUROSCI.2345-13.2013>
- Seeley, W. W. (2019). The Salience Network: A Neural System for Perceiving and Responding to Homeostatic Demands. *Journal of Neuroscience*, 39(50), 9878–9882. <https://doi.org/10.1523/JNEUROSCI.1138-17.2019>
- Segal, A., Tiego, J., Parkes, L., Holmes, A. J., Marquand, A. F., & Fornito, A. (2024). Embracing variability in the search for biological mechanisms of psychiatric illness. *Trends in Cognitive Sciences*, S1364661324002535. <https://doi.org/10.1016/j.tics.2024.09.010>
- Sha, Z., Wager, T. D., Mechelli, A., & He, Y. (2019). Common Dysfunction of Large-Scale Neurocognitive Networks Across Psychiatric Disorders. *Biological Psychiatry*, 85(5), 379–388. <https://doi.org/10.1016/j.biopsych.2018.11.011>
- Sherman, L. E., Rudie, J. D., Pfeifer, J. H., Masten, C. L., McNealy, K., & Dapretto, M. (2014). Development of the Default Mode and Central Executive Networks across early adolescence: A longitudinal study. *Developmental Cognitive Neuroscience*, 10, 148–159. <https://doi.org/10.1016/j.dcn.2014.08.002>
- Solmi, M., Radua, J., Olivola, M., Croce, E., Soardo, L., Salazar de Pablo, G., Il Shin, J., Kirkbride, J. B., Jones, P., Kim, J. H., Kim, J. Y., Carvalho, A. F., Seeman, M. V., Correll, C. U., & Fusar-Poli, P. (2022). Age at onset of mental disorders worldwide: Large-scale meta-analysis of 192

epidemiological studies. *Molecular Psychiatry*, 27(1), 281–295. <https://doi.org/10.1038/s41380-021-01161-7>

Sripada, C., Angstadt, M., Taxali, A., Kessler, D., Greathouse, T., Rutherford, S., Clark, D. A., Hyde, L. W., Weigard, A., Brislin, S. J., Hicks, B., & Heitzeg, M. (2021). Widespread attenuating changes in brain connectivity associated with the general factor of psychopathology in 9- and 10-year olds. *Translational Psychiatry*, 11(1), 575. <https://doi.org/10.1038/s41398-021-01708-w>

Stevens, M. C. (2009). The developmental cognitive neuroscience of functional connectivity. *Brain and Cognition*, 70(1), 1–12. <https://doi.org/10.1016/j.bandc.2008.12.009>

Woodward, N. D., & Cascio, C. J. (2015). Resting-state functional connectivity in psychiatric disorders. *JAMA Psychiatry*, 72(8), 743–744. <https://doi.org/10.1001/jamapsychiatry.2015.0484>

Xia, C. H., Ma, Z., Ciric, R., Gu, S., Betzel, R. F., Kaczkurkin, A. N., Calkins, M. E., Cook, P. A., García de la Garza, A., Vandekar, S. N., Cui, Z., Moore, T. M., Roalf, D. R., Ruparel, K., Wolf, D. H., Davatzikos, C., Gur, R. C., Gur, R. E., Shinohara, R. T., ... Satterthwaite, T. D. (2018). Linked dimensions of psychopathology and connectivity in functional brain networks. *Nature Communications*, 9(1), 3003. <https://doi.org/10.1038/s41467-018-05317-y>

Table 1. Study Variable Descriptive Statistics and Participant Comparisons by Follow-Up Completion.

