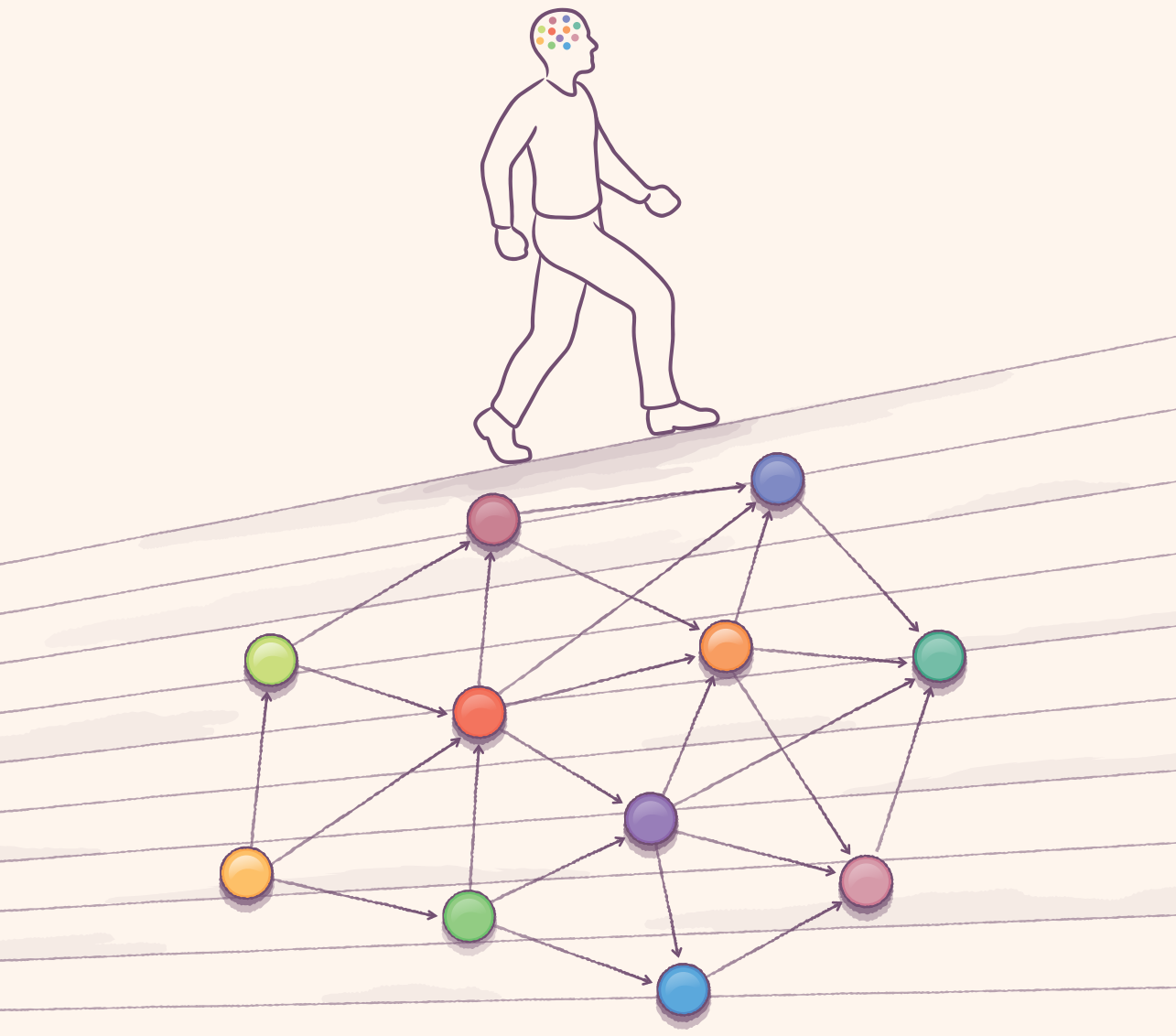
Executive Functioning and Developmental Psychopathology

Integrating Dynamics, Experimental and Modeling Approaches



René Freichel

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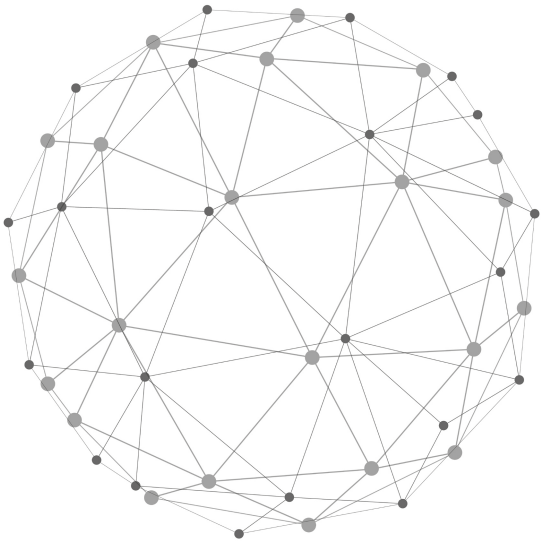

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Executive Functioning and Developmental Psychopathology: Integrating Dynamics, Experimental and Modeling Approaches

ACADEMISCH PROEFSCHRIFT

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aan de Universiteit van Amsterdam
op gezag van de Rector Magnificus
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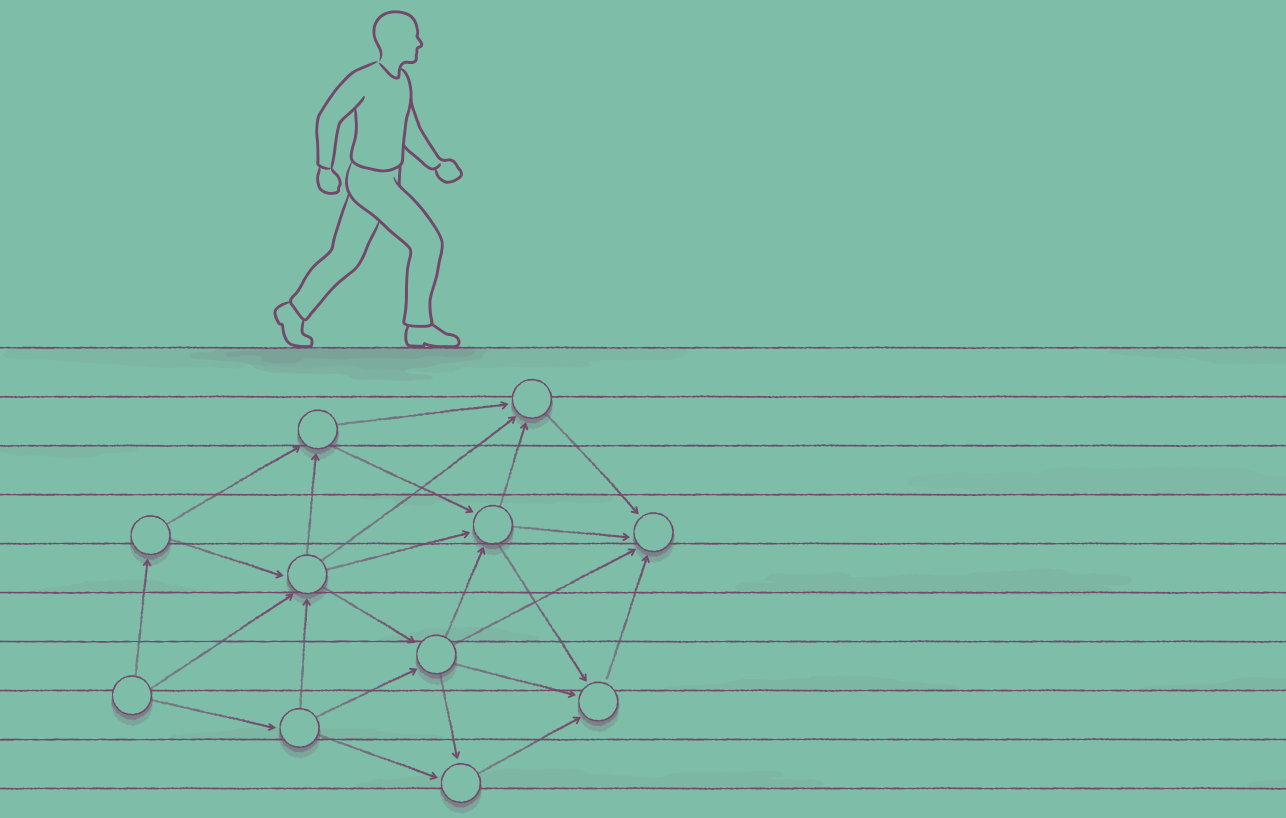

CHAPTER 1

General Introduction

Developmental Psychopathology is a field at the crossroads of clinical and developmental psychological science. It aims to provide answers to fundamental questions concerning the developmental processes that contribute to the emergence of psychopathology (Rutter & Sroufe, 2000). This interdisciplinary field views mental health problems as developmental phenomena that result from the interplay of distal and proximal risk and protective factors, ranging from psychological (e.g., comorbidities) and cognitive (e.g., inhibitory control) to genetic, biological, and social factors. By adopting a lifespan perspective, developmental psychopathology emphasizes how early experiences, traits, or characteristics may shape mental health outcomes later in life. Importantly, adolescence represents a key window for the development of mental health problems: A recent meta-analytical review of 192 epidemiological studies found that the peak age of onset for any mental disorder was 14.5 years (Solmi et al., 2022), highlighting early and middle adolescence as a critical window. Another study showed that one-fifth of the total disability burden globally was driven by mental health conditions in 5- to 24-year-olds (Kieling et al., 2024). These striking statistics highlight the importance of preventing and treating adolescent mental health conditions to reduce the overall disease burden and move individuals towards healthier life trajectories.

Symptoms of mental disorders can be categorized along two transdiagnostic dimensions that go beyond specific diagnoses listed in the International Classification of Diseases (ICD) or the Diagnostic and Statistical Manual of Mental Disorders (DSM): internalizing symptoms (manifested inward), such as depressive or anxiety symptoms, and externalizing symptoms (manifested outward), such as oppositional-defiant behaviors (Achenbach & Edelbrock, 1981; Caspi et al., 2014; Kotov et al., 2017). These descriptive dimensions show different developmental trajectories during adolescence. From early to mid-adolescence, internalizing symptoms increase, particularly among girls, while externalizing symptoms typically peak earlier and gradually decline over time for both sexes (Leve et al., 2005). These divergent trends highlight the importance of identifying risk factors of adolescent psychopathology to prevent mental health problems from persisting throughout life.

Paralleling this emergence of adolescent mental health problems is the development and maturation of cognitive control during adolescence. Cognitive control, an umbrella term that is sometimes used synonymously with executive functioning, refers to all processes that allow individuals to direct attention and to achieve goal-directed thoughts or behaviors (Diamond, 2013). Prominent models of cognitive control feature separate but correlated functions, such as inhibitory control, shifting attention, and working memory (Miyake et al., 2000). Impairments in cognitive control are common in multiple mental disorders, thus potentially representing a transdiagnostic risk factor of psychopathology (McTeague et al., 2016). Analogous to the development of the p-factor, a general liability to develop mental disorders (Caspi et al., 2014), Abramovitch and colleagues (2021) showed that the C-factor (cognitive dysfunction) represents a universal transdiagnostic marker of mental disorders. The transdiagnostic relevance of cognitive control has reached contemporary frameworks for studying mental health. For instance, the National Institute of Mental Health (NIMH) developed the Research Domain Criteria (RDoC), a framework that replaces diagnostic categories with functional domains that may explain specific symptoms. One of the six

functional domains is the cognitive system that includes, among others, cognitive control, working memory, and attention as relevant dimensions (Insel et al., 2010).

To advance our understanding of the emergence of adolescent psychopathology, it is important to examine dynamic developmental processes rather than static time-invariant risk factors. One promising avenue to capture these dynamic developmental processes is by using network analysis, a relatively novel methodological approach. Network analysis, as a statistical toolkit, was inspired by the network theory of mental disorders (Borsboom, 2017; Borsboom & Cramer, 2013; Cramer et al., 2010) that views mental disorders as systems of dynamically interacting symptoms rather than latent entities. Networks consist of nodes (variables) that are connected through edges representing statistical relations. The ability to examine partial associations (while controlling for all other nodes in the network) represents a compelling advantage of network analysis to identify which symptom-symptom or cognitive control-symptom association is strong and may indicate a direct relationship. In the context of developmental psychopathology, network models may thus be able to capture 'developmental cascades' – temporal links between symptoms and cognitive control functions that help explain the emergence of adolescent psychopathology.

This thesis aims to further our understanding of the interplay of cognitive control and adolescent psychopathology using innovative assessment paradigms and network analytical approaches. Specifically, the thesis aims to (1) apply advanced statistical network modeling approaches to characterize the dynamic, longitudinal interplay of cognitive control functions and symptoms, (2) introduce novel paradigms to assess cognitive control, with a focus on attentional control, and (3) identify methodological challenges particularly concerning the use of symptom network analysis in clinical-developmental science.

The thesis is organized into three main parts, followed by an integrative discussion. **Part 1** of the thesis focuses on the longitudinal associations between executive functioning and transdiagnostic symptom domains within the context of developmental psychopathology. Chapter 2 examines the prospective associations between different executive functions at age 11 and internalizing and externalizing symptoms at ages 13 and 15. Chapter 3 shifts focus to adolescent substance use, examining how impulsivity, closely related to cognitive control, and other more distal risk factors (personality traits and life stressors) predict adolescent alcohol use. This chapter uses longitudinal network models to test developmental theories of alcohol use risk that describe drinking motives as proximal risk factors on which more distal factors (i.e., personality traits, stressful life events) converge. Chapter 4 tests competing theories in developmental psychopathology that view executive function impairments as risk factors versus consequences of psychopathology. Chapter 5 introduces a novel moderated cross-lagged panel network approach (mCLPN) to examine how outside factors, such as cognitive control, may moderate the temporal dynamics between substance use and externalizing and internalizing symptoms.

Part 2 of the thesis complements findings from longer time scales (i.e., years, see Part 1) by examining short-term dynamics at the level of hours and days. Chapter 6 uses ecological momentary assessment (EMA) data to examine daily mood fluctuations and their associations with executive functioning at the hour-to-hour and day-to-day level. Chapter 7

presents a novel paradigm for assessing attentional control, specifically attentional capture - the phenomenon that stimuli with certain features (high-value reward signals) capture more attention (Pearson et al., 2024). This chapter presents a novel VMAC task with both reward and punishment contexts and examines cross-sectionally how these attentional biases relate to mental health outcomes.

The final **Part 3** of the thesis (Chapters 8-10) addresses broader methodological challenges in using network analysis for studying developmental psychopathology. Chapter 8 introduces brain-symptom network models to better understand the associations between depressive symptoms, including cognitive dysfunction, and relevant neural markers. Chapter 9 offers an overview of relevant longitudinal network analysis and structural equation modeling approaches for studying dynamic developmental processes. Chapter 10 presents the first preregistration checklist for longitudinal network models to increase transparency and the robustness of findings derived from longitudinal network analysis in developmental psychopathology. Chapter 11 concludes the thesis by integrating results from the different chapters of the thesis. This chapter highlights relevant clinical implications and points to methodological challenges and future directions.

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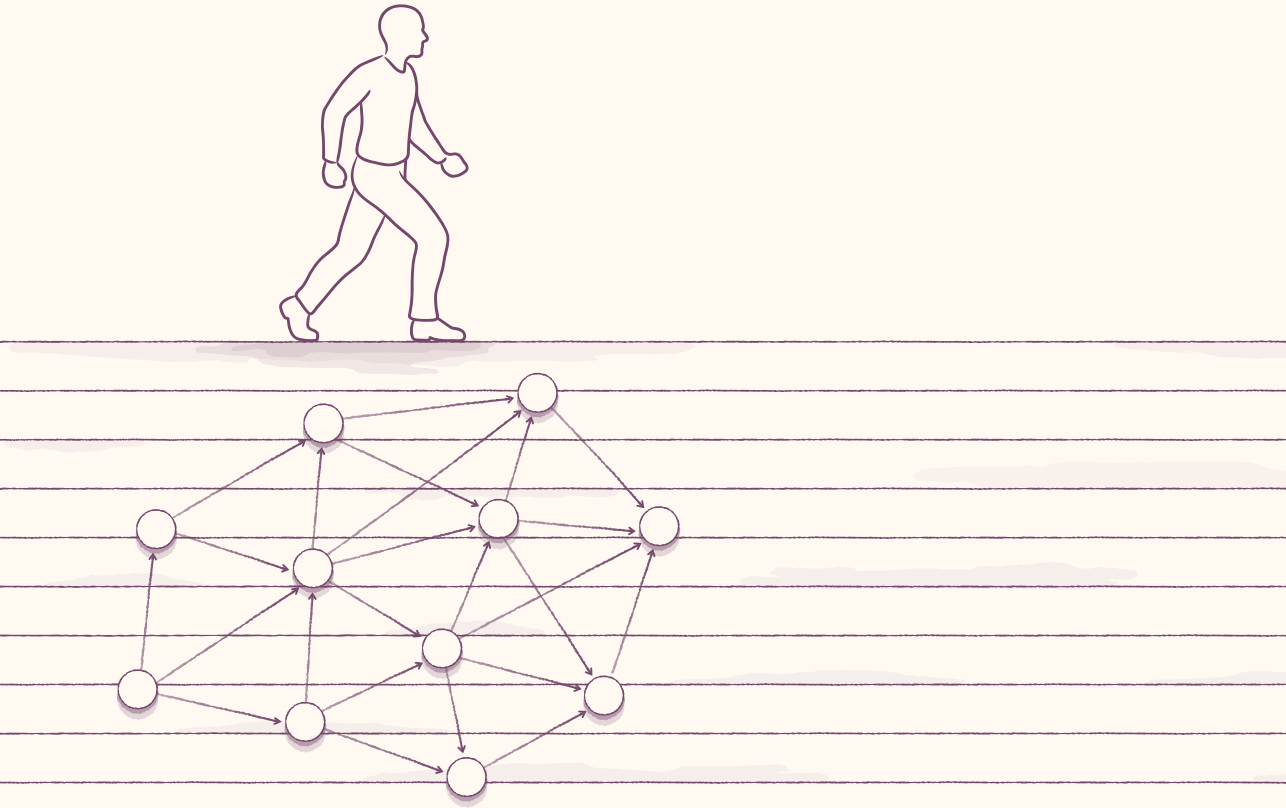
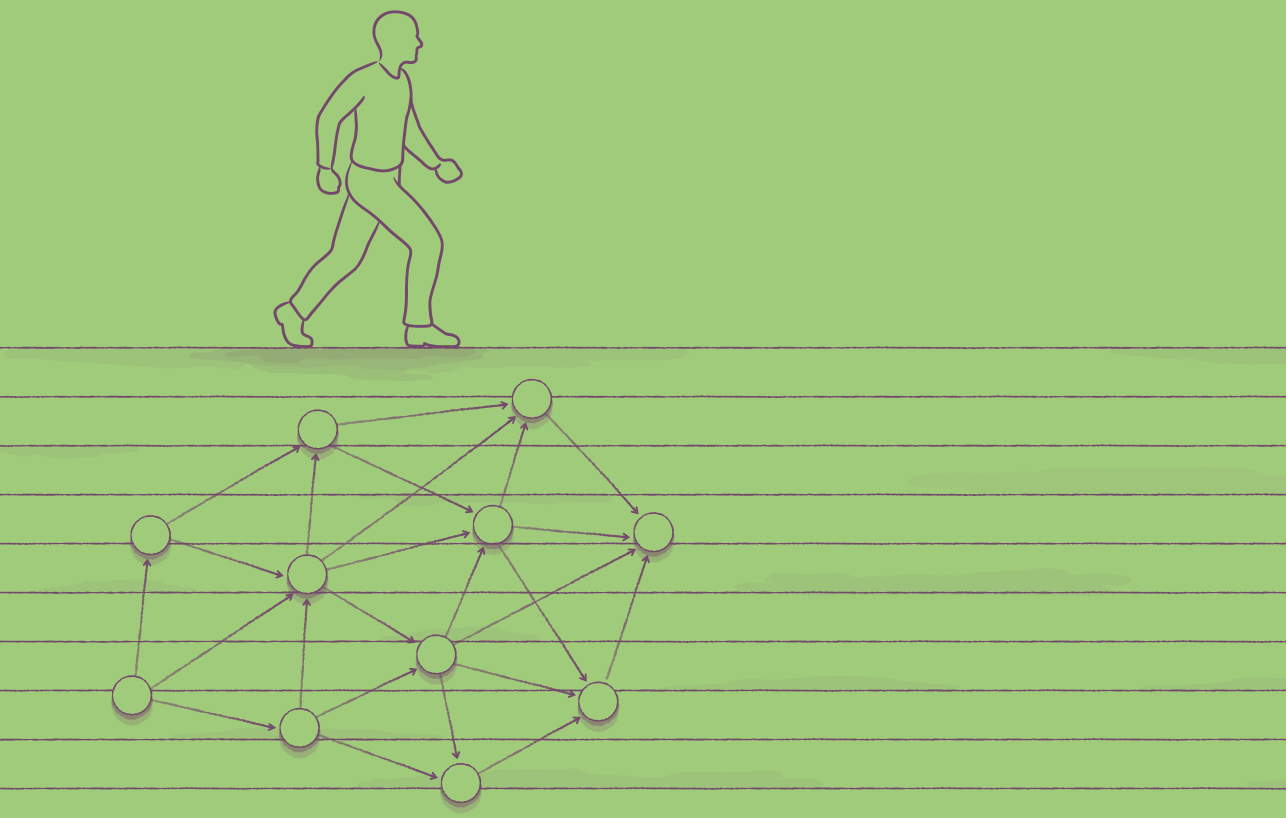


Part 1

Executive Functioning in Developmental Psychopathology



CHAPTER 2

Executive functioning, internalizing and externalizing symptoms: Understanding developmental dynamics through panel network approaches

This chapter is adapted from:
Freichel, R., Pfirrmann, J., de Jong, P. J., Cousijn, J., Franken, I. H. A., Oldehinkel, A. J., Veer, I. M., & Wiers, R. W. (2023). Executive functioning, internalizing and externalizing Symptoms: Understanding developmental dynamics through panel network approaches. *JAACAP Open*, 2(1), 66–77. https://doi.org/10.1016/j.jaacop.2023.11.001

Abstract

Objective: Early adolescence is a transition period during which many mental health disorders emerge. The interplay between different internalizing and externalizing mental health problems in adolescence is poorly understood at the within-person level. Executive functioning (EF) in early adolescence has been shown to constitute a transdiagnostic risk factor, but the specificity of the associations between different domains of EF and mental health problems remains unclear.

Method: Network dynamics (i.e., temporal effects) of different internalizing and externalizing symptoms were investigated leveraging data from the Tracking Adolescents' Individual Lives Survey (TRAILS), a large longitudinal panel study of adolescents (> 1,641 participants) assessed at ages 11, 13, and 15. Two novel methodological panel network approaches were used: cross-lagged panel network models and graphical vector autoregressive models. Hierarchical regression models were used to investigate prospective associations between different measures of EF and broadband transdiagnostic dimensions.

Results: Depressive problems predicted a range of other internalizing symptoms (i.e., panic, somatic problems, separation anxiety, general anxiety, social phobia) over time, particularly during early adolescence. Important feedback loops with reciprocal associations between different anxiety symptoms were identified. Different facets of EF assessed at age 11, particularly sustained attention, showed weak but significant prospective associations with internalizing and externalizing symptoms at age 13.

Conclusion: The present findings emphasize the importance of targeting depressive problems in early adolescence to prevent a spiral of different internalizing symptoms from arising later on.

Key words*:* executive functioning; externalizing; internalizing; network

Introduction

Early adolescence is a key transition period during which the prevalence of a range of mental health problems increases and in which top-down executive functions have been proposed to play a role (Davidson et al., 2015). Globally, approximately 25 to 31% of adolescents experience a common mental health disorder (Silva et al., 2020), thereby making adolescent psychopathology a substantial burden of disease and a growing source of disability (Twenge et al., 2019). The vast majority of mental health disorders diagnosed in adults, including internalizing and externalizing problems, emerge during adolescence and have been associated with a range of negative long-term consequences (Kessler et al., 2007). Thus, studying the emergence of different mental health problems during adolescence is crucial to better identify early risk factors and the deleterious cycles that may contribute to persistence of symptoms.

Symptoms of adolescent mental health problems are highly comorbid and have been shown to predict each other over time (Kessler et al., 2012). This high level of comorbidity and interdependence between symptoms of different disorders motivated the development of novel methodological frameworks, such as the symptom network approach (Borsboom & Cramer, 2013). The network approach poses that psychopathology arises from complex interactions between symptoms, both within the same time window and across time. The symptom interactions (edges) between distinct symptom clusters (e.g., internalizing – externalizing) are called 'bridge symptoms' and might have an important role in the emergence of comorbidities (Jones et al., 2021). Therefore, panel network models are uniquely equipped to provide mechanistic insights into the development of psychopathology during key transition periods, such as adolescence.

To date, numerous studies have examined cross-sectional associations between internalizing and externalizing symptoms (Funkhouser et al., 2021; McElroy et al., 2018). Developmental process models, such as the developmental cascade model (Moilanen et al., 2010) have been proposed to explain symptom dynamics according to which externalizing problems primarily predict subsequent internalizing symptoms, rather than the reverse direction. Adolescent externalizing behaviors, such as delinquency and aggression, may give rise to negative responses from the adolescent's environment, such as social rejection, academic difficulties, or punishment, which, in turn, may precede internalizing symptoms, such as anxiety or depression (Patterson & Capaldi, 1990; Weeks et al., 2016). Countless studies found sex differences in the prevalence of adolescent internalizing (i.e., higher prevalence among girls) (Mojtabai et al., 2016) and externalizing (i.e., higher prevalence among boys) symptoms (Eme, 2016). Moreover, there is evidence suggesting sex differences in the developmental trajectories of externalizing problems across adolescence (Cleverley et al., 2012). Little is known about sex differences in developmental cascades (i.e., predictive associations between internalizing and externalizing symptoms). Moreover, most studies testing within-person theories have failed to disentangle within- and between-person effects in the symptom associations over time, and thus little is known about the dynamic changes

within the network structure, and which symptoms bridge the internalizing and externalizing domains at the different stages of adolescence.

Existing studies on the associations between different internalizing and externalizing symptoms show 3 important limitations that we aim to address in the present study. First, most network analysis studies are cross-sectional studies and consequently lack temporal precedence. For instance, a recent network analysis study showed that generalized anxiety disorder symptoms are most central in largely similar symptom network constellations across different age groups (i.e., ages 7.5-14) (McElroy et al., 2018). However, little is known about how different internalizing and externalizing symptoms predict each other over time - temporal associations that are crucial for informing causal inferences. Second, prior evidence for the co-occurrence of heterogeneous symptoms of different disorders is primarily based on between-person relationships. As widely noted, understanding mechanistic pathways traditionally entails an examination of isolated within-person (change within individuals) effects that form the basis for intervention efforts (within-person change) (Curran & Bauer, 2011). Third, existing studies on the co-occurrence and interplay between internalizing and externalizing symptoms are mostly restricted to the later stages of adolescence and early adulthood. Our study is the first to examine contemporaneous (within the same time window) and temporal associations across a 6-year period in early adolescence (ages 11-16).

A core risk factor for the development of internalizing and externalizing mental health problems may be relatively suboptimal executive functioning (EF) in early adolescence. Impairments of EF, including working memory, response inhibition, cognitive flexibility, and sustained attention (Tillman et al., 2015) have been associated with elevated internalizing and externalizing symptoms. Moreover, a general psychopathology factor ('p factor', Caspi et al., 2014) has been associated with poorer EF (Romer & Pizzagalli, 2021). However, existing studies primarily examined associations between single executive functions (e.g., working memory) and symptoms in isolation. Little is known about the specificity of associations between executive functions and the development of internalizing and externalizing symptoms at different stages of adolescence.

Using a longitudinal panel dataset of more than 2,100 adolescents, our study aimed to examine the dynamic interplay between a range of internalizing and externalizing mental health problems. Based on developmental cascade models, we predicted that externalizing problems should largely predict internalizing problems over time. We used 2 different panel network analytical approaches in parallel. First, cross-lagged panel network (CLPN) models were used to examine age-specific and wave-by-wave changes in the network structure while conflating within- and between-person effects. Second, panel graphical vector autoregressive (GVAR) models were used to separate within- and between-person effects in the overall network structure. The parallel use of both approaches provided us with a unique perspective for understanding developmental cascades at both the intraindividual level (i.e., individuals' within-person deviations from the mean) and the interindividual level (i.e., individuals' scores on a variable relate to scores of others on another variable). Both statistical approaches serve different goals, namely, the understanding of within-person mechanisms (i.e., panel GVAR model) as well as the prediction of future outcomes based on the combined within- and

between-person effects (i.e., CLPN analysis). Finally, we connected this development of 2 broadband transdiagnostic dimensions (internalizing, externalizing) to different domains of EF assessed at age 11.

Method

Data Source and Procedure

We used data from the Tracking Adolescents' Individual Lives Survey (TRAILS) study, a longitudinal cohort study of Dutch (pre-)adolescents assessed every two to three years from ages 10-11 (wave 1) to ages 28-30 (wave 7). The study recruited a representative general population sample from urban and rural areas across five municipalities in the Netherlands. The study design, procedure, and sample characteristics are described in detail elsewhere (de Winter et al., 2005; Oldehinkel et al., 2015). The present study used data from the first three assessment waves (ages 10-16), considering that all relevant outcome measures have been assessed consecutively with the same instrument during these waves. At each wave, a range of self-report questionnaires and clinical interviews were administered. Neuropsychological and cognitive tasks were administered during the first wave only.

Measures

Youth Self-Report. The Youth Self-Report (YSR; Achenbach, 2001) is a commonly used and well-validated measure of behavioral and emotional problems of children aged 11-19 years old. The YSR consists of 112 items that assess internalizing and externalizing mental health problems on a three-point scale (0 = not true, 1 = somewhat or sometimes true, 2 = very or often true). The YSR includes two broadband domains, namely Internalizing (31 items; Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints scales) and Externalizing (32 items; Rule-Breaking Behavior and Aggressive Behavior scales) symptoms. Moreover, there are specific DSM-oriented scales for Depressive Problems, Anxious Problems, Somatic Problems, Attention Deficit Hyperactivity Problems, Oppositional Defiant Problems, and Conduct Problems. The respective items were averaged to construct continuous scale scores ranging between 0 (no symptoms present) and 2 (all symptoms always or very present). Our analyses focused on all subscales, excluding the anxiety subscale as anxiety was assessed in more detail using the Revised Child Anxiety and Depression Scale (RCADS).

Revised Child Anxiety and Depression Scale. The RCADS (Chorpita et al., 2000) is a self-report measure of five anxiety subtypes and depression symptoms. The questionnaire consists of 47 items that are scored on a four-point Likert scale (0 = never, 1 = sometimes, 2 = often, 3 = always). The RCADS assessment comprises six scales (separation anxiety, generalized anxiety, social phobia, panic disorder, obsessive-compulsive disorder (OCD), major depressive disorder) corresponding largely to the DSM-5 dimensions of anxiety and depressive disorders. The depression scale was not assessed at wave 3. Satisfactory psychometric properties of the RCADS have been well documented, including a replication of the factor structure in the TRAILS sample (Ferdinand et al., 2006).

Executive Functioning. EF was assessed at ages 10-12 (wave 1) using the Amsterdam Neuropsychological Task battery (De Sonneville, 1999). The following tasks were included: Sustained Attention Dots (SAD) task, measuring sustained attention, Shifting Attentional Set-visual (SAD-v) task measuring response inhibition and cognitive flexibility, as well as the Memory Search Letters (MSL) task measuring working memory maintenance. Following previous recommendations on these tasks (Brunnekreef et al., 2007), we removed all reaction-time (RT) and accuracy measures (outlier) with an absolute z-score larger than or equal to 4.

Sustained Attention Dots (SAD). The SAD task assesses the ability to sustain attention over time (De Sonneville, 1999). On each trial, participants are randomly presented with a three-, four-, or five-dot pattern and instructed to press the "yes" button for a target (four-dot), and the "no" button for all other (three-dot or five-dot) patterns. An auditory feedback signal is provided for erroneous responses. The task consists of a total of 600 visual dot patterns that are presented across 50 series of 12 trials each. There are 200 trials for each type of stimulus (three-, four-, or five-dot pattern), resulting in a 1:2 target to non-target ratio. The primary outcome measure of the task is the within-person standard deviation of the mean reaction time of 50 series (fluctuation in tempo), as well as the overall percentage of errors. Higher scores on these performance measures indicate worse sustained attention.

Shifting Attentional Set–Visual (SAD-V). The SAD-V task measures two components of shifting attention: response inhibition and cognitive flexibility (De Sonneville, 1999). It consists of three parts in which participants mimic the direction of jumping squares by clicking the left or right mouse button. In part one, one square is green and jumps randomly (fixed-compatible condition); in part two, the square is red and participants must mirror the direction (fixed-incompatible condition); and in part three, the square can be green or red and participants must adapt their response based on the color of the square (random condition). Response inhibition is calculated as the difference in mean reaction time/percentage of errors between part two and one (Bloemen et al., 2018). Cognitive flexibility is computed by subtracting the mean reaction time/percentage of errors of part one from part three. Higher difference scores indicate weaker cognitive flexibility.

Memory Search Letters (MSL). The MSL assesses working memory maintenance in varying memory load and distraction (De Sonneville, 1999). It consists of three parts in which participants memorize one, two, or three target letters. Participants must decide whether four letters on the screen contain the target letter. Target and non-target trials alternate in random order. Working memory maintenance is computed as the difference between part three (3 target letters) and part one (1 target letter) mean reaction times/percentage of errors (Bloemen et al., 2018). Higher difference scores indicate worse working memory maintenance.

Data Analysis

To examine how different internalizing and externalizing symptoms predict each other over time, we used two distinct panel network analytical approaches. First, we implemented a CLPN approach following the study by Zainal and Newman (2022) to examine age-specific developmental changes. Second, we used graphical vector autoregressive (GVAR) models that allowed us to separate within- and between-person effects in the network structure, and

discern contemporaneous and temporal associations. These two approaches offer unique advantages (CLPN: wave-by-wave analysis, panel GVAR: within- and between-person effects separation) in studying the symptom interplay. To examine how EF measures assessed at the first wave relate to the development (waves 2-3) of internalizing and externalizing mental health problems, we used hierarchical regression models.

CLPN Models. CLPN models (Rhemtulla et al., 2022) were used to examine the interrelationship between all ten internalizing and externalizing symptoms during the three waves of data. CLPN models examine lag-1 cross-lagged relationships between all nodes after incorporating autoregressive effects (i.e., a node predicting itself over time). Regularized regressions that included the Least Absolute Shrinkage and Selection Operator (LASSO) with 10-fold cross-validation were used to calculate autoregressive and cross-lagged estimates between two consecutive waves (i.e., wave 1 to wave 2; wave 2 to wave 3). The LASSO regularization in the model implements a penalty procedure (using a tuning parameter γ) through which weak coefficients (i.e., edges) are set to zero. This estimation method leads to sparser networks with a lower probability of obtaining false-positive edges in the network. Cross-validation was used to select the optimal γ parameter. Directed cross-lagged edges in the temporal networks represent associations between different nodes across time while controlling for all other nodes in the network. Cross-sectional networks were estimated using the 'EBICglasso' algorithm that uses Bayesian information criterion (BIC) model selection and LASSO regularization with a hyperparameter set to 0.5 to remove spurious edges from the network structure.

GVAR Model. We used panel GVAR network models(Epskamp et al., 2018) to discern temporal (predictions over time) and contemporaneous (within the same time window) associations in the network structure. The panel GVAR model is structurally similar to a random-intercept cross-lagged panel model(Epskamp, 2020; Hamaker et al., 2015) and separates within-person (changes within individuals) and between-person (relationship between means) effects. We detrended the data for linear and quadratic effects of time and standardized across waves to ensure stationarity, as is commonly done in panel GVAR approaches that focus on the correlational structure of interest (Freichel et al., 2023; Speyer et al., 2021). First, we fitted a saturated model (with all edges) that was pruned with a step-up model search (alpha=0.05) to remove false positives (Blanken et al., 2022). Model fit was evaluated according to standard criteria of good fit as indicated by the Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and the Tucker-Lewis index (TLI). This novel panel GVAR modeling approach yielding average within-person effects ensures that trait-like (between-person) effects are accounted for in the estimation of contemporaneous and temporal networks.

Network Centrality Metrics. For the temporal network, we computed the in-strength (i.e., sum incoming edge weights) and out-strength (i.e., sum outgoing edge weights) for every node. These commonly used measures capture the degree to which variables exert their influence (out-strength, influence) and are being influenced (in-strength, predictability) by other variables in the network. To examine the extent to which different symptoms act as important bridge symptoms in the contemporaneous networks, we computed measures of

bridge centrality using the networktools package (Jones, 2018). Bridge centrality describes the sum of edge weights between a node in one community (e.g., internalizing symptoms) and all other nodes from a different community (e.g., externalizing symptoms).

Estimation and Model Stability Analysis. There was a substantial drop-out of participants throughout the waves (participation rate: wave 2: 96%; wave 3: 76%). We used Full-Information-Maximum-Likelihood (FIML) estimation in both the panel GVAR network and regression analyses. FIML is a gold-standard approach (Enders, 2001) that provides unbiased estimates that are similar to multiple imputation procedures assuming data missing at random. We used a case-dropping bootstrapping analysis to examine the stability of the estimated edge weights and centrality measures. Panel network models were estimated and visualized using the R packages glmnet, psychonetrics, and qgraph (Epskamp, 2021; Epskamp et al., 2012; Friedman et al., 2017).

Hierarchical Regression Models. We used hierarchical regression models to investigate how executive functions (measured at wave 1) affect the development of internalizing and externalizing symptoms at wave 2 and wave 3. We first included the aggregate measures of symptoms (at the previous wave) and sex as predictors, then added all EF measures. We compared the variance explained by the two models. For instance, for the prediction of internalizing symptoms at wave 2, the nested two-step models follow the specification, and the same was done for wave 3:

Model 1: Internalizing Symptoms wave 2 = Sex + Internalizing Symptoms wave 1 + Externalizing Symptoms wave 1

Model 2: Internalizing Symptoms wave 2 = Sex + Internalizing Symptoms wave 1 + Externalizing Symptoms wave 1 + Working memory RT wave 1 + Working memory Errors wave 1 + Response Inhibition Errors wave 1 + Response Inhibition RT wave 1 + Fluctuation Tempo wave 1 + Fluctuation Errors wave 1 + Cognitive Flexibility RT wave 1 + Cognitive Flexibility Errors wave 1

Results

At the group level, we observed an increase in the average level of externalizing symptoms, and a decrease in the average level of internalizing symptoms throughout early and middle adolescence (see Supplement 1, available online). In particular, there was a trend of increasing prevalence of attention-deficit/hyperactivity disorder (ADHD) and decreasing prevalence of separation anxiety during adolescence. See Tables S1 and S2 (available online) for further details regarding the significant changes and proportion of missingness across waves. Table 1 provides reports relevant sample characteristics. According to the cutoff scores for the YSR, 23% of individuals showed a borderline (subclinical) or clinical score for any internalizing mental health problems at wave 1. 12% of individuals showed a borderline or clinical score for any externalizing mental health problem at wave 1.

Table 1

Descriptive Sample Characteristics Information

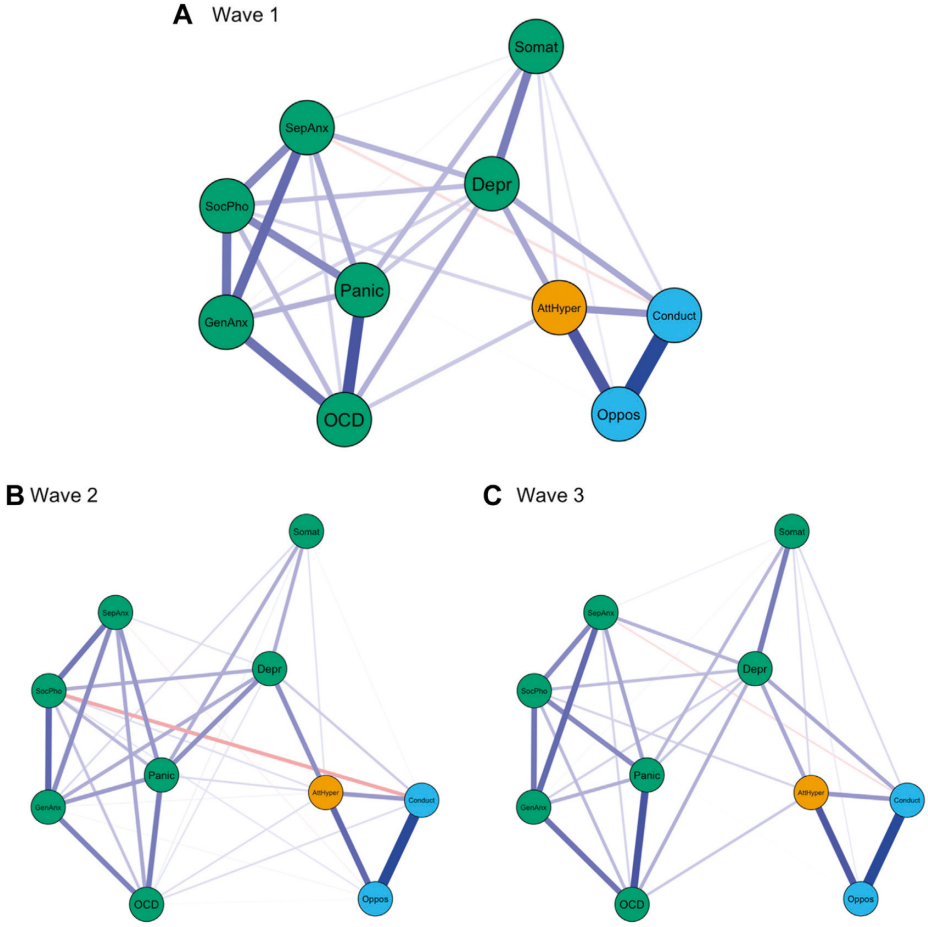
	Wave 1		Wave 2		Wave 3	
	Mean	SD	Mean	SD	Mean	SD
N	2170		2074		1641	
Sex (% female)	50.92		51.30		53.26	
Age	10.61	0.65	13.07	0.61	15.78	0.77
Internalizing scale score (YSR)	0.36	0.24	0.33	0.24	0.31	0.25
Externalizing scale score (YSR)	0.27	0.20	0.29	0.20	0.31	0.21
Depressive Problems (YSR)	0.29	0.25	0.27	0.26	0.30	0.27
Somatic Problems (YSR)	0.46	0.33	0.32	0.29	0.26	0.28
Attention Deficit Hyperactivity Problems (YSR)	0.59	0.36	0.67	0.38	0.68	0.38
Oppositional Defiant Problems (YSR)	0.45	0.35	0.46	0.35	0.46	0.35
Conduct Problems (YSR)	0.23	0.20	0.23	0.19	0.24	0.20
General anxiety Disorder (RCADS)	0.66	0.45	0.48	0.43	0.52	0.42
Social Phobia (RCADS)	0.78	0.43	0.68	0.46	0.73	0.50
Separation Anxiety (RCADS)	0.37	0.35	0.24	0.29	0.22	0.26
Panic Disorder (RCADS)	0.43	0.36	0.30	0.32	0.28	0.29
Obsessive-compulsive Disorder (RCADS)	0.60	0.44	0.34	0.35	0.29	0.36

Note. SD = Standard Deviation, YSR = Youth Self-Report; RCADS = Revised Child Anxiety and Depression Scale.

Contemporaneous Associations Between Internalizing and Externalizing Symptoms

Figure 1 displays cross-sectional associations among internalizing and externalizing symptoms at waves 1, 2, and 3. The overall network structure showed strong similarities across the three waves. As expected, internalizing and externalizing symptoms clustered together and showed strong positive associations within their respective cluster. Interestingly, depressive problems emerged as a key bridge symptom connecting a range of internalizing and externalizing symptoms at all three waves. Moreover, attention/hyperactivity problems and conduct problems showed moderately strong positive associations with depressive problems and somatic problems. We observed a negative association between social phobia and conduct problems only at wave 2.

Figure 1. Cross-sectional Internalizing-Externalizing Networks



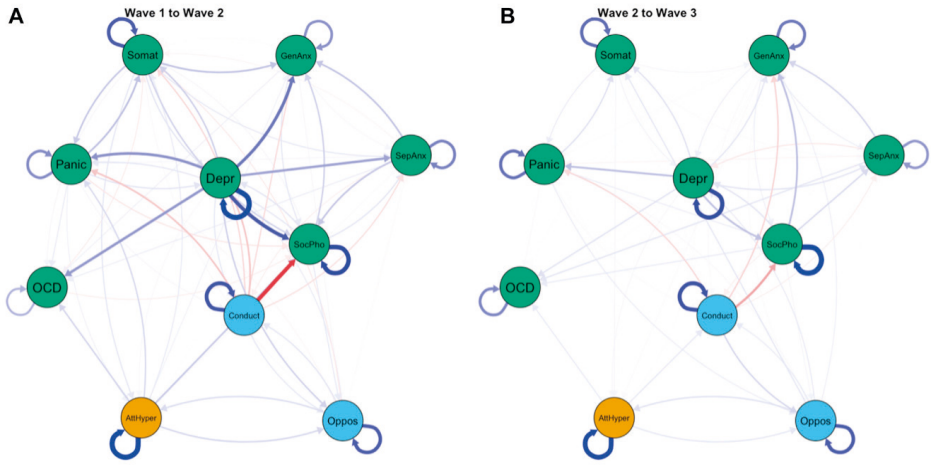
Note: (A-C) Waves 1-3. Attention problems abbreviations: AttHyper = attention-deficit/hyperactivity disorder; Externalizing abbreviations: Oppos = oppositional defiant problems; conduct = conduct problems; Internalizing abbreviations: Depr = depressive problems; Somat = somatic problems; GenAnx= Generalized Anxiety Disorder; SocPho = social phobia; SepAnx = separation anxiety disorder; Panic = panic disorder; OCD = obsessive-compulsive disorder.

To quantify the importance of different bridge symptoms, connecting internalizing and externalizing symptom clusters, we computed a measure of bridge centrality for the contemporaneous networks at all three waves (see Figure S1, available online). Consistent with the visual inspection, Depressive Problems constitute the most important internalizing bridge symptom It should be noted that the importance of Depressive Problems as bridge symptoms appears to be decreasing across the three waves.

Internalizing – Externalizing Symptom Dynamics Over Time

We examined temporal associations (see Figure 2) separately between waves (i.e., wave 1 – wave 2; wave 2 – wave 3) in the cross-lagged panel network model. Overall, we observed a complex interplay between different internalizing and externalizing symptoms that predicted each other over time through numerous pathways. During early adolescence (Figure 2A), depressive problems emerged as a key predictor of other internalizing problems (e.g., panic disorder, somatic problems, separation anxiety, general anxiety disorder, social phobia, OCD). This influence of depressive problems on other nodes is also shown in the high out-strength (strength of outgoing edges) of depressive problems. The degree of influence of depressive problems on other symptoms was substantially lower during the later stages of adolescence (waves 2-3). Importantly, social phobia symptoms were predicted by a range of other symptoms (e.g., lower conduct problems, depressive problems, separation anxiety symptoms). This high level of predictability of social phobia symptoms during later adolescence can also be seen in the high in-strength (strength of incoming edges) (see Figures S2-S3, available online). The temporal networks at both change points (wave 1 to wave 2 and wave 2 to wave 3) indicated various reinforcing feedback loops (e.g., attention-deficit hyperactivity problems – oppositional defiant problems) in which two symptoms predict each other over time.

Figure 2. Temporal Internalizing-Externalizing Networks from Cross-Lagged Panel Model

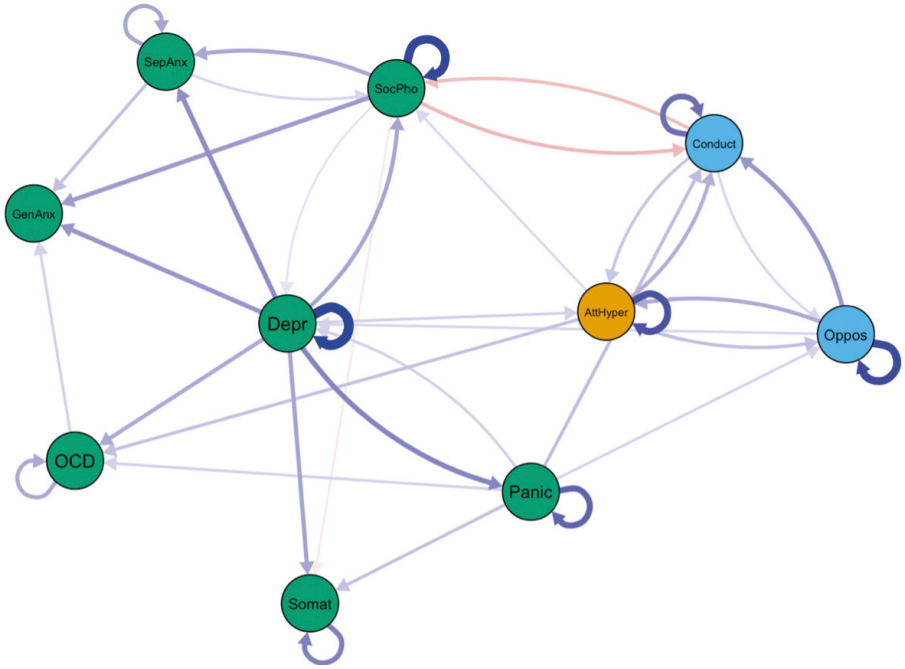


Note: A) Wave 1 to 2. (B, C) Wave 2 to 3**.** AttHyper = Attention Deficit Hyperactivity, Oppos = Oppositional Defiant Problems, Conduct = Conduct Problems, Depr = Depressive Problems, Somat = Somatic Problems, GenAnx = Generalized Anxiety disorder, SocPhob = Social Phobia, SepAnx = Separation Anxiety Disorder, Panic = Panic Disorder, OCD = Obsessive-compulsive Disorder.

To validate the findings from our cross-lagged panel network analyses described above, we used a novel panel GVAR approach that can separate within- and between-person effects in the network structure. The pruned panel GVAR models showed a good fit to the data (root mean square error of approximation = 0.043, comparative fit index = 0.95, Tucker-

Lewis index = =0.94). The contemporaneous (Figures S4-S5, available online) and temporal networks (Figure S6, available online) from the panel GVAR analyses generally replicated the central findings from our CLPN analysis at a within-person level. Despite the large similarities, we found some differences between the methods as the directionality of a few associations (e.g., Conduct Problems – Social Phobia) changed in the panel GVAR model (see Figure 3).

Figure 3. Pruned Temporal Network from Panel Graphical Vector Autoregressive Model



Note: AttHyper = attention deficit hyperactivity; Oppos = oppositional defiant problems; Conduct = conduct problems; Depr = depressive problems; Somat = somatic problems; GenAnx = generalized anxiety disorder; SocPhob = social phobia; SepAnx = separation anxiety disorder; Panic = panic disorder; OCD = obsessive-compulsive disorder.

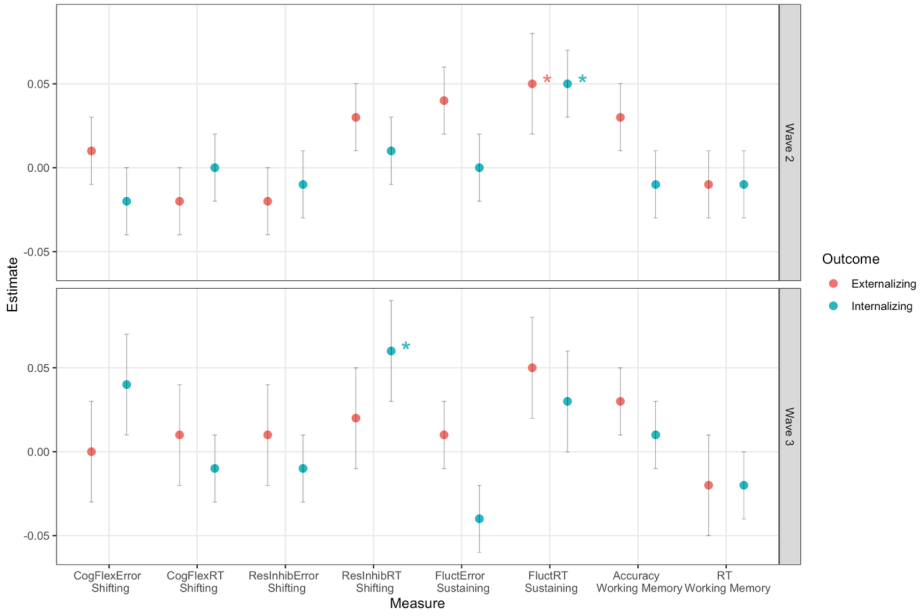
The models presented above included five different subscales of anxiety assessed through the RCADS measure. As shared method variance may be an issue that could explain the greater within-measure associations found, we conducted sensitivity analyses in which we replaced the five RCADS anxiety subscales with the single YSR anxiety subscales (see Supplement 2, available online). The results can be found in Figures S7-S9, available online. These models largely replicate some of the most important temporal associations (e.g., depressive symptoms predicting most other anxiety symptoms) found above.

Prospective Associations Between EF and Internalizing and Externalizing Symptoms

Hierarchical regression models were used to estimate associations between specific executive functions (see Figure S10, available online) assessed at age wave 1 (mean age 10.61) and the development of internalizing and externalizing symptoms at wave 2 (mean age 13.07)

and wave 3 (mean age 15.78). The EF measures were assessed only at wave 1, and thus they could not be integrated into the panel network approaches that require assessments at all respective waves. Overall, the regression models showed significant associations between behavioral measures of sustained attention (speed) at the first wave and future externalizing and internalizing symptoms (see Figure 4 for a visualization of regression coefficients). Higher fluctuations in tempo throughout the Sustained Attention Dots task at wave 1 (indicating worse sustained attention) were associated with higher scores of externalizing and internalizing symptoms at waves 2 and 3. Moreover, slower response inhibition/shifting was associated with internalizing symptoms at wave 3, but not at wave 2. All the effect sizes are small (all betas <.07) and the total variance explained in the second-step models that included these EF measures was not substantially larger than the first-step models that included only sex and mental health problems at the previous wave as predictors (see Tables S3-S10, available online).

Figure 4. Regression Estimates for Different Executive Functioning Measures



Note: The regression estimates refer to the regression models (in step 2) that include sex, and previous internalizing and externalizing symptoms as predictors. The standard errors for the regression estimates are shown in vertical bars. CogFlex = cognitive flexibility, ResInhib = response inhibition, Fluct = fluctuations in tempo (sustained attention), RT = reaction times. *p < .05.

No other task measure of attentional shifting (response inhibition accuracy, cognitive flexibility speed, cognitive flexibility accuracy) or working memory was significantly associated with prospective internalizing or externalizing symptoms. Tables S1-S8 (available online) provide an overview of all regression coefficients.

Discussion

The present study is the first to our knowledge to investigate the network dynamics of internalizing and externalizing symptoms in early adolescence at the within-person level. Mental health problems showed dynamic patterns of interactions throughout adolescence, in which depressive problems predicted various other internalizing symptoms in particular. Executive functions assessed in early adolescence were differentially associated with the development of internalizing and externalizing symptoms later in adolescence.

Our investigation revealed mostly stable cross-sectional symptom network constellations throughout different stages of adolescence (ages 11-16). Our bootstrapping analysis confirmed sufficient stability of these networks (see Figures S11-S13, available online). In line with previous network analysis studies (Blondino & Prom-Wormley, 2022) and the recent Hierarchical Taxonomy of Psychopathology (HiTOP) classification system (Conway et al., 2022), our results support the presence of internalizing and externalizing symptom clusters in which different symptoms of the same domain are strongly co-occurring. Across all three waves, we found robust cross-construct associations between ADHD symptoms and anxiety disorder (OCD and social phobia) symptoms (internalizing). This co-occurrence is in line with epidemiological evidence suggesting substantial rates of comorbidity between ADHD and OCD across the life span (Abramovitch et al., 2015). Moreover, we found a negative association between social phobia and conduct problems that is consistent with observations from clinical practice. Individuals with an overwhelming fear of social situations show low levels of aggressive behavior (DeWall et al., 2010) out of fear of negative judgments by others.

A crucial finding from our results concerns the role of depressive problems as a key bridge symptom (cross-sectional networks) and predictive marker (temporal networks). Depressive problems emerged as the most important bridge symptom, connecting various internalizing symptoms to other externalizing symptoms in the contemporaneous network. The importance of depressive problems as a bridge symptom slightly decreased over the course of adolescence. This finding aligns with reports showing that early-onset depression is associated with high rates of comorbidity with other mental disorders, including anxiety disorders in adolescents (Rohde et al., 1991).

The temporal networks emphasize the influential role of depressive problems in predicting a range of other internalizing symptoms in early adolescence. This is consistent with the high centrality observed in the contemporaneous network. During the change from age 10 to age 13 (wave 1-2) in particular, depressive problems predicted higher levels of social phobia, OCD, general anxiety, separation anxiety, panic symptoms, and somatic complaints. Our results are consistent with a recent cross-lagged panel analysis indicating depressed mood as an important influencer of other symptoms (Funkhouser et al., 2021). However, we replicate and extend this finding by separating within- and between-person effects that are conflated in CLPN models (Rhemtulla et al., 2022). Likely, different cognitive-behavioral processes prevalent in depression may explain this catalyst role of depressive symptoms on other symptom states. For instance, rumination as a core symptom of depression has been shown to predict anxiety symptoms and characterize comorbid depression-anxiety

(Nolen-Hoeksema, 2000). Moreover, prior diagnosis of major depressive disorder and negative affect predicted the onset of panic attacks in high-school students (Hayward et al., 2000). Particularly in early adolescence, low energy, avoidance, and social isolation triggered by depression may lead to the emergence of various anxiety problems that persist throughout adolescence and adulthood. Thus, our findings suggest that targeting adolescent depressive symptoms in clinical practice may prove viable in preventing various other internalizing symptoms arising later in life. To the best of our knowledge, this is one of the first study showing this catalyst effect of depression on a range of outcomes at the within-person level in early and middle adolescence.

In addition to the prominent role of depressive problems, we identified important feedback loops (reciprocal associations) between key symptoms. For instance, panic symptoms and somatic problems predicted each other over time in both panel network models. The increased body vigilance common in panic attacks may exacerbate negative or threatening interpretations of physical sensations that manifest in somatic problems. This in turn may lead to increased anxiety sensitivity which constitutes a major risk factor for panic disorder (McNally, 2002). Interestingly, we also observed reciprocal associations (in both methods) between ADHD symptoms and oppositional defiant problems that are consistent with bidirectional associations reported in prior literature (Burns & Walsh, 2002). These findings further complement discussions regarding the developmental precursor model of ADHD symptoms (Harvey et al., 2016), according to which ADHD symptoms predict argumentative/defiant symptoms.

We complemented our analysis of symptom network dynamics during adolescence by examining how EF assessed at age 10-11 plays into the development of broadband transdiagnostic dimensions of internalizing and externalizing symptoms. Our results suggest that primarily sustained attention, and no other executive functions such as working memory or attentional shifting, might represent a transdiagnostic risk factor for the development of internalizing and externalizing mental health problems. Our findings extend prior studies that showed cross-sectional or domain-specific (i.e., affective problems) associations with sustained attention (Bastiaansen et al., 2015; van Deurzen et al., 2012). The ability to sustain attention to relevant stimuli or information despite distractions constitutes a core executive function that plays a major role in daily functioning of individuals. Impairments in sustained attention have been associated with a range of mental disorders, including internalizing conditions, such as depression (Keller et al., 2019), and externalizing conditions, such as ADHD (Avisar & Shalev, 2011). Our results suggest that sustained attention is an important executive function in early adolescence with negative clinical transdiagnostic repercussions. Shifting attention was associated with more internalizing symptoms only at wave 3. This is consistent with previous links between difficulties shifting attention, response inhibition, and psychopathological processes, such as rumination (Chuen Yee Lo et al., 2012). Importantly, we found that the executive functions, including sustained attention, explained no additional variance over and beyond internalizing and externalizing mental health problems at the previous wave. Likely, associations between sustained attention and internalizing/externalizing mental health problems at the previous waves already accounted for these

effects. Other executive functions, such as attentional inhibition and working memory, showed no significant associations with the symptom measures. It is possible that these executive functions are associated with mental health problems at other time lags (i.e., later adolescence) or show solely disorder-specific associations. Future studies should also consider integrating a common EF factor at each time point into symptom panel networks - a crucial step towards understanding the dynamic interplay that was not feasible in our study considering that EF was assessed only at the first assessment wave. For instance, integrating cognitive measures of sustained attention in the dynamic network models may bridge our understanding of transdiagnostic risk factors as well as transdiagnostic network dynamics.

Our findings should be interpreted in light of several limitations. First, our analyses relied on self-report of adolescents, which may naturally be biased. Moreover, to foster model estimation and identification, we used scale scores that do not account for measurement error. Future studies should include both parent- and teacher-reports to validate these findings, and model all multiple-indicator constructs as latent variables. Second, the two methodological panel network approaches are limited by several methodological constraints. A common concern in all network modeling approaches is the assumption of multivariate normality. Restricted variance in some nodes (e.g., OCD at wave 3) may affect the estimation of temporal effects (Epskamp et al., 2018). The CLPN model cannot separate within- and between-person effects, which limits the interpretation of directed temporal effects as mechanistic pathways. The panel GVAR approach overcomes this limitation because it separates within- and between-person effects in the network structure. The resulting temporal associations are Granger-causal and fulfill the criteria of temporal precedence, however, these associations might not necessarily indicate causal effects (Borsboom et al., 2021). Moreover, the panel GVAR modeling approach assumes lag-1 linear dynamics between all variables and an approximately stationary time series, and thus it cannot capture (non-linear) processes that operate on shorter or broader time scales. Further methodological developments, including methods for formal model comparisons are needed to better understand the differences in results (e.g., the role of oppositional problems) obtained from CLPN and panel GVAR models which likely concern the separation of within- and between-person effect. Lastly, future studies should adopt confirmatory network modeling strategies to directly replicate and test the predictions emerging from developmental cascade models. We have used FIML estimation to account for missingness in the GVAR and regression models. Although FIML is widely considered an appropriate tool that can produce unbiased parameter estimates (Enders & Bandalos, 2001), it cannot overcome the assumption of Missing Completely At Random (MCAR), and some evidence suggests that attrition in the TRAILS sample is associated with baseline levels of psychopathology (de Winter et al., 2005; Huisman et al., 2008).

Our study is the first to our knowledge to investigate the within-person network dynamics between a range of internalizing and externalizing symptoms throughout early adolescence. We showcase the use of two novel methodological panel network approaches in parallel, namely the CLPN and panel GVAR models, that are uniquely equipped to explore dynamic patterns of interaction in longitudinal datasets while controlling for many variables.

Adolescence remains a consequential period of sensitivity in which different internalizing and externalizing problems emerge, influence each other, and contribute to symptom persistence in adulthood. Our findings identified sustained attention as a transdiagnostic risk factor, and we pinpointed key catalyst symptoms (e.g., depressive problems) in early adolescence. Future empirical investigations of these target points in intervention studies may potentially lead to effective intervention efforts that may prevent a deleterious cycle of symptom enhancement from arising.

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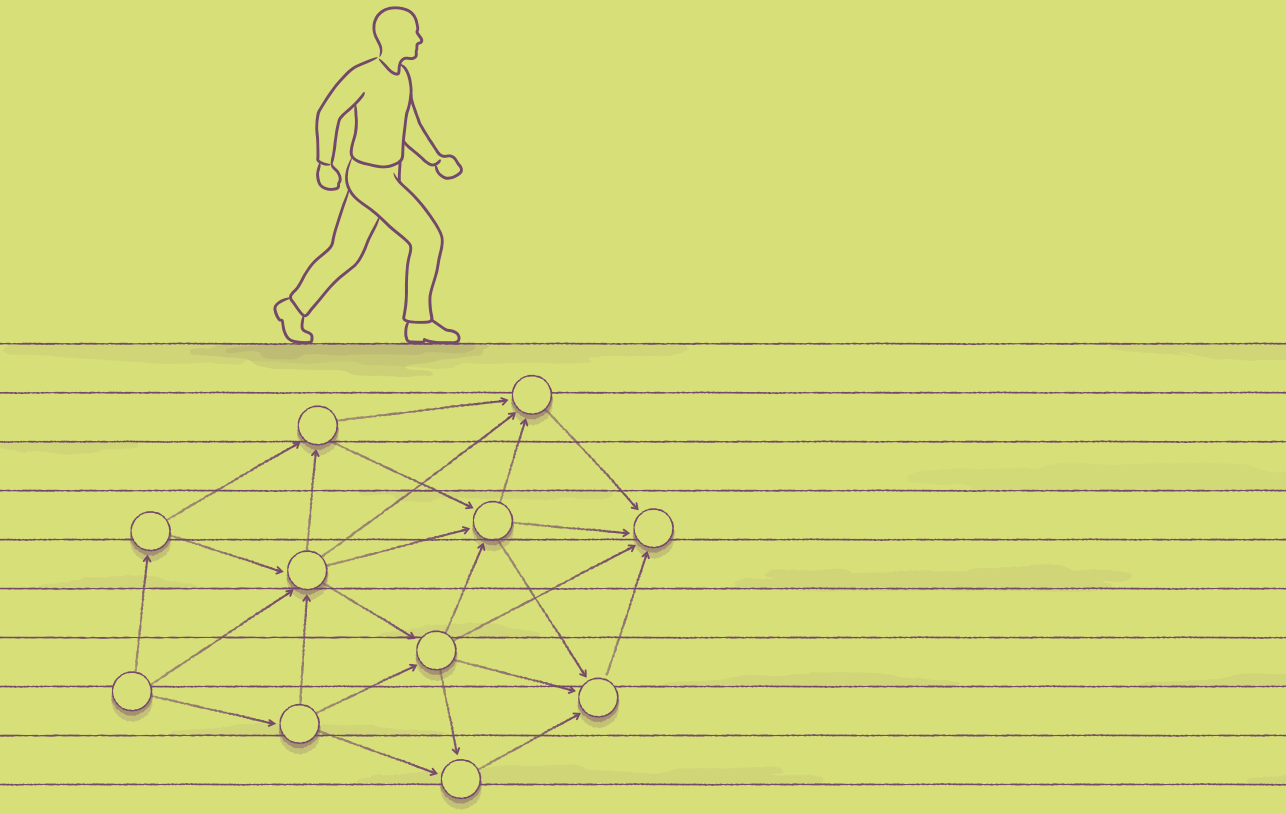


CHAPTER 3

Drinking motives, personality traits and life stressors—identifying pathways to harmful alcohol use in adolescence using a panel network approach

This chapter is adapted from:
Freichel, R., Pfirrmann, J., Cousijn, J., de Jong, P., Franken, I., Banaschewski, T., Bokde, A. L. W., Desrivières, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Martinot, J. L., Martinot, M. P., Artiges, E., Nees, F., Orfanos, D. P., Poustka, L., Hohmann, S., Fröhner, J. H., … IMAGEN Consortium (2023). Drinking motives, personality traits and life stressors-identifying pathways to harmful alcohol use in adolescence using a panel network approach. Addiction (Abingdon, England), 118(10), 1908–1919. https://doi.org/10.1111/add.16231

Abstract

Background and aims: Models of alcohol use risk suggest that drinking motives represent the most proximal risk factors on which more distal factors converge. However, little is known about how distinct risk factors influence each other and alcohol use on different temporal scales (within a given moment versus over time). We aimed to estimate the dynamic associations of distal (personality and life stressors) and proximal (drinking motives) risk factors, and their relationship to alcohol use in adolescence and early adulthood using a novel graphical vector autoregressive (GVAR) panel network approach.

Design, setting, and cases: We estimated panel networks on data from the IMAGEN study, a longitudinal European cohort study following adolescents across three waves (aged 16, 19, 22 years). Our sample consisted of 1829 adolescents (51% females) who reported alcohol use on at least one assessment wave.

Measurements: Risk factors included personality traits (NEO-FFI: neuroticism, extraversion, openness, agreeableness, and conscientiousness; SURPS: impulsivity and sensation seeking), stressful life events (LEQ: sum scores of stressful life events), and drinking motives [drinking motives questionnaire (DMQ): social, enhancement, conformity, coping anxiety and coping depression]. We assessed alcohol use [alcohol use disorders identification test (AUDIT): quantity and frequency] and alcohol-related problems (AUDIT: related problems).

Findings: Within a given moment, social [partial correlation (pcor) = 0.17] and enhancement motives (pcor = 0.15) co-occurred most strongly with drinking quantity and frequency, while coping depression motives (pcor = 0.13), openness (pcor = 0.05), and impulsivity (pcor = 0.09) were related to alcohol-related problems. The temporal network showed no predictive associations between distal risk factors and drinking motives. Social motives (beta=0.21), previous alcohol use (beta = 0.11), and openness (beta = 0.10) predicted alcohol-related problems over time (all $P < 0.01$).

Conclusions: Heavy and frequent alcohol use, along with social drinking motives, appear to be key targets for preventing the development of alcohol-related problems throughout late adolescence. We found no evidence for personality traits and life stressors predisposing towards distinct drinking motives over time.

Keywords: Adolescence, alcohol use, alcohol-related problems, panel network, risk factors, drinking motives

Introduction

Substance use disorders, including alcohol use disorders, present severe psychiatric conditions that have been linked to all-cause mortality and cardiovascular disease (Wood et al., 2018), thereby causing a substantial health and economic burden (Effertz & Mann, 2013). The transition from adolescence to emerging adulthood is characterized by rapidly increasing rates of alcohol use, as well as significant biological, cognitive, and social changes (Brown et al., 2008; Squeglia & Gray, 2016). Harmful alcohol use during this important developmental period may interfere with the normative course of development, and consequently, increase the risk of future alcohol-related problems and dependence (de Goede et al., 2021; Grant et al., 2006; McCambridge et al., 2011). Identifying pathways towards harmful alcohol use in late adolescence could therefore help to develop more effective prevention and early intervention strategies.

Several risk factor domains for the initial onset and maintenance of harmful alcohol use during adolescence and early adulthood have been identified. Among those, early onset of drinking, personality traits, environmental life stressors, and drinking motives received particular empirical support (DeWit et al., 2000). There is consistent evidence linking personality traits, such as impulsivity and sensation seeking, to adolescent binge drinking (i.e., consumption of high quantities of alcohol in short time periods) (Adan et al., 2017; Mackinnon et al., 2014; Spear, 2018). With respect to the 'big five' classification of personality traits, a recent meta-analysis (Lui et al., 2022) showed that higher levels of extraversion and lower levels of conscientiousness were most consistently associated with binge drinking among a predominantly young adult sample. Both longitudinal and cross-sectional research has implicated stressful life events as a major risk factor for the onset and degree of alcohol use throughout adolescence and early adulthood (Fenton et al., 2013; Kirsch & Lippard, 2022; Peltier et al., 2019; Shin et al., 2018; Thompson et al., 2020; Tschorn et al., 2021). A recent study of a community sample of adolescents demonstrated that high or repeated exposure to early life stressors (before the age of 17 years) was associated with an increased risk for alcohol-related problems in late adolescence and early adulthood (Shin et al., 2018).

In addition to personality and life stressors, a growing body of evidence highlights the role of drinking motives in adolescent alcohol consumption. According to Cooper's four-factor model (1994), four distinct motivations to drink emerge from the valence (i.e., to reduce negative affect or increase positive affect), as well as the source (i.e., internal or external) of the expected reinforcement of alcohol consumption. The four resulting drinking motives are social (positive, external) motives, enhancement (positive, internal) motives, conformity (negative, external) motives, and coping (negative, internal) motives. Grant and colleagues (2007) extended the four-factor model and further distinguished between motives of coping with anxiety and with depression. It has been suggested that drinking motives constitute the most proximal predictors of alcohol consumption on which more distal factors converge (Kuntsche et al., 2005). That is, distal risk factors (e.g., personality traits, life stressors) may give rise to distinct drinking motives, which in turn influence alcohol use behavior as proximal risk factors. Indeed, ample research has supported drinking motives to be a mediator in the

relationship between personality traits and alcohol consumption (Chinneck et al., 2018; Curcio & George, 2011; Kuntsche et al., 2010; Littlefield et al., 2010; Loose et al., 2018; Poelen et al., 2022). Although research examining the relationship between life stress, drinking motives, and alcohol use is largely restricted to adulthood, some studies have also provided support for the mediator role of drinking motives in adolescents and young adults (Rice & Van Arsdale, 2010; Shin et al., 2020; Temmen & Crockett, 2020).

Despite a substantial body of literature highlighting the role of personality traits, life stressors, and drinking motives for adolescent alcohol consumption, research has primarily examined specific risk factor domains (e.g., personality traits) in isolation (Chinneck et al., 2018; Loose et al., 2018; Temmen & Crockett, 2020). As a consequence, potentially complex associations between different personality traits, life stressors, and drinking motives remain poorly understood, both with respect to their co-occurrence and potential temporal dynamics. Moreover, existing studies that focused upon the interplay of distal and proximal risk factors of alcohol use are primarily of cross-sectional nature and thus, cannot discern within- and between-person effects. However, understanding such within-person (change within individuals) and between-person (individual differences) effects is crucial (Curran & Bauer, 2011), given that interventions targeting specific risk factors will lead to within-person change.

In the current study, we therefore applied a novel methodological approach, a panel graphical multilevel network model (Epskamp, 2020), to longitudinal data from the IMAGEN cohort, a large-scale (n > 1800) study assessing alcohol use and associated risk factors (personality, life events, and drinking motives) throughout adolescence and early adulthood (16-22 years). A longitudinal network approach allowed us to a) investigate complex (inter-) relations among alcohol risk factor domains, b) discern undirected contemporaneous from directed temporal effects, and c) separate within- and between-person effects. (Deserno et al., 2021; Höltge et al., 2022).

The current study aimed to identify normative developmental pathways to harmful alcohol use in late adolescence and early adulthood using a novel panel data network approach. Our approach was guided by two main research questions: 1) How are multiple personality traits and life stressors related to each other, and different drinking motives? and 2) How are these relations linked to late adolescent alcohol use and related problems (over time)? Drawing upon previous literature (Chinneck et al., 2018; Cooper et al., 2016; Kuntsche et al., 2005, 2010; Loose et al., 2018), we predicted that different patterns of personality traits and life stressors would give rise to distinct drinking motives over time, and that drinking motives would present the most proximal predictors of alcohol use in adolescence and early adulthood. We also hypothesized that positive drinking motives (social, enhancement) would predict alcohol use, while negative coping motives would be predictive of alcohol-related problems.

Method

Data source

We acquired data from the IMAGEN project, a large-scale, longitudinal, multicenter cohort study of adolescents (Schumann et al., 2010). The IMAGEN cohort included a large group of adolescents who were recruited across eight European research centers, including sites in Germany (Berlin, Dresden, Hamburg, and Mannheim), the United Kingdom (London and Nottingham), Ireland (Dublin), and France (Paris). Personality, stressful life events, drinking motives, and alcohol consumption were assessed at the ages 16 (wave 2), 19 (wave 3), and 22 (wave 4) years. The study was approved by all local ethics committees in accordance with the declaration of Helsinki. Written informed consent was obtained by the legal guardian of the adolescent participant prior to the age of 18, and by the participant thereafter. A more detailed description of the sample composition and study design is provided elsewhere (Schumann et al., 2010). All network analyses were based on data acquired at waves, 2, 3, and 4, and restricted to adolescents who reported consuming alcohol on at least one of the three assessment waves (n = 1829).

Measures

Alcohol use and related problems

Adolescent alcohol use and related problems were assessed using the Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993). The AUDIT is a self-report based 10-item screening instrument for hazardous and harmful alcohol consumption. We used sum scores of the two AUDIT subscales (Verhoog et al., 2020) in our network analysis: quantity and frequency of alcohol use (item 1-3; possible subscale scores: 0 to 12), and alcohol-related problems (item 4-10; possible subscale scores: 0 to 28). Both AUDIT subscales were simultaneously included in the model. An overview of all Cronbach's alpha estimates can be found in the Supporting information (see Supporting Information, Table S3).

Drinking motives

A modified version of the Drinking Motives Questionnaire-Revised (DMQ-R, Cooper, 1994) was used to assess motives for alcohol use. The questionnaire comprises 28 items (see Supporting information, Table S1 and S2) that measure five distinct drinking motives (Grant et al., 2007): enhancement (5 items), social (5 items), conformity (5 items), coping anxiety (4 items), and coping depression (9 items). Each item on the DMQ-R questionnaire asks participants to rate on how many occasions a specific reason motivated them to use alcohol in the past 12 months on a 5-point Likert scale (1 = (almost) never, 2 = seldom, 3 = sometimes, 4 = often, 5 = always). We calculated subscale scores for each motive as the mean of relevant item scores.