	Baseline Full Sample				Non-Missing Sample		Missing Sample		χ^2/df	P-value
	n	Min	Max	Mean (SD) or %	n	Mean (SD) or %	n	Mean (SD) or %		
<i>Demographics and Covariates</i>										
Age (months)	9348	107	133	119.25 (7.52)	7743	119.28 (7.55)	1605	119.10 (7.39)	0.88	0.377
Sex (% Female)	9348			49.74	7743	49.53	1605	50.77	0.783	0.376
Non-Hispanic White (%)	9346			53.60	7743	56.79	1603	38.18	2860.1	<0.001
Black (%)	9346			13.72	7743	11.66	1603	23.64	216.15	<0.001
Asian (%)	9346			2.00	7743	1.95	1603	2.18	72.344	<0.001
Hispanic (%)	9346			20.23	7743	19.20	1603	25.20	623.2	<0.001
Other (%)	9346			10.46	7743	10.40	1603	10.79	408.41	<0.001
Average FD	9336	0.02	2.49	0.23 (0.22)	7732	0.23 (0.22)	1604	0.26 (0.23)	4.68	<0.001
No Medication Use (%)	9335			53.50	7736	52.51	1599	58.29	4.256	<0.001
<i>P-Factor Scores</i>										
P1	9344	0	57.14	7.03 (7.46)	7743	6.82 (7.23)	1601	8.03 (8.42)	5.37	<0.001
P2	8840	0	50.63	6.75 (7.16)						
P3	8618	0	53.65	6.43 (7.04)						
P4	8034	0	54.02	6.46 (7.04)						
	All Waves				Non-Missing Sample		Missing Sample		χ^2/df	P-value
	n	Min	Max	Mean (SD) or %	n	Mean (SD) or %	n	Mean (SD) or %		
<i>Resting-State Functional Connectivity</i>										
<i>Hypothesized Network Connectivity</i>										
DMN-DMN	9336	0.04	0.49	0.24 (0.06)	7732	0.24 (0.06)	1604	0.24 (0.06)	1.25	0.211
DMN-FPN	9336	-0.13	0.23	0.05 (0.04)	7732	0.05 (0.04)	1604	0.05 (0.04)	1.46	0.144
DMN-SN	9336	-0.21	0.33	0.07 (0.06)	7732	0.08 (0.06)	1604	0.07 (0.06)	2.28	0.023
FPN-SN	9336	-0.17	0.38	0.09 (0.06)	7732	0.09 (0.06)	1604	0.09 (0.06)	1.50	0.134
<i>Exploratory Network Connectivity</i>										
DMN-VAN	9335	-0.09	0.30	0.09 (0.05)	7731	0.09 (0.05)	1604	0.09 (0.05)	2.97	0.003
DMN-DAN	9336	-0.37	0.06	-0.13 (0.05)	7732	-0.13 (0.05)	1604	-0.13 (0.06)	4.55	<0.001

DMN-CON	9336	-0.13	0.23	0.05 (0.04)	7729	-0.11 (0.05)	1604	-0.11 (0.06)	1.91	0.056
FPN-FPN	9336	0.04	0.48	0.21 (0.06)	7732	0.21 (0.06)	1604	0.22 (0.06)	0.97	0.334
FPN-VAN	9335	-0.15	0.18	0.02 (0.04)	7731	0.02 (0.04)	1604	0.02 (0.04)	0.94	0.348
FPN-DAN	9336	-0.11	0.25	0.06 (0.04)	7732	0.06 (0.04)	1604	0.06 (0.04)	2.16	0.031
FPN-CON	9333	-0.22	0.16	-0.01 (0.05)	7729	-0.01 (0.04)	1604	-0.01 (0.05)	2.85	0.004
SN-SN	9336	-0.05	0.85	0.40 (0.12)	7732	0.40 (0.12)	1604	0.39 (0.12)	0.87	0.383
SN-VAN	9335	-0.19	0.36	0.09 (0.07)	7731	0.09 (0.07)	1604	0.08 (0.06)	2.53	0.011
SN-DAN	9336	-0.31	0.18	-0.04 (0.06)	7732	-0.04 (0.06)	1604	-0.04 (0.06)	4.78	<0.001
SN-CON	9333	-0.12	0.46	0.13 (0.07)	7729	0.12 (0.07)	1604	0.13 (0.07)	1.07	0.283
VAN-VAN	9335	0.04	0.51	0.24 (0.06)	7731	0.24 (0.06)	1604	0.23 (0.06)	1.36	0.175
VAN-DAN	9335	-0.32	0.08	-0.08 (0.05)	7731	-0.08 (0.05)	1604	-0.08 (0.05)	4.81	<0.001
VAN-CON	9333	-0.17	0.27	0.03 (0.05)	7728	0.02 (0.05)	1604	0.03 (0.05)	0.81	0.417
DAN-DAN	9336	0.05	0.55	0.27 (0.07)	7732	0.28 (0.07)	1604	0.27 (0.07)	2.71	0.007
DAN-CON	9333	-0.06	0.33	0.09 (0.05)	7729	0.09 (0.05)	1604	0.09 (0.05)	2.57	0.010
CON-CON	9333	0.09	0.60	0.31 (0.07)	7729	0.31 (0.07)	1604	0.30 (0.07)	3.75	<0.001