Personality measures

Personality traits were assessed by means of two self-report questionnaires: the Neuroticism-Extraversion-Openness Five Factor Inventory (NEO-FFI; (Costa & McCrae, 1995; McCrae &

John, 1992) and the Substance Use Risk Profile Scale (SURPS) (Woicik et al., 2009). The NEO-FFI contains 60 items that measure the Five-Factor personality dimensions: neuroticism, extraversion, openness, agreeableness, and conscientiousness. Each item on the NEO-FFI presents a self-descriptive statement to which participants must indicate their agreement on a 5-point Likert scale (1 = strongly disagree, 2 = disagree, 3 = neutral, 4 = agree, 5 = strongly agree). We computed total scores for each personality dimension as the sum of 12 item scores in accordance with the inventory's five-factor structure (score range: 12-60). The SURPS is a brief, 23-item self-report scale that assesses four personality risk dimensions for specific patterns of substance use: hopelessness, anxiety sensitivity, impulsivity, and sensation seeking. Participants must rate their agreement with each of the 23 items on a 4-point Likert scale from 1 (= strongly disagree) to 4 (= strongly agree). We included sum scores of the SURPS subscales impulsivity (5 items, score range: 5-20) and sensation seeking (6 items, score range: 6-24) in our network analysis, as those have been most consistently related to adolescent binge drinking (Adan et al., 2017; Spear, 2018).

Stressful life events

The Life Events Questionnaire (LEQ) (Newcomb et al., 1981) is a 39-item scale that assesses the perceived desirability and lifetime occurrence of stressful life events across seven life domains: parents/family, accident/illness, sexuality, autonomy, deviance, relocation, and distress. Perceived desirability is assessed by asking participants how happy or unhappy each item would make them feel on a 5-point Likert scale (-2 = very unhappy, -1 = unhappy, 0 = neutral, 1 = happy, 2 = very happy). To ensure that the experience of life stressors was perceived as negative, we first categorized each item based on its rated desirability as negative (desirability < 0), neutral (desirability = 0), or positive (desirability > 0) (Newcomb et al., 1981). We then selected all negative valence items (desirability < 0) for each participant separately and computed the sum score of their life-time occurrence (0 = no, 1 = yes; score range: 0-39) at each wave.

Statistical analysis and modeling

We used a panel graphical vector autoregression (GVAR) model (Epskamp, 2020) for network estimation. The panel GVAR is a multi-level lag-1 GVAR model (Epskamp et al., 2018) that is structurally similar to a random intercept cross-lagged panel data model to fit data from independent subjects assessed on a few measurement occasions. The VAR part of the model predicts each variable as a combined function of the variable's own, and all other variables' cross-lagged values (lag-1), thereby accounting for the temporal dependencies of repeated intra-individual assessments. The graphical part subsequently estimates a Gaussian Graphical Model (GGM) on the residual (co)variances of the VAR to uncover the relation between variables within a specific measurement occasion (Epskamp et al., 2018). As such, the panel GVAR allows for the estimation of temporal effects (i.e., directed partial correlations derived from standardized regression coefficients), contemporaneous effects (i.e., partial contemporaneous correlations) and between-subjects effects (i.e., partial between-subjects correlations). The directed temporal network describes how variables

predict each other across waves while the undirected contemporaneous network describes symmetric bidirectional associations within the same measurement period. Importantly, the estimated temporal and contemporaneous parameters in the panel GVAR encode fixed effects—that is, within-person effects of an average person in the population (Epskamp, 2020). Before estimating the panel networks, we detrended the data for possible linear and non-linear effects of time and standardized assessment scores across waves. This approach is considered appropriate in panel network analytical approaches, in which the focus of interest is on the correlational, and not the mean structure (Speyer et al., 2021). We first estimated a saturated model structure (i.e., all edges included) and used a full information maximum likelihood (FIML) estimator to account for missing data. Following initial model estimation, we applied standard pruning procedures to remove non-significant edges and performed a step-up model-search along modification indices that is common practice in the network analytical literature (Blanken et al., 2022). The pruning process removes all non-significant edges (using alpha = 0.05) and then re-estimates the model with all non-significant edges fixed to zero. This ensures that all estimates in the final model are based on a pruned model that excludes non-significant edges.

Model fit was evaluated based on the root mean squared error (RMSEA), comparative fit index (CFI), and the Tucker-Lewis index (TLI) according to standard criteria (RMSEA < 0.05, CFI > 0.95, TLI > 0.95 (Kline, 2015; Sivo et al., 2006). We used the psychonetrics package (Epskamp, 2021) for modeling and the qgraph package (Epskamp et al., 2012) for network visualization. To assess the stability of the final network, we employed a bootstrapping procedure (N = 1000). Strength centrality measures were computed to quantify the relative node importance in the network. For the temporal network, we calculated each node's in-strength (i.e., sum of all ingoing absolute edge weights) and out-strength (i.e., sum of all outgoing absolute edge weights). For the contemporaneous networks, we estimated the node strength, which is defined as the sum of all absolute edge weights that are connected to a given node (McNally, 2016). All analyses were carried out using the software R version 4.1.2 (R Core Team, 2024). This study was not pre-registered, and our results should be considered exploratory.

Results

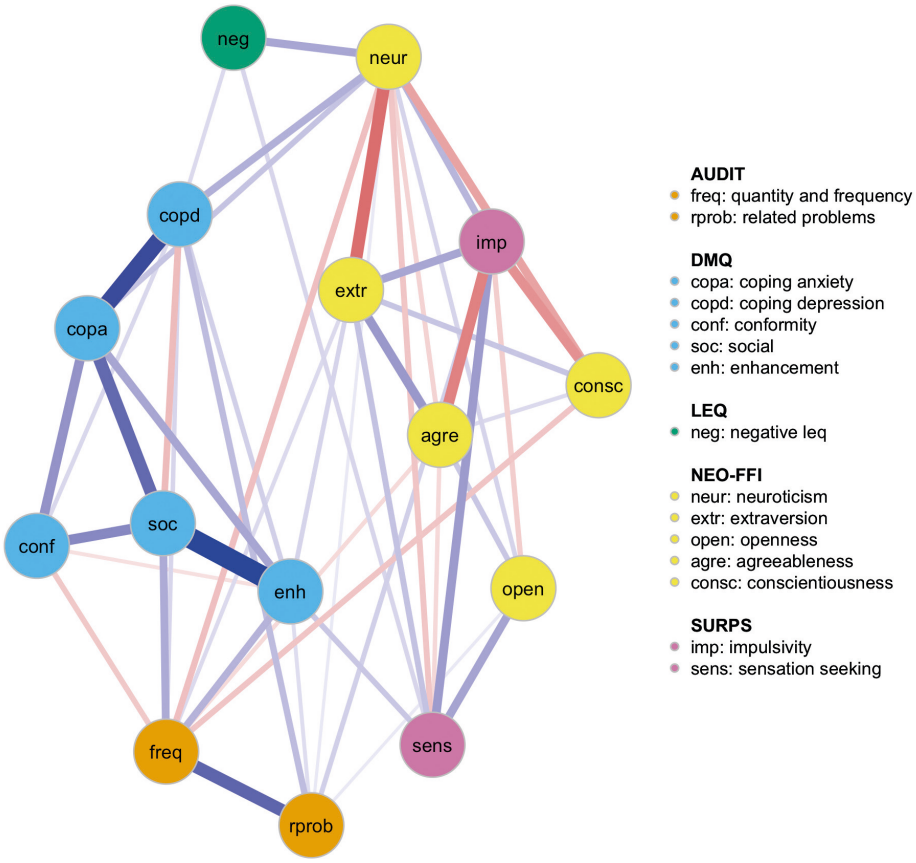
The sample included 1829 participants that were recruited among eight European research sites: Berlin (n = 206), Dresden (n = 234), Hamburg (n = 231), Mannheim (n = 218), London (n = 234), Nottingham (n = 299), Dublin (n = 187), and Paris (n = 220). Our sample consisted of 51% (n = 929) female, 46% (n = 850) male, and 3% (n = 50) without available or consistent data on sex. Among the 1829 eligible participants (i.e., alcohol use on at least one of the three assessment waves), 1630 (89.12%) provided data at wave 2, 1471 (80.43%) at wave 3, and 1333 (72.89%) at wave 4. Participants showed an average increase in alcohol use and related problems throughout the assessment period, with moderate levels of drinking (AUDIT quantity and frequency: mean = 4.25, standard deviation (SD) = 2.19; AUDIT-related problems: mean = 2.00, SD = 3.01) at the last wave. A detailed description of sample characteristics and

missing values for each measure is provided in the Supporting information (see Supporting information, Tables S4 and S5).

The saturated panel network model provided an excellent fit to the data (BIC = 151280.89, RMSEA = 0.03, CFI = 0.97, TLI = 0.95). We applied standard pruning procedures (alpha = 0.05) to make the networks robust against false positive findings and facilitate interpretation. The pruned model showed a similarly good fit (BIC = 149843.30, RMSEA = 0.03, CFI = 0.95, TLI = 0.95).

The contemporaneous network shown in Figure 1 depicts undirected partial correlations between variables within a given moment, after accounting for their temporal dependencies. Overall, the network revealed associations between all five drinking motives, personality traits, and different facets of alcohol use. There was a strong association between alcohol use quantity and frequency and alcohol-related problems. Alcohol use quantity and frequency further showed positive associations with the social, enhancement, and, to a lesser extent, coping depression motives, as well as negative associations with conformity, conscientiousness, agreeableness, and neuroticism. The enhancement motive was additionally associated with the social drinking motive, extraversion, sensation-seeking, and the two coping motives (anxiety and depression). Alcohol-related problems were associated with the coping depression motive and impulsivity, and, to a lesser extent, with neuroticism and openness. Stressful life events showed positive associations with neuroticism, the coping depression motive, and sensation seeking. Node strength centrality analysis (see supporting information, Figure S1) revealed that among all personality traits included, neuroticism showed the highest relative importance in the network. Among the five drinking motives, the social motive was identified as the most central, although closely followed by enhancement and coping (anxiety and depression) motives.

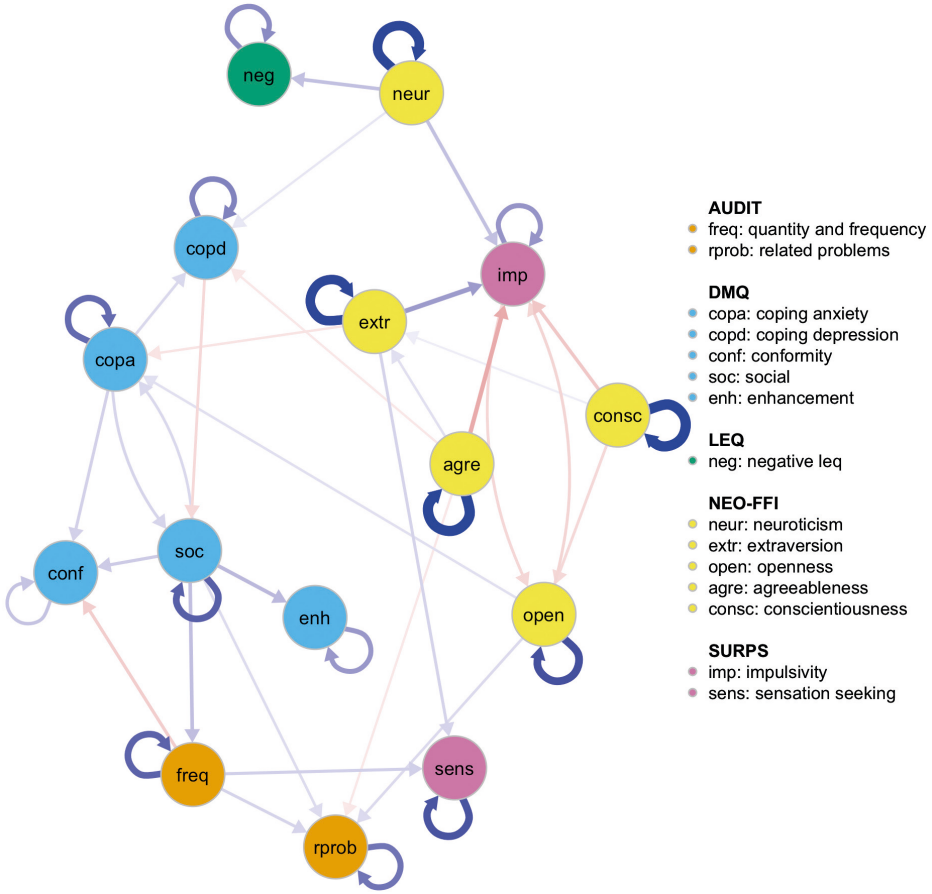
Figure 1. Fixed-effect contemporaneous associations within the same time window.



Note. The thickness and color (blue = positive, red = negative) of the edges represent the strength and direction of the associations, respectively.

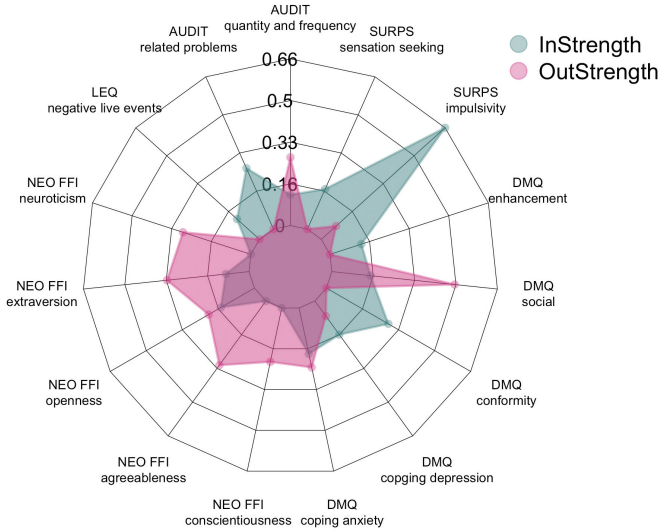
The temporal network depicts directed predictive relationships between drinking motives, personality domains, negative life events, and alcohol use (see Figure 2). Overall, directed temporal associations revealed a complex pattern of unidirectional, bi-directional (i.e., feedback loops), and autoregressive effects, in which four pathways toward alcohol use and related problems emerged. First, previous alcohol use and related problems predicted future drinking and related problems respectively (autocorrelations). Secondly, alcohol use quantity and frequency predicted alcohol-related problems at the next time point. Thirdly, the social drinking motive directly predicted alcohol use quantity and frequency, as well as alcohol-related problems over time. Fourthly, higher levels of openness predicted more alcohol-related problems.

Figure 2. Fixed-effect directed temporal associations.



Our node centrality analysis (see Figure 3) revealed that impulsivity had the highest in-strength, while social drinking motives showed the highest out-strength. In other words, impulsivity was strongly predicted by most NEO-FFI factors, namely higher levels of extraversion and neuroticism, as well as lower levels of conscientiousness, agreeableness, and openness. Conversely, social drinking motives positively predicted alcohol use, alcohol-related problems, and a range of other drinking motives (enhancement, conformity, and coping anxiety) at the next measurement occasion.

Figure 3. Outgoing and incoming strength of all nodes.



Note. The radar chart visualizes the degree (y-axis) to which variables in the temporal network influence other variables (out-strength) and are being influenced by other variables (in-strength) over time.

All associations central to the interpretation of the networks are sufficiently stable as indicated by our bootstrapping analysis (Supporting information, Figures S3 and S4). Contemporaneous and temporal edge weights are provided in the Supporting information (see Tables S6 and S7).

Discussion

The aim of our study was to explore the complex interrelationships between distal (personality risk profiles, stressful life events) and proximal (drinking motives) risk factors of late adolescent alcohol use and problems using a novel panel network methodology. Applying panel GVAR models to data of a large-scale cohort study, we disentangled within- from between-person relations, and modeled the contemporaneous and temporal interrelations between distinct risk factor domains and adolescent alcohol use. Our findings describe normative developmental patterns in the general population. Overall, the panel GVAR model suggested that the various domains of risk factors were dynamically related and associated with alcohol use and related problems throughout adolescence and early adulthood. The resulting contemporaneous and temporal networks revealed both overlapping and distinct structures, thus highlighting the importance of understanding risk factors for alcohol use in the context of different temporal scales.

At the contemporaneous level, we identified two main patterns of associations that evolved around the expected valence of drinking (i.e., to increase positive affect versus to decrease negative affect) (Cooper, 1994). The first pattern involved a strong relation between the two positive valence motives: drinking for social reasons and drinking to enhance positive mood or well-being. Importantly, the two positive valence motives (i.e., social and

enhancement) showed the strongest associations with drinking frequency but were unrelated to alcohol use related problems. These findings are well aligned with existing literature in which social and enhancement motives have been most consistently related to frequent and heavy alcohol use (Kuntsche et al., 2005; Loose et al., 2018; Sjödin et al., 2021). Within the positive reinforcement pattern of associations, we also observed positive relations between extraversion, sensation seeking, and the enhancement motive. Supporting evidence for these associations comes from previous studies reporting that more extraverted and sensation-seeking adolescents are more likely to drink for enhancement motives (Adams et al., 2012; Comeau et al., 2001; Curcio & George, 2011; Loose et al., 2018; Woicik et al., 2009).

Within the second pattern of associations, the negative valence pattern, the role of neuroticism, coping depression, stressful life events, and alcohol-related problems warrants a more detailed inspection. Neuroticism was positively associated with stressful life events, the two coping motives, impulsivity, and alcohol-related problems, but negatively with alcohol use frequency. These findings are consistent with research on this topic suggesting that more neurotic adolescents and young adults tend to show a higher reactivity to stressful situations (Wrzus et al., 2021; Xin et al., 2017), more impulsive behavior (Fetterman et al., 2010), and higher tendencies to use drinking as a coping mechanism for anxiety or depression (Chinneck et al., 2018). Importantly, among the neuroticism-centered associations, only the coping depression motive also covaried with stressful life events and alcohol-related problems, thereby further supporting the importance of contextual factors and potentially separate motivational processes (coping anxiety versus coping depression) in neuroticism-associated drinking patterns (Woicik et al., 2009).

Among the various personality traits, impulsivity showed the strongest association with alcohol-related problems, which fits the general characterization of impulsivity as an inability to control behavior when facing immediate reinforcers (such as alcohol) (Woicik et al., 2009). Surprisingly, impulsivity did not co-occur with any of the five drinking motives. This finding diverges from previous work showing a non-specific pattern of associations between impulsivity and drinking motives (Adams et al., 2012; Poelen et al., 2022). One potential explanation for this inconsistency might arise from the application of different analysis methods across studies. That is, whereas studies reporting a relationship between impulsivity and drinking motives primarily relied upon zero-order correlations (Adams et al., 2012; Mackinnon et al., 2014; Poelen et al., 2022), the use of partial correlations has failed to reveal such associations (Woicik et al., 2009). In the current study we replicated that pattern, finding significant Pearson's correlations ($r = 0.16$ to 0.29, all $p < 0.05$) between mean scores of impulsivity and all five drinking motives (see Figure S2 in supplementary materials) on a cross-sectional level, but not in our contemporaneous network representing partial correlations after accounting for temporal dependencies. Although to a lesser extent than impulsivity, openness to experience covaried with alcohol-related problems, which is in contrast to previous research reporting no association between openness and alcohol-related problems and dependence (Kotov et al., 2010).

The temporal network revealed dynamic associations among personality traits, stressful life events, drinking motives, and alcohol use. Overall, associations were predominantly, but not exclusively, restricted within risk factor domains, which is in contrast to our hypothesis

that personality traits and stressful life events might predispose towards specific drinking motives over time (Kuntsche et al., 2005; Loose et al., 2018). Our findings highlight three key pathways to alcohol use and related problems throughout adolescence and early adulthood. First, social drinking motives emerged as the node with the highest out-strength centrality, predicting a) the quantity and frequency of alcohol use, b) alcohol-related problems (directly and indirectly through alcohol use frequency and quantity), and c) various other drinking motives. These findings indicate that the external social reinforcement effects of alcohol use might have more far-reaching implications than typically assumed (Grant et al., 2007). That is, higher levels of social motives for drinking may increase alcohol use and related problems (directly and indirectly), which, in turn, drives the development of alcohol dependence at a later stage. These findings are in line with a previous cross-lagged panel study in young adult men showing that social motives predicted heavy alcohol use and related consequences 15 months later (Labhart et al., 2017). Moreover, initial alcohol use for social motives may heighten the acceptability of drinking, thereby risking transcendence to other motives driving alcohol use and related problems on a contemporaneous level. Secondly, previous alcohol use quantity and frequency predicted future alcohol use quantity and frequency, as well as alcohol-related problems. In combination with the first pathway, these findings do not support the importance of a range of coping motives for the development of alcohol-related problems (cf. Cooper, 1994), but rather suggest that alcohol use, possibly harmful use (Kuntsche et al., 2013), during adolescence is the driving force in developing future alcohol-related problems (de Goede et al., 2021; Rehm et al., 2013). Third, higher levels of openness predicted more alcohol-related problems over time. This finding is somewhat surprising, given the mixed evidence from cross-sectional studies. That is, while most studies reported no relation between openness and alcohol use and related problems (Ernst-Linke et al., 2023; Lui et al., 2022), others suggested that openness may even attenuate the risk of heavy alcohol consumption (Luchetti et al., 2018), but also reduce the probability of abstinence (Hakulinen et al., 2015). Lastly, impulsivity emerged as the node with the highest in-strength centrality, indicating that impulsivity was the risk factor being most influenced by other factors in the network. While impulsivity was associated with alcohol-related problems at the contemporaneous level, it was not influenced by any of the alcohol use measures at the previous measurement, nor did it predict alcohol use quantity and frequency or related problems at the next time point. These findings are in contrast with prior research consistently reporting a bidirectional temporal relationship between impulsivity and the development of alcohol use disorders over time (Verdejo-García et al., 2008; White et al., 2011). Several factors may contribute to the observed discrepancy, including the use of a predominantly healthy sample recruited in non-clinical settings, as well as our ability to control for a range of other risk factors (e.g., the level of previous alcohol use) in the temporal network.

The current findings should be interpreted in light of several limitations. First, the personality trait impulsivity was assessed as a single construct on the SURPS questionnaire in the current study (Woicik et al., 2009). However, according to the UPPS-P model of impulsivity (Cyders et al., 2007; Whiteside & Lynam, 2001), impulsivity presents a multi-dimensional construct with different facets of impulsivity (i.e., negative urgency, positive urgency, lack of premeditation,

lack of perseverance, and sensation seeking) relating to different aspects of alcohol involvement (Coskunpinar et al., 2013; Tran et al., 2018). As the SURPS's impulsivity scale seems to relate most strongly to the positive and negative urgency facets (Blanchard et al., 2020), future studies might benefit from the inclusion of all impulsivity-related facets in the model. Secondly, we used an adapted version of the DMQ that included subtle changes to the original item wordings for the social, enhancement, and conformity subscales. Despite the high levels of internal consistencies found for all subscales, future studies should validate our findings using the original measure (Grant et al., 2007). Thirdly, the use of self-report measures for the assessment of stressful life events and alcohol use may be subject to biases common in retrospective recall. Fourthly, the current study did not account for potential sex, gender, or cultural (i.e., recruitment centers) differences in the contemporaneous and temporal panel networks. However, mounting evidence points to the existence of sex-specific risk profiles for adolescent alcohol use and alcohol-related problems (Dir et al., 2017; Peltier et al., 2019). Future studies might thus benefit from the estimation of separate sex- or gender-specific networks. Fifthly, it should be emphasized that while the within-person temporal associations found in the panel GVAR model describe temporally ordered relations between variables that fulfill the criteria of Granger causality (Granger, 1969), associations may not necessarily reflect causal effects (Borsboom et al., 2021). Moreover, existing panel GVAR panel models assume linear lag-1 relationships in an approximately stationary time series. With a linear lag of three years in the IMAGEN cohort, the estimated temporal network might not capture relations that operate on more granular or longer time scales. Future studies could thus benefit from the use of different time scales, especially in the context of trait-motive convergence. Lastly, our findings were based on a group of largely healthy adolescents who were first assessed at age 16 years, a time when the majority had already started using alcohol. It is possible that stronger temporal connections between coping motives and alcohol-related problems might emerge in subclinical samples.

To conclude, our resulting panel networks revealed a complex pattern of associations among different distal (personality traits, life stressors) and proximal (drinking motives) alcohol use risk factors throughout adolescence and early adulthood. The contemporaneous and temporal networks showed structural differences, highlighting the importance of examining the interplay of alcohol risk factor domains at different temporal scales. In the context of temporal predictions, the prior quantity and frequency of alcohol use, openness, and social motives emerged as the most important predictors of future alcohol use and alcohol-related problems. In contrast to our expectations, distal risk factors (personality traits and stressful life events) did not converge on different drinking motives over time. After controlling for temporal dependencies, drinking to increase positive affect (social and enhancement motives) uniquely covaried with drinking quantity and frequency, while drinking to cope with negative affect (coping depression motives) also co-occurred with alcohol use problems. In this context, impulsivity emerged as the distal factor that co-occurred most strongly with alcohol-related problems within a given moment. Our findings outline specific risk factor patterns that may offer ground for time-sensitive intervention and prevention efforts aimed at targeting harmful alcohol use and alcohol-related problems. In particular, interventions targeting heavy and frequent drinking, and social motives in late adolescence may prove to be effective in preventing a negative spiral of alcohol-related problems from arising in the future.

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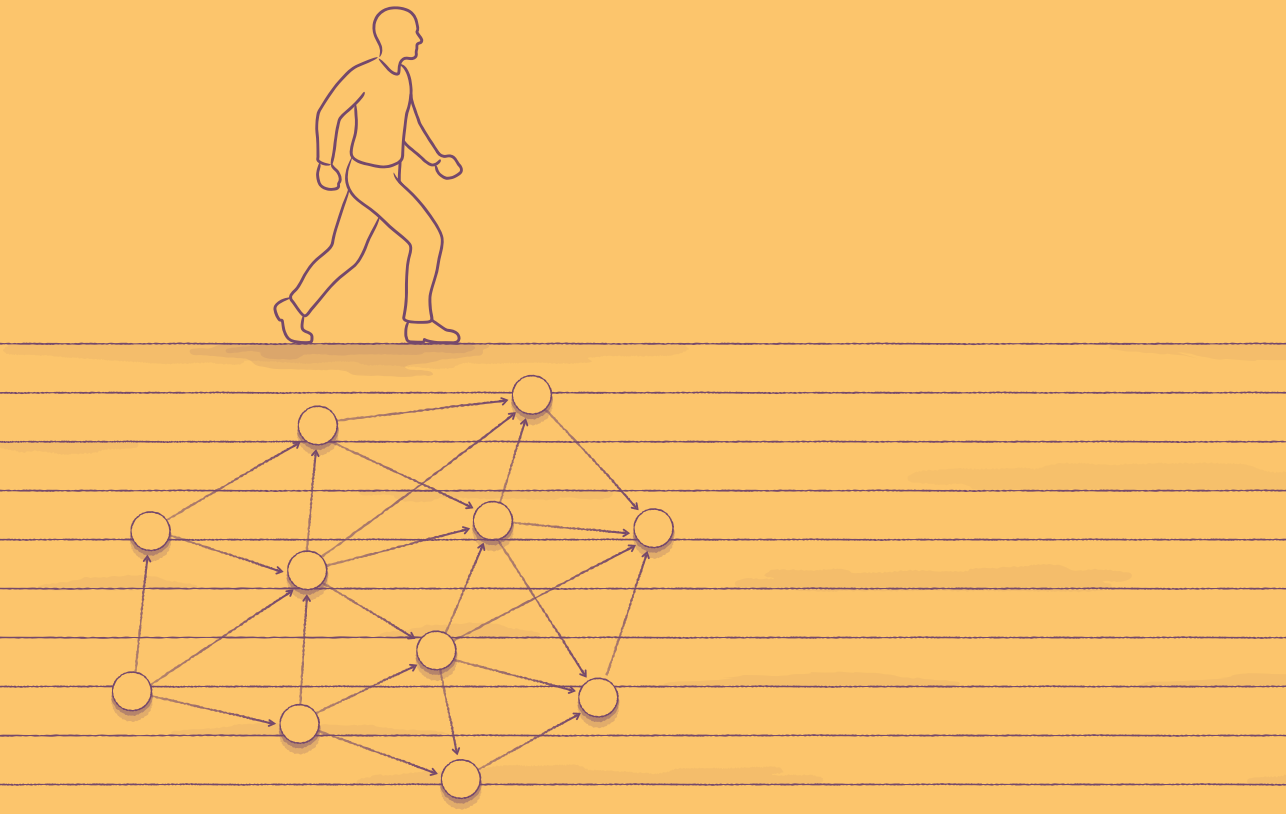

CHAPTER 4

Investigating risk factor and consequence accounts of executive functioning impairments in psychopathology: An 8-year study of at-risk individuals in Brazil

This chapter is adapted from:
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Abstract

Background. Executive functioning (EF) impairments are widely known to represent transdiagnostic risk factors of psychopathology. However, a recent alternative account has been proposed, according to which EF impairments emerge as consequences of psychopathology.

Methods. Using a longitudinal cross-lagged panel network analysis approach, we tested these competing theoretical accounts at different stages during adolescence. We used data from the Brazilian High Risk Cohort Study for the Development of Childhood Psychiatric Disorders, in which 61% of individuals at wave 1 were selected due to their high risk for psychopathology. Participants were assessed across three assessment waves during early (wave 1: n = 1,992, mean age = 10.20 years) and middle adolescence (wave 2: n =1,633, mean age = 13.48 years; wave 3: n = 1,439, mean age = 18.20 years). We examined associations between working memory, inhibitory control, and broad-band measures of psychopathology.

Results. During early adolescence, lower inhibitory control was a risk factor for externalizing problems that, in turn, predicted lower working memory capacity. During middle adolescence, bidirectional associations became more prominent: inhibitory control and working memory functioned as both risk factors and consequences. Externalizing problems both predicted and were predicted by poor inhibitory control. Internalizing and externalizing symptoms showed bidirectional associations over time. Externalizing problems predicted more internalizing symptoms, whereas internalizing symptoms predicted fewer externalizing problems during middle adolescence.

Conclusions. Our results corroborate dynamic theories that describe executive dysfunctions as precursors and consequences of psychopathology in middle adolescence.

Introduction

Adolescence represents a critical period marked by neurobehavioral changes, brain maturation, and cognitive-emotional development (Dahl et al., 2018). As widely noted, this period of sensitivity may set the stage for developing adolescent mental health problems that often continue into adulthood (Lee et al., 2014; Paus et al., 2008). Three-fourths of all lifetime cases of mental disorders have an onset before the age of 24 years (Kessler et al. 2005), hence, onset in childhood, adolescence, or emerging adulthood. Different cognitive functions, in particular inhibitory control and working memory, have been proposed to play dual roles as being both risk factors and consequences of psychopathology in adolescence and emerging adulthood (Goschke, 2014; Huang-Pollock et al., 2017; Liu & Pérez-Edgar, 2019; Verdejo-García et al., 2008; White et al., 2011). However, little is known about the specificity of these roles with respect to a) different stages during adolescence and b) different broad-band symptom domains of psychopathology (internalizing/externalizing). Understanding age-specific cognitive antecedents of developmental psychopathology is crucial for developing time-sensitive interventions and prevention efforts.

Executive functions are defined as a broad class of cognitive control components (Miyake et al., 2000) that include inhibitory control, shifting attention, and working memory. Different developmental profiles with a general improvement of these functions across adolescence and young adulthood have been described (e.g., Ferguson et al., 2021). Executive functions rapidly develop in mid-adolescence (ages 10-15) before stabilizing in early adulthood (Tervo-Clemmens et al., 2023). These developments are accompanied by neural changes in various brain regions, including the prefrontal cortex, as well as hormonal shifts for heightened social and affective processing (Crone & Dahl, 2012). Some evidence points to variations in trajectories of different executive functions (Steinberg et al., 2008) – with linear decreases in impulsivity from age 10 but a non-linear change in sensation-seeking (increasing until age 10 before stabilizing).

Cognitive processes and their development have been more broadly associated with mental health (see RDoC framework, Insel et al., 2010). Recent studies proposed that global deficits in EF increase the transdiagnostic vulnerability to psychopathology during adolescence for at-risk individuals, such as children who experienced neglect (Schäfer et al., 2023; Wade et al., 2020). Similarly, global executive functions prospectively predicted the general psychopathology factor ('p factor'; Caspi et al., 2014) during early (i.e., 10-14 years) adolescence (Martel et al., 2017; Romer & Pizzagalli, 2021). In addition to these associations with overall psychopathology, some evidence points to a potential specificity between different executive functions and broad problem domains of psychopathology (internalizing, externalizing). For instance, working memory constitutes one of the most central executive functions (Kane & Engle, 2002) and has been specifically associated with externalizing disorders in children (Huang-Pollock et al., 2017). Inhibitory control, described as the ability to control impulses or automatic behaviors, has been established as a core aspect of emotion regulation and was implicated in both externalizing (Berger & Buttelmann, 2022) and internalizing problems (Sætren et al., 2021) during childhood and adolescence.

The past two decades of research in developmental psychopathology have led to different theoretical approaches to integrate these findings. The risk factor theory proposes that EF impairments precede and increase individuals' vulnerability to developing social, emotional, and behavioral problems later in life. According to the model by Carver et al. (2017), low inhibitory control may lead to internalizing or externalizing problems depending on the level of incentive sensitivity (low: internalizing, high: externalizing). Conceptually related, Wiers and colleagues (2018) suggested that relatively weak executive functions constitute a general risk factor for psychopathology and that temperament determines the primary area of problems (internalizing or externalizing). In the field of addiction, there is strong evidence in favor of a pre-existing cognitive vulnerability, particularly with respect to impulsive behaviors (e.g., Verdejo-García et al., 2008; White et al., 2011), going back to the classical work of Gorenstein and Newman (1980). Another study on executive functioning deficits in daily life (Letkiewicz et al., 2014) found that poor EF prospectively predicted depressive symptoms, while the reverse pathway was not present.

A contrasting theory, namely the 'complication' (Maasalo et al., 2021), 'consequence', or 'scar' account, suggests that EF impairments may represent consequences of psychopathological processes present in internalizing and externalizing disorders. Thus, weak EF may also constitute a consequence of psychopathology. A recent study among 7-9-year-old children found evidence in favor of this consequence account for externalizing symptoms that constrained the development of inhibitory control (Maasalo et al., 2021). In addiction, there is also some evidence in favor of a consequence account, with early substance use negatively impacting the development of executive functions in adolescence, with the strongest evidence from animal studies (e.g., Spear, 2018), although the evidence is weaker in human development (e.g., see the systematic review by de Goede et al., 2021). At the level of broad-band symptom domains, more internalizing and externalizing symptoms among 13-14 year old adolescents predicted lower EF 3-4 years later on (Brieant et al., 2022). A recent study using the Adolescent Brain Cognitive Development Study showed that p-factor scores, which represent a general dimension of psychopathology across internalizing and externalizing symptoms, prospectively predicted change in EF, but the reverse direction was also present (Romer & Pizzagalli, 2021). This suggests that EF may serve a dual role, acting both as a risk factor for the development of psychopathology and as a consequence of it.

Nevertheless, the majority of studies in humans explicitly focused on the 'risk factor' account by including different executive functions as predictors and symptom measures as outcomes (Freichel, Pfirrmann, de Jong, et al., 2023). Thus, less is known about how various executive functions are longitudinally associated with internalizing and externalizing symptoms at different stages of development. Existing studies on the prospective associations between different EFs, on the one hand, and both internalizing and externalizing symptoms, on the other hand, show several characteristics that constrain the possibility to differentiate between the risk factor versus consequence account of EFs in psychopathology: (1) Most studies focused on late childhood and early adolescence, and little is known about associations during middle adolescence; (2) through the use of traditional statistical approaches, executive functions have been commonly treated as sole predictors; (3) studies spanning longer time periods

during adolescence typically examined average changes across time, and they thus failed to capture potential changes in the structure of associations ('covariances') at different change points during adolescence (Freichel, Pfirrmann, Cousijn, et al., 2023).

With data from the Brazilian High-Risk Cohort Study for Mental Conditions, a large longitudinal dataset of adolescents (Salum et al., 2015) assessed during three waves, the present study aimed to fill these critical caveats by addressing two key research questions: (1) Are particular domains of EF, specifically working memory and inhibitory control, risk factors for or consequences of both internalizing and externalizing symptoms? Based on prior studies (e.g., Yang et al., 2022), we predicted that inhibitory control would be a general risk factor for both. Moreover, we predicted that working memory would be a risk factor specific to externalizing symptoms. (2) Do these associations differ between different stages of adolescence? We investigated the associations between cognitive measures and internalizing/externalizing symptoms through the use of a novel methodological approach, namely cross-lagged network analysis (Wysocki et al., 2022). This exploratory, methodological approach allowed us to study the dynamic interplay (of risk factors and consequences) between specific waves without presupposing the role of EFs as either predictors or outcomes in symptom development.

Materials and Methods

Data source and procedure

We used data from the Brazilian High Risk Cohort Study for the Development of Childhood Psychiatric Disorders (BHRCS). This longitudinal panel study recruited a school-based community sample across two cities in Brazil (São Paulo, Porto Alegre). The recruitment targeted both high-risk children (i.e., based on extensive family history of mental disorders screening) and a randomly selected community sample. Our analyses included the entire sample, which combines both high-risk and randomly selected participants, to ensure sufficient variability in all symptom and cognitive measures. This approach was essential as network analyses require variability to accurately capture patterns of covariance between different levels of cognitive functioning and symptom development. Participants were invited to three study visits (wave 1: years 2010-2011, wave 2: years 2013-2014, wave 3: years 2017-2019) that included the administration of clinical interviews and neuropsychological assessments. The study design and sample selection are described in more detail elsewhere (Salum et al., 2015). The study was approved by the ethical committees of the universities at each site, and all parents of participants provided informed consent.

Measures

Psychopathology

CBCL. The Child Behavior Checklist (CBCL, Brazilian version) (Bordin et al., 1995) was used as a parent-report questionnaire to assess psychopathology symptoms. A parent or caregiver completed the checklist with 120 items that assessed emotional-behavioral problems

(Bordin et al., 2013), specifically the syndrome scales: anxious/depressed, withdrawn, somatic complaints, social problems, thought problems, attention problems, rule-breaking problems, aggressive behavior, and other problems. These dimensions can be grouped into two broad-band scales (Cohen et al., 1985): internalizing symptoms (withdrawal, somatic complaints, anxiety/depression) and externalizing symptoms (rule-breaking problems, aggressive behavior). The CBCL was shown to be a valid assessment tool across cultures (Ivanova et al., 2007).

ABCL. At the third measurement wave, participants aged 18 and above were administered the Adult Behavioral Checklist (ABCL, Achenbach & Rescorla, 2003). The ABCL is part of the Achenbach System of Empirically Based Assessment and consists of 118 items that are completed by a close informant (e.g., partner, parents, friends). Similar to the CBCL (for participants below 18 years of age), we derived two broad-band scales (internalizing: withdrawn, somatic complaints, anxious/depressed; externalizing: rule-breaking problems, aggressive behavior) as well as separate empirically derived scales.

Executive functions

Digit span task. A digit span task (forwards/backwards) from the Wechsler Intelligence Scale for Children (Wechsler, 2002) was used to assess short-term working memory capacity in the context of verbal information. Participants were instructed to listen to a sequence of numbers and repeat it either forward or backward. As the sequence got increasingly longer, the task became more difficult.

Corsi blocks. A corsi blocks (forwards/backwards) task (Vandierendonck et al., 2004) assessed the short-term working memory capacity of visual-spatial information. Participants were instructed to repeat a spatial sequence a researcher indicated by tapping up to nine identical blocks. We first standardized the backward digit span scores from both tasks (digit span and corsi block span) and then averaged these scores for every participant to create one aggregate measure of working memory. This was considered appropriate as both tasks' backward digit span scores correlated moderately ($r = 0.46$, $p < 0.01$). This process also increased the reliability of the measure and also allowed us to prevent issues of multicollinearity (i.e., two strongly interconnected nodes) that are common problems in network analysis (Borsboom et al., 2021). Higher averaged digit span scores indicate better working memory capacity.

Inhibitory control.

A Go/no-go task (Bitsakou et al., 2008) was used to assess inhibitory control. Participants were instructed to press buttons indicating the direction of arrows as soon and as accurately as possible. When a double-headed arrow appeared, participants were instructed to stop pressing the button (no-go). There were 75 go-trials and 25 no-go trials. All stimuli were displayed for 100ms, and the intertrial period was 1500ms. As relevant measures of inhibitory control, we included participants' average RT of correct Go trials and the number of commission errors. The use of these two distinct measures was considered appropriate as they capture different dimensions of inhibitory control and may account for a possible speed-

accuracy tradeoff. We used observed scores for the cognitive task measures, rather than latent variables, to avoid issues related to disattenuation that can arise when measurement error is removed from only some variables in the model. We removed outlier reaction times and accuracy scores with an absolute z-score equal to or larger than 4 (Brunnekreef et al., 2007). To facilitate interpretation, we inverted the two indices of inhibitory control (reaction times and error) such that higher scores indicate better inhibitory control (lower reaction times and higher accuracy).

Data preprocessing

For all analyses, we only included individuals with no missing data at the first wave for the CBCL measures and cognitive tasks (digit span, corsi-blocks, Go/NoGo task). In the Supplementary Table S1, we provide the proportion of missingness for all individual subscales/measures. The second analysis (cross-lagged network analysis from wave 2 to wave 3) included individuals with available CBCL (age below 18 years) and ABCL (age above 18 years) at the third wave. The distributions for all subscales of the available CBCL and ABCL measures appeared similar, and thus, we first standardized the CBCL/ABCL subscales separately before integrating them into the model.

Cross-lagged network analysis

To examine the temporal associations between cognitive markers and symptoms, we used cross-lagged network analysis (Wysocki et al., 2022). This approach, as used in extant literature (Freichel, Pfirrmann, de Jong, et al., 2023; Zainal & Newman, 2022b), implements a series of regularized regressions to estimate cross-lagged (different nodes predicting each other over time) and autoregressive (node predicting itself over time) effects at every change point. The Least Absolute Shrinkage and Selection Operator (LASSO) with 10-fold cross-validation (to find the optimal γ-value) was used to shrink weak estimates to zero. The cross-lagged estimates describe temporal associations between nodes while controlling for all other variables in the network. This cross-lagged network analysis method has two key benefits that are important in the present application: (1) The model identifies temporal associations without requiring an a-priori specification of predictors and outcomes that are based on theoretical assumptions about the nature of EF in symptom development; (2) By controlling for all other variables in the network, the model can inform conclusions about the relative importance of different measures, such as executive functions. Considering the variance in the age range of our sample (within every wave), we included age as a covariate in the model following the approach of Funkhouser et al., (2021) and Zainal and Newman (2022). This meant that age was an additional predictor (covariate); however, it was not predicted by any other variable in the model. Similarly, we conducted a sensitivity analysis including gender as a covariate. The models were estimated using the glmnet (Friedman et al., 2017) and visualized using the qgraph (Epskamp et al., 2012) R packages. We used non-parametric bootstrapping procedures (Epskamp et al., 2018) with 1,000 bootstraps to examine the accuracy of the estimated edge weights.

Results

Sample characteristics

Table 1 summarizes demographic and clinical characteristics at all three waves separately for the high-risk and randomly selected population samples. The high-risk group comprised 60.89% of the sample. Boys were slightly overrepresented overall, but there were no significant differences in gender distribution between samples. At the aggregate level, we found notable trends with increases in internalizing symptoms (in particular withdrawn-depressed problems) across waves, while externalizing symptoms peaked during the second wave. Participants in the high-risk sample were slightly older (<0.3 years). The high-risk group exhibited significantly higher levels of psychopathology across all CBCL subscales, with internalizing and externalizing symptoms showing consistent differences between the groups at all three waves.

Participants' cognitive performance also steadily improved throughout the three waves, with higher speed and accuracy in both the inhibition and working memory tasks during the later waves. The high-risk and randomly selected samples showed significant differences in inhibitory control, where the randomly selected group showed fewer errors at wave 2 and faster reaction-times at wave 3. Moreover, individuals in the high-risk group exhibited lower working memory capacity at wave 3.

There was substantial attrition throughout the three waves, with 35.14% of individuals from the first wave dropping out during the study. A supplemental analysis (see Supplementary Table S2) revealed that age, gender, externalizing symptoms, and cognitive performance at wave 1 were not associated with dropout. However, higher internalizing symptoms at wave 1 predicted a lower probability of dropout.

Associations During Early Adolescence

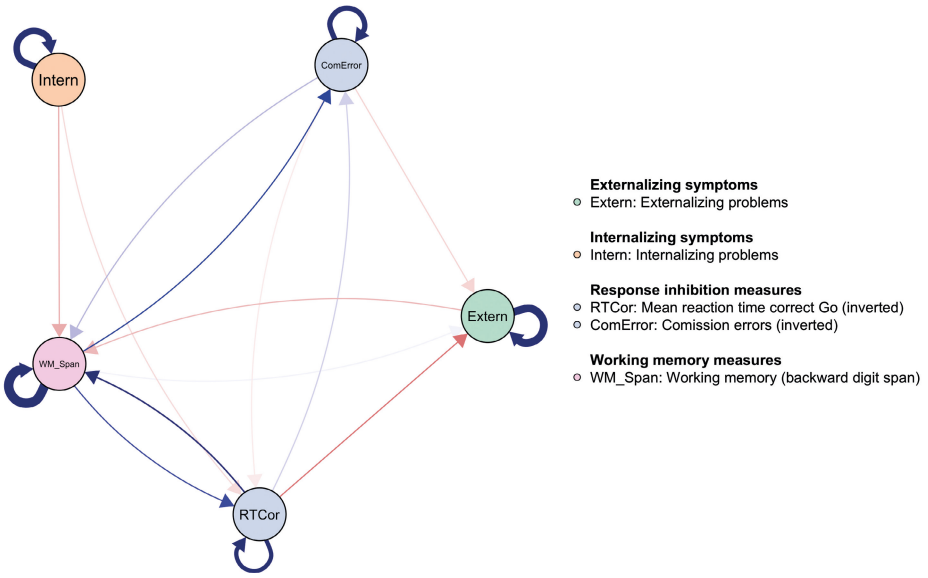
Figure 1 visualizes the temporal associations occurring during early adolescence (from wave 1 to wave 2, age: $M_{wave\ 1}$=10.2, $M_{wave\ 2}$= 13.5). Directed edges (i.e., arrows) indicate temporal associations between nodes from wave 1 predicting nodes at wave 2. At the aggregate level, both indices of inhibitory control (lower commission errors and faster reaction time) predicted fewer externalizing problems. Commission errors and the index of working memory capacity predicted each other over time. A higher working memory capacity predicted fewer commission errors, and, to a lesser extent, the opposite pathway was present. Both externalizing and internalizing problems predicted a lower working memory span over time. There was also a strong positive association between working memory capacity and speed of inhibitory control. Higher working memory capacity predicted faster reaction times in the inhibitory control task, and the reverse direction was present as well. The estimated networks were sufficiently stable (see Supplementary Figures S1-S2 for the results from our bootstrapping stability analysis).

Table 1. Sample characteristics at all waves

	Wave 1		Wave 2		Wave 3		Significance
	High-risk	**Randomly selected**	**High-risk**	**Randomly selected**	**High-risk**	**Randomly selected**	
N	1213	779	997	636	874	565	
Sex: % Female	43.94	47.5	43.33	45.75	41.6	46.18	
Age in Years	10.31 (1.92)	10.02 (1.90)	13.57 (1.94)	13.32 (1.89)	18.31 (2.02)	18.03 (1.97)	w1, w2, w3
Internalizing sum score	9.53 (8.95)	7.11 (7.60)	9.98 (8.85)	7.92 (7.91)	10.76 (9.19)	9.31 (8.53)	w1, w2, w3
Externalizing sum score	9.53 (9.56)	6.91 (8.15)	9.43 (8.90)	7.42 (8.20)	8.99 (8.79)	7.12 (7.52)	w1, w2, w3
Anxiety subscale	4.49 (4.22)	3.42 (3.75)	4.25 (4.07)	3.46 (3.83)	4.46 (4.17)	3.98 (4.17)	w1, w2
Withdrawn-depressed subscale	2.24 (2.89)	1.56 (2.34)	3.03 (3.29)	2.26 (2.73)	3.53 (3.31)	3.01 (3.08)	w1, w2, w3
Somatic problems subscale	2.80 (3.35)	2.12 (2.79)	2.70 (3.07)	2.20 (2.73)	2.82 (3.30)	2.34 (3.05)	w1, w2
Thought problems subscale	2.33 (3.33)	1.62 (2.64)	1.98 (2.79)	1.55 (2.51)	2.25 (2.89)	1.55 (2.28)	w1, w2, w3
Attention problems subscale	5.45 (4.80)	3.83 (4.13)	5.08 (4.54)	4.02 (4.32)	4.46 (4.38)	3.23 (3.74)	w1, w2, w3
Rule-breaking problems subscale	2.31 (3.03)	1.73 (2.65)	2.50 (3.07)	1.93 (2.89)	2.91 (3.36)	2.27 (2.71)	w1, w2, w3
Aggressive problems subscale	7.23 (7.04)	5.18 (5.92)	6.93 (6.37)	5.48 (5.85)	6.04 (6.03)	4.82 (5.33)	w1, w2, w3
GoNoGo mean RT correct Go	450.85 (126.83)	445.69 (119.83)	347.65 (91.04)	339.98 (79.36)	305.24 (70.01)	292.52 (62.13)	w3
GoNoGo percentage commission errors	25.56 (21.78)	24.40 (22.00)	20.23 (21.33)	17.79 (20.28)	10.36 (14.05)	9.70 (12.51)	w2
WM Digit Span Task regular	3.54 (1.60)	3.55 (1.52)	4.36 (1.78)	4.46 (1.67)	4.45 (1.92)	4.55 (1.76)	
WM Digit Span Task corsi	4.73 (2.15)	4.82 (2.04)	5.90 (2.14)	5.97 (2.12)	6.79 (2.37)	7.09 (2.25)	w3

Note. High-risk refers to the high-risk sample. Randomly selected refers to the randomly selected community sample. The range of the CBCL scales differs between syndromes. N refers to the number of participants with no missing data on the CBCL/ABCL internalizing and externalizing measures. w1 = wave 1, w2 = wave 2, w3 = wave 3. To facilitate data interpretation, we display only the CBLC scores and the raw scores (RT and errors) of the inhibitory control tasks. Chi-squared tests were used to compare gender distributions, while two-sided t-tests were used to assess differences in all symptom and cognitive measures. The significance columns refers to $p < 0.05$. The Supplementary table S3 includes all relevant test statistics. Abbreviations: CBCL = Child Behavior Checklist, RT= Reaction Time, M = Mean, SD = Standard Deviation, ms = milliseconds, w = wave.

Figure 1. Wave 1 to wave 2 temporal associations (broad-band scales).

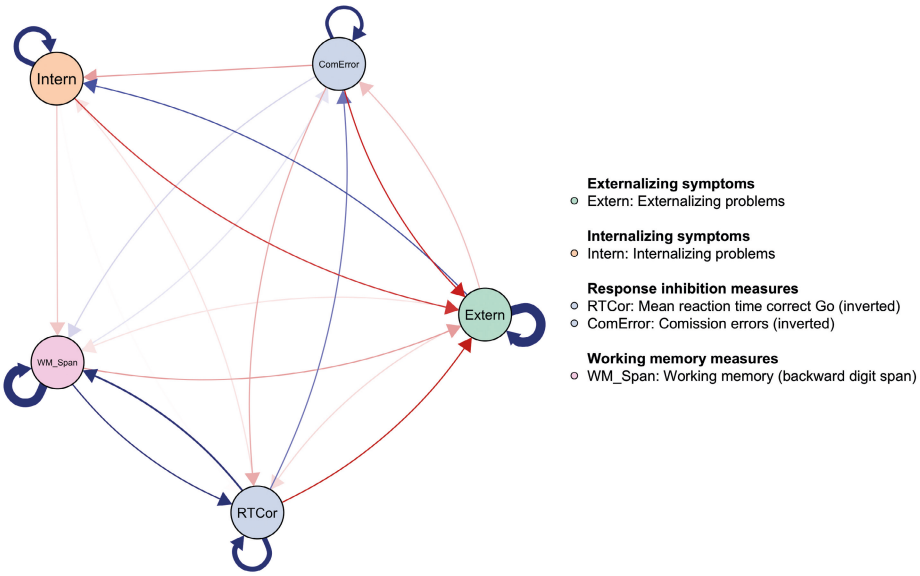


Note. The network depicts temporal associations from wave 1 to wave 2. Each node represents a construct measured at both waves. Outgoing edges (arrows) reflect how a construct at wave 1 predicts another construct at wave 2, while incoming edges reflect how a construct is predicted by other constructs from wave 1. The outcome measures corresponding to the inhibitory control tasks have been inverted to facilitate interpretation. Higher scores on all cognitive control measures (RTCor, ComError, WM_Span) indicate better performance. The colors (blue = positive; red = negative) and thickness of the edges represent the direction and strength of associations, respectively. The edge weights are scaled based on the highest absolute edge weight in the network. The circular arrows on top of each node indicate autoregressive effects (i.e., the extent to which a construct predicts itself over time from wave 1 to wave 2).

Predictive Associations from Early to Middle Adolescence

Next, we examined the associations between cognitive functioning measures and symptoms during the change from early to middle adolescence (age: $M_{wave\ 2}$ = 13.5, $M_{wave\ 3}$ = 18.20). The aggregate temporal network (see Figure 2) is more dense (proportion of non-zero edge: 92% for waves 2-3, 72% for waves 1-2) and contains more reciprocal associations than the previous network during early adolescence. Consistent with the associations observed during early adolescence, better inhibitory control (commission errors and reaction time) predicted fewer externalizing problems. However, we also found effects in the opposite direction, with more externalizing problems predicting worse inhibitory control. There was a weak negative association between internalizing symptoms and working memory span. Moreover, better inhibitory control (fewer commission errors) predicted fewer internalizing problems. Finally, we observed reciprocal associations between externalizing and internalizing symptoms with different signs: A higher level of externalizing symptoms predicted more internalizing symptoms, whereas higher levels of internalizing symptoms at wave 2 predicted fewer externalizing problems later on (wave 3).

Figure 2. Wave 2 to wave 3 temporal associations (broad-band scales).



Note. The network depicts temporal associations from wave 2 to wave 3. Each node represents a construct measured at both waves. Outgoing edges (arrows) reflect how a construct at wave 2 predicts another construct at wave 3, while incoming edges reflect how a construct is predicted by other constructs from wave 2. The outcome measures corresponding to the inhibitory control tasks have been inverted to facilitate interpretation. Higher scores on all cognitive control measures (RTCor, ComError, WM_Span) indicate better performance. The colors (blue = positive, red = negative) and thickness of the edges represent the direction and strength of associations, respectively. The edge weights are scaled based on the highest absolute edge weight in the network. The circular arrows on top of each node indicate autoregressive effects (i.e., the extent to which a construct predicts itself over time from wave 2 to wave 3).

Sensitivity analyses

In addition to the cross-lagged network analysis, we estimated the panel graphical vector-autoregression panel model (Epskamp, 2020) on all three waves of data to examine whether we may be able to obtain an average within-person temporal network (across the three waves). The panel GVAR model is similar to a random-intercept cross-lagged panel model, requires three waves of data, and yields partial (directed) within-person correlations. The model assumes stationarity, and given the substantial trends, we standardized the data at each time point, which inflated the model fit statistics. The panel GVAR model estimated on the detrended data showed poor fit (TLI = 0.56, CFI = 0.73, RMSEA = 0.084), further suggesting that the network structure changes over the course of the three waves. Thus, we considered a cross-lagged network analysis approach examining wave-by-wave associations defensible. Moreover, we re-estimated the cross-lagged network analyses with gender as an additional covariate. This sensitivity analysis (see Supplementary Figures S3-S4) confirmed that key findings remain consistent when controlling for gender.

Discussion

The present study identified dynamic associations between EF measures, namely working memory and inhibitory control, and broadband scales of internalizing and externalizing symptoms during different stages of adolescence. Our findings revealed two key pathways through which executive functions play into transdiagnostic symptom development.

Relatively weak inhibitory control is a risk factor for externalizing symptoms throughout adolescence

During both early and middle adolescence (average ages 10-14, 14-18), relatively weak inhibitory control, indicated by high scores on commission errors and higher reaction times, predicted more externalizing symptoms. The specificity of this association has been found in prior work (Bohlin et al., 2012; Quach et al., 2020). Our study replicated and extended these findings because we examined a longer time frame ranging from ages 10 to 18 and controlled for a range of other variables, including working memory capacity and internalizing symptoms. Low inhibitory control may lead to emotion regulation difficulties and more impulsive behaviors. In line with this risk-account, a recent study by Hentges et al. (2020) showed that an intervention on inhibitory control during early childhood was associated with a reduction in externalizing symptoms at age 14. Further research is needed to better understand the time frame at which the negative repercussions of low inhibitory control in early adolescence can still be mitigated. Our results further indicated that externalizing symptoms predicted future internalizing symptoms during the change from early to middle adolescence. Likely, externalizing behaviors, such as delinquency or aggressive behaviors, may lead to adverse reactions from peers, with a potential loss of social status. This may include disciplinary actions, peer rejection, and academic challenges – all constituting stressors that could explain the increase in internalizing symptoms later on (Weeks et al., 2016).

Adolescent externalizing and internalizing problems predict lower working memory capacity

A novel finding from our analyses (at both change points) is that internalizing and externalizing problems predicted a lower working memory capacity over time. There may be multiple underlying mechanisms that could explain these associations. Emotional and behavioral problems may lead to extensive worrying or rumination that could tax cognitive resources, such as working memory (Levens et al., 2009). Our analyses also revealed a weak positive association between working memory capacity and externalizing problems during early adolescence (see Figure 1). Relatively high working memory capacity was associated with higher scores on externalizing symptoms during early adolescence, specifically with more rule-breaking behavior. We provide possible explanations for this time-sensitive role of working memory: Likely, higher working memory at age 10 allows adolescents to engage in more sophisticated, complex behaviors and social settings that may, in turn, lead to situations during which rule-breaking or externalizing behaviors can be shown. This aligns with the proposed view of social working memory as a key cognitive competency associated with

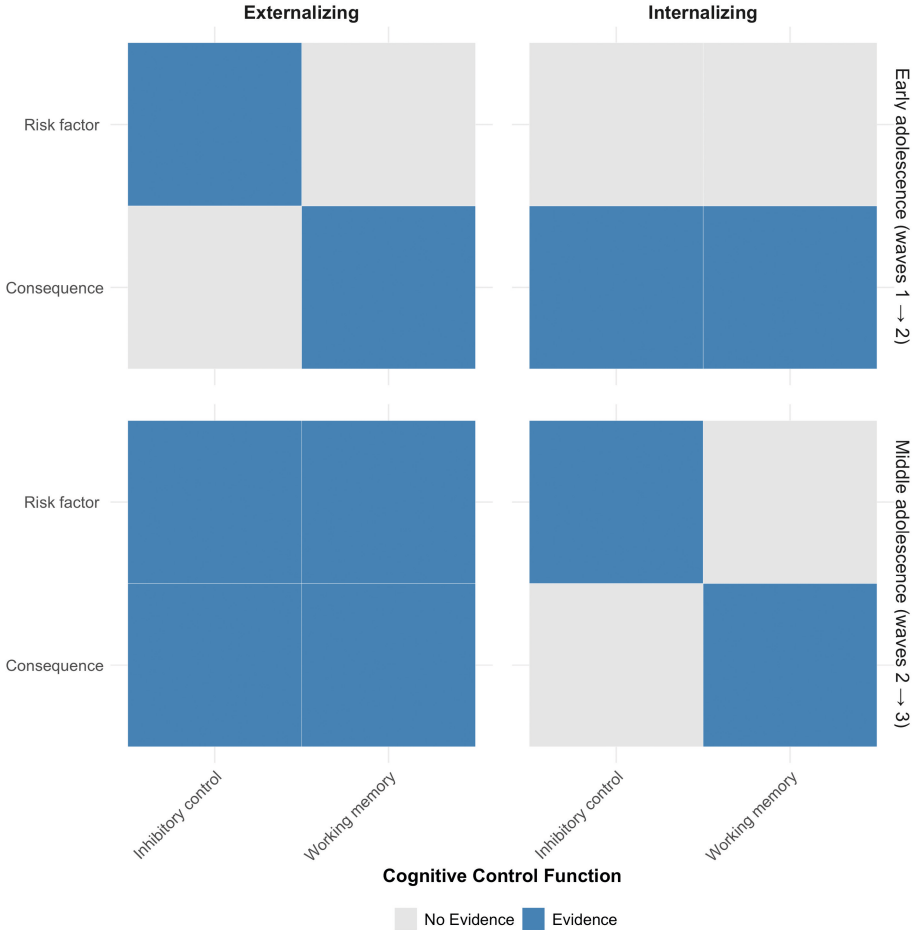
individuals' social network size (Krol et al., 2018). In particular, high working memory at age 10 may put adolescents into contact with social environments that increase the likelihood of externalizing symptoms (aggressive and rule-breaking behaviors) later on. These speculations warrant further research into potential mediating factors, such as the role of peer relationships and family dynamics.

In addition to the predictive associations between EF and symptoms outlined above, we found a complex pattern of associations within the EF and internalizing/externalizing symptom domains. In both early and middle adolescence, we found that a higher working memory capacity predicted lower speed during the inhibition task and more attention problems. This is in line with previous research showing that these different cognitive functions are interrelated during development (Beattie et al., 2018). In addition, our findings regarding the evolving dynamic relationships among executive functions during adolescence, the distinct roles of inhibition and working memory, and the overall growth of cognitive abilities throughout align with the cognitive mutualism theory (Kievit, 2020). This theory describes that positive associations between EFs contribute to general cognitive ability during sensitive developmental windows. Future studies should further integrate these dynamic associations between different EFs in developmental psychopathology theories.

Risk factor and consequence accounts depend on the developmental stage

Altogether, our findings underscore the importance of understanding the role of executive functions in the context of different developmental stages. The conceptual Figure 3 illustrates the evidence in favor of the aforementioned theoretical accounts (risk factor versus consequence theory of cognitive dysfunction) based on our cross-lagged network models. We found that evidence for the risk factor theory of inhibitory control, according to which cognitive dysfunction (see 'C factor', Abramovitch et al., 2021) precedes the development of externalizing symptoms in early adolescence. However, working memory in early adolescence appears to be a cognitive function that is a 'complication' (Maasalo et al., 2021) of this transdiagnostic symptom development. In contrast, during middle adolescence (waves 2 to 3), we observed more reciprocal relationships, with working memory and inhibitory control being both risk factors and consequences. Likely, psychopathology in early life may impact the normative development of executive functions (Rudd et al., 2021), which, in turn, exert their influence as catalysts of more or less adaptive developmental processes. Our study used a novel panel network analytical approach that allowed us to test different theoretical approaches by studying working memory and inhibitory control as both predictors and outcomes in parallel.

Figure 3. Evidence for risk factors and consequence accounts for different cognitive functions, transdiagnostic dimensions, and stages during adolescence.



Note. This figure was based on the presence of directed edges between cognitive control functions (working memory, inhibitory control) and transdiagnostic dimensions (internalizing, externalizing symptoms) at different stages during adolescence (Figure 1: early adolescence, Figure 2: early to middle adolescence).

There are, however, several limitations that should be noted. First, cross-lagged panel network analysis conflates within- and between-person effects (Hamaker et al., 2015) and may result in a low specificity in identifying true within-person temporal effects (Freichel et al., 2024). The relevant network model for identifying within-person associations (i.e., the panel GVAR model) showed a poor model fit, even when the temporal trends were removed. This provided further evidence that the network structure indeed changes during adolescence. However, as we did not separate within- and between-person effects, these temporal estimates should not be interpreted as causal, mechanistic processes. Second, there was a significant attrition rate, in particular, during the second assessment wave, and thus, our findings may be biased.

Third, we used two well-validated measures of EF, namely working memory and inhibitory control. Future studies could test temporal associations between transdiagnostic symptom measures and a wider range of executive functions, including measures of cognitive flexibility, shifting, updating, and verbal and motor speed. Deriving a common EF factor and integrating it in models of symptom dynamics may provide additional insights. Lastly, our analysis of the last assessment wave combined information from the CBCL/ABCL assessment. While this is commonly done in studies with a wide age range (Savage et al., 2015), it is important to acknowledge that these two measures, though designed to be analogous, may assess somewhat different aspects of behavior and emotional problems in children versus adults. This could introduce a degree of measurement invariance that may affect the accuracy and interpretation of the observed cross-lagged effects.

A greater understanding of the dynamic associations between EF and psychopathology may provide valuable insights into the sensitive time windows in adolescence, during which interventions may be useful. Our findings showed that during early adolescence 1) lower inhibitory control is a risk factor for externalizing symptoms, and 2) working memory capacity is a consequence of externalizing symptoms. During middle adolescence, both working memory and inhibitory control serve as both risk factors and consequences of symptom development. Further research is needed to tailor cognitive developmental cascade theories to specific phases of adolescent development. The ultimate objective is to develop early intervention strategies that target relevant EFs in time to prevent an 'escalation' of negative symptom dynamics from arising during adolescence.

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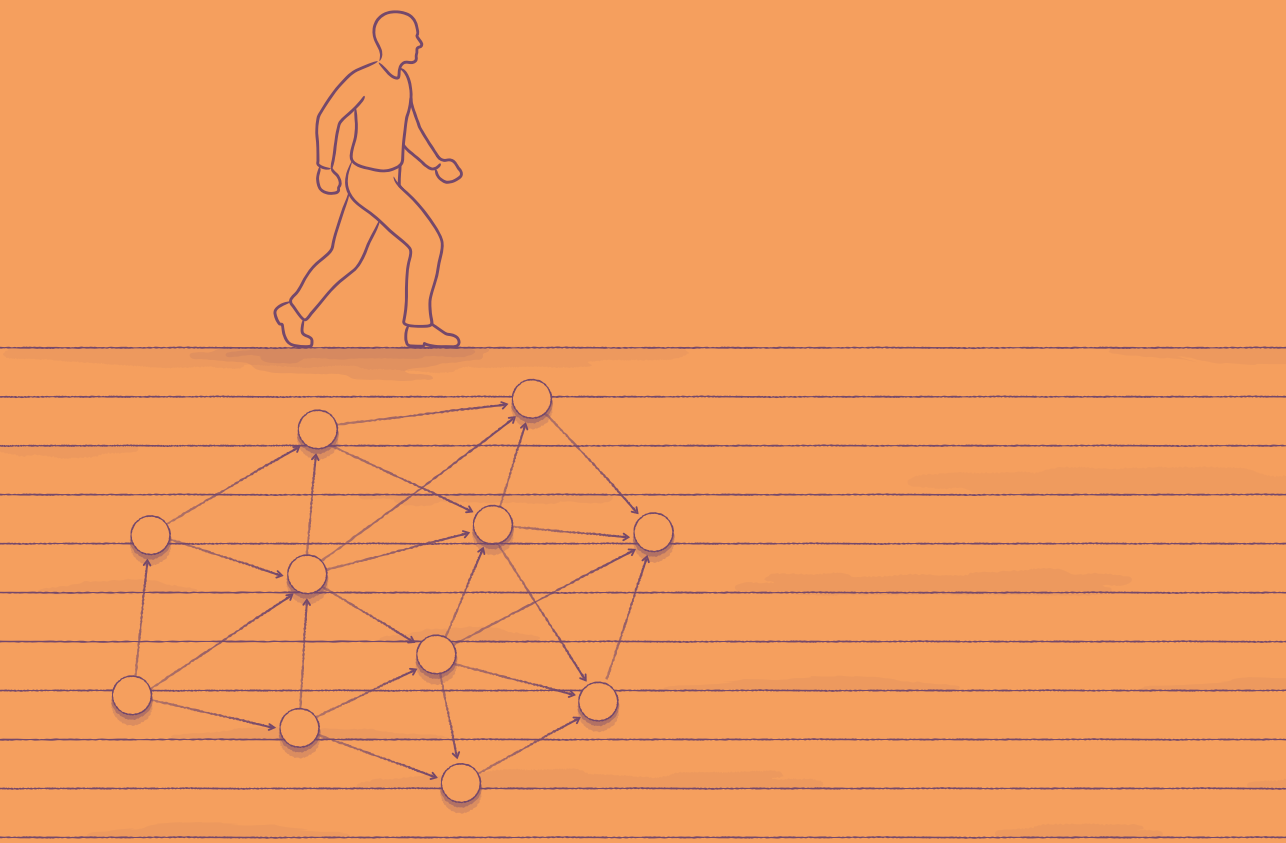

CHAPTER 5

Impaired working memory and risk-taking predict detrimental symptom dynamics in adolescence – A moderated cross-lagged panel network approach

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Abstract

Background: Recent models in developmental psychopathology emphasize the dynamic interplay between substance use and internalizing and externalizing symptoms. This interplay may be moderated by known risk factors, such as impaired working memory capacity and high-risk taking behavior. This study introduces an adaptation of the cross-lagged panel network approach (CLPN) to examine how these factors moderate the temporal associations between substance use and internalizing and externalizing symptoms.

Methods: Using data from the IMAGEN study (N = 1,364), we tested how working memory and risk-taking at age 14 moderated temporal associations between internalizing/externalizing symptoms and substance use over two years (ages 14 → 16).

Results: Alcohol use showed reciprocal associations with externalizing symptoms and predicted heavier tobacco and cannabis use at age 16. Externalizing symptoms at age 14 predicted more externalizing symptoms and substance use at age 16. Poor working memory and high risk-taking moderated the temporal associations between both symptom domains and substance use. When risk-taking was high, the link between internalizing and externalizing symptoms at age 14 and cannabis use at age 16 was stronger.

Conclusions: These findings highlight cognitive risk factors in the substance use/ symptom dynamics and illustrate the value of the moderated CLPN approach in clinical-developmental science.

Keywords: executive functioning; cross-lagged panel network; substance use; internalizing symptoms; externalizing symptoms

Introduction

The incidence of mental health conditions among adolescents has been increasing, with peak onset around age 15 (McGorry et al., 2024), and substance use disorders being among the most prevalent (Sacco et al., 2024). Alcohol, tobacco, and cannabis use are the most commonly used substances by adolescents – with earlier age of onset being a risk factor for developing substance-use-related problems (Robins & Przybeck, 1985; Sjödin et al., 2024). For instance, drunkenness among 15-year olds was shown to predict a range of different problem behaviors across North American and European samples (Kuntsche et al., 2013), such as injuries and fights. The period between ages 14 and 16 is also marked by increased risk-taking and sensation-seeking (Steinberg, 2004) and is critical for the initiation of substance use and the development of behavioral problems, including internalizing and externalizing problems (Spear, 2000).

Recent models on the development of child-adolescent psychopathology emphasize the dynamic interplay between substance use and internalizing/externalizing symptoms, highlighting how these domains mutually influence and predict each other over time (Freichel et al., 2023; Freichel, Pfirrmann, et al., 2024; Speyer et al., 2022). Complex bidirectional relationships between psychiatric symptoms have been integrated in developmental cascade models that, for instance, identified externalizing symptoms as predictors of internalizing symptoms later on (Moilanen et al., 2010). A study by Colder et al. (2013) showed that externalizing symptoms in 12-year-old adolescents predicted alcohol, tobacco, and cannabis use two years later. This externalizing risk pathway for adolescent substance use has been well-documented in the extant literature (Rothenberg et al., 2020; Cox et al., 2021; Goodman, 2010).

Relatively weak, still developing, executive functions (EFs) during early adolescence may serve as a potential risk factor for developing substance use-related problems, as well as internalizing and externalizing symptoms (Freichel, Pfirrmann, et al., 2024). Impaired EF may lead to emotional dysregulation, increasing the susceptibility to dysfunctional behavior patterns, in particular impulsive behaviors (Verdejo-García et al., 2008; White et al., 2011), throughout the lifespan (East-Richard et al., 2020). While relatively weak EF has long been associated with an increased vulnerability for externalizing disorders (Gorenstein & Newman, 1980), more recently it has also been related to a vulnerability for internalizing disorders (Yang et al., 2022) and as a transdiagnostic marker for mental health problems in general (Goschke, 2014). Working memory (i.e., the ability to store and manipulate information over short time windows) constitutes one of the most central EFs (Baddeley, 1992) and has been associated with the general psychopathology (p) factor (Caspi et al., 2014). Conceptually related but distinct from core executive functions are measures of self-control and risk-taking that were shown to predict a range of outcomes related to substance dependence and health (Adlaf & Smart, 1983; Moffitt et al., 2011).

In summary, prior literature has established two critical findings: First, there are temporal pathways linking internalizing and externalizing symptoms with substance use during adolescence. Second, distal associations exist between impaired EFs, risk-taking, and internalizing/externalizing symptoms in adolescence. Despite these significant advances, the

intersection between these two domains (i.e., temporal symptom interplay and relationship with EFs) has not been thoroughly explored. Thus, it remains largely unclear whether relatively weak EFs (compared with peers) in early adolescence may not only directly heighten the risk for the development of substance use and internalizing/externalizing symptoms but may also have more indirect effects via moderating the temporal associations between adolescent internalizing/externalizing symptoms and substance use. A more comprehensive understanding of how (weak) EFs are involved in the dynamic interplay between substance use and internalizing/externalizing symptoms is crucial as it may help us better grasp the impact of low EFs for predicting dysfunctional cycles of symptom escalation (e.g., stronger temporal associations between externalizing symptoms and substance use).

To model the dynamic symptom interplay, symptom network analysis has gained considerable traction over the past decade. This approach is rooted in the network theory of psychopathology, which posits that mental disorders emerge out of the dynamic interactions between symptoms (Borsboom & Cramer, 2013; Cramer et al., 2010). A significant advantage of network analysis lies in its ability to (i) estimate partial correlations, thereby accounting for a range of factors, and (ii) identify the best-fitting temporal patterns of associations within the data without predefining predictors and outcomes. Various network analytical approaches have been developed to estimate temporal associations across extended periods, often examined through panel studies (Borsboom et al., 2021; Freichel, 2023). One of the more recent methodological advancements in this field is cross-lagged panel network analysis (CLPN; Wysocki et al. 2022), which implements a series of regularized regressions for estimating temporal networks based on two assessment waves (i.e., time points) of panel data. This approach has previously been used to examine temporal associations between symptoms of psychopathology in youth and adolescence (Freichel, Pfirrmann, et al., 2024; Funkhouser et al., 2021). However, no study has yet adapted this approach to examine how executive functioning and risk-taking may interact with these temporal associations.

The primary objective of the present study is to explore how working memory and risk-taking may play into the temporal dynamics of internalizing/externalizing symptoms and substance use. In order to explore these moderated effects, we introduce an extension to the CLPN approach, which we refer to as moderated cross-lagged panel network analysis (mCLPN). The mCLPN allows us to examine how individual differences in EF might influence the temporal associations between symptoms and substance use at a later age. Based on prior literature (Colder et al., 2013; Freichel, Pfirrmann, et al., 2024), we outlined several predictions for the temporal associations in early adolescence: (1) Externalizing symptoms predict later internalizing symptoms and substance use; (2) risk-taking predicts substance use and externalizing symptoms; (3) relatively weak working memory and high risk-taking are associated with stronger temporal associations between externalizing symptoms and adolescent substance use, suggesting that deficits in EF may exacerbate the development of problematic substance use through externalizing symptoms.

Methods

Data Source and Procedure

Data were obtained from the IMAGEN study (Schumann et al., 2010), a longitudinal cohort study that followed a community-based sample of young adolescents at eight different locations across Europe. The IMAGEN study was approved by all local ethics committees, and informed consent was obtained from all participants or their legal guardians. Exclusion criteria consisted of neurological conditions (e.g., epilepsy), treatment for schizophrenia or bipolar disorder, an IQ below 70, and specific MRI contraindications (e.g., metal implants). See Schumann et al. (2010) for more details on the sample characteristics. The IMAGEN study included both home assessments and several study visits to the local lab site. All participants were recruited at the age of 14 (wave 1) in local high schools and invited to three follow-up visits after two-year periods (wave 2: age 16; wave 3: age 19, wave 4: 22). During each wave, participants completed a battery self-report questionnaires and clinical interviews. Several behavioral tasks related to EFs were administered during and after functional magnetic resonance imaging at the first wave. We only used data from waves 1-2 to (i) capture EFs at early adolescence and (ii) as we expect the effect of EFs on the interplay between symptoms and substance use to be strongest at more proximal time windows (i.e., waves 1 → 2).