Note. Groups were compared using chi-square tests and independent samples t-tests for categorical and continuous variables, respectively. P-values are unadjusted; *p*-values that survived FDR correction ($q < 0.05$) for the 31 tests are bolded. Slight differences in sample size are due to missing data on certain variables. P = general psychopathology factor scores; 1 = baseline Wave 1; 2 = one-year follow-up Wave 2; 3 = two-year follow-up Wave 3; 4 = three-year follow-up Wave 4; DMN = default mode network; FPN = frontoparietal network; SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Table 2. Unconditional Quadratic Growth Model of P-Factor Scores over Four Waves.

	b	SE	95% CI
Fixed Effects			
Intercept	7.068***	0.199	[6.654, 7.482]
Wave	-0.416***	0.060	[-0.535, -0.298]
Wave ²	0.083***	0.019	[0.046, 0.120]
Random Effects			
	Variance	SD	95% CI
Level 2 Intercept	19.322	4.396	[4.396, 4.400]
Level 2 Linear Slope	3.943	1.986	[1.626, 2.290]
Level 2 Intercept-Linear Slope Correlation	-0.410		[-0.497, -0.327]
Level 2 Quadratic Slope	0.202	0.450	[0.269, 0.577]
Level 2 Intercept-Quadratic Slope Correlation	0.060		[-0.104, -0.197]
Level 1 Residual	11.450	3.384	[3.381, 3.424]

Note. Unstandardized estimates are shown. Level 1 Residual = intraindividual variability (within-person repeated measures); Level 2 = interindividual variability (between-person). SE = standard error; CI = confidence interval. *** $p < 0.001$.

Table 3. Within- and Between-Network Resting-State Functional Connectivity Relations with Intercepts and Slopes of P-Factor Scores Over Wave.

Resting-State Functional Connectivity Networks	Intercept (Main Effect)		Slope (Interaction Effect)	
	β	95% CI	β	95% CI
Hypothesized Networks				
DMN-DMN	-0.027*	[-0.049, -0.006]	-0.003	[-0.008, 0.002]
DMN-FPN	0.025*	[0.005, 0.045]	-0.002	[-0.007, 0.003]
DMN-SN	0.013	[-0.007, 0.033]	-0.004	[-0.010, 0.001]
FPN-SN	0.010	[-0.011, 0.030]	-0.002	[-0.007, 0.003]
Exploratory Networks				
DMN-VAN	-0.030**	[-0.050, -0.009]	-0.008**	[-0.013, -0.003]
DMN-DAN	0.055***	[0.034, 0.076]	0.002	[-0.003, 0.007]
DMN-CON	0.041***	[0.020, 0.062]	0.000	[-0.005, 0.006]
FPN-FPN	-0.013	[-0.033, 0.008]	-0.001	[-0.006, 0.004]
FPN-VAN	0.025*	[0.005, 0.045]	0.004	[-0.001, 0.009]
FPN-DAN	-0.019	[-0.039, 0.001]	-0.006*	[-0.011, -0.001]
FPN-CON	0.014	[-0.006, 0.034]	-0.001	[-0.006, 0.004]
SN-SN	0.021*	[0.000, 0.041]	0.006*	[0.000, 0.011]
SN-VAN	0.012	[-0.009, 0.032]	0.003	[-0.002, 0.008]
SN-DAN	-0.004	[-0.024, 0.016]	0.001	[-0.004, 0.006]
SN-CON	0.027**	[0.007, 0.047]	0.008**	[0.003, 0.013]
VAN-VAN	0.004	[-0.016, 0.025]	-0.002	[-0.008, 0.003]
VAN-DAN	0.036***	[0.016, 0.057]	0.007**	[0.002, 0.012]
VAN-CON	0.043***	[0.022, 0.063]	0.004	[-0.001, 0.009]
DAN-DAN	-0.032**	[-0.053, -0.012]	-0.004	[-0.009, 0.001]
DAN-CON	-0.017	[-0.037, 0.003]	0.002	[-0.003, 0.007]
CON-CON	-0.006	[-0.027, 0.015]	0.001	[-0.004, 0.006]