Measures

Strengths and Difficulties Questionnaire (At Waves 1-2)

The strengths and difficulties questionnaire (SDQ) is a behavioral screening measure to assess social and emotional strengths and behaviors among children and adolescents (Goodman, 1997). The short-form questionnaire consists of 25 items (see Table S1 in the Supplementary Materials) that assess the five subscales: emotional symptoms, conduct problems, inattention/hyperactivity, peer relationship problems, and prosociality. Single items are rated on a 3-point Likert scale (not true, somewhat true, certainly true). Conduct problems and hyperactivity form the externalizing subscale, and emotional problems and peer-relationship problems form the internalizing subscale (A. Goodman et al., 2010). Sum scores on both the internalizing and externalizing subscales range from 0 to 20 with higher scores indicating more severe problems. The SDQ showed good structural and construct validity in non-clinical and community samples (Dahlberg et al., 2019; Hawes & Dadds, 2004).

Alcohol Use Disorders Identification Test (At Waves 1-2)

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is the WHO gold-standard screening instrument for alcohol use and problems with good psychometric properties (Källmén et al., 2019). The first three items of the AUDIT (typically referred to as AUDIT-C) were used to assess the frequency of drinking. This AUDIT-C total score (0-12) was previously shown to be valid in identifying individuals with hazardous and harmful drinking behavior (Verhoog et al., 2020).

Tobacco and Cannabis Use (At Waves 1-2)

Tobacco (past month) and cannabis (past year) use was measured through items from the European School Survey Project on Alcohol and Drug (ESPAD, Hibell et al., 2004). Respondents indicated their tobacco use ("How frequently have you smoked cigarettes during the last 30 days?") on a six-point Likert scale (0 = 'Not at all', 1 = 'Less than 1 cigarette per week', 2 = 'Less than 1 cigarette per day', 3 = '1-5 cigarettes per day', 4 = '6-10 cigarettes per day', 5 = '11-20 cigarettes per day', 6 = 'More than 20 cigarettes per day'). Cannabis use was assessed using the question "On how many occasions over the last 12 months have you used marijuana (grass, pot) or hashish (hash, hash oil)?" and respondents indicated their use on a six-point Likert scale (0 = '0', 1 = '1-2', 2 = '3-5', 3 = '6-9', 4 = '10-19', 5 = '20-39', 6 = '40 or more'.

Executive Functioning Tasks (At Wave 1)

Executive functioning was assessed using neuropsychological tests, including the Cambridge Neuropsychological Test Automated Battery (CANTAB, Robbins et al., 1994). We used two cognitive tasks (Spatial Working Memory Task, Cambridge Gambling Task) that were all administered only at wave 1 and, relevant for the aims of our study, spanned different domains of EF, specifically spatial working memory and risk-taking.

Spatial Working Memory Task. The Spatial Working Memory (SWM) task assesses working memory with respect to visuospatial information that participants are instructed to retain, manipulate, and use according to a heuristic strategy. A number of colored squares ('boxes') are displayed on a screen and participants are asked to find blue tokens in a number of boxes to fill up an empty column. The number of boxes on the screens increases until all eight boxes need to be used in the search. Both the color and the position of the boxes are changed in every trial. 'Between search errors' occur when participants touch boxes that have already been searched on that trial. More between errors indicate lower short-term spatial working memory. The SWM task has been commonly used in both clinical populations and typically developing adolescent samples (Faridi et al., 2015).

Cambridge Gambling Task. The Cambridge Gambling Task (CGT) was used to assess impulsive and risky decision-making (Rogers et al., 1999). A number of red and blue boxes were shown on the screen and participants were instructed to indicate whether a target stimulus ('yellow token') was hidden behind one of the red or blue boxes. A range of points was shown to participants to bet on their guess. If participants picked the wrong color, these points would be lost. However, in the case that their guess was correct, the points would be gained. We derived a commonly used measures of risk-taking (overall betting ratio) from the CGT task. A higher overall proportion bet indicates more risk-taking behavior and a greater proportion of points that the participants bet on the trials.

Statistical Analysis

Moderated Cross-Lagged Panel Network Analysis

The CLPN uses a series of regularized regression models to estimate autoregressive (e.g., internalizing symptoms at t1 predicting themselves at t2) and cross-lagged effects (e.g., internalizing symptoms at t1 predicting externalizing symptoms at t2) between two consecutive time points. In this framework, each variable serves as both a predictor (at t1) and outcome (at t2). The model uses 10-fold cross-validation to select the tuning parameter lambda (λ) that produces the lowest mean squared error. The tuning parameter λ determines the level of penalization and minimizes overfitting. This model specification has been used in empirical investigations in the extant literature (Freichel, Pfirrmann, et al., 2024; Funkhouser et al., 2021; Zainal & Newman, 2023). For detecting potential moderation effects of EF measures (at t1) on the temporal associations between symptoms and substance use measures (t1 → t2), we developed an extension of the cross-lagged panel network analysis (CLPN, Wysocki et al., 2022) approach. We expanded this standard CLPN approach by integrating two time-invariant predictor variables (EF measures), assessed only at t1. We included these EF measures as predictor variables and their respective interaction terms (i.e., moderation effects) with all other variables measured at t1. Contrary to the other variables in the CLPN that are both added as predictors (at t1) and outcome (at t2), these EF measures were not treated as outcomes/nodes in the model. This extended approach allowed us to examine how (1) EF predicts all outcome measures at t2 directly (e.g., higher risk-taking at t1 predicting more substance use at t2) and (2) EF moderates the autoregressive and temporal cross-lagged associations between nodes (e.g., externalizing symptoms at t1 predicting substance use at t2, and this temporal association is stronger for higher risk-taking individuals at t1). Before estimating the mCLPN, we standardized the EF measures (at t1), and standardized all symptom and substance use measures across waves separately. This standardization was necessary as all measures were on different scales (e.g., number of errors, symptom scores), and standardization across time points ensured that the trends across time were retained.

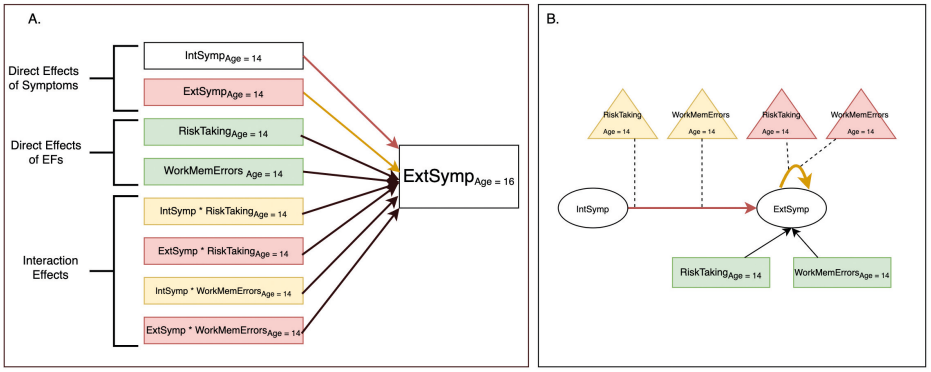
Moderated CLPN Visualization

As the adapted/moderated CLPN model estimates more parameters than typically used in CLPN networks, we introduce an extended method of visualization: Figure 1, panel A, illustrates the path diagram for a single regularized regression model in our mCLPN model. For this visualization, we included only one variable as the outcome, whereas typically all variables in the CLPN are treated as outcomes. In this example, externalizing symptoms (at age 16) are being predicted by themselves (at age 14, autoregressive effects), internalizing symptoms (at age 14, cross-lagged effect), the two EF measures (at age 14), and their respective interactions with both externalizing (moderated autoregressive effects) and internalizing (moderated cross-lagged effects) symptoms. Figure 1, panel B, illustrates the resulting extended network visualization. The nodes represent internalizing and externalizing symptoms, respectively. As in this example illustration, internalizing symptoms predict externalizing symptoms, these two nodes are connected through an edge (internalizing → externalizing) following the standard

approach for visualizing CLPN. We extend the standard CLPN visualization by representing the direct effects of the two EF measures as green rectangles, with arrows pointing toward their respective outcomes. This visualization indicates that higher levels of these EF measures (e.g., increased risk-taking and working memory) at t1 are associated with more externalizing symptoms at t2. The moderation effects between the EF measures and the relevant symptom/substance use measures are represented as triangles. These interactions can apply to both cross-lagged effects (e.g., internalizing at t1 predicting externalizing at t2, shown in yellow) and autoregressive effects (e.g., internalizing at t1 predicting internalizing at t2, shown in red).

Figure 1

Illustration of the moderated CLPN path diagram and the corresponding network visualization.



Note. Panel A shows the path diagram of the mCLPN for one single outcome (externalizing symptoms) at t2. Panel B shows an illustration of the moderated network visualization that includes relevant estimates from the mCLPN shown in Panel A. The nodes in Panel B represent both predictors and outcomes. The rectangles represent only predictors. The top red arrow in panel A corresponds to the directed edge in panel B. The second to the top yellow arrow in panel A corresponds to the autoregressive circular arrow in panel B. The direct effects of the EF measures are shown in rectangles (green). Triangles indicate moderation effects for the cross-lagged temporal associations (yellow) and the autoregressive effects (red). Positive moderation effects (i.e., strengthening) will be indicated by a '+' sign inside the triangle, whereas negative moderation effects (i.e., weakening) will be indicated by a '-' sign. EFs = executive functions; IntSymp = internalizing symptoms; ExtSymp = externalizing symptoms; RiskTaking = risk-taking EF measure; WorkMemErrors = working memory EF measure.

Simulation Study

Since the proposed moderated CLPN approach is an extension to the CLPN methodology and involves estimating more parameters, we conducted a preliminary simulation study. We assessed the effectiveness of the mCLPN approach (i.e., sensitivity, specificity, estimation error) at varying sample sizes (n = 100-1,000). Our simulation results indicated high sensitivity and moderate specificity of the mCLPN for detecting moderation effects (set to 0.1) at large sample sizes (n = 1,000), with lower sensitivity at smaller sample sizes (n = 100-500). See supplementary materials section 2 for more details.

Network Stability

To examine the stability of the estimates derived from the model, we use a non-parametric bootstrapping approach (Epskamp et al., 2018) in which the model is re-estimated 1,000 times. In each iteration, the sample is randomly split into training and test sets, ensuring our results not overly dependent on a specific data partition. We computed bootstrapped confidence intervals around the edge weights and the percentage of estimates being non-zero (in all bootstrapped samples). All analysis scripts were made public on the Open Science Framework (https://osf.io/bt2e7/?view_only=1fca6eed92bb4badbbf31d06f001c22f).

Results

Sample Characteristics

Our analyses were based on a sample of 1,364 individuals (51.5% female) who provided complete data for all relevant measures of symptoms, substance use, and cognitive tasks. Participants were adolescents, on average, 13.93 years old (median = 14, SD = 0.46) at wave 1 and 16.02 years old (SD = 0.74) at wave 2. There was substantial variability in the reported levels of alcohol use; more than half of all individuals reported consuming any alcohol during the first wave, and this substantially increased by the second wave ($t = -31.70$, $p < .001$; see Figure S1 for the distribution of the AUDIT-C score).

At age 14, 9.2% of individuals reported any tobacco use, which increased to 27.3% at age 16. This indicates a notable rise in tobacco use over time (McNemar test based on use/non-use: $\chi^2 = 204.73$, $p < .001$). There was a low prevalence of cannabis use in this sample: At age 14, 2.1% of individuals reported any cannabis use, whereas at follow-up (T2), this proportion increased significantly (McNemar test based on use/non-use: $\chi^2 = 124.66$, $p < .001$), with 12.8% of individuals (n = 175) reporting cannabis use.

The prevalence of internalizing/externalizing symptoms showed a diverging pattern over time: Internalizing symptoms slightly increased from ages 14 to 16 (age 14: M = 4.44, SD = 2.90; age 16: M = 4.77, SD = 3.16; $t = -4.061$, $p < .001$). In contrast, the level of externalizing symptoms slightly decreased over time (age 14: M = 5.86, SD = 3.00; age 16: M = 5.16, SD = 3.03; $t = 8.98$, $p < .001$).

Participants demonstrated moderate levels of risk-taking as indicated by the overall betting ratio (M = 48.92, SD = 13.15), which was consistent with other adult comparison samples (Romeu et al., 2020). Participants committed, on average, 18.21 between search errors (SD = 13.30) during the working memory task. The spatial working memory performance in our sample was slightly better than that previously reported for non-clinical samples in the IMAGEN data (Nemmi et al., 2018).

Temporal Relations Between Substance Use and Broad-band Symptoms

Figure 2 visualizes the mCLPN results for the temporal associations from age 14 (wave 1) to age 16 (wave 2). The bootstrapping analysis indicates sufficient stability and can be found in Figure S4 in the supplementary materials. Edges identified in at least 50% of all bootstrap samples may be regarded as stable (Zainal & Newman, 2022). Here, we only visualize and interpret those moderation effects that were retrieved in more than 50% of bootstrapped samples.

Cross-lagged and Autoregressive Effects (Typical CLPN Estimates)

Externalizing symptoms at age 14 were associated with increases in internalizing symptoms at age 16, while the reverse direction was not included. Moreover, externalizing symptoms were associated with increases in tobacco, cannabis, and alcohol use from age 14 to 16. Alcohol use specifically showed reciprocal associations with externalizing symptoms and was associated with increases in tobacco and cannabis use from age 14 to 16. Internalizing symptoms were associated with less alcohol and cannabis use over time.

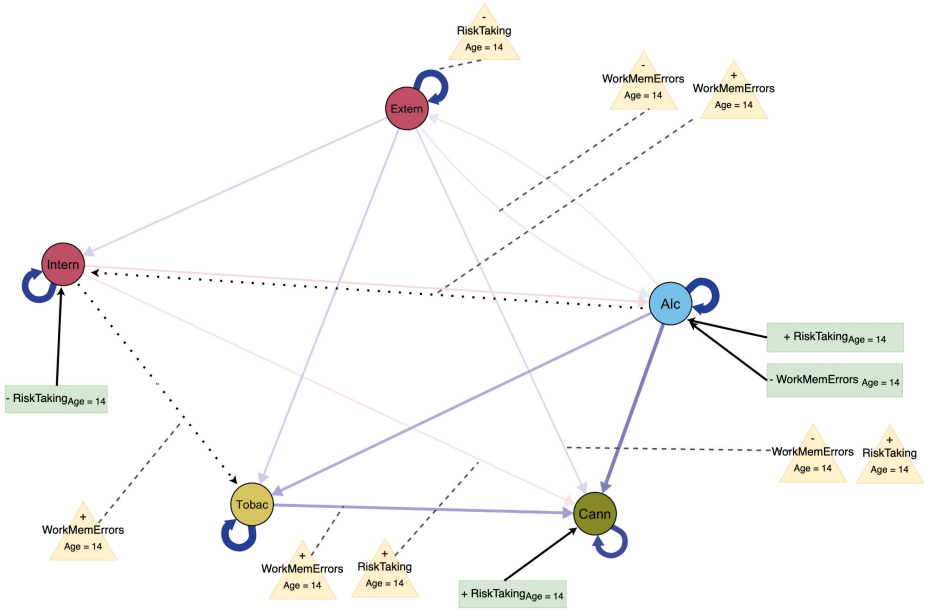
Direct and Moderation Effects (Extended CLPN Estimates)

There were several direct associations between the EF measures and symptom scores and substance use: Higher risk-taking at age 14 was associated with increases in cannabis and alcohol use at age 16, and with fewer internalizing symptoms at age 16. Surprisingly, fewer working memory errors at age 14 (i.e., relatively strong WM) were associated with more alcohol use at age 16.

We observed several moderation effects involving both EF measures: The association between externalizing symptoms at age 14 and cannabis use at age 16 was stronger for higher levels of risk-taking and better working memory at age 14. Notably, there were several positive interaction effects involving working memory: More working memory errors (i.e., relatively poor working memory) at age 14 were associated with stronger associations between alcohol use at age 14 and internalizing symptoms at age 16. Similarly, poorer working memory was associated with a stronger lagged association (wave 1 → wave 2) between (i) internalizing symptoms and tobacco use, as well as between (ii) tobacco use and cannabis use. Moreover, there was a negative interaction effect of working memory errors on the association between externalizing symptoms and alcohol use: The positive association between externalizing symptoms and alcohol use was weaker for individuals with poorer working memory. Finally, higher risk-taking was associated with a stronger (i.e., more positive) association between internalizing and externalizing symptoms at age 14 and cannabis use at age 16. Interestingly, we also found risk-taking to moderate a lower autocorrelation of externalizing symptoms, suggesting that individuals' level of externalizing symptoms was less stable over time when risk-taking at age 14 was high.

Figure 2

Moderated CLPN for Associations Between Age 14 (Wave 1) and Age 16 (Wave 2)



Note. WorkMemErrors = working memory errors; RiskTaking = risk-taking. Higher scores on WorkMemErrors indicate worse working memory; higher scores on RiskTaking indicate more risk-taking. The direction of the direct and interaction effects is indicated through a + (positive) or – (negative). A positive interaction effect (+) indicates that the relationship between two nodes strengthens in a positive direction, meaning the association becomes more positive. Conversely, a negative interaction effect (–) indicates that the relationship weakens or shifts in a negative direction, meaning the association becomes more negative. Dashed arrows between nodes represent indirect associations where no direct effect was found, however, these relationships were part of an interaction effect. Triangles indicate moderation effects for the cross-lagged temporal associations and the autoregressive effects. The figure displays only those interaction effects that were non-zero more in than 50% of the bootstrapped samples.

Discussion

The current study introduced a novel extension of the cross-lagged panel network (CLPN) approach, which was then used to address a central research question in the field of developmental psychopathology: Do impaired working memory and risk-taking predict broad-band symptom domains and substance use? Additionally, how do these factors moderate the temporal relationships between symptoms and use of different substances during adolescence? Our findings (i) advance our understanding of impaired working memory and risk-taking as transdiagnostic risk factors for symptom development and (ii) illustrate the potential of moderated CLPN in future research.

Externalizing Pathway to Substance Use and Internalizing Problems

First, we observed a normative pattern of substance use and symptom development during adolescence, characterized by increased use of all three substances, rising internalizing symptoms, and slight decreases in externalizing symptoms. This trajectory, occurring between the ages of 14 and 16, aligns with established developmental trends (Palmer et al., 2009). We then used an extended moderated CLPN approach to investigate the role of EF in the dynamic interplay between internalizing/externalizing symptoms and use of different substances.

Our findings revealed a relatively dense temporal network of associations between ages 14 and 16. Alcohol use at age 14 predicted higher levels of alcohol use at age 16 as well as cannabis and tobacco use. This finding may be due to alcohol being the most frequently tried substance by adolescents at age 13 or younger in European samples (ESPAD Group, 2020), and use of any substance was shown to be related to the involvement in other substances. Externalizing symptoms predicted later internalizing symptoms and the use of cannabis, tobacco, and alcohol. This externalizing pathway into substance use fits with prior results (King et al., 2004). For instance, approximately 50% of adolescents who begin substance use before age 15 have a history of conduct problems (Odgers et al., 2008). Further, the prediction of internalizing symptoms based on externalizing problems aligns with developmental cascade models (Moilanen et al., 2010). Different factors may explain this association, for instance, adolescents with conduct problems may face social rejection from peers that in turn may drive loneliness and depressive symptoms (Rotenberg, 2020). Interestingly, internalizing symptoms at age 14 were associated with less alcohol use at age 16. This novel finding may suggest that internalizing symptoms may as a protective factor against increased alcohol use during adolescence. As shown, alcohol use increases during this developmental period and is primarily driven by social drinking motives (Freichel et al., 2023) and peer influence. Adolescents with higher levels of internalizing symptoms, albeit not meeting the criteria for an internalizing disorder, may be more withdrawn and less likely to socially motivated drinking.

Impaired Working Memory and Risk-taking as a Predictors and Catalysts of Symptom Development

Our empirical investigation of the role of EF risk factors, namely working memory and risk-taking, showed evidence for specificity in the link between these factors and broad-band symptom domains. Higher risk-taking was associated with more cannabis and alcohol use. This replicates a well-established association in the literature between risk-taking and substance use and aligns with a previous study showing greater risk-taking in individuals with potentially problematic substance use in the IMAGEN sample (Schneider et al., 2012).

We believe the novelty of our study lies in analyzing how impaired cognitive functions not only predict an increase in substance use and symptom burden but also serves as a catalyst in moderating the temporal associations between substance use and internalizing/externalizing symptoms. By integrating EF as a moderator, we were able to examine how higher risk-taking and poorer working memory intensify the temporal associations between externalizing symptoms and subsequent substance use. Our findings revealed two key patterns: First,

relatively poor working memory was associated with stronger temporal associations between (i) alcohol use and internalizing symptoms, (ii) internalizing symptoms and tobacco use, and (iii) tobacco use and cannabis use. Second, high levels of risk-taking were associated with stronger outgoing links from both internalizing and externalizing symptoms towards cannabis use. This patterns of findings may speak to the potential role of relatively poor or delayed development of working memory in the development of psychopathology (Huang-Pollock et al., 2017) and elevated risk-taking as transdiagnostic risk factors implicated in the symptom development. Importantly, our findings suggest that elevated risk-taking acts as a vulnerability factor, that not only contributes to increased substance use independently (i.e., alcohol and cannabis use) but also magnifies their interconnections with broad-band psychopathology symptoms (i.e., externalizing/internalizing → cannabis use) over time. The present study further highlights the importance of expanding symptom network analysis to integrate behavioral measures alongside biological measures and neural biomarkers (Blanken et al., 2021; Freichel, Lenartowicz, et al., 2024; Piazza et al., 2024) to better describe the dynamic symptom interplay that characterize psychopathology (Borsboom, 2017).

Our results also showed several unexpected findings: Prior work has linked poor working memory performance to externalizing (Huang-Pollock et al., 2017), depression and anxiety symptoms (Moran, 2016; Snyder, 2013). While our results showed that poor working memory moderated temporal associations, we found no direct effect of low working memory on internalizing or externalizing symptoms. Similarly, better working memory at age 14 was associated with more alcohol use at age 16. One possible explanation is that adolescents with better working memory at this age may also be exhibit higher levels of social engagement and participate more in social drinking (Freichel et al., 2023)

Utility of the moderated CLPN approach

We extended the CLPN approach to include moderation effects of time-invariant factors such as baseline factors or demographic characteristics. In a first simulation study we showed that the mCLPN approach is equally effective (i.e., achieving comparable levels of sensitivity and specificity) in detecting moderation effects as it is in identifying direct cross-lagged effects provided the sample size is large (e.g., n = 1000). However, at smaller sample sizes (100-500), the model showed low levels of sensitivity, indicating that it cannot reliably recover all main and interaction effects. The need for larger sample sizes to detect moderation effects (that are typically of smaller magnitude) is unsurprising and fits with long-standing knowledge about design and sample size decisions in regression models (Gelman, 2023; McClelland & Judd, 1993). Therefore, researchers employing moderated CLPN should ensure adequate power for accurate model estimation. Moreover, our simulation study revealed only moderate levels of specificity even at larger sample sizes, indicating that it may likely falsely identify interaction effects. This is consistent with a prior simulation study on the CLPN (Freichel, Veer, et al., 2024), that found low specificity in recovering the true within-person temporal associations. Thus, there may be a higher risk of false positives, underscoring the importance of interpreting these exploratory results, particularly the unexpected findings, with caution.

We introduced a first extension of moderated CLPN, that could be adapted in multiple ways. For example, applied researchers may include an additional pruning and re-estimation step in which estimates that were shown to be zero are fixed to zero (Wysocki et al., 2022). This may lead to a higher specificity in correctly identifying moderation effects. While moderated symptom network models exist for cross-sectional (Haslbeck, 2022) and time-series data (Bringmann et al., 2024), the present study is, to the best of our knowledge, the first to employ a moderated CLPN approach in panel data. This served as a proof-of-concept and more extensive simulation studies are needed to validate this approach and establish the conditions under which moderated CLPN perform optimally. We believe that moderated CLPN may in principle hold promise with respect to advancing clinical psychological science in two key domains: First, it allows investigators to study the effect of baseline variables assessed only once, as demonstrated in the present study, or other stable, time-invariant factors, such as personality traits, trauma, chronic illness, financial difficulties on the symptom interplay. For example, consider the case of rumination as a well-established cognitive vulnerability factor that has been associated with the development of depressive symptoms over time (Nolen-Hoeksema, 2000). Rumination may also promote sleep problems (Clancy et al., 2020) m which are closely linked to depressive symptoms (Bao et al., 2017). Using moderated CLPN, researchers could examine whether trait rumination moderates the associations between sleep problems and depressive symptoms while controlling for other relevant factors, such as anxiety symptoms, within a network approach.

Another potential for using moderated CLPN may involve studying intervention effects. In studying intervention effects, we are primarily interested in modelling changes over time. A commonly used panel network model, such as the panel graphical vector-autoregression (VAR) models (Epskamp, 2020) operates under the assumption of stationarity and does not allow to include time-invariant factors. According to this model and assumption there should be no changes in the means and variances across the assessment period, which is directly at odds with the objective of investigating treatment effects over time. To overcome the assumption of stationarity Blanken et al. (2019) introduced network intervention analysis in which a network is estimated for each assessment and in which a treatment allocation variable (e.g., cognitive behavioral therapy or control condition) is included in the network. While this approach allows to explore which symptoms are directly affected by treatment, by estimating separate networks for each assessment wave, all effects happening over time are lost. Our extended moderated CLPN may close this gap as it allows to include a time-invariant factor (i.e., treatment allocation) while still modelling the effects over time. As such, moderated CLPN that include treatment variables may provide an additional lens through which to examine the effects of treatment on the temporal symptom interplay across extended periods.

Limitations

Several limitations should be noted when interpreting our findings. First, our core analyses rely on data based on adolescents' self-report on several measures, including the SDQ. Future studies should further validate the patterns found using both teacher- and parent-rated reports (on the SDQ). Moreover, the skewed distribution of AUDIT scores, with a significant proportion of individuals reporting no alcohol use at the first wave, may impact the accuracy of the estimates. Second, due to the limited data available, our measures of frequency of use for tobacco (i.e., cigarette use) and cannabis differed with respect to the time scales (occasions per year/week), and thus, they cannot capture differences in use on a granular level. Third, we focused on two commonly used EF measures, namely working memory and risk-taking. To fully capture the transdiagnostic relevance of EFs, future studies should include additional EFs, particularly inhibition and attention-shifting (Miyake et al., 2000). It is important to highlight that while our findings reflect typical developmental patterns in the general adolescent population, the observed relationships between cognitive measures and broad-band measures of psychopathology may differ in clinical populations with internalizing or externalizing disorders. Fourth, the CLPN approach only identifies linear associations and does not distinguish between within- and between-person effects (see Curran & Bauer, 2011). Thus, the resulting temporal associations should not be interpreted as causal, mechanistic processes. Moreover, as symptom network models focus on partial effects while controlling for many other factors, the absolute strength of the temporal effects remains difficult to interpret or quantify. Lastly, we used a novel adaptation of the CLPN approach to detect moderation effects between baseline EF measures and temporal symptom-substance use associations. Our supplemental stimulation study provided evidence for the effectiveness of this approach in retrieving interaction effects at large sample sizes. However, an extended set of simulations is necessary to evaluate the robustness of these findings across different effect sizes and model complexities.

Concluding Comments

In conclusion, this article introduced a novel extension of the CLPN approach, allowing us to explore how impaired EF not only predicts substance use and symptom burden but also moderates the temporal relationships between internalizing, externalizing symptoms, and substance use. For instance, we showed evidence for the role of risk-taking as a transdiagnostic risk factor as it may intensify the associations between broad-band symptom domains and cannabis use. While the moderated CLPN approach holds promise for advancing our understanding of developmental psychopathology, further validation through simulation studies are necessary to fully establish its robustness and applicability across different contexts.

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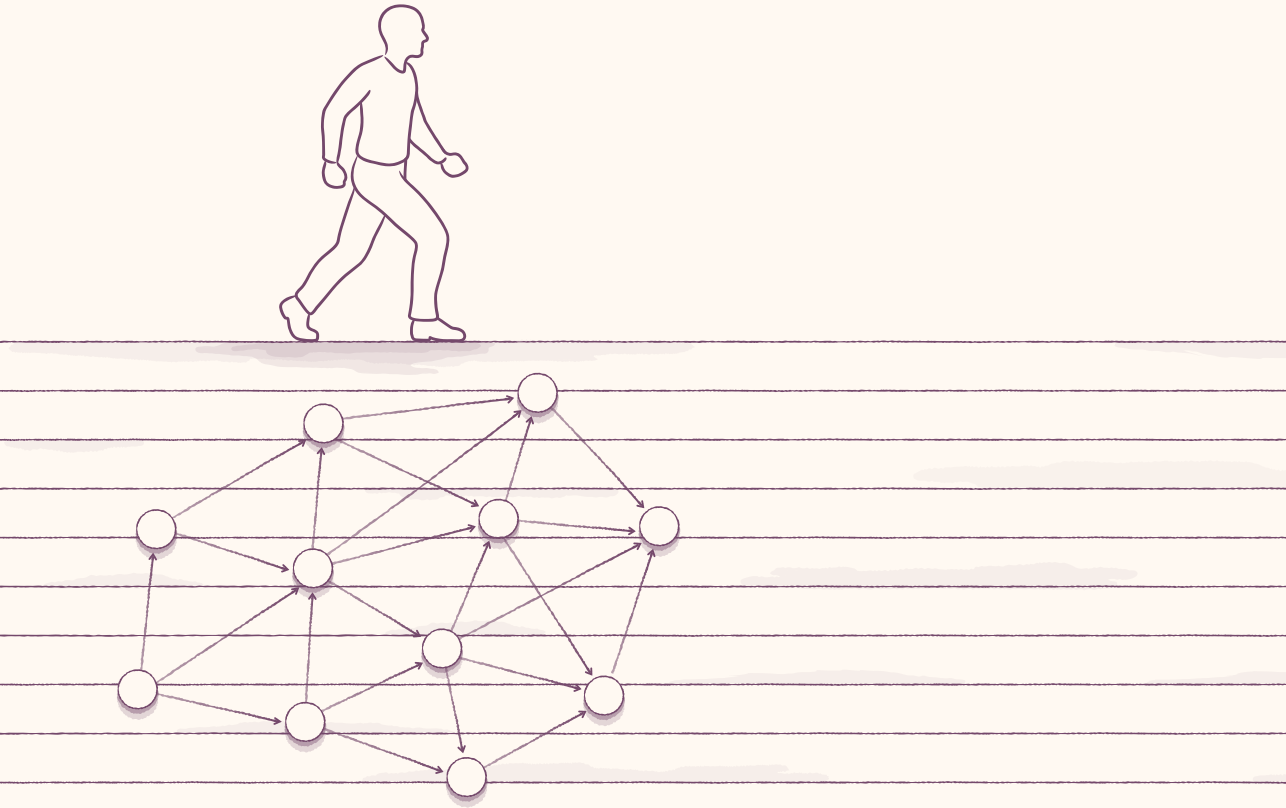
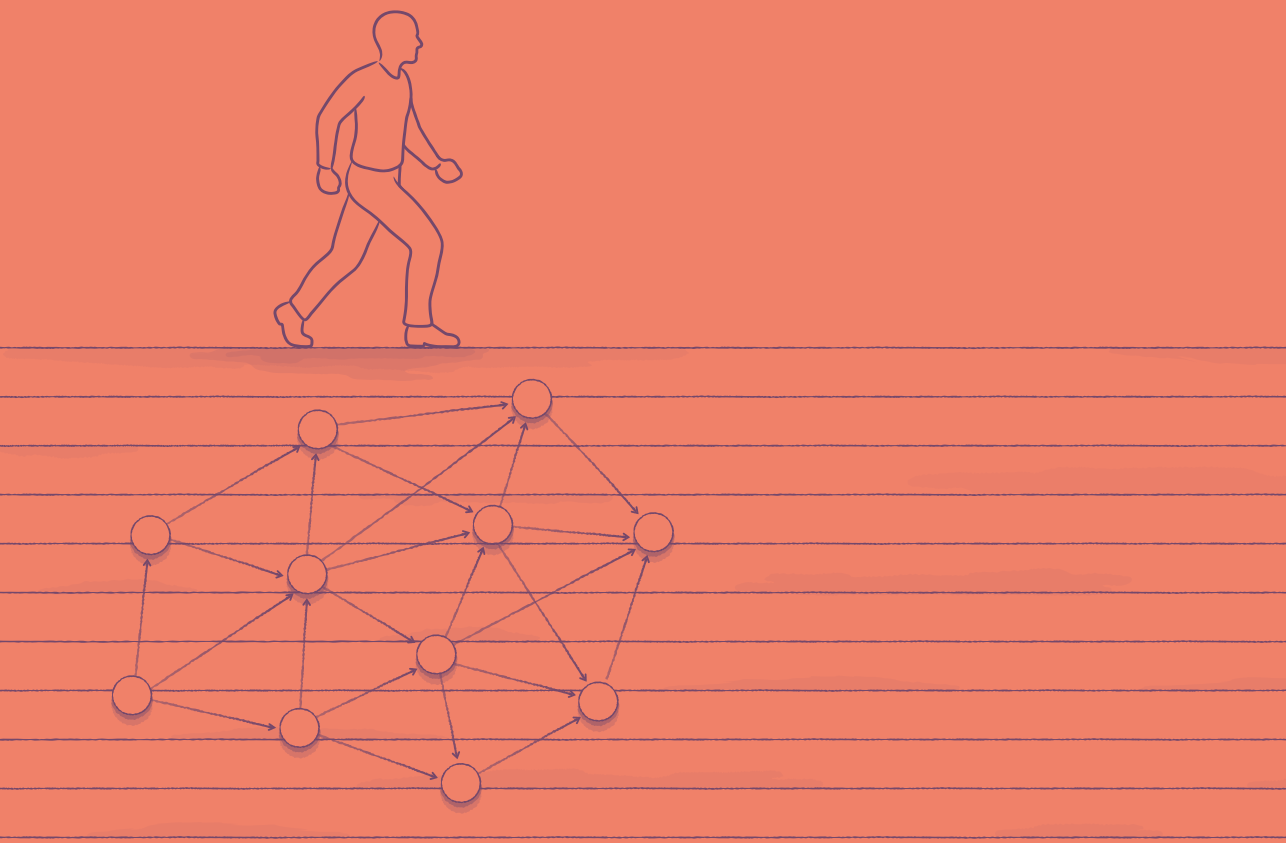


Part 2

Short-term dynamics and experimental designs



CHAPTER 6

Executive Functioning and Daily Mood Dynamics: A Multi-Method EMA Study

This chapter is adapted from:
Freichel, R., Karami Motaghi, A., Ruppin, S., Hamlett, G. E., de Jong, P., Cousijn, J., Wiers, R. W., & Veer, I. M. Executive functioning and daily mood dynamics: A multi-method EMA study. Manuscript in preparation.

Abstract

Executive functions (EF) play an important role in regulating emotions and affective states. EF can be assessed with behavioral tasks or with self-report instruments. It can be conceptualized as a trait, and measured at a single time point, or as a state that fluctuates over time. This exploratory multi-method ecological momentary assessment (EMA) study aimed to examine (1) how different aspects of EF predict daily dynamics in affect (i.e., variability and inertia), (2) to what extent perceived problems with EF predict positive and negative affect, and (3) the relationship between daily EF performance (i.e., Stroop task) and affect. Forty-six undergraduate students completed EF assessments (digit span and go/no-go tasks) before and after a two-week EMA period. The EMA included measures of positive and negative affect (four times a day, every three hours), and a short smartphone-based Stroop color word task every morning. We found no significant links between EF as indexed by daily Stroop performance and affect states. Yet, inattention as indexed by higher omission rates at the go/no-go task during pre-test (before the EMA period) was associated with greater variability in negative affect and lower inertia of positive affect. Multi-level vector-autoregression (VAR) network models identified contemporaneous associations between self-reported EF indicators and affective states: Better attentional control co-occurred with greater positive affect. Greater difficulties disengaging from repetitive thoughts were related to higher negative affect and lower positive affect within the same time window. At the temporal level, increases in negative affect and decreases in positive affect preceded greater difficulty disengaging from repetitive thoughts over time. Limitations of this study include the modest sample size and the use of a mobile Stroop task, which may not have reliably captured attentional/inhibitory control. We conclude that affect regulation is related to self-reported EF, but affect also appears to modulate self-reported EF facets at the level of hours.

Introduction

Difficulties in regulating emotions are a core feature underlying various mental health problems (Sheppes et al., 2015). Depression, for instance, is characterized by high levels of daily negative affect, including sadness, depressed mood, feelings of hopelessness and worthlessness, excessive or inappropriate guilt, and diminished pleasure in activities (American Psychiatric Association, 2013). Such affective experiences are commonly conceptualized along two distinct but related dimensions: positive and negative affect (Watson & Clark, 1994). This distinction has been incorporated in various theoretical frameworks, including the Tripartite Model of Anxiety and Depression (Clark & Watson, 1991) and Research Domain Criteria (RDOC), which considers affective valence systems as transdiagnostic markers of psychopathology (Insel et al., 2010).

Positive and negative affect are not static; rather, they fluctuate constantly, varying across hours and days (Golder & Macy, 2011). With the rise of smartphone-based ecological momentary assessment (EMA), researchers have been able to capture these fluctuations and investigate their links with symptoms of psychopathology (Myin-Germeys et al., 2009; van de Leemput et al., 2014). No longer limited to static properties of emotions, such as intensity or valence, we can characterize processes that unfold over time, and identify patterns, or certain dynamics that are linked to psychopathology. Within this field, two key metrics are commonly used to quantify affect fluctuations: variability and inertia (Koval et al., 2013). Variability quantifies the level of change in affect states over time, with higher variability indicating a greater range in emotional states. Inertia describes the extent to which affect persists over time, with higher levels of inertia indicating greater stability in emotions, meaning that an individual's current affect is strongly predicted by their previous affective state. Greater affect variability has been linked to poorer well-being (Houben et al., 2015), while heightened inertia of negative affect has been specifically associated with symptoms of depression (Koval et al., 2013; Kuppens et al., 2012).

Despite extensive research on the relationship between these affect state characteristics and symptoms of psychopathology, the cognitive mechanisms underlying these affect fluctuations remain unclear. Executive functions (EF), including inhibitory control, cognitive flexibility, or shifting, and working memory (Miyake et al., 2000), have been linked to the ability to regulate positive and negative affect states. Different EF functions are highly interconnected (Diamond, 2013; Ferguson, 2022) and share common neural substrates, primarily within the prefrontal and cingulate cortex (Salehinejad et al., 2021). Impairments in EFs may hamper adaptive affect regulation and represent transdiagnostic markers of psychopathology (McTeague et al., 2016). While EF impairments can be assessed through performance metrics on behavioral tasks, complementary self-report measures (Snyder et al., 2021) provide valuable insight into how individuals experience EF difficulties in their daily lives. One such measure, the Webexec measure of problems with EF (Buchanan et al., 2010), assesses self-perceived difficulties with EF, including attentional control, impulsivity, and difficulties disengaging from repetitive thoughts. Relatively poor self-reported EF has been associated with a range of maladaptive problem behaviors (Freichel, Christensen, et al.,

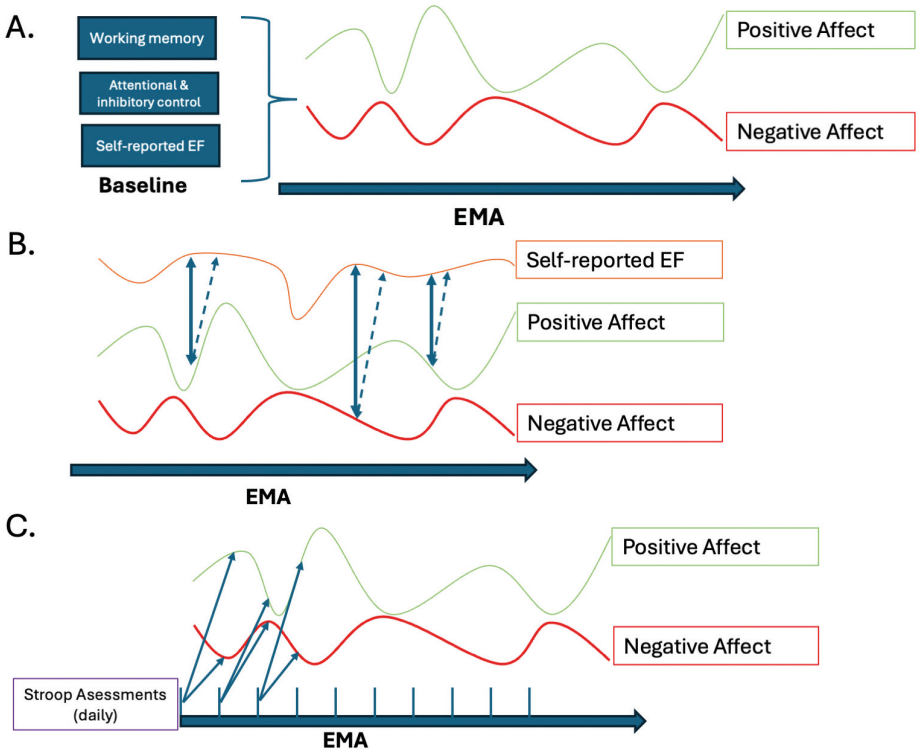
2024). However, despite extensive evidence that EF deficits are transdiagnostic markers of psychopathology, their role in daily positive and negative affect dynamics remains unclear. Moreover, it remains an important question what type of EF measure (behavioral vs. self-report) is most predictive of relevant outcomes observed in everyday life.

Most studies examined associations between trait-level EF, typically assessed at a single point, and their association with long-term outcomes, such as changes in affect or symptom severity (Bardeen et al., 2022; Freichel, Pfirrmann, et al., 2024). However, EF may also operate on much shorter time scales, ranging from hours to weeks, and it may also determine properties other than static outcomes, such as variability and inertia. Additionally, some evidence suggests that EF performance itself varies within and across days (Bennett et al., 2008), including aspects such as working memory capacity (Neubauer et al., 2019). Evidence that EF has a state-like component, in addition to its well-established trait-like qualities, opens new avenues for understanding its role in psychopathology. Rather than focusing solely on individual differences in EF, we can examine fluctuations within the same person over time and explore whether periods of reduced EF are also marked by increased vulnerability to psychological difficulties (e.g., as reflected in persistent negative affect or difficulties in disengaging from repetitive negative thinking).

To address these gaps, the present study employs a multi-method EMA approach that integrates performance-based, self-reported, and momentary assessments of EF. Our study aimed to examine three key questions (see Figure 1): (1) regarding EF as a trait, how do different aspects of EF - specifically, working memory, inhibitory control, and self-reported problems with EF, assessed at baseline, predict daily dynamics in affect (i.e., variability and inertia)? (2) How are levels of perceived problems with EF associated with positive and negative affect, both within the same time window and also several hours later? (3) What is the relationship between daily EF performance (measured via the Stroop task) and daily positive and negative affect?

Figure 1

Visual Representation of the Three Study Aims Examining The Association Between EF and Affect



Note. EF = executive functions. The three panels refer to specific research questions: (1) How do baseline EF measures (working memory, inhibitory control, and self-reported EF problems) predict affect dynamics (variability and inertia) across the EMA period? (2) How do self-reported EF problems (i.e., attentional control, impulsivity, and difficulty disengaging from repetitive thoughts) and momentary positive and negative affect predict each other across time (hours, dashed lines) and within the same time window (solid lines)? (3) How does a measure of attentional control (Stroop Task) predict next-day positive and negative affect?

Methods

Participants and Procedure

51 participants were recruited through the University of Amsterdam (UvA) Behavioral Science Lab subject pool. Participants were eligible for the study if they were between 18 and 60 years old and proficient in English. They were excluded from the study if they failed to complete the pre-test or start with the EMA assessments. After obtaining informed consent, participants attended a 10–15-minute meeting, either in person or online, with a researcher to review the study procedures. During this meeting, participants were instructed to download and set up the Avicenna EMA platform (https://avicennaresearch.com). The study consisted of three phases: a pre-test (one hour), an experience sampling (EMA) period (cumulative four hours in total across all days), and a post-test (one hour), amounting to a total study duration

of six hours. The pre- and post-tests consisted of multiple self-report clinical assessments and two cognitive tasks: the go/no-go task and the backward digit span task. All self-report assessments were conducted online via Qualtrics (Qualtrics, 2020) , while cognitive tasks were administered online using Inquisit (Inquisit, 2023). Participants could select the time of day to complete the pre- and post-tests. The order of the go/no-go and backward digit span tasks was randomized. Starting the day after the pre-test, participants received four daily notifications via the Ethica app for a duration of 15 days, with Sundays excluded.

Participants received course credits based on their compliance rate. For the EMA phase, participants earned two credits for a compliance rate above 50%, three credits for a compliance rate above 75%, and four credits for a compliance rate exceeding 95%. The study was approved by the ethics committee at the Department of Psychology at UvA (ethics IRB number: 2023-DP-15935).

Measures

Measures at Pre- and Post-test

Backward Digit Span Task. Working memory was assessed using the visual digit span test (backward only; Woods et al., 2011). Participants were shown a sequence of numbers, beginning with two digits, and were instructed to mentally retain the presented digits and avoid using external aids or strategies to enhance their performance. Participants were asked to recall and enter the numbers in reverse order. If they provided the correct response, the subsequent sequence increased by one digit; if they made two consecutive errors, the following sequence was shortened by one digit. The task ended after 14 trials. During the initial practice phase, participants completed between two and eight practice trials, continuing until they provided a correct response. During the practice phase, participants received on-screen feedback after each correct response. If they did not provide a correct response within the allowed number of practice trials, the task was terminated. The numbers were presented for 1s one after another. Participants were instructed to provide their answer by selecting the digits from a circle of digits. The estimated test-retest reliability of the mean digit span based on the pre- and post-test measures was acceptable ($r = .64$, $p < .001$).

Go/no-go Task. Inhibitory control and inattention were assessed using a Go/No-Go task (adapted version of Fillmore et al., 2006). Participants were shown green ('go-signal') or blue ('no-go-signal') rectangles in either vertical or horizontal orientation on the screen. When a go-signal appeared, participants were instructed to press the spacebar as quickly as possible. When a no-go signal appeared, participants were instructed to refrain from pressing the space bar. We primarily examined the percentage of commission errors (incorrect response during no-go trials), omission errors (no response during go-trials), and the reaction times during successful go trials. More commission errors indicate poorer inhibitory control, while omission errors may reflect inattention/lapses in attention (Meule, 2017). The test-retest reliability estimates for omission and commission errors were acceptable (commission errors: $r = .59$, $p < .01$; omission errors: $r = .37$, $p = 0.07$).

EMA Measures

Our EMA study included a morning survey (at 9:00 AM), followed by four mood surveys (at 10:00 AM, 1:00 PM, 4:00 PM, 7:00 PM), and an evening survey (at 9:00 PM) for 15 days. Participants were able to complete the surveys within a 30-minute time window. The morning survey included a brief Stroop task (see description below) and several items assessing sleep quality and substance use on the previous day. The evening survey assessed participants' overall daily satisfactions and expectations for the following day.

Stroop Task. The Stroop color word test (Stroop task) included congruent (text and font color match), incongruent (different text and font color), and neutral stimuli (a row of hashtags). This mobile Stroop task has been used in prior work with EMA designs (Gignac et al., 2022). Before starting the main sessions, participants completed three practice rounds. The task lasted 60 seconds and included the colors red, blue, green, and yellow. The number of trials was not fixed, but the task always concluded after 60 seconds. Participants were instructed to indicate whether the two colors (text and font) are identical by choosing 'Yes' or 'No'. During the task, participants received visual and audio feedback for correct and incorrect responses. A timer indicating the remaining time was shown on the screen. The screen also displayed the number of correct answers they have provided. We computed a measure of mean response accuracy that indicates the average proportion of correct responses. Higher levels of response accuracy indicated better attentional control. We also computed the within-person variance of response accuracy, indicating the variability. Higher levels of within-person accuracy variance indicated less stable attentional control.

Positive and Negative Affect. We assessed participants' mood four times per day using selected items adapted from the Positive and Negative Affect Schedule - Expanded Version (PANAS-X; Watson & Clark, 1994). Participants responded to the items on a scale from 1 (very slightly or not at all) to 5 (extremely). At each beep, we computed a sum score of negative affect based on negative affect items (sad; nervous; guilty; afraid; tired; hopeless; anxious; annoyed; restless). We also computed a sum score for positive affect at each beep based on the respective positive affect items (happy; calm; energetic; concentrated; determined).

Self-reported Problems with Executive Functions. To assess self-reported EF problems, we used the Webexec (Buchanan et al., 2010) measure of executive functioning problems. This self-report has been developed for online use and includes six items assessing distinct EF problems. The items are scored on a four-point scale (1 = No problems experienced, 2 = A few problems experienced, 3 = More than a few problems experienced, 4 = A great many problems experienced). The measure has been developed for online use and has been linked to broad-band symptom domains (Freichel, Christensen, et al., 2024). We computed a total score (range 6-24), with higher scores reflecting greater difficulties in EF. During the pre-test, we included the measure in its original form. For the EMA assessment, we developed three items that were adapted from the Webex measure. The selected items included "I get stuck on certain issues and can't move on" (difficulty with disengagement from repetitive thoughts or low cognitive flexibility), "When I want to deliberately concentrate on something, I am capable of ignoring environmental distractions" (attentional control), and "I find myself acting on impulses" (impulsivity). These three items were selected given that

they reflect key EF components. Participants responded to these items on a seven-point scale ranging from “Strongly disagree” to “Strongly agree”.

Statistical Analysis

Affect fluctuations

We obtained two measures of affect dynamics: (1) Affect variability: We computed within-person affect variance separately for positive and negative affect, which captures the level of affect fluctuations in an individual’s time-series. It is a widely used measure of affect variability (Schoevers et al., 2021), and it does not take the temporal dependency of the assessments into account. Higher within-person variances indicate greater affect fluctuations. (2) Inertia: We followed the procedure outlined by Hawes and Klein (2024) and computed person-mean centered lagged values of positive and negative affect. We then estimated multi-level models (with intercept and autoregressive terms as random effects) predicting positive and negative affect values based on their respective lagged values while ignoring overnight lags. Inertia was defined as the resultant autoregressive estimates derived from the multi-level models. Higher levels of inertia indicate that individuals’ level of negative or positive affect states carry over to the next assessment point. The lme4 package (Bates et al., 2015) was used for multi-level model estimation. We used linear regression models to examine associations between affect dynamics (variability and inertia) and the baseline EF measures. For each affect dynamic (separately for positive and negative affect), we included all EF measures (working memory, inhibitory control, and the sum score of self-reported problems EF) as predictors within the same model.

Network estimation

All analyses were conducted in R (version 4.4.0; R Core Team, 2024). We estimated a multi-level vector-autoregression network model (mlVAR), using the mlVAR (Epskamp et al., 2018) R package for network estimation, and the qgraph (Epskamp et al., 2012) R package for network visualization. This approach allowed us to estimate temporal, contemporaneous, and between-person networks. The temporal network indicates the average within-person autoregressive (i.e., node predicting itself) and cross-lagged temporal lag-1 associations (i.e., node A predicting node B over time). Across individuals, the contemporaneous network describes instantaneous associations within the same time window after accounting for the temporal associations. The between-person network captures individual differences, representing trait-like associations between participants’ means across the study period. Prior to network estimation, we detrended the time-series to remove a cumulative linear trend across the study period. To do so, we fitted separate regression models for each variable using a consecutive event number as the predictor and removed the estimated linear trend from the observed values. This procedure is commonly applied in stationary VAR-models given their focus on short-term fluctuations rather than overall trends (Ebrahimi et al., 2021). To facilitate interpretation, we used the layout (i.e., node positioning) of the temporal network structure for all other network structures.

Analyses of Stroop Performance and Affect

Next, we examined the associations between a daily measure of attentional control (Stroop task performance accuracy) and affect fluctuations. We first aggregated positive and negative affect scores at the daily level by computing participants' average positive and negative affect scores across all beeps within each day. We centered participants' Stroop accuracy, positive, and negative affect scores around participants' own mean to be able to examine daily fluctuations. We fitted four multi-level models to examine temporal lag-1 associations using the within-person centered predictor variables. Specifically, we tested whether (1) Stroop accuracy predicted next-day positive affect, and (2) Stroop accuracy predicted next-day negative affect. In an exploratory fashion, we also examined whether (3) positive affect predicted next-day Stroop accuracy, and (4) negative affect predicted next-day Stroop accuracy. We included random intercepts for participants to capture individual differences in overall affect levels and Stroop performance.

Results

Sample Characteristics

The final sample comprised 46 participants, with a mean age of 20.81 years (SD = 2.16), of whom 73.81% were female (26.19% male). Participants completed 2048 unique EMA responses, with an average of 44.52 responses per person (SD = 9.79). Participants completed on average 14.48 days (SD = 0.86), with an average of 3.06 responses (SD = 0.58) per day. The compliance rate based on four prompts was 76.96%. There was significantly more missingness for the daily Stroop Task assessment: Participants on average completed the Stroop Task on 7.74 days (SD = 4.37), with a compliance rate of 51.36%.

Table 1 presents descriptive statistics for relevant pre-test and EMA measures. There was substantial variability in the reported positive and negative affect and self-reported EF measures. Participants showed high response accuracies in the daily Stroop task. At the pre-test, participants showed relatively high levels of working memory (average digit span = 7.1) and attentional/inhibitory control as indicated through omission and commission rates.

Table 1

Descriptive Information on Pre-test and EMA Measures

Variable	Mean	SD
Positive Affect	9.33	2.88
Negative Affect	13.04	5.22
Webexec Total Score	13.49	3.67
Stroop Accuracy	0.95	0.04
Digit Span Length	7.1	1.36
Go/No-Go Omission Rate	0.04	0.06
Go/No-Go Commission rate	0.03	0.03

The Association Between Baseline EF and Affect Dynamics

The first goal of our study was to examine the association between different cognitive measures assessed at the pre-test and positive and negative affect dynamics (inertia and variability). See Table 2 for all relevant estimates from the separate regression models. For positive affect variability and negative affect inertia, none of the cognitive predictor variables were significant (all p > .05). However, for negative affect variability, a higher Go/No-Go omission rate was significantly, but weakly associated with greater variability (p = .021). No significant associations emerged for self-reported problems with EF, working memory, or Go/No-Go commission errors in predicting negative affect variability (all p > .05). For positive affect inertia, a higher Go/No-Go omission rate was significantly associated with lower inertia (p = .008). This indicates that participants with more omission errors showed less stability in their positive affect over time. No other EF measures predicted positive affect inertia.

Table 2

Regression Models Predicting Affect Variability and Inertia

Outcome	Predictor	Estimate	SE	t	p
Positive Affect Variability	Webexec Total Score	2.5	3.837	0.652	0.519
	Digit Span Length	0.886	3.679	0.241	0.811
	Go/No-Go Omission Rate	-0.49	3.763	-0.13	0.897
	Go/No-Go Commission rate	-1.777	3.659	-0.486	0.63
Negative Affect Variability	Webexec Total Score	11.067	7.481	1.479	0.148
	Digit Span Length	8.599	7.174	1.199	0.239
	Go/No-Go Omission Rate	17.804	7.338	2.426	**0.021**
	Go/No-Go Commission rate	-6.474	7.135	-0.907	0.37
Positive Affect Inertia	Webexec Total Score	-0.019	0.012	-1.59	0.121
	Digit Span Length	-0.007	0.012	-0.593	0.557
	Go/No-Go Omission Rate	-0.033	0.012	-2.818	**0.008**
	Go/No-Go Commission rate	0.001	0.012	0.083	0.935
Negative Affect Inertia	Webexec Total Score	-0.002	0.009	-0.208	0.837
	Digit Span Length	-0.001	0.009	-0.068	0.946
	Go/No-Go Omission Rate	-0.016	0.009	-1.859	0.072
	Go/No-Go Commission rate	<0.001	0.009	-0.008	0.994

Note. SE = standard error; t = t-statistic; p = p-value. Significant p-values (p < 0.05) are highlighted in bold. WebExec score = self-reported executive function problems. Variability refers to the within-person variability. The omission and commission rates were based on the Go/No-Go task. All predictors were standardized prior to analysis.

The Role of Self-Reported EF Problems in Daily Positive and Negative Affect Dynamics

When examining cross-sectional correlations between baseline EF measures and individuals' average levels of the respective EMA item measures, we found no significant associations between Go/No-Go measures (omission/commission errors) and the respective self-reported EF items. However, surprisingly, a higher digit span length was associated with a higher score of the item concerning difficulty disengaging ($r=0.40$, $p=0.01$). The second goal of our study was to examine the role of self-reported problems with EF (attentional control, impulsivity, and difficulty disengaging from repetitive thoughts) in daily positive and negative affect dynamics.

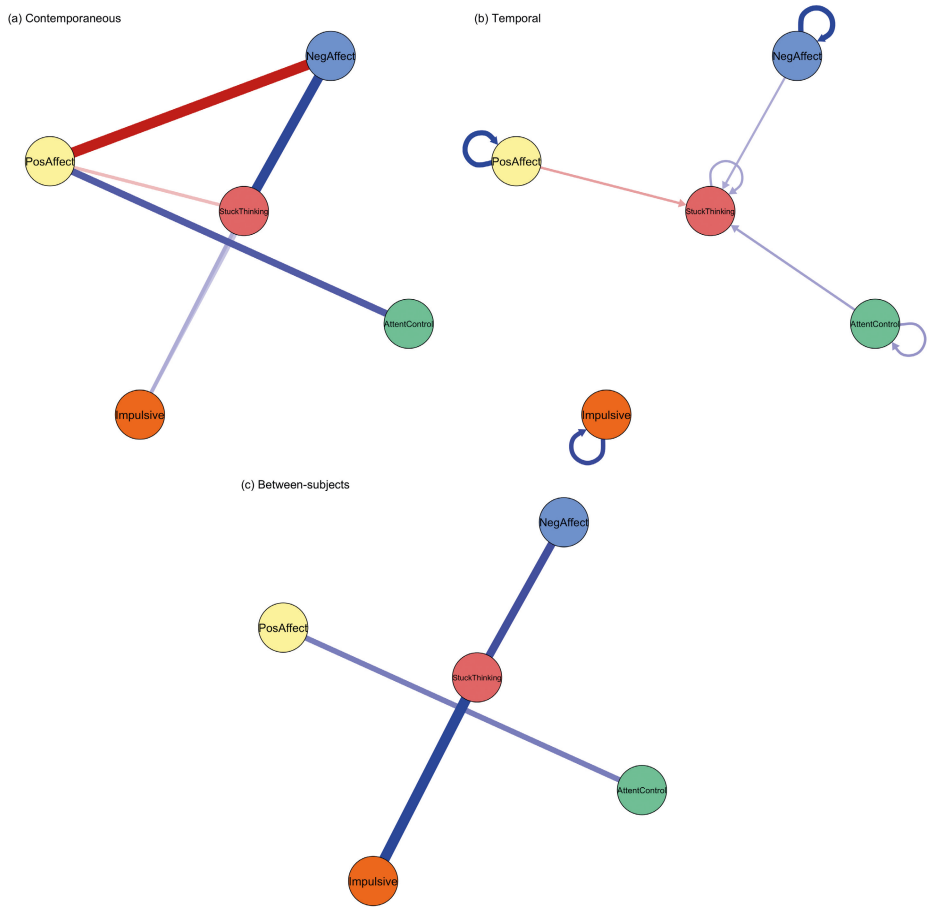
The contemporaneous network is shown in Figure 2, panel A. Within the same time window, better attentional control co-occurred with more positive affect. Impulsivity was associated with more negative affect and more problems disengaging from repetitive thoughts. Moreover, difficulties disengaging from repetitive thoughts was related to higher negative affect and lower positive affect within the same time window.

When examining within-person temporal associations (see Figure 2, panel B), we found a sparse temporal network. More negative affect and less positive affect predicted more difficulties disengaging from repetitive thoughts. Better attentional control also predicted more difficulties disengaging from repetitive thoughts over time.

The undirected between-person network (Figure 2, panel C) shows trait-like time-invariant associations across the sample. Individuals who on average reported more negative affect also reported more difficulties disengaging from repetitive thoughts compared to other participants. Across the sample, higher impulsivity was linked to increased difficulty in disengaging from repetitive thoughts. Similarly, there was a positive association between positive affect and attentional control, indicating that individuals with better attentional control also appeared to report more positive affect.

Figure 2

Network Models of Positive Affect, Negative Affect, and Self-Reported EF Problems



Note. (a) Contemporaneous network: associations within the same time window; (b) Temporal network: temporal lag-1 associations across time; (c) Between-subjects network: associations between participants' average level of different variables. PosAffect = positive affect; NegAffect = negative affect; StuckThinking = difficulty disengaging from repetitive thoughts; AttentControl = ability to focus despite distractions; Impulsive = impulsivity. The thickness and color saturation of the edges indicate the magnitude of associations (positive = blue, negative = red).

The Association Between Daily Attentional Control and Positive/Negative Affect

The third goal of our study was to explore how a daily direct measure of attentional control (smartphone-based Stroop task) was associated with positive and negative affect. Our multi-level analyses examining lag-1 temporal associations did not reveal any significant associations between Stroop accuracy and positive/negative affect ($p > 0.05$). When examining aggregate correlations, we found a significant association between the within-person Stroop accuracy variability and variability in negative affect ($r = .47$, $p < 0.05$). Greater

fluctuations in attentional control were associated with greater fluctuations in negative affect. The average Stroop accuracy was not significantly associated with positive or negative affect variability or inertia ($p > 0.05$).

Discussion

The present study aimed to study the role of EF in mood dynamics from three distinct perspectives: First, we investigated how baseline trait-like measures of EF, including working memory, inhibitory control, and self-reported EF difficulties, predict fluctuations in daily positive and negative affect. Second, we examined the temporal and contemporaneous associations between momentary measures of self-reported EF problem and positive and negative affect. Third, we assessed whether daily task-based measures of inhibitory-attentional control predict daily positive/negative affect.

Altogether, this multi-method pilot study found no evidence for the utility of behavioral measures of attentional control (mobile Stroop task) in predicting positive/negative affect. Similarly, at a lag of three hours, self-reported EF measures also did not predict affect. As shown by Snyder et al. (2021), self-report and behavioral measures assess largely distinct dimensions of executive functioning with different predictive utility. Self-report measures are easy to administer, efficient, and capture individuals' perceived levels of EF, but they are also prone to various biases related to self-evaluation (e.g., social desirability, self-perception). In contrast, behavioral measures require increased effort to administer, capture more automatic response processes, and are vulnerable to other cognitive biases and practice effects (Hohl & Dolcos, 2024). This study showed that neither approach predicted short-term affect measures; however, task-based measures at pre-test predicted aggregate affect dynamics.

Attentional lapses (omission rate) predict mood dynamics

Our study showed links between attentional lapses (higher omission rates in the Go/No-Go task) and greater negative affect variability. This broadly fits with models of emotion regulation that view attentional and cognitive control as a top-down system for regulating emotions (Shomstein, 2012). Our study is one of the first to show such links between inattention (attentional lapses) at baseline and repeatedly assessed negative affect at the daily level. Unable to downregulate negative emotions when they emerge, individuals may find that their emotions intensify more suddenly and drastically, amounting to a pattern of instability. This finding may indicate that a lower ability to exert control over attention may make it more difficult to disengage from negative thoughts (Yip et al., 2023), thus contributing to the instability of negative affect. There is also evidence for interventions targeting attentional control (e.g., mindfulness or cognitive bias modification) as a coping strategy for dealing with negative emotions (Li et al., 2023).

Dynamic interplay between self-reported EF problems and affect

Our VAR-network models indicated that self-reported EF problems co-occur with more negative and less positive affect within the same time window. Better attentional control

(specifically the ability to ignore environmental distractions) co-occurred with more positive affect. Similarly, greater difficulty disengaging from repetitive thoughts was associated with less positive and more negative affect. These links fit with a broader literature on the benefits of emotion regulation skills (including distraction, Brans et al., 2013; Wante et al., 2018) and mindfulness (Hill & Updegraff, 2012) for regulating affective states. In line with our findings, a recent experimental study showed that the association between emotion regulation abilities and positive affect was mediated by increased attention to happy faces (Suslow et al., 2022). Moreover, we found that higher levels of impulsiveness (specifically acting on impulses) also co-occurred with greater negative affect and more difficulty disengaging from repetitive thoughts. This fits with prominent theoretical models, such as the UPPS-P model (Cyders et al., 2007) that conceptualizes negative urgency, the tendency to act rashly when experiencing negative mood, as a unique facet of impulsivity.

At the temporal level, we found directed links from affect towards difficulty disengaging from repetitive thoughts. Contrary to our expectations, the reverse direction was not present. Positive affect predicted better disengagement from repetitive thoughts, while negative affect was associated with more difficulty letting go of them. Prior work has shown bidirectional associations between rumination, a process closely related to difficulties disengaging from repetitive thoughts or lack of cognitive flexibility, and depressive symptoms over longer time periods (Whisman et al., 2020). Thus, the nature of the association between negative affect and disengagement from repetitive thoughts may change depending on the time scale. At the present three-hour lag, negative affect may be too persistent to disengage from, while positive affect could represent a source of distraction that could be used to shift attention.

No association between daily attentional-inhibitory control and affect dynamics.
We showed no significant association between daily Stroop task performance and positive/negative affect. There may be multiple explanations accounting for the lack of associations: First, the performance on the smartphone-based Stroop task may be influenced by environmental distractions, learning effects, lack of engagement, and other sources of measurement error, and it may thus not represent a reliable indicator of inhibitory-attentional control. Second, participants showed high levels of overall accuracy (M = 0.95, SD = 0.04), indicating a ceiling effect that limited the variability in performance. Other metrics, such as reaction time or reaction time variability, may be more informative indicators in this sample. Lastly, it is possible that changes in attentional-inhibitory control may have cumulative long-term effects on positive/negative affect over extended periods, and thus our analyses examining short time-scales may not have been able to detect such effects.

Limitations
There are several limitations of our study. First, our analytical sample (n = 46) was relatively small, and while the EMA data provided sufficient power for estimating the multi-level VAR model, other analyses of associations between baseline EF and affect dynamics may have been underpowered. Our sample, consisting of undergraduate students, showed high levels of task accuracy. Replicating these effects in general population or subclinical samples would

be necessary to better judge the predictive utility of EF measures. Second, the smartphone-based Stroop task assessment, though used in prior studies (Gignac et al., 2022), may not be as reliable as laboratory-based administration. Third, there was only little variance (ceiling effect) in the Stroop performance, which may have limited our ability to identify associations with affect measures. Fourth, the EMA items used for assessing self-reported EF problems were adapted from an established self-report measure but have not been formally validated. Lastly, our analyses of temporal dynamics did not consider relevant contextual factors, such as sleep, stress, substance use, history of psychopathology or psychotropic medication use, or other environmental factors, which may have influenced affect states and self-reported EF problems.

Concluding comments

This multi-method EMA study explored multiple avenues for linking EF (both behavioral measures and self-reported) with positive and negative affect. We found that inattention, as indexed by higher omission rates at the go/no-go task during pre-test (before the EMA period), was associated with greater variability in negative affect and lower inertia of positive affect. Daily Stroop performance was unrelated to next-day positive and negative affect. Multi-level VAR models indicated that concurrently, affect regulation is related to self-reported EF, but at the temporal level, affect also appears to modulate EF. This proof-of-principle pilot study illustrated complementary perspectives to link baseline, hourly self-reported, and daily EF measures. to affect regulation. Future research should adapt these perspectives to evaluate the predictive utility of EF in predicting affect regulation in sub-clinical and clinical populations.

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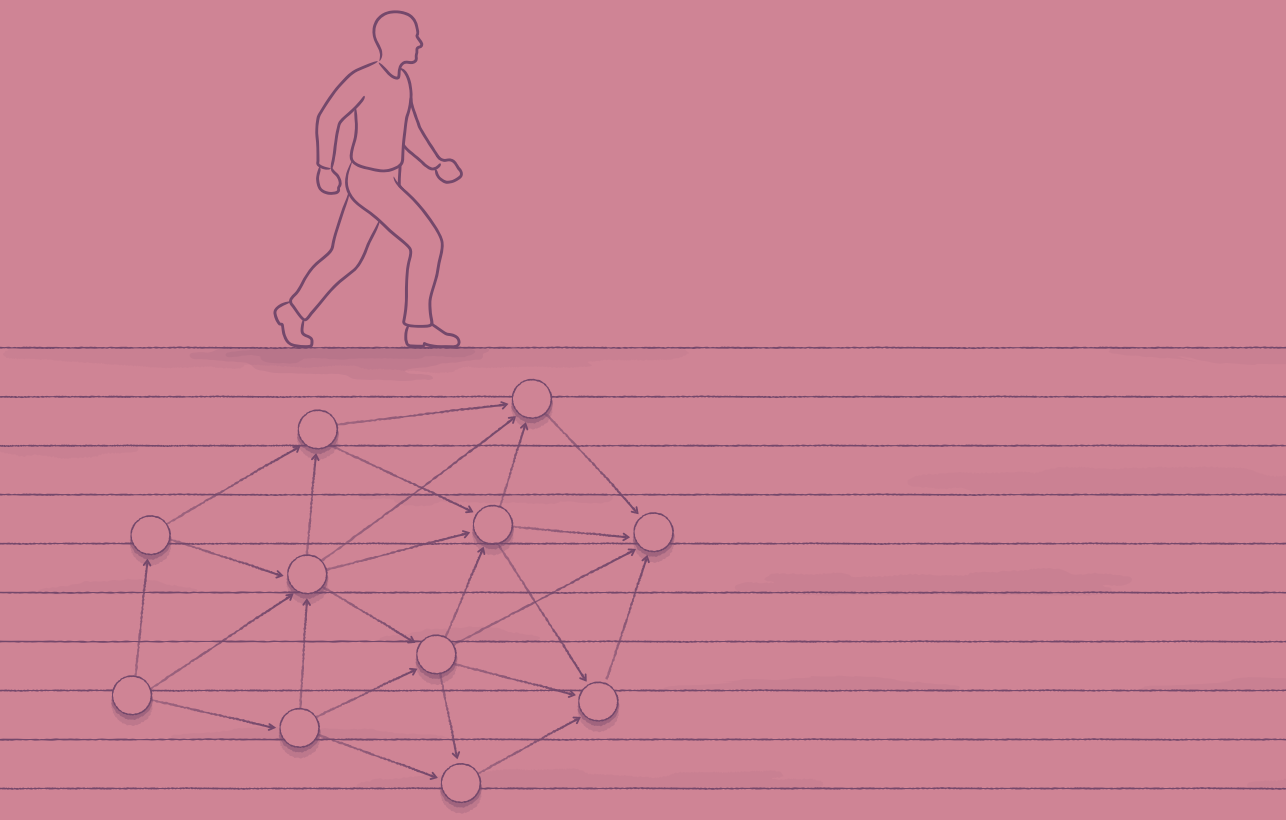


CHAPTER 7

Value-modulated attentional capture in reward and punishment contexts, attentional control, and their relationship with psychopathology

This chapter is adapted from:
Freichel, R., Mrkonja, L., de Jong, P. J., Cousijn, J., Franken, I., Ruiter, T. A., Le Pelley, M., Albertella, L., Watson, P., Veer, I. M., & Wiers, R. W. (2023). Value-modulated attentional capture in reward and punishment contexts, attentional control, and their relationship with psychopathology. Journal of Experimental Psychopathology, 14(4), 20438087231204166. https://doi.org/10.1177/20438087231204166

Abstract

Attentional bias towards rewards has been extensively studied in both healthy and clinical populations. Several studies have shown an association between reward value-modulated attentional capture (VMAC) and greater substance use. However, less is known about the association between these VMAC effects and internalizing symptoms. Moreover, while VMAC effects have also been found in punishment contexts, the association between punishment VMAC and psychopathology has not been studied so far. In the present two-part preregistered study, we adapted a novel VMAC task to also include a punishment context and examined associations with internalizing symptoms and substance use. Our results showed consistent VMAC effects in reward contexts across two separate studies. Attentional capture was stronger for distractors associated with high rewards than for low rewards. We replicated and extended previous findings by showing such VMAC effects in a substantially shorter task that also included alternating punishment blocks. Contrary to our expectations, we found no VMAC effects in punishment contexts and no direct associations between VMAC and symptom measures. Our results speak to the feasibility of assessing VMAC effects using a scalable and short behavioral online task, but the relationship with the development of internalizing and externalizing psychopathology remains uncertain.

Keywords: psychopathology, punishment, reward, value-modulated attentional capture

Introduction

It has been well-documented that our attention can be automatically directed towards stimuli that have been associated with positive or negative outcomes (Watson et al., 2019; Wentura et al., 2014). Individuals are more likely to look at stimuli predicting higher rewards, compared to neutral or low-reward stimuli, even when such stimuli are completely irrelevant to the current task (Anderson et al., 2011), or when doing so is counterproductive and results in a monetary loss (Le Pelley et al., 2015). Similarly, threat or punishment-related stimuli capture our attention even when attending to them results in an unpleasant electric shock (Anderson & Britton, 2020; Mikhael et al., 2021; Schmidt et al., 2015), monetary loss, or loud noise. This automatic attentional bias towards distractors signaling high rewards or punishments is known as value-modulate attentional capture (VMAC). Little is known about the test-retest reliability of these VMAC tasks and their utility for studying punishment-related attentional capture.

Such capture effects are argued to be evolutionary adaptive processes that ensure potential threats or rewards are quickly detected in order to be avoided or approached. However, substantial evidence also shows that attentional biases for rewards and punishments can become maladaptive and have been associated with psychopathology (Anderson, 2021). For example, individuals with a history of substance use problems often show an attentional bias towards substance-related stimuli (Field et al., 2016; Wiers et al., 2023). On the other hand, studies reported that individuals with moderate to severe depressive symptoms show no such reward-driven attentional capture (Anderson et al., 2014, 2017). Greater value-modulated attentional capture has been associated with the severity of addictive and obsessive-compulsive behaviors (Albertella, Chamberlain, et al., 2020; Albertella, Le Pelley, et al., 2019, 2020; Anderson et al., 2013). This effect of reward on attentional capture may be particularly persistent in individuals with alcohol use disorder (Albertella, Watson, et al., 2019): a higher persistence of learned attentional capture following reversal of stimulus-reward contingencies predicted risky patterns of alcohol use. In other words, individuals who were quicker and better able to adapt to the changed reward contingencies, were less likely to exhibit risky alcohol use.

Individual differences in cognitive control may explain the propensity for these automatic attentional capture effects. For instance, Albertella and colleagues (2017) showed that VMAC is associated with illicit substance use only among individuals with low cognitive control (Albertella et al., 2017). Similarly, a study by Houben and Wiers (2009) showed that stronger implicit associations between alcohol and positive affect predicted increased alcohol use and alcohol-related problems only in individuals with low response inhibition. This interaction between cognitive control and attentional capture is in line with dual-process theories (Gladwin et al., 2011) that conceptualize the competition of automatic and reflective processes in the development of addictive behaviors.

Most studies on the association between VMAC and psychopathology have focused on addictive behaviors, and little is known about the transdiagnostic value of attentional capture. Specifically, the link between attentional capture and internalizing symptoms or general distress is not yet fully understood. Various studies have emphasized the importance

of decreased sensitivity to reward in depression, especially in individuals exhibiting anhedonia (Pizzagalli, 2014; Zald & Treadway, 2017), but to date, only a handful of them (Anderson et al., 2014, 2017) have studied blunted reward processing specifically in terms of value-driven attentional capture. Additionally, hypersensitivity to negative stimuli has been associated with depression, such that individuals with depression have difficulties in shifting attention away from negative stimuli (Gotlib & Joormann, 2010; Grahek et al., 2018). Similarly, this hypersensitivity to punishment has been linked to anxiety disorders (Bar-Haim et al., 2007). Other studies using the spatial orienting task have found no evidence for cross-sectional or temporal associations between attentional bias for cues signaling reward or punishment and anxiety or behavioral problems (Kreuze et al., 2020, 2022).

While research on VMAC and anxiety in the punishment context is still lacking, Kim & Anderson (2020) have recently shown that threat-induced anxiety (through electrical stimulation) reduces reward-related attentional capture in a healthy population. Nonetheless, similarly to reward-related attention, little research has been done to assess the link between punishment-related attentional capture and anxiety and depressive symptoms.

Individual differences in attentional control as a clinical assessment tool for psychopathology would only be useful if tasks measuring such attentional control or biases show to have high test-retest reliability. So far, to the best of our knowledge, no studies have explored the reliability of the VMAC task as used in the current study. However, a notable exception by Anderson & Kim (2019) has shown that a similar version of the VMAC task has in fact very low test-retest reliability when using RT measures. In this paper, we aim to shed light on the reliability of the novel VMAC task.

Understanding the reward and punishment processing specifically in the context of VMAC is important considering its clinical implications with respect to attentional biases in different mental health conditions (e.g., addiction) but also its theoretical importance as it may represent a direct test of valence (reward/punishment) processing at a low and automatic level.

The present preregistered study aimed to investigate the association between reward- and punishment-related attentional capture, general cognitive control, and substance use and internalizing symptoms. First, we aimed to replicate the VMAC reward effects found in Le Pelley et al. (2015) and Albertella et al. (Albertella, Watson, et al., 2019). We extended these studies by (1) testing a novel punishment variation and (2) assessing the test-retest reliability of the VMAC task, and (3) investigating associations between VMAC and internalizing symptoms and substance use. In an exploratory (non-preregistered) fashion following the approach by Albertella et al. (2017), we aimed to investigate if general cognitive control, as assessed by a Stroop Deadline Task, would moderate the relationship between VMAC and substance use.