Note. Intercept effects represent the main effect of baseline RSFC on between-person levels of p-factor scores. Slope effects represent the interactions between baseline RSFC and wave, or within-person trajectories of p-factor scores over time. Covariates that were included in the models but are of no interest (i.e., age, sex, scanner type) are not shown here. * unadjusted $p < 0.05$; ** unadjusted $p < 0.01$; *** unadjusted $p < 0.001$. Standardized estimates are shown.

Bolded estimates survived FDR correction ($q > 0.05$) for the 42 tests. CI = confidence interval;

DMN = default mode network; FPN = frontoparietal network; SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Table 4. *Follow-Up Tests of Relations between Resting-State Functional Connectivity and the General Psychopathology Factor at Follow-up Waves.*

Resting State Functional Connectivity	Std. β	95% CI
DMN-DMN		
<i>Wave 1</i>	-0.030*	[-0.054, -0.006]
<i>Wave 2</i>	-0.033*	[-0.060, -0.006]
<i>Wave 3</i>	-0.036*	[-0.066, -0.005]
DMN-VAN		
<i>Wave 1</i>	-0.037***	[-0.061, -0.014]
<i>Wave 2</i>	-0.046***	[-0.072, -0.020]
<i>Wave 3</i>	-0.054***	[-0.084, -0.024]
DMN-DAN		
<i>Wave 1</i>	0.057***	[0.033, 0.081]
<i>Wave 2</i>	0.059***	[0.032, 0.086]
<i>Wave 3</i>	0.061***	[0.030, 0.091]
DMN-CON		
<i>Wave 1</i>	0.041***	[0.017, 0.065]
<i>Wave 2</i>	0.042**	[0.015, 0.068]
<i>Wave 3</i>	0.042**	[0.011, 0.073]
SN-CON		
<i>Wave 1</i>	0.035**	[0.012, 0.058]
<i>Wave 2</i>	0.043***	[0.017, 0.069]
<i>Wave 3</i>	0.051***	[0.021, 0.081]
VAN-DAN		
<i>Wave 1</i>	0.043***	[0.020, 0.067]
<i>Wave 2</i>	0.050***	[0.024, 0.077]
<i>Wave 3</i>	0.058***	[0.028, 0.088]
VAN-CON		
<i>Wave 1</i>	0.047***	[0.023, 0.070]
<i>Wave 2</i>	0.051***	[0.024, 0.077]
<i>Wave 3</i>	0.055***	[0.025, 0.085]
DAN-DAN		
<i>Wave 1</i>	-0.036**	[-0.060, -0.013]
<i>Wave 2</i>	-0.041**	[-0.067, -0.014]
<i>Wave 3</i>	-0.045**	[-0.075, -0.015]

Note. For the hypothesized and exploratory models that demonstrated a significant association between baseline resting state functional connectivity and between-person levels of psychopathology (intercepts) but not within-person rates of change (slopes) in the main analyses (see Table 3), follow-up tests were conducted to determine whether baseline connectivity was associated with psychopathology scores at Waves 1, 2, and 3 (i.e., one-, two- and three-year follow-ups, respectively). Standardized estimates are shown. ***unadjusted $p < 0.001$;

**unadjusted $p < 0.01$; *unadjusted $p < 0.05$. DMN = default mode network; SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Table 5. Sensitivity Analyses of Within- and Between Network Resting State Functional Connectivity Relations with the Intercept and Slope of P-Factor Scores Over Wave.

Resting State Functional Connectivity Networks	Intercept (Main Effect)		Slope (Interaction Effect)	
	β	95% CI	β	95% CI
<i>Medication Covariate</i>				
DMN-DMN	-0.026*	[-0.047, -0.005]	-0.003	[-0.008, 0.002]
DMN-VAN	-0.029**	[-0.049, -0.009]	-0.008**	[-0.013, -0.003]
DMN-DAN	0.055***	[0.034, 0.076]	0.002	[-0.003, 0.007]
DMN-CON	0.039***	[0.019, 0.060]	0.000	[-0.005, 0.006]
SN-CON	0.025*	[0.005, 0.045]	0.008**	[0.003, 0.013]
VAN-DAN	0.036***	[0.015, 0.056]	0.007**	[0.002, 0.012]
VAN-CON	0.038***	[0.018, 0.059]	0.004	[-0.001, 0.009]
DAN-DAN	-0.031**	[-0.051, -0.011]	-0.004	[-0.009, 0.001]
<i>Excluding for High-Motion</i>				
DMN-DMN	-0.032*	[-0.059, -0.005]	-0.006	[-0.012, -0.001]
DMN-VAN	-0.028*	[-0.055, -0.002]	-0.011**	[-0.017, -0.004]
DMN-DAN	0.060***	[0.034, 0.087]	0.009**	[0.002, 0.015]
DMN-CON	0.035*	[0.008, 0.061]	0.001	[-0.005, 0.008]
SN-CON	0.050***	[0.025, 0.075]	0.007*	[0.001, 0.014]
VAN-DAN	0.030*	[0.004, 0.056]	0.012***	[0.005, 0.018]
VAN-CON	0.058***	[0.031, 0.083]	0.006	[-0.001, 0.012]
DAN-DAN	-0.035**	[-0.061, -0.010]	-0.007*	[-0.014, -0.001]
<i>Excluded for Completely Missing Follow-Up Data</i>				
DMN-DMN	-0.033**	[-0.054, -0.011]	-0.003	[-0.008, 0.002]
DMN-VAN	-0.031**	[-0.052, -0.010]	-0.008**	[-0.014, -0.003]
DMN-DAN	0.059***	[0.037, 0.080]	0.002	[-0.003, 0.007]
DMN-CON	0.044***	[0.022, 0.065]	0.001	[-0.005, 0.006]
SN-CON	0.028**	[0.008, 0.049]	0.008**	[0.003, 0.013]
VAN-DAN	0.040***	[0.019, 0.061]	0.007**	[0.002, 0.013]
VAN-CON	0.042***	[0.021, 0.063]	0.004	[-0.001, 0.009]
DAN-DAN	-0.036***	[-0.056, -0.015]	-0.004	[-0.010, 0.001]

Note. * unadjusted $p < 0.05$; ** unadjusted $p < 0.01$; *** unadjusted $p < 0.001$; DMN = default mode network; SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Figure Legends.

Figure 1. *Flowchart of Participant Exclusion for Present Study.*

Note: Figure 1 details how participants were excluded from the larger ABCD study sample for inclusion in the present study. The full ABCD baseline sample includes $n = 11,876$. The present study includes baseline data from $n = 9,344$ participants. fMRI = functional magnetic resonance imaging; QC = quality control; CBCL = childhood behavior checklist; Wave 2 = one-year follow-up; Wave 3 = two-year follow-up; Wave 4 = three-year follow-up.