Methods

The present paper consists of two separate studies that follow a similar procedure and design. In Study 1, our goal was to examine value-modulated attentional capture effects in both reward and punishment contexts in a student population. We aimed to replicate these VMAC effects in Study 2 in which the task contained more blocks and the condition (reward/punishment) of the first block was randomized across participants. We preregistered the study design, variable selection, and analytical strategy before data collection for both studies. Complete results of the preregistered analyses that are not reported below can be found in the Supplementary Materials. The preregistrations can be accessed via the Open Science Framework (Study 1: https://tinyurl.com/7wbyhky6; Study 2: https://tinyurl.com/yckczxjr).

Study 1

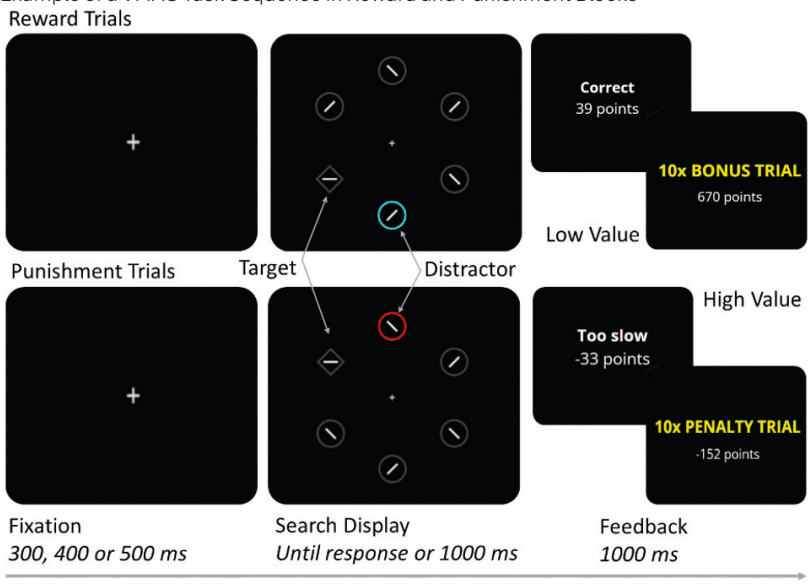
Participants. Eighty-four undergraduate psychology students were recruited from the University of Amsterdam in exchange for course credits. Participants also had the opportunity to enter a raffle for an additional 50 EUR voucher based on their performance on the VMAC task. Participants were informed they would receive one raffle ticket for every 250 points earned, therefore increasing their chances of winning a voucher with better performance. All participants reported normal or corrected-to-normal vision, normal color vision, and fluency in English. The assessments (in English) were conducted online twice, two weeks apart, in a full within-subject design. The study was approved by the Ethics Committee of the Psychology Department of the University of Amsterdam (2022-DP-14766; 2022-COP-15790). The final sample size (n = 84) was smaller than the originally planned and preregistered target sample size, as student recruitment encountered various difficulties.

Materials

Value-modulated attentional capture task. The VMAC task, based on (Albertella, Watson, et al., 2019), consisted of a short practice block, followed by eight blocks of the actual task. In study 1, the task always started with a reward block, in which participants could gain points, followed by a punishment block in which they would lose points. The rest of the task continued to alternate between the reward and punishment blocks in the same way. All stimuli were generated using jsPsych (de Leeuw, 2015) and presented online on a black background through Pavlovia (https://pavlovia.org/). Each block comprised 30 trials, and each trial began with a fixation cross at the center of the screen (for 300-500ms), followed by the search display (for 1000ms) and feedback (for 1000ms). The search display (see Figure 1) consisted of four gray circles (non-targets), one gray diamond (target), and one colored circle (distractor), arranged evenly around the center of the screen.

Figure 1

Example of a VMAC Task Sequence in Reward and Punishment Blocks



Note. The top panel displays a correct response on a high- or low-value distractor in the reward block. The bottom panel displays an incorrect/slow response on high- or low-value distractor trials in the punishment block. The distractor colors in the figure are exemplary and represent only one of the color pairs. In the actual task, there were four colors in total; two for reward (high and low) and two for punishment (high and low).

Participants were instructed to respond to the direction (vertical or horizontal) of the line inside a diamond while ignoring the colored distractor (all shapes other than the target contained line segments tilted randomly 45° to the left or right). Fast and correct responses to the line inside the target resulted in a greater reward and a smaller loss in the reward and punishment blocks respectively (+ 0.1 point for each millisecond the RT was below 1000ms in reward blocks; -0.1 point per millisecond in punishment blocks). Incorrect or slow responses resulted in zero points in reward, and maximum loss of points in punishment blocks. The distractor on each trial was rendered in one of the four colors; either a high (e.g., cyan), or low (e.g., red) color in reward blocks, and high (e.g., yellow), or low (e.g., purple) color in the punishment blocks. The colors of the distractors were counterbalanced across participants. A high-colored distractor signaled a bonus trial in which 10 times more points could be earned or lost. The location of the shapes, and the orientation of the target line were randomly and evenly counterbalanced across trials. During practice no rewards or punishments were given. Figure 1 depicts an example of a VMAC task trial sequence.

We recorded RT and response accuracy on each trial. Of particular interest was the extent to which distractors interfered with responding to the target as a function of their motivational status (high vs. low value), since this would imply an influence of value on the likelihood that distractors captured participants' attention (i.e., a VMAC effect). Following previous VMAC studies (Albertella, Le Pelley, et al., 2019), the VMAC-Reward score was calculated as the

difference between high reward and low reward RTs, and the VMAC-Punishment score as the difference between high punishment and low punishment RTs. Scores closer to zero indicate little difference in attentional capture between high and low distractors, while scores larger than zero indicate greater attentional capture by high-value distractors.

Stroop Deadline Task. In the Stroop Deadline task (SDL; Burgoyne & Engle, 2020), participants were instructed to respond to the color of a word presented on-screen, and to ignore the meaning of the word. The novel version of this task meant that the response deadline adapted to participants' accuracy; the task got more difficult (i.e., shorter response deadline) with each correct response. Conversely, the task got easier (i.e., longer response deadline) with incorrect responses. The SDL score was calculated as the response deadline of the last (18th) block. Better performance at the end of the task (lower SDL score) indicated better attentional control. Further details on the SDL task procedure can be found elsewhere (Burgoyne & Engle, 2020).

Clinical Measures

Alcohol Use. The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a gold-standard 10-item self-report screener to classify alcohol use and related problems. The total score (0-40) of the AUDIT assessed alcohol use-related problems.

Cannabis Use. The Cannabis Use Disorders Identification Test-Revised (CUDIT-R; (Adamson et al., 2010) is an 8-item self-report screening measure of cannabis use and cannabis-related problems in the past six months. The CUDIT-R is a commonly used measure with good psychometric properties in college students (Schultz et al., 2019). The CUDIT-R total score ranged from 0 to 32, with higher scores representing higher levels of cannabis use-related problems.

Internalizing Problems. The 21-item short-form version of the Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995) consists of three 7-item subscales that assess depression, anxiety, and stress symptoms on a four-point Likert scale (0 = Did not apply to me at all, 3 = Applies to me very much). Scores on each scale ranged from 0 to 21 with higher scores representing greater symptom levels. A recent cross-country investigation of the factor structure and reliability of the DASS-21 provided support for the validity of the DASS-21 as a general indicator of distress (Zanon et al., 2021). Furthermore, DASS-21 scores have previously been associated with addictive behaviors and used to control for psychological distress in studies using the same reward VMAC task paradigm (e.g., Albertella, Chamberlain, et al., 2020; Albertella, Le Pelley, et al., 2019)

Procedure. After consenting to the study and providing demographic information (gender, age, and nationality), participants completed the two cognitive tasks in Study 1 followed by the clinical questionnaires in a single 1-hour session. The order of the two cognitive tasks was randomized across participants, with half of the participants starting with the VMAC task and the other half with the Stroop Deadline Task (SDL). After two weeks, participants were invited to participate again, and they followed the same procedure.

Data Analysis. Eighty-three participants finished the first session of Study 1. Following the specifications in the preregistration, participants with overall accuracy below 65% on

the VMAC task (n = 9) were excluded from all analyses. Several participants (n = 3) were furthermore excluded as they responded in less than 150ms on more than 25% of the VMAC trials, preventing calculation of the mean RTs for some of the blocks. Following Albertella et al. (2019) the first two trials of each block of the VMAC task were discarded and reaction times (RTs) less than 150ms (0.07% of all RTs) were excluded. Analyses of RTs were restricted to correct responses only (81.43% of all RTs). Accuracy and RTs of the VMAC task were analyzed on the rest of the sample (n = 72) separately for reward and for punishment blocks using a 4 × 2 analysis of variance (ANOVA) with Block (1–4) and distractor type (high, low) as factors. We included session (1/2) as an additional factor. Separate regression models that included gender and the VMAC reward/punishment scores as predictors were used to examine associations with clinical measures.

Additionally, participants with accuracy below 70% on the SDL task (n = 3), and those who did not respond correctly to at least two out of three attention check items in the clinical questionnaires (n = 1) were excluded from analyses of associations with clinical measures. A multiple regression with VMAC scores, SDL score, and their interaction as independent variables, and AUDIT/CUDIT sum scores and DASS-21 subscales as dependent variables, was conducted on the rest of the sample (n = 68).

Out of the 72 participants who successfully finished the first session, 44 returned for session 2. The same exclusion criteria applied to the second session; participants were excluded based on VMAC task accuracy (n = 1), SDL accuracy (n = 0), and attention check item (n = 0), leaving a final sample of 43 for the second session. Analyses of accuracy and RTs of the VMAC task of session 2 were analyzed using the same procedures as session 1.

Study 2

Participants. In Study 2, 144 undergraduate psychology students were recruited from the University of Amsterdam in exchange for course credits and the opportunity to win an additional 50 EUR voucher. All participants reported normal or corrected-to-normal vision, normal color vision, and fluency in English. All assessments were conducted online.

Procedure. To replicate and more accurately interpret the findings of Study 1, the procedure of the second study was mostly kept identical to Study 1, but with the following exceptions: First, the number of blocks in the VMAC task was increased from eight to twelve, with half of the blocks being reward and the other half punishment. Second, half of the participants started the VMAC task with a punishment block, and the other half with reward, to test for order effects. Finally, the SDL task and the second assessment session were not included in Study 2.

Data Analysis. Out of 144 participants, 112 successfully finished all assessments. Following the same procedures as in Study 1, participants with overall accuracy below 65% on the VMAC task (n = 3), and those that had RTs lower than 150ms on more than 25% of the trials (n = 3) were excluded. For the rest of the sample (n = 106), RTs less than 150ms (0.18% of all RTs) were excluded, and analyses of RTs were restricted to correct responses only (82.59% of all RTs). Furthermore, participants who did not respond correctly to the attention check item in the clinical questionnaires (n = 1) were excluded from analyses of clinical measures.

The RTs of the Reward VMAC task were analyzed using a 6 x 2 x 2 analysis of variance (ANOVA) with Block (1–6) and Distractor Type (high, low) as within-subject, and Block Order (reward first, punishment first) as between-subject factors.

Results

Sample Characteristics

On average, more than half of the participants responded in the lower range of the AUDIT, CUDIT-R, and DASS-21 questionnaires (i.e., low alcohol/cannabis use; normal depressive/anxiety/stress symptoms) in both studies, indicating a relatively healthy overall sample. Only about 5-15% of the sample recorded responses in the severe range (i.e., likelihood of dependence; severe depressive/anxiety/stress symptoms). Table 1 describes the sample characteristics for both studies in more detail. We report Cronbach's alpha coefficients as a measure of internal consistency in Table S8 in the supplementary materials.

Table 1

Sample Characteristics in Both Studies

	Study 1 – Session 1	Study 1 – Session 2	Study 2
Sample Size (N)	72	43	106
Gender (%)	56.34% Female	58.14% Female	80.19% Female
	42.25% Male	39.53% Male	16.98% Male
	1.41% Other	2.33% Other	2.83% Other
Age range (Mean, SD)	18-33 (20.92, 2.56)	18-33 (21, 2.93)	17-35 (19.78, 2.24)
AUDIT Range (Mean, SD)	0-27 (8.11, 5.62)	N/A	0-27 (7.16, 5.16)
CUDIT-R Range (Mean, SD)	0-24 (5.07, 6.51)	N/A	0-28 (3.84, 5.96)
DASS-21 (Mean, SD)		N/A	
Total Score	0-84 (31.13, 19.34)		2-96 (36.42, 22.52)
Depression Subscale	0-40 (10.34, 9.13)		0-36 (11.85, 9.83)
Anxiety Subscale	0-26 (7.3, 6.35)		0-32 (9.7, 7.85)
Stress Subscale	0-30 (13.49, 7.2)		0-38 (14.87, 8.5)

Notes. The N/A (Not Applicable) refers to measures that have not been assessed during the second session of Study 1. SD = Standard Deviations; AUDIT = Alcohol Use Disorders Identification Test; CUDIT-R = Cannabis Use Disorders Identification Test-Revised; DASS-21 = Depression Anxiety Stress Scales-21.

Study 1

VMAC Effects. Table 2 summarizes all relevant ANOVA effects when including session as a factor. For the reward VMAC task, we found significant effects of both Block and Distractor Type across both sessions. The main effect of Block was significant, with lower RTs as participants progressed through the task. The main effect of Distractor Type was significant,

with higher RTs on trials with a high-reward distractor (session 1: M = 670.08, SD = 70.75; session 2: M = 604.76, SD = 71.06) compared to the RTs on trials with a low-reward distractor (session 1: M = 655.71, SD = 74.5; session 2: M = 592.87, SD = 65.09). The Block x Distractor Type interaction was also significant, indicating that participants took longer to respond to high-reward than low-reward distractors depending on the block (i.e., there was a significant RT difference between high- and low-reward distractors starting from the third block).

For the VMAC Punishment task, we found a significant main effect of Block (lower RTs as participants progressed through the task) and a significant main effect of Distractor Type (higher RTs on trials with a high-reward distractor compared with low-reward distractor). There were no significant Block x Distractor Type interaction effects.

In both reward and punishment VMAC tasks, there were also main effects of session (i.e., faster responses during the second session) and significant block * session interaction indicating that the change in RT across blocks depends on the session (i.e., learning effects). Only in the punishment VMAC, we found a significant block * distractor type * session interaction which may indicate stronger VMAC effects (i.e., difference between high and low reward) during the later blocks of session 2. ANOVAs conducted for reward and punishment at both sessions separately can be found in the supplementary table S2.

Table 2

ANOVA Results for VMAC Reaction Time Effects At Both Sessions of Study 1

Analysis	F	p	DF	η^2
Reward VMAC				
Block	34.73	< 0.001**	2.45, 103	0.453
Distractor Type	20.17	< 0.001**	1, 42	0.324
Session	105.8	< 0.001**	1, 42	0.716
Block * Distractor Type	3.51	0.017*	3, 126	0.077
Block * Session	5.3	0.002*	3, 126	0.112
Distractor Type * Session	0.059	0.81	1, 42	0.001
Block * Distractor Type * Session	1.049	0.373	3, 126	0.024
Punishment VMAC				
Block	28.65	< 0.001**	2.46, 103.2	0.405
Distractor Type	7.248	0.01*	1, 42	0.147
Session	81.83	< 0.001**	1, 42	0.661
Block * Distractor Type	0.169	0.883	2.44, 102.4	0.004
Block * Session	10.53	< 0.001**	3, 126	0.201
Distractor Type * Session	2.011	0.164	1, 42	0.046
Block * Distractor Type * Session	3.099	0.029*	3, 126	0.069

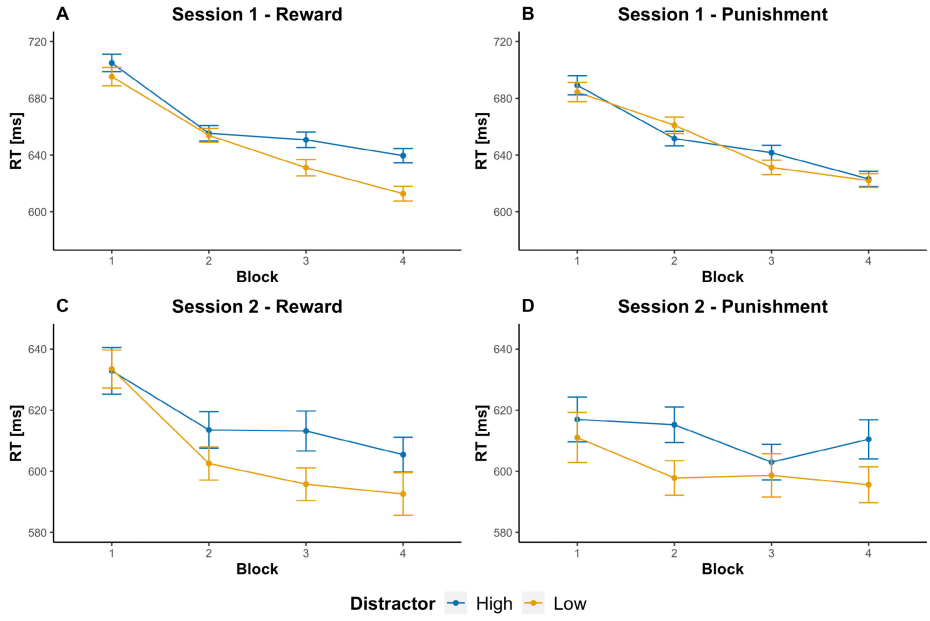
Note. Degrees of freedom (DF) reported are corrected for sphericity. Effect sizes reported are partial eta squared (η^2). ** denotes p-values below 0.001, and * denotes p-values below 0.05.

Slower responses on high-value trials than low-value trials in the VMAC task could reflect attentional capture by the high-value distractor interfering with search for the target, or could reflect a strategic slowing by participants in order to respond more accurately when more points were at stake. To assess this latter possibility, an additional exploratory analysis was conducted to test for a possible speed-accuracy tradeoff. Response accuracy in the VMAC task was analyzed using a 4 x 2 x 2 ANOVA with Block (1–4), Distractor Type (high, low), and Session (1, 2) as factors. Across sessions and both reward and punishment blocks (see supplementary Table S3), the main effect of Block was significant, with higher accuracy as participants progressed through the task. For reward, there were no main effects of Distractor Type, and no significant interaction effects (Block* Distractor Type) at either session, indicating no speed-accuracy tradeoff. However, we found a significant main effect of distractor type on accuracy in the punishment VMAC task (see Table S3), with higher accuracies in the low distractor compared with the high distractor conditions. That is, participants were less accurate in responding to the target when the display contained a high-value (vs low-value) distractor. However, when running ANOVAs separately for the two sessions, we found no more main effect of distractor type on the accuracy in the punishment VMAC task (see Table S4).

Figure 2 summarizes the reaction times for different distractor types and VMAC task blocks (reward/punishment) at both sessions. The significant VMAC Reward effect (RT difference between high and low reward) appears mostly after the third block during both sessions.

Figure 2

VMAC Effects for Reward and Punishment in Study 1 (For Both Session 1 and Session 2)



Note. The vertical bars represent within-participant standard errors. RT = reaction times.

Clinical Measures. Following the preregistration, we examined associations between attentional capture in reward/punishment blocks and alcohol/cannabis use and depression/anxiety respectively in session 1. None of the models were significant (see Figure S1 in the supplementary materials). We also found no significant interaction effects between the VMAC reward scores and the SDL measure ($p > 0.05$). In an exploratory fashion following the approach by (Albertella et al., 2017), we examined interaction effects between the SDL score, the VMAC reward effect, and the association with internalizing symptoms. We found a significant interaction effect ($p = 0.03$) for the DASS-21 anxiety symptom score (see Figure S2 in the supplementary materials). A higher VMAC-Reward score was positively associated with more anxiety symptoms in individuals with poorer attentional control (long response deadline as assessed through the SDL task). We found no significant interaction effects for the stress and depression subscales ($p > 0.05$).

Test-retest Reliability. The bivariate correlation between the VMAC-Reward scores (i.e., difference in RT for high-value vs. low-value trials) at session 1 and session 2 was non-significant, $r(41) = 0.086$, $p = 0.585$. The correlation between the VMAC-Punishment scores at session 1 and session 2 was also non-significant, $r(41) = -0.021$, $p = 0.893$. The separate RT measures for high and low reward/punishment at session 1 and session 2 showed statistically significant moderate to strong correlations (see Figure S3 in the supplementary materials). The test-retest reliability for the SDL response deadline was moderate; SDL scores at session 1 and session 2 showed a significant association ($\underline{r} = 0.56$, $p < 0.01$).

Study 2

VMAC Effects. All estimates from the ANOVA are reported in Table 3 below. For the Reward VMAC task, the main effect of Block was significant, with lower RTs as participants progressed through the task. The main effect of Distractor Type was significant, with higher RTs on trials with high-reward distractor ($M = 653.02$, $SD = 78.4$) compared to the RTs on trials with low-reward distractor ($M = 641.94$, $SD = 77.86$). The Block x Block Order interaction was also significant, indicating that participants were significantly better at completing subsequent blocks of the same type (reward/punishment) that they started with due to practice effects. There was no effect of Block Order, and no other two-way (i.e., Block Order x Distractor Type; Block x Distractor Type), or three-way interactions.

The analysis conducted on the RTs of the Punishment VMAC task revealed similar results as Study 1. A significant main effect of Block was found, with lower RTs as participants progressed through the task. There was no effect of Distractor Type, and no effect of Block Order, but a significant Block x Block Order interaction was found. No other two-way (i.e., Block Order x Distractor Type; Block x Distractor Type), or three-way interactions were found.

Table 3

ANOVA Results for VMAC Task Effects for Study 2

Predictor	***F***	***p***	***DF***	η^2
Reward VMAC (N = 106)				
Block	103.278	< 0.001 **	4.26, 442.9	0.498
Distractor Type	23.470	< 0.001 **	1, 104	0.184
Block Order	0.181	0.672	1, 104	0.002
Block * Distractor Type	0.656	0.657	5, 520	0.006
Block * Block Order	8.250	< 0.001 **	4.26, 442.9	0.073
Distractor Type * Block Order	0.250	0.618	1, 104	0.002
Block * Distractor Type * Block Order	0.954	0.446	5, 520	0.009
Punishment VMAC (N = 106)				
Block	100.063	< 0.001 **	4.31, 447.93	0.49
Distractor Type	0.003	0.958	1, 104	<0.0001
Block Order	2.604	0.11	1, 104	0.024
Block * Distractor Type	1.500	0.188	5, 520	0.014
Block * Block Order	2.636	0.0299 *	4.31, 447.93	0.025
Distractor Type * Block Order	0.033	0.855	1, 104	<0.0001
Block * Distractor Type * Block Order	1.881	0.096	5, 520	0.018

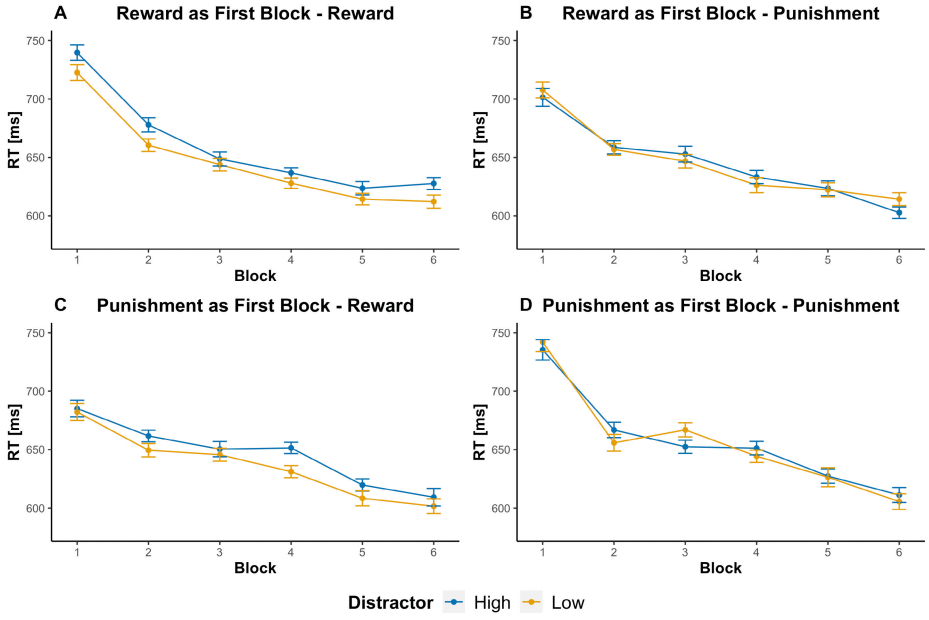
Note. Degrees of freedom (DF) reported are corrected for sphericity. Effect sizes reported are partial eta squared (η^2). ** denotes p-values below 0.001, and * denotes p-values below 0.01.

Furthermore, the analysis of accuracy of the Reward VMAC task revealed no speed-accuracy tradeoff. A 6 x 2 x 2 ANOVA with Block (1–6) and Distractor Type (high, low) as within-subject, and Block Order (reward first, punishment first) as between-subject factors revealed a significant main effect of Block, with higher accuracy as participants progressed through the task. There was no effect of Distractor Type, and no effect of Block Order. A Block x Block Order interaction was found. No other interactions were found (see Table S5). As we did not find any significant differences in RTs of the Punishment VMAC task, we did not conduct the corresponding speed-accuracy tradeoff test.

Figure 3 shows the RT for both distractor types (low/high) and study conditions (reward as first block, punishment as first block). The VMAC reward effects (difference in RT between high and low) are present regardless of whether reward or punishment appears as a first block.

Figure 3

VMAC Effects for Reward and Punishment in Study 2



Note. The vertical bars represent within-person standard errors.

Clinical Measures. We found no significant associations between the VMAC reward/punishment scores and clinical measures (see Figure S3 for an overview of all regression estimates). In an exploratory fashion, when controlling for DASS-21 total score, all findings stayed the same ($p < 0.05$).

Discussion

The present two-study report investigated the associations between reward- and punishment-modulated attentional capture effects and internalizing symptoms and substance use in a student sample. As predicted, we found reward-related VMAC effects across both studies. However, contrary to our predictions, test-retest reliability of the VMAC effect was very low, we did not find punishment-related VMAC effects across sessions, and there were no meaningful associations between attentional capture and clinical measures.

During the reward VMAC trials in both studies participants took longer to respond to high-reward compared to low-reward distractor trials, indicating they were more likely to attend to the distractor that signaled a high reward, even though such distraction resulted in less money earned. These findings are consistent with several value-modulated attentional capture studies in which reward-associated distractors capture attention even when task-irrelevant (e.g., Anderson et al., 2011; Le Pelley et al., 2015). We extend these previous studies by showing that the reward-related attentional capture persists in a shorter task

setup, and despite the presence of alternating punishment blocks. This suggests that reward contingencies are maintained despite the alternating punishment blocks. Interestingly, while most of the previous VMAC tasks contained at least 400 (and up to 2,000) trials, our current task in study 1 consisted of only 120 trials for reward and punishment blocks. The difference in capture between the high and low reward distractors can already be seen after three reward blocks, at which point participants have only gone through 90 reward trials. Furthermore, our exploratory accuracy analyses showed that these effects are not a result of a speed-accuracy tradeoff, indicating that participants did not simply take longer to respond to the high-reward distractor in order to be more accurate.

A range of studies have shown that aversively conditioned stimuli similarly capture attention (e.g., Schmidt et al., 2015). However, we did not find such effects in the current VMAC punishment task in session 1 of Study 1 or Study 2. In this novel punishment adaptation, we found no significant differences in participants' response time between high- and low-punishment distractor trials. Importantly, previous studies that used a similar task setup as the current VMAC task, almost exclusively focused on threat- and fear-related attentional capture (e.g., electrical shock or loud noises). Likely, this may activate punishment contingencies that are different from processes relevant in monetary loss. Threatening stimuli, as opposed to losing money, could indicate a biological adaptation, as life-threatening dangers should quickly be seen and avoided. Moreover, those studies that have focused on monetary loss specifically did not use this particular VMAC design in which a distractor is merely a signal of punishment, rather than an association between a response and monetary loss. For example, Wentura et al. (2014) found attentional capture effects related to monetary loss using a task design that included a variation of the classic training and test phases, that have often been used in value-driven attentional capture tasks by Anderson and colleagues (e.g., Anderson et al., 2011). This could potentially make a difference in interpreting the results of the current and previous punishment-modulated capture effects. Considering the paucity of research on VMAC effects in punishment settings, future studies should test other variations of the VMAC task which could include threat stimuli (e.g., loud noises or electric shocks), rather than monetary signals of punishment. It remains yet to be tested whether indeed such punishment effects can only be detected in instrumental conditioning designs with a separate training phase.

Another possible explanation for the absence of punishment VMAC effects in the present studies may be the small number of trials. While it is striking how quickly reward contingencies can be established, it is possible that participants need more time to learn punishment contingencies compared to the reward ones. Although this might seem at odds with evolutionary theories which suggest that threatening stimuli capture attention faster (Öhman & Mineka, 2001, 2003), the present study does not use evolutionary-related stimuli but goal-related stimuli (i.e., monetary loss). Some recent research suggests that indeed participants needed fewer trials to learn reward compared to punishment and neutral associations (Wang et al., 2018) which may speak to this alternative explanation. Indeed, in Study 1, we did find significant punishment VMAC effects two weeks later, which could not be explained by selective attrition. However, these punishment effects of session 2 should

be interpreted with caution due to the smaller sample size, and different sets of distractor colors used for the second session. In fact, increasing the number of trials within the same VMAC task in Study 2, still did not reveal punishment-related attentional capture effects. Future studies should aim to compare different task lengths and times between task sessions.

Several limitations should be considered when interpreting our results. First, we used a sample of college students that showed substantial variability with respect to alcohol use, but little variability with respect to internalizing symptoms and cannabis use. This lack of variability may also explain the absence of associations between VMAC effects and substance use. Future studies should investigate whether heightened reward-processing is associated with greater drug use only in individuals diagnosed with substance use disorders. Moreover, in our analyses of the associations between VMAC and substance use and related problem (AUDIT, CUDIT-R total scores), we did not control for the recency of use. Thus, it is possible that acute substance use impairs individuals' attentional control making it more difficult to differentiate between high and low distractor stimuli. This may explain the lack of an association. To better understand the relationship between VMAC and substance use, future studies should consider controlling for the time elapsed since last substance use.

We found that individuals with low attentional control (high SDL score) showed a positive association between VMAC reward scores and anxiety symptoms. Although this pattern is broadly consistent with previous findings that suggest interactions between cognitive control and attentional capture (Albertella et al., 2017), it should be interpreted with caution considering a) the low test-retest reliability of the VMAC reward score, and b) the exploratory nature of this analysis which included many contrasts.

Second, the primary outcome measure of our VMAC task (reward and punishment difference scores) showed low test-retest reliability. This is consistent with a previous report of low test-retest reliability of a different VMAC version by Anderson & Kim (2019) and it could also explain the lack of associations between VMAC scores and clinical measures in our study. Prior research suggests that low test-retest reliability may be related to factors such as low between-subject variability (Hedge et al., 2018) or the use of reaction time difference scores as measures of attentional control (Draheim et al., 2019). Nonetheless, the VMAC reward RT difference score remains a commonly used metric in the attentional bias literature and our study is one of the first to report test-retest reliability estimates.

The present study replicates previously established reward-related attentional capture effects (Le Pelley et al., 2015) in which individuals are slower to respond to a target when a distractor that signals a high reward is present. The study also provides additional insights into how these capture effects can be established fairly rapidly, and with a fewer number of trials. Moreover, our findings show how these reward-related effects persist throughout the task, despite several interruptions of similar punishment trials. Further research on punishment-related attentional capture is needed in order to establish the nuances of how such punishment attentional biases occur. Accurately assessing reward- and punishment-modulated attentional capture effects using a scalable and short behavioral online task may provide a window of opportunity to better grasp the cognitive-motivational processes underlying the development of mental health problems.

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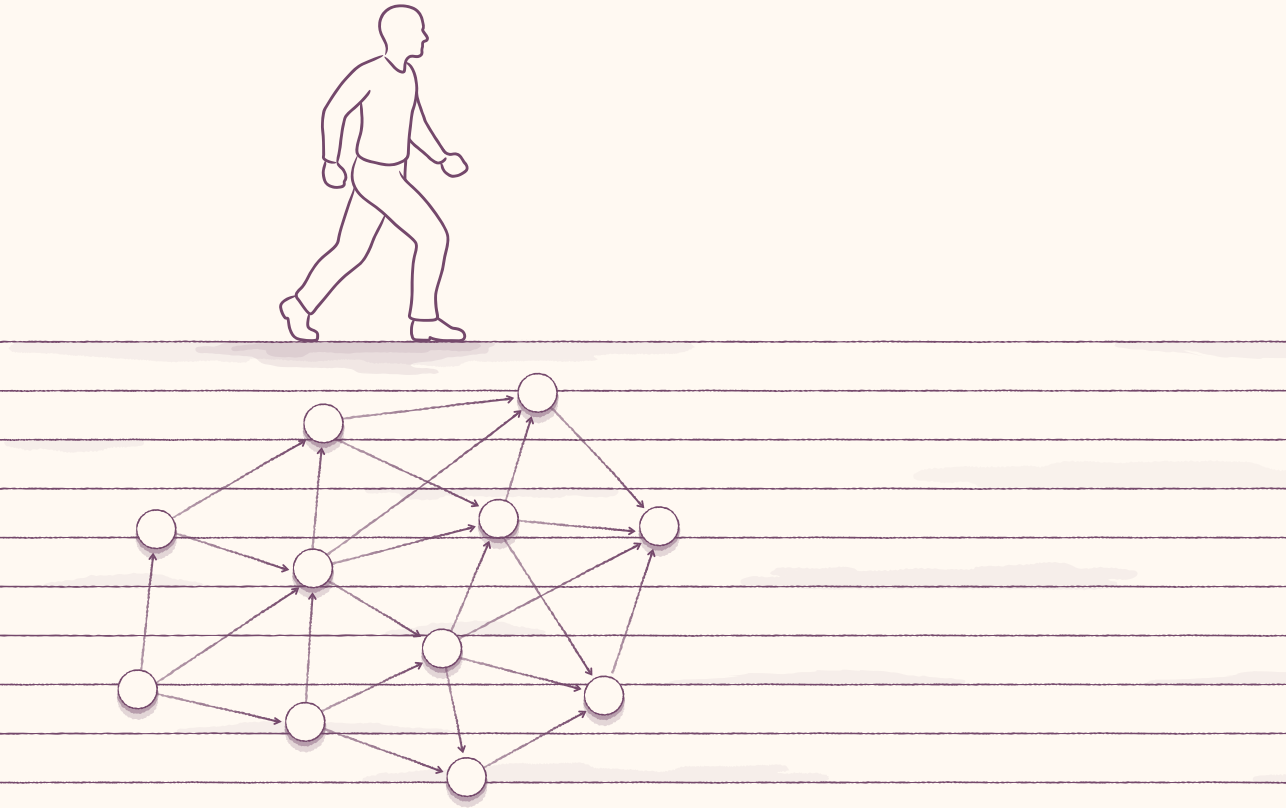
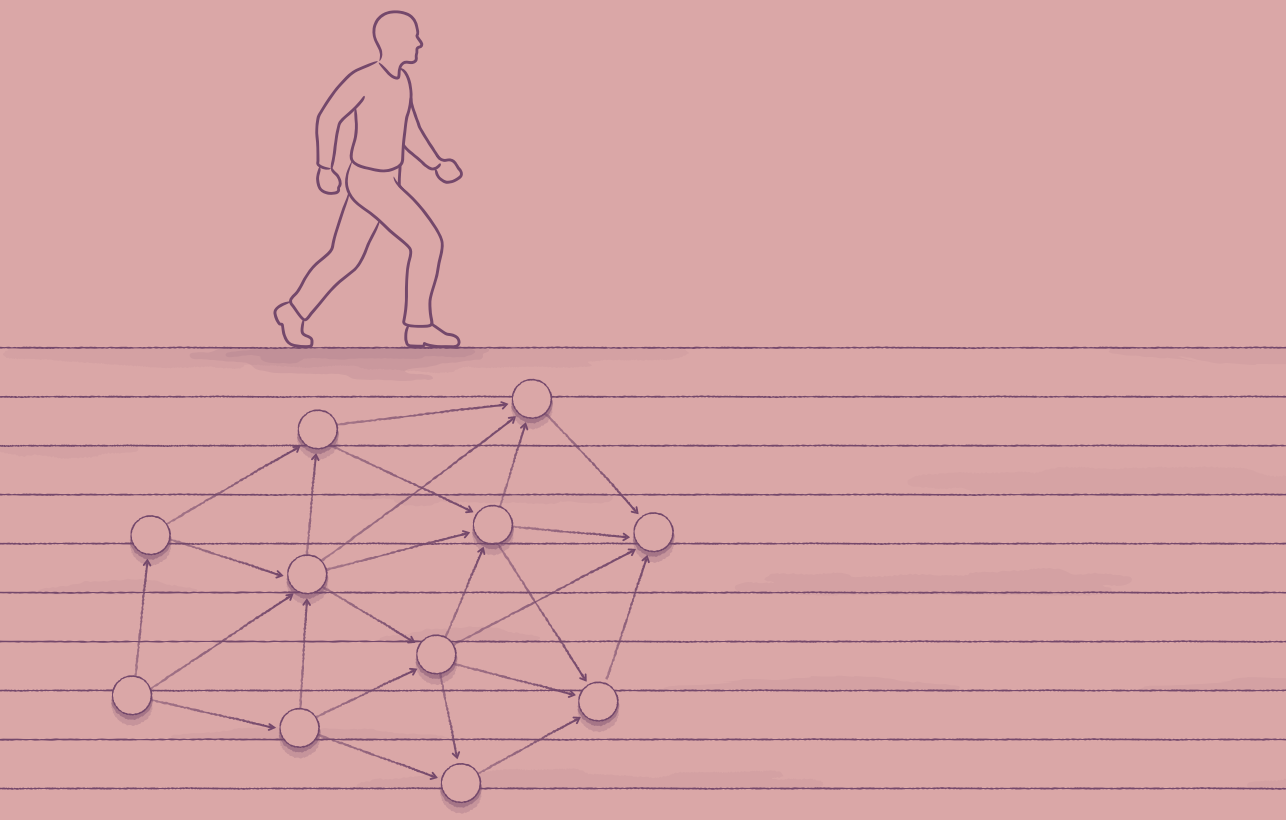


Part 3

Methodological innovations



CHAPTER 8

Unraveling robust brain-behavior links of depressive complaints through granular network models for understanding heterogeneity

This chapter is adapted from:
Freichel, R., Lenartowicz, A., Douw, L., Kruschwitz, J. D., Banaschewski, T., Barker, G. J., Bokde, A. L. W., Desrivières, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Brühl, R., Martinot, J. L., Martinot, M. P., Artiges, E., Nees, F., Orfanos, D. P., Paus, T., Poustka, L., … Blanken, T. F. (2024). Unraveling robust brain-behavior links of depressive complaints through granular network models for understanding heterogeneity. Journal of Affective Disorders, 359, 140–144. https://doi.org/10.1016/j.jad.2024.05.060

Abstract

Background: Depressive symptoms are highly prevalent, present in heterogeneous symptom patterns, and share diverse neurobiological underpinnings. Understanding the links between psychopathological symptoms and biological factors is critical in elucidating its etiology and persistence. We aimed to evaluate the utility of using symptom-brain network models to parse the heterogeneity of depressive complaints in a large adolescent sample.