Figure 2. *General Factor of Psychopathology (Romer et al., 2023; Romer & Pizzagalli, 2021).*

Note: Figure 2 represents the structure of general psychopathology (i.e., p-factor) identified previously in the ABCD sample. The higher-order factor “p” captures the shared variance across different dimensions of psychopathology. There are five lower-order factors related to specific dimensions of psychopathology. Sample items (but not all items) from the CBCL that load onto each specific factor are provided. P = general factor of psychopathology or “p-factor”; EXT = externalizing factor; INT = internalizing factor; ND = neurodevelopmental factor; SOMAT = somatization factor; DETACH = detachment factor.

Figure 3. *Intercept and Slope Effects of Altered Connectivity within and between Resting-State Functional Connectivity Networks.*

Note: Figure 3A illustrates the intercept effects and Figure 3B illustrates the slope effects of the association between altered baseline RSFC and p-factor scores over time, over and above age, sex, scanner type, and in-scanner motion. Link width and color are scaled by strength of association (wider, darker links = stronger effects). Intercept effects ranged from $\beta = -0.032$ to 0.055 . Slope effects ranged from $\beta = -0.008$ to 0.008 . Negative associations are shown in blue and positive associations are shown in red. Effects that were significant after FDR correction ($q < 0.05$) are outlined in black dashed lines. DMN = default mode network; FPN = frontoparietal network;

SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Figure 4. *Baseline RSFC Relations with the Rate of Change in P-Factor Scores over Wave.*

Note: The interaction between baseline RSFC and wave (slope) for p-factor scores is illustrated. Estimated marginal mean analysis revealed that preadolescents with lower DMN-VAN connectivity (1SD below mean) and higher VAN-DAN and SN-CON connectivity (1SD above mean) showed the steepest rates of u-shaped change in p-factor scores over time. All simple slopes are $p < 0.001$. SD = standard deviation; DMN = default mode network; FPN = frontoparietal network; SN = salience network; VAN = ventral attention network; DAN = dorsal attention network; CON = cingulo-opercular network.

Figure 1.

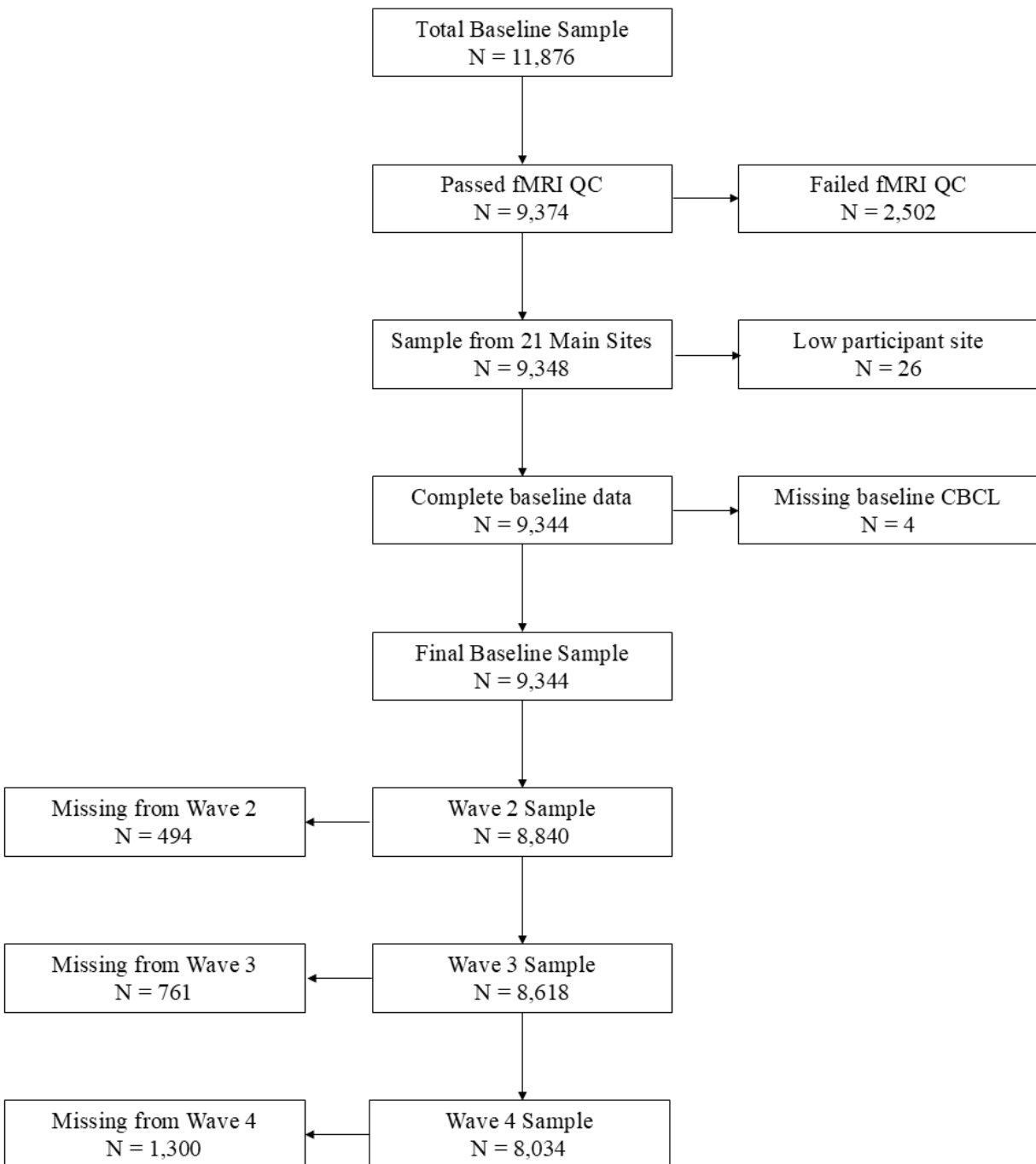


Figure 2.

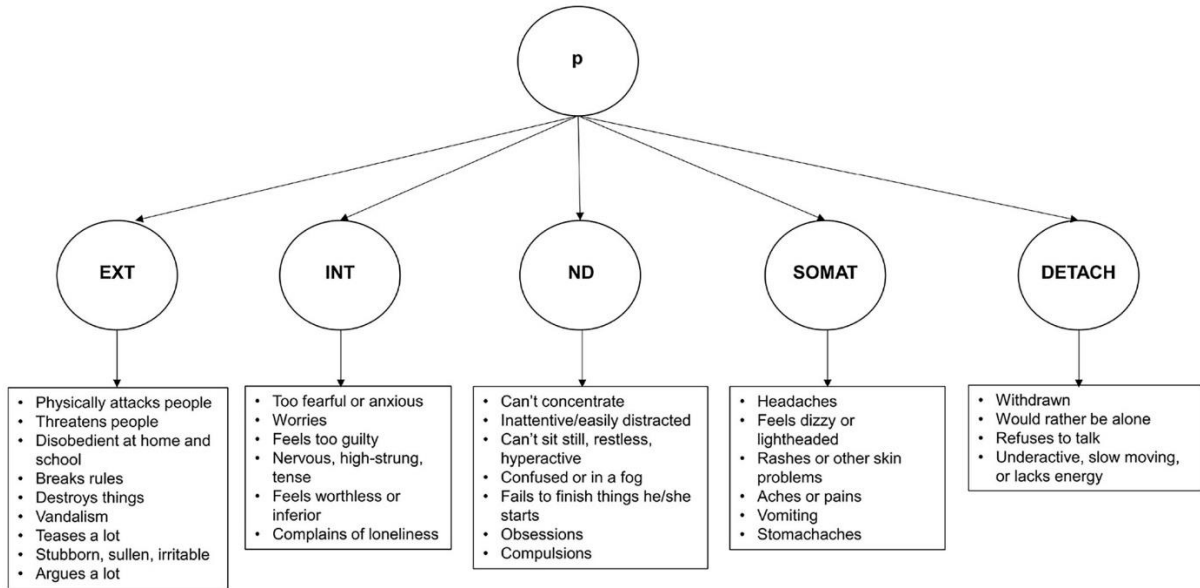


Figure 3.

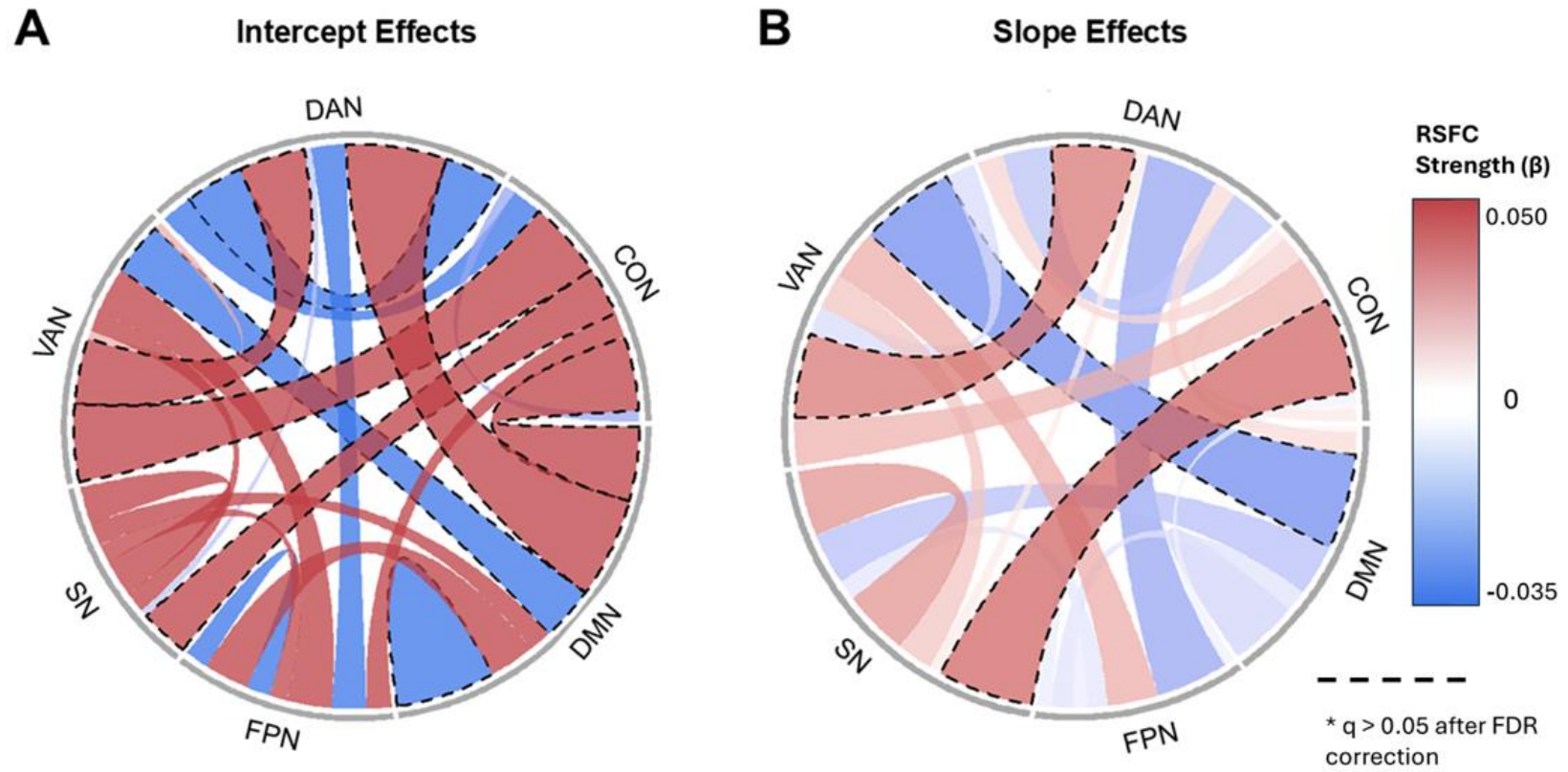


Figure 4.