Methods: We used data from the third wave of the IMAGEN study, a multi-center panel cohort study involving 1317 adolescents (52.49% female, mean±SD age=18.5±0.7). Two network models were estimated: one including an overall depressive symptom severity sum score based on the Adolescent Depression Rating Scale (ADRS), and one incorporating individual ADRS item scores. Both networks included measures of cortical thickness in several regions (insula, cingulate, mOFC, fusiform gyrus) and hippocampal volume derived from neuroimaging.

Results: The network based on individual item scores revealed associations between cortical thickness measures and specific depressive complaints, obscured when using an aggregate depression severity score. Notably, the insula's cortical thickness showed negative associations with cognitive dysfunction (partial cor. = -0.15); the cingulate's cortical thickness showed negative associations with feelings of worthlessness (partial cor. = -0.10), and mOFC was negatively associated with anhedonia (partial cor. = -0.05).

Limitations: This cross-sectional study relied on the self-reported assessment of depression complaints and used a non-clinical sample with predominantly healthy participants (19% with depression or sub-threshold depression).

Conclusions: This study showcases the utility of network models in parsing heterogeneity in depressive complaints, linking individual complaints to specific neural substrates. We outline the next steps to integrate neurobiological and cognitive markers to unravel MDD's phenotypic heterogeneity.

Keywords: depression symptoms; neural markers; network analysis; heterogeneity

Introduction

Depressive symptoms continue to be highly prevalent across the globe, with increasing rates among adolescents and young people (Goodwin et al., 2022). Depression is a highly heterogeneous disorder (Goldberg, 2011) diagnosed based on the presence of five out of nine DSM-5 symptoms. These symptoms are, however, diverse, ranging from weight loss or gain to depressed mood, and contribute to disorder heterogeneity that poses challenges for treatment. Symptom network models have been used to capture this heterogeneous symptom expression as they conceptualize mental disorders as systems of interacting symptoms. The heterogeneity observed at the level of depression symptoms is mirrored in the disorder's heterogeneous neurobiological underpinnings (Buch & Liston, 2021): depression has been associated with a wide range of alterations in brain structure and function (Gray et al., 2020; Marx et al., 2023), changes in neurotransmitter systems (Kennis et al., 2020), and genetic variations (Kendall et al., 2021). At the level of neuroanatomical alterations, meta-analytical evidence in adult samples points to lower hippocampal volume (Schmaal et al., 2016) and lower cortical thickness in several regions, including the insula, cingulate, orbitofrontal cortex, and fusiform gyrus (Schmaal et al., 2020). Modeling this interplay between symptom expression and biology is crucial for understanding depression's etiology and, ultimately, treatment (Remes et al., 2021).

However, when both domains (i.e., psychological/biological) are combined, then typically, at least one domain is simplified in the process (Blanken et al., 2021), often to a single aggregate dimension. Most studies examining associations between structural and functional neural alterations and depressive symptoms, either use depression sum scores or subscales (aggregating the psychological level) or they use aggregate measures derived from neuroimaging, such as overall cortical thickness, or structural or functional connectivity, (aggregating the biological level). This abstraction potentially obscures more fine-grained associations, that could potentially account for the symptom heterogeneity.

While many studies have revealed close relationships between depression and brain structure and function (e.g., Schmaal et al., 2020), fewer studies have examined this link for specific depressive complaints. For instance, social anhedonia symptoms have been associated with reduced (gray) matter volume in the bilateral caudate nucleus (Enneking et al., 2019). Similarly, there is evidence for associations between disturbed white matter microstructure and cognitive dysfunction in depression (Meinert et al., 2022)." One recent pilot study that included both brain and individual symptom measures into one network model did reveal cross-modal (i.e., brain-symptom) relations even in a small sample of depressed and never-depressed adults (Hilland et al., 2020). This finding suggests that fine-grained associations could indeed be obscured when using aggregate measures, but this was not evaluated directly.

We believe that network analysis (Borsboom et al., 2021) offers distinct advantages for studying granular cross-modal associations between individual symptoms and brain markers. First, network analysis can identify unique, conditional (i.e., partial) associations while controlling for the influence of all other symptom nodes or brain markers in the model. Given

the many and strong relations between the depressive symptoms themselves, this provides the opportunity to distinguish direct from indirect effects. Second, from a conceptual perspective, network analysis maps onto the complex organization of mental disorders that consists of interconnected symptoms, cognitive, and neurobiological features (Blanken et al., 2021).

In the present study, we replicate the approach by Hilland et al. (2020) in a substantially larger sample to identify relations between depressive complaints and five a-priori selected (based on Hilland et al., 2020) brain markers (cortical thickness measures for insula, cingulate, mOFC, fusiform gyrus, and hippocampal volume). In addition, we will extend the previous study by directly evaluating whether parsing heterogeneity into individual item scores relative to an overall severity measure reveals cross-modal relations that otherwise would remain hidden.

Methods

Participants, procedure, and outcomes

We have used data from the third wave of IMAGEN study (Schumann et al., 2010), a multi-center panel cohort study of adolescents. Our final sample included 1,317 adolescents (52.49% female, M±SD = 18.5±0.7 years old, range: 18-23 years old) that completed the Adolescent Depression Rating Scale (ADRS) and 3D T1-weighted gradient-echo Magnetic Resonance Imaging (MRI) scans. The ADRS is a validated 10-item self-report scale to assess the presence (present/ not present) of adolescent depression symptoms (Revah-Levy et al., 2007). The scale consists of 10 items that assess the presence of different depressive complaints on a binary scale (1 = True/Present, 0 = False/Not present). A total ADRS depression severity (sum) score above 6 is commonly used as a cut-off for a clinically relevant diagnosis of MDD as it ensures maximum sensitivity and specificity (Revah-Levy et al., 2007, 2011; Vulser et al., 2015). ADRS scores of 3 to 5 indicate 'sub-threshold depression' (Revah-Levy et al., 2011).

The present sample showed substantial variability in the presence of all complaints (see Table S1 in supplementary materials (SM) section 1), with 12% (n = 155) of individuals being in the 'sub-threshold' depression group, and an additional 7% of individuals (n=89) meeting the criteria (score ≥ 6) for MDD. The magnetic resonance imaging (MRI) data was acquired using standard protocols to ensure homogeneity across scanners, including a 3D T1-weighted gradient echo volume (see SM1 and Schumann et al., 2010 for more details). Cortical thickness of insula, cingulate, mOFC, fusiform and hippocampal volume were estimated using the FreeSurfer software. We selected the same five brain regions (see Figure 1) as Hilland et al. (2020) and followed their exact procedures: we averaged left/right hemispheres and used z-residuals for hippocampal volume (regressing out sex, intracranial volume). Age was not included as a covariate in the model considering that our sample was based on one assessment wave from a cohort study comprising adolescents of a comparable age group, with an average age of 18.5 years and a standard deviation of 0.7 years.

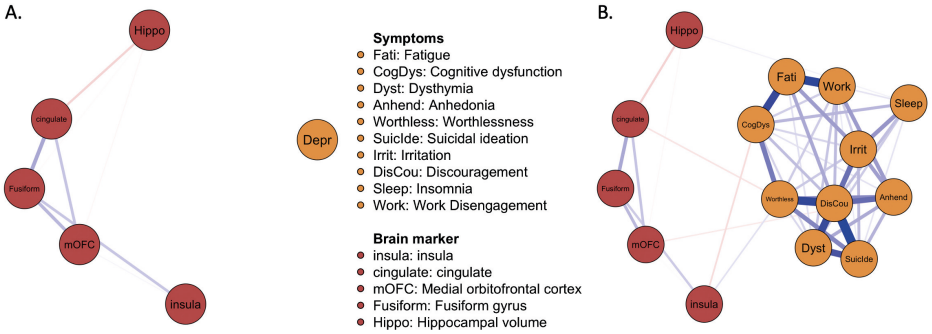
Statistical analysis

To investigate whether the abstraction of symptoms as sum scores obscures more fine-grained relations between brain regions and depression complaints, we estimated two network models. Both networks contained the same brain measures (i.e., cortical thickness measures, hippocampal volume); however, one included the ADRDS sum score, indicating overall depression severity, and one included all individual ADRS items, representing different depressive complaints. The network estimation includes a nodewise regression approach in which every node is predicted by all other nodes. To minimize false positive edges, we have used LASSO (Least Absolute Shrinkage and Selection Operator) regularization with cross-validation (determines optimal level of penalization) to shrink estimates towards zero and avoid overfitting (see SM2 for more details). For the network including depression severity, we estimated a Gaussian Graphical Model, including all variables as continuous, whereas for the network including the single depression items, we estimated a Mixed Graphical Model, including the items as binary variables (present/not present) and the brain measures as continuous, taking the different variable types into account (MGM, Haslbeck & Waldorp, 2020). The resulting connections in both networks ('edges') represent pairwise conditional associations (similar to partial correlations) that control for all other nodes in the network. While traditional statistical significance is not defined in these models, edges are included based on model fit. Included edges thus improve the fit of the model to the data. We assessed the edge weights' accuracy using bootstrapping (n=1,000, see SM2).

Results

The network including depression severity is shown in Figure 1A. We found no cross-modal associations between any of the neural markers and overall depression severity. In contrast, we found many positive associations within the respective domains (i.e., among depressive complaints and cortical thickness measures) in the network estimated on the separate depression complaints (Fig. 1B). The networks were sufficiently stable, and all cross-modal links were retrieved in at least half of the bootstrapped samples (range 53-85%). Interestingly, we found cross-modal associations between cortical thickness measures and specific complaints: cingulate was negatively associated with worthlessness (retrieved in 59%), insula was negatively associated with cognitive dysfunction (85% retrieved), and mOFC was negatively associated with anhedonia (53% retrieved). We found positive associations between insula and worthlessness (61% retrieved) and between hippocampal volume and sleep problems (60% retrieved). An additional subgroup analysis of individuals with either sub-threshold depression or meeting all criteria for a depression diagnosis showed differences but replicated two cross-modal associations (worthlessness – cingulate; insula – worthlessness).

Figure 1. Depressive complaints - brain network model.



Note. The thickness of the lines indicates the strength of association. The connections (edges) in the network represent pairwise, partial associations between different complaints and brain markers. Positive conditional associations are colored in blue, negative conditional associations are colored in red. Panel A includes the ADRS severity score (Depr). Panel B includes all ADRS depression complaints. The nodes for the four brain regions (i.e., insula, cingulate, mOFC, Fusiform) refer to cortical thickness. Hippo =hippocampal volume; mOFC =medial orbitofrontal cortex. All edge weights can be found in supplementary Tables S2–S3. The cut argument has been set to 0. Both networks were visualised using the same maximum edge weight for scaling.

Discussion

The present study is one of the first to pinpoint granular associations between neural substrates of overall depressive symptomatology and specific depression complaints using an integrated network approach. Crucially, we showed that these robust associations remain hidden when only including overall depression severity, concealing the heterogeneous complaints. The negative associations shown (between regional cortical thickness and complaints) align with prior evidence for cortical thinning as a depression biomarker (Suh et al., 2019), prompting us to speculate about the mechanisms at play. The link between cortical thinning of the insula and cognitive dysfunction could reflect the insula's pivotal role in high-level cognitive control and emotional processing (Menon & Uddin, 2010). This interpretation about altered affective processing in depression may be particularly relevant, in light of our findings regarding the negative association between cingulate's cortical thickness and feelings of worthlessness, given the prominent role of the cingulate cortex in emotional processing (Etkin et al., 2011). At the same time, we want to stress that such interpretations are highly speculative, as our estimated links are undirected and estimated cross-sectionally. Interestingly, our results also uncovered novel links, such as positive associations between insula and worthlessness.

We believe that our findings have dual implications; with respect to guiding future brain-behaviour research and bear relevance for clinical practice. First, our comparative analysis of networks estimated on an aggregate measure of depression severity (Fig. 1A) and specific depression complaints (Fig. 1B) showed stark differences. The heterogeneity underlying the association between neural substrates and depressive complaints was obscured when using an aggregate score. This suggests that networks estimated at the level of individual symptoms

and neural makers have the potential to dissect these hidden associations and may allow us to better grasp the heterogeneity of depression.

Second, while primarily exploratory, symptom-brain networks, as showcased in this report, may inspire research that could eventually be used for potential clinical applications. The current diagnostic heterogeneity of depression complicates an effective treatment (Buch & Liston, 2021). Thus, identifying specific symptom-brain biomarker connections, such as the link between the thinning of insula and cognitive dysfunction, may pave the way for delineating distinct psychobiological subtypes of depression. This may potentially lead to more accurate diagnostic and tailored treatment approaches, moving us closer to a personalized medicine model in psychiatry. However, this is a more conceptual point as we refrain from drawing a direct line from a cross-sectional association study in a particular sample to real-world clinical applications.

Limitations

A limitation of our study is the self-reported assessment of depressive complaints that may naturally be biased. In addition, our sample was relatively healthy (only 19% of participants with depression or sub-threshold depression), and thus, the cross-modal links should be understood as associations describing how variability in depressive complaints is linked to variability in the selected brain markers. Future studies should replicate our findings using clinical samples with a higher number of individuals with MDD. Lastly, the cross-sectional nature of our study precludes any conclusions about the directionality and causal nature of the associations between neural markers and depressive complaints. The present study serves as a 'proof-of-principle' that may inspire future work to validate the mapping of symptoms and neural markers in clinical samples.

Conclusions

Altogether, this brief report showcases the utility of brain-symptom networks in the case of depressive complaints. Moving forward, future research should adopt such approaches and integrate neurobiological and cognitive markers to parse the phenotypic heterogeneity of depressive symptomatology both at a cross-sectional and developmental level.

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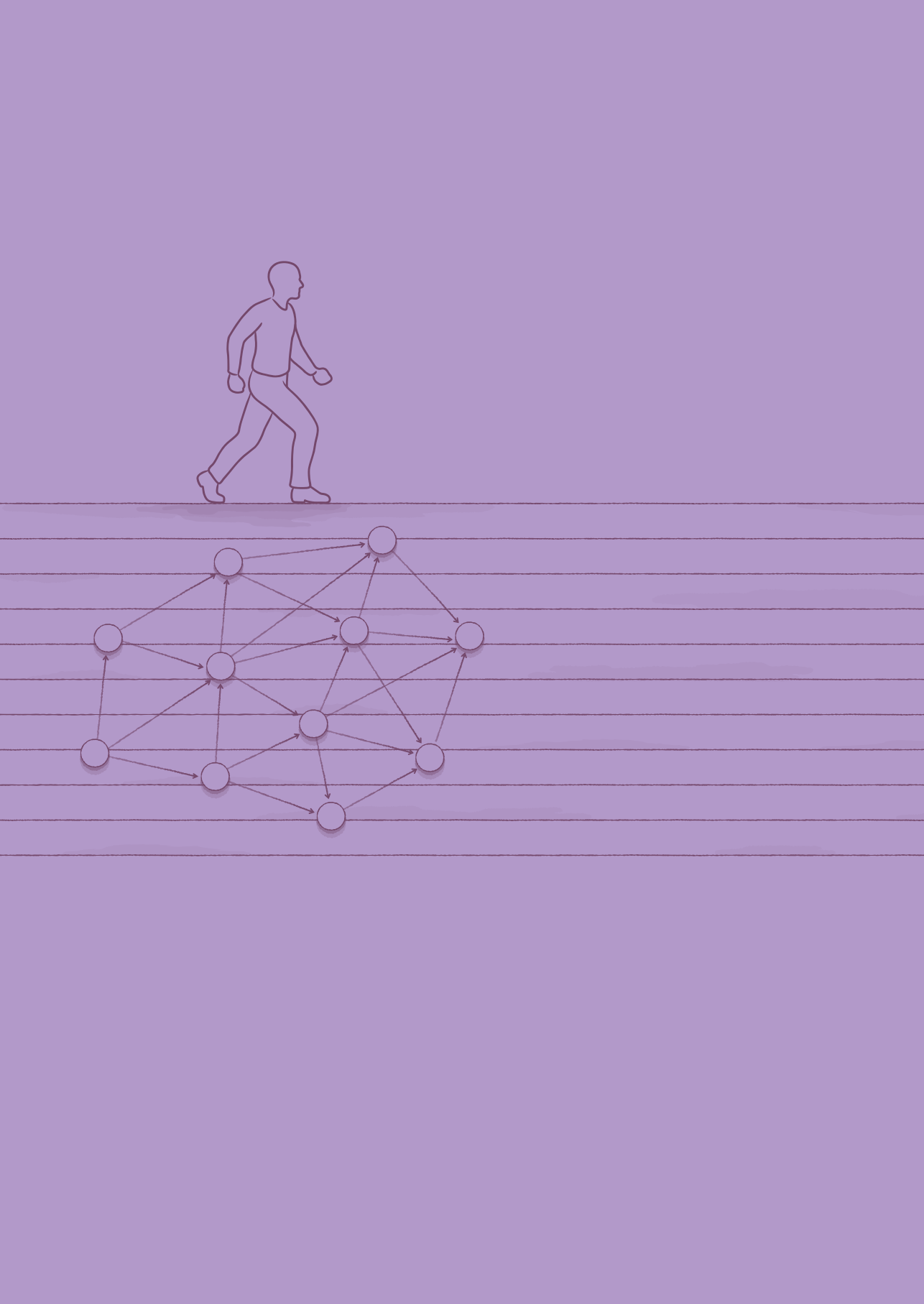


CHAPTER 9

Cross-lagged panel models for studying psychopathology: A comparative overview of structural equation and panel network approaches

This chapter is adapted from:
Freichel, R., Veer, I.M., Wiers, R., McNally, R. J., Epskamp, S. Cross-Lagged Panel Models for Studying Psychopathology: A Comparative Overview of Structural Equation and Panel Network Approaches. Manuscript under review. Preprint available at: https://osf.io/preprints/psyarxiv/b94qt

Abstract

Researchers have increasingly adopted complex methodological approaches to investigate the co-development of symptoms over longer time frames, such as months and years. Panel studies assess a typically large group of individuals at multiple time points over an extended period. Various analytical approaches exist for examining the co-development of variables in panel data, including long-standing Structural Equation Models (SEM) and network models. This paper provides a detailed review and application of two recent panel network approaches, namely the cross-lagged panel network model (CLPN) and the panel graphical vector autoregression model (panel GVAR). We describe how these approaches compare to the two most relevant SEM approaches, specifically the cross-lagged panel model (CLPM) and the random-intercept cross-lagged panel model (RI-CLPM). We describe each method's distinct characteristics, advantages, and limitations. To illustrate these, we applied these models to a panel dataset of adolescents and young adults (NSPN 2400 cohort study), examining the relationships between impulsivity and symptoms of depression and anxiety. Results showed varying temporal associations, highlighting the importance of model selection based on research objectives and data characteristics. A simulation study demonstrated that models separating within- and between-person effects (panel GVAR, RI-CLPM) reproduced true within-person temporal effects more accurately. Our review highlights the value of using multiple approaches in multiverse analyses to assess the sensitivity of findings to different analytical methods. Ultimately, the choice of analytical method greatly influences how dynamic cross-lagged processes in developmental psychopathology are interpreted, affecting the development and refinement of relevant clinical theories.

Key words: panel data models; network analysis; within-person effects; developmental psychopathology

Introduction

Recent years have witnessed a trend towards complex systems approaches for understanding developmental and dynamic processes underlying psychopathology (Hayes & Andrews, 2020; Hofmann et al., 2016). To study long-term dynamics across time, researchers have been using 'panel' studies, a design in which a typically large group of individuals (N) are assessed at a limited set of time points (t) at approximately equal time-intervals, across a period ranging from several weeks to decades. This data source allows researchers to identify associations between variables at discrete time points as well as intraindividual differences across time (Borsboom et al., 2021). Unlike ecological momentary assessment (EMA), the data collection is less frequent and intensive, but the number of assessed variables is often numerous, and the study duration is sufficiently long to capture long-term alterations and progressions over time. Typically, panel data focuses on large sample sizes (N > t), whereas EMA studies typically have small to moderate sample sizes (t > N). Secondary longitudinal datasets on a wide range of factors and age spectra have become readily available online (Kievit et al., 2022). Panel data has proven valuable for answering clinically meaningful questions about symptom chronicity or development (Schlechter et al., 2022), and for testing long-standing theories, such as Moffitt's (1993) developmental taxonomy.

To study (symptom) dynamics from panel data, investigators have developed a range of analytical tools (Freichel, 2023), including cross-lagged structural equation (SEM) models and recent panel network adaptations. To date, researchers have employed these approaches independently, and a comprehensive review describing differences between models and the unique characteristics, advantages, and disadvantages of each method is lacking. The present paper aims to provide a comparative overview of two recent panel network approaches for studying cross-lagged associations, including the cross-lagged panel network model (CLPN) and the panel graphical vector autoregression (panel GVAR) model. We explain how these differ from two appropriate SEM equivalents, namely the cross-lagged panel model (CLPM) and the random-intercept cross-lagged panel model (RI-CLPM). While many other models are available for exploring cross-lagged associations in panel data, we restricted our comparison to the two most prominent SEM approaches to provide a clear and focused report for applied researchers.

This paper is organized in four sections. We first elaborate on two principal problems (i.e., separation within-/between-person effects, and partial or zero-order estimation) that help explain the differences between these four approaches. The second section describes all models in detail with respect to their model architecture, estimation process, and it provides a comparative review of unique advantages and limitations. The third section illustrates the use of all models in an easily accessible, empirical dataset when investigating a substantive research question in developmental psychopathology. In the fourth section, we employ a simulation approach to test the extent to which the models can uncover true within-person relations.

Separation of Within- and Between-Person Effects

The discussion concerning separating within and between-person effects dates back to Allport's separation of nomothetic (generalizable) and idiographic (specific to individuals) research (Allport, 1937, 1962), and it remains a topic of longstanding debate within the last decade (Curran & Bauer, 2011; Hamaker et al., 2015; Lüdtke & Robitzsch, 2021). As discussed by Molenaar (2004), findings from the large-scale sample approach may not generalize to individuals and thus, a paradigm shift towards 'thinking within-person' is necessary (Hamaker, 2012). Within-person processes describe changes occurring within the same individual unfolding over time. Most theories in (developmental) psychopathology are inherently within-person and focus on intraindividual processes related to symptom development or resilience. For example, inspired by Hierometer theory, researchers have found that individuals experienced lower symptoms of depression and anxiety on days when their self-esteem was higher (than their average level; Mahadevan et al., 2023).

In contrast to such within-person processes, between-person associations are inter-individual associations that apply across a group of individuals. These between-person associations are assumed to be stable within individuals. For example, the diathesis-stress model (Zuckerman, 1999) emphasizes the interplay of stressful life events and between-person differences. That is, individuals differ in their vulnerabilities to develop mental disorders based on biological-genetic factors, personality traits, or a combination of these. In certain situations, within- and between-person effects may show opposite directions, and thus, generalizing between-person effects to an individual may lead to faulty inferences (Curran & Bauer, 2011). A famous example from medical science illustrates this case: People who exercise have a lower risk for heart attacks (between-person effect), however, when exercising, individuals' risk for a heart attack is higher (Curran & Bauer, 2011; Mittleman et al., 1993). As widely noted, there are several strict assumptions under which between-person effects may generalize to the individual (Molenaar, 2004). Assuming an ergodic system, patterns found at a group level generalize to individuals when the system is homogeneous (i.e., equivalence of groups and individuals) and stationary (i.e., not changing in means or variance over time). Thus, if ergodicity holds, then there are no between-person relations (i.e., relations between stable averages) or between-person variances. In psychology, where individual differences in stable traits are the norm, these assumptions are naturally untenable.

Stationarity refers to the stability of statistical properties (i.e., mean, variance, covariance) over time. When studying developmental processes occurring over long time frames (e.g., adolescence) or critical periods (e.g., treatment), the assumption of stationarity is less likely to be met. In practice, the theoretical distinction between within- and person-effects dictates the choice of researcher's analytical approach when analyzing panel data. Integrating random intercepts or latent variables in the model may capture stable between-person differences when studying cross-lagged associations (i.e., different factors predicting each other) across time. This means that individual differences are modeled as stable averages that are not represented in the temporal (network) structures. Thus, the four panel data models discussed in this overview either treat individuals as homogeneous (i.e., models that do not contain random intercepts) or retrieve fixed-effects structures that describe the average (person's)

effects in the network structure (see Epskamp, 2020). It is important to note that such fixed within-person effects describe associations for the average subject and thus, they may not map onto idiographic (i.e., single-subject) within-person models.

Partial Correlations or Zero-Order Correlations

In addition to the separation of within- and between-person effects, panel data models differ with respect to their focus on partial or marginal/zero-order estimates (correlations). In recent years, the use of pairwise Markov random fields to model psychological network have become increasingly popular (Epskamp, Borsboom, et al., 2018). In such a model, nodes (variables) are connected through edges (links between variables) that represent conditional associations controlling for (i.e., partialing out) all other included variables. In the case of continuous variables, these edges represent undirected or directed partial correlations (Epskamp & Fried, 2018; Wild et al., 2010). Network analytical approaches have been applied in various domains of clinical psychology (McNally et al., 2015; Robinaugh et al., 2020) and were in part motivated by network theoretical approaches (Borsboom, 2017; Borsboom & Cramer, 2013) according to which mental disorders are emergent phenomena that arise from interactions between symptoms. In contrast to latent variable modeling, the focus is on the unique, rather than the shared variance of variables, leading to distinct benefits and shortcomings (Epskamp et al., 2017). It has been argued that this focus on pairwise conditional (in-)dependencies through partial estimates may facilitate speculations about causal relations among the variables (Haslbeck et al., 2022; Pearl, 2000). Network models thus typically focus on lower-level variables, such as symptoms or questionnaire items as nodes rather than latent variables.

In the context of panel data models, these usually consist of sets of exogenous variables that covary, and temporal effects that model the prediction over time. More precisely, panel models can opt to model the first wave of data as exogenous, meaning its variance-covariance structure is not structurally modeled, but model all subsequent waves of data as exogenous. In addition, exogenous terms explain new 'input' in the model at each time point, termed innovation. For example, take a simple model $X_{t=1} \rightarrow X_{t=2} \rightarrow X_{t=3,}$ the innovation at t=1 represents the variance of $X_{t=2}$ that is not explained by $X_{t=1}$. Note, we do not term this residual, as the variance propagates further to $X_{t=3}$. The covariance between innovation terms represents contemporaneous covariance that is unique within a wave of data. In addition to the innovation variance-covariance structure, approaches that separate within- and between-person variance include the innovation variance-covariance structure (then representing within-person variance) and the variance-covariance structure of the random intercept / mean per person (representing between-person variance). SEM approaches (RI-CLPM, CLPM) typically choose to model these variance-covariance structures through marginal/zero-order correlations/covariances, whereas a network approach (e.g., panel GVAR) may instead model these variance-covariance structures using partial correlations.

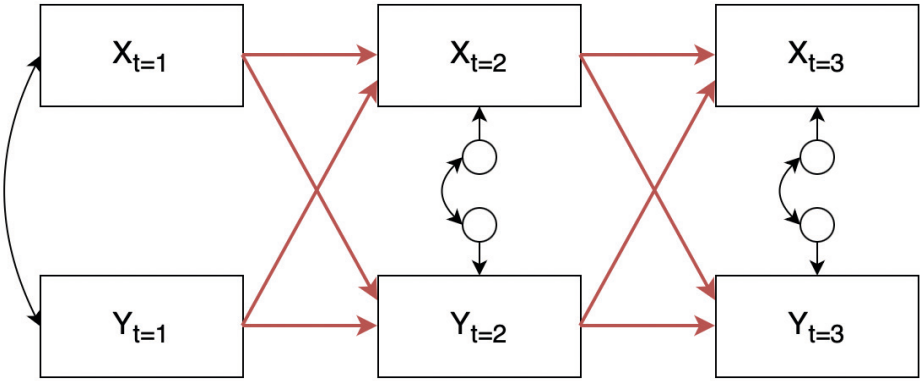
Panel Data Models

Panel data models differ with respect to their structure (e.g., use of random intercepts) and the underlying model estimation procedure (e.g., use of regularization). We describe differences in structure (see path diagrams) and estimation for each model below.

(1) Cross-lagged panel model (CLPM)

The CLPM consists of two core parts: (1) Autoregressive effects (i.e., one variable predicting itself at a later time point) describing the stability of a measure, and (2) linear cross-lagged effects that denote the effect of one variable on another variable within the same time frame and at a later one (see Figure 1). These cross-lagged effects (i.e., $X_{t=1} \rightarrow Y_{t=2}$) are regression coefficients that control for the prior level of the predicted variable ($Y_{t=2}$). The model requires at least two time points of data. For a detailed description of the CLPM, see Selig & Little (2012). Its main limitation is the inability to separate within- and between-person effects. Thus, it is unclear whether the resulting temporal effects reflect true within-person effects or whether they may be due to differences between people. Depending on the extent of true within- and between-person sources of variance, the temporal effects may reflect a mixture of effects that is difficult to interpret (Berry & Willoughby, 2017). The model fit can be evaluated using standard SEM indices of model fit, including the Root Mean Squared Error (RMSEA), comparative fit index (CFI), and the Tucker-Lewis Index (TLI). Standard criteria of good model fit (e.g., RMSEA < 0.05, CFI > 0.95, TLI > 0.95) are described elsewhere (Kline, 2005; Sivo et al., 2006). Alternatively, the model parameters can be estimated by performing a series of multiple regression analyses (one for each variable measured at t=2 with all variables measured at t=1 as predictor).

Figure 1. Cross-lagged panel model path diagram.

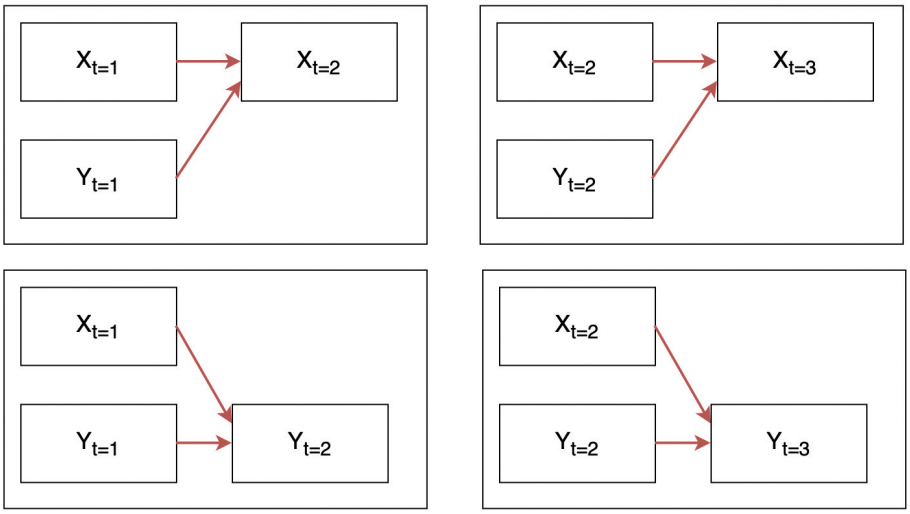


Note. Observed variables are included as squares. Small circles represent innovation terms. t refers to the measurement time point. Directed curved arrows represent marginal covariances. Network visualizations typically visualize the autoregressive and cross-lagged estimates (see red arrows) in a directed temporal network.

(2) **Cross-lagged panel network model (CLPN)**

The CLPN (Wysocki et al., 2022) is structurally similar to cross-lagged panel models (see Figure 2). By regressing every variable on itself and all other at the previous time point, the model estimates autoregressive and cross-lagged estimates. The model requires at least two time points and estimates cross-lagged and autoregressive effects separately for each changepoint (i.e., $t_1 \rightarrow t_2$). Considering the typically large number of parameters being estimated, CLPN also includes Least Absolute Shrinkage and Selection Operator (LASSO) regularization to shrink parameters to zeros, thus resulting in a sparse temporal network structure networks with a lower probability of false-positive edges (Funkhouser et al., 2021; Wysocki et al., 2022). This process of regularization is common in network models that estimate many parameters (i.e., conditional associations between nodes) with only a limited number of observations. There are two common techniques for selecting the appropriate level of regularization (lambda parameter λ): (1) selecting the λ that yields the lowest Extended Bayesian Information Criterion (EBIC), or (2) using cross-validation to choose the λ-value that performs best across the validation sets. CLPN uses LASSO regularization with 10-fold cross-validation is used to determine the optimal tuning parameter in the regularization process. This initial version of the CLPN (with code first published on OSF in 2017) has been used widely in the extant literature (e.g., see Freichel et al., 2024; Funkhouser et al., 2021; Schlechter et al., 2022). A recently revised version of the CLPN (Wysocki et al., 2022, version 2 uploaded in June 2024) also includes an additional SEM pruning step to obtain non-regularized estimates. In this pruning step, the paths that the regularized model identifies as zero are set to zero, and the model is re-estimated and inspected for statistically significant paths.

Figure 2. Cross-lagged panel network model path diagram.



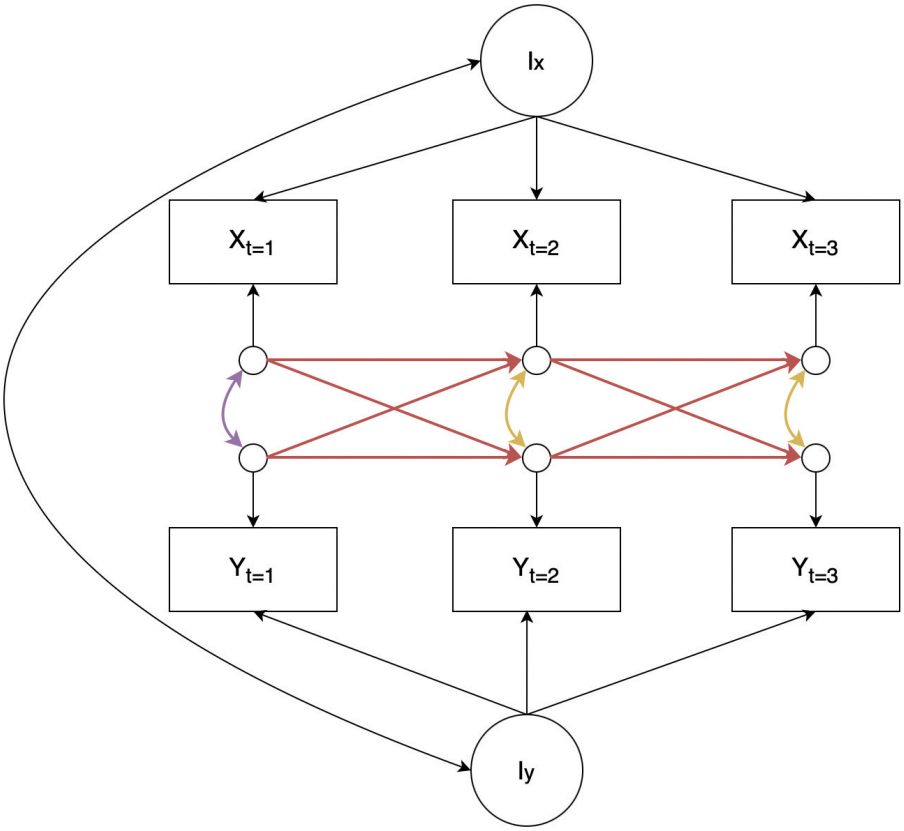
Note. Squares represent observed variables. t refers to the measurement time point. The figure shows a CLPN for studying changes from t1 to t2, and from t2 to t3. This consists of separate models per changepoint (left panel: t1-t2; right panel: t2-t3). Every node at the second time point is regressed on the nodes from the first time point. Network visualizations typically visualize the autoregressive and cross-lagged estimates (see red arrows) in temporal networks (t1-t2; t2-t3) .

(3) **Random-intercept cross-lagged panel model** *(RI-CLPM)*

The RI-CLPM is an extended CLPM that includes a random intercept (see Figure 3) for each individual (Hamaker et al., 2015) and requires typically at least three time points of data for model identification. The random-intercept captures stable, time-invariant between-person differences, representing variance that does not vary within but only between people. The resulting within-person temporal effects (i.e., cross-lagged and autoregressive estimates) describe fluctuations around individuals' mean scores. The measurement error variances for these within-person latent effects are typically constrained to zero to facilitate model identification. The random intercepts are included as latent variables with the individual observations as indicators. The factor loadings can be fixed to 1 to ensure that the model is identifiable, and the random intercept can be interpreted as the average score of the individual across all time points. Both the mean structure (i.e., zero intercept or free mean on intercepts) and the parameters (variant/invariant across time) can either be fixed or be freely estimated. Mulder and Hamaker (2021) recently introduced three key extensions of the RI-CLPM, namely models that allow for 1) integrating time-invariant predictors, 2) multiple-group model estimation, and 3) multiple-indicator estimation. Concerning the stationarity of processes, the RI-CLPM offers flexibility in its specification. The model either specifies the grand means of variables (across participants) as time-varying or as stationary/invariant over time (Mulder & Hamaker, 2021). Comparing the constrained (i.e., time-invariant means) and unconstrained models using chi-square difference tests (Kline, 2005) may allow researchers

to judge whether this stationarity in means is appropriate. Similarly, it is possible to test whether the cross-lagged effects change over the measurement period (i.e., time points) by estimating unconstrained and constrained (i.e., time-invariant cross-lagged effects) models. These sequential model comparisons may inform model specification decisions regarding which constraints should be included in the model, and whether stationarity of means and covariances is appropriate. Empirical researchers may consider starting with unconstrained model and then continually add constraints to compare the model fit.

Figure 3. Random-intercept cross-lagged panel model path diagram.

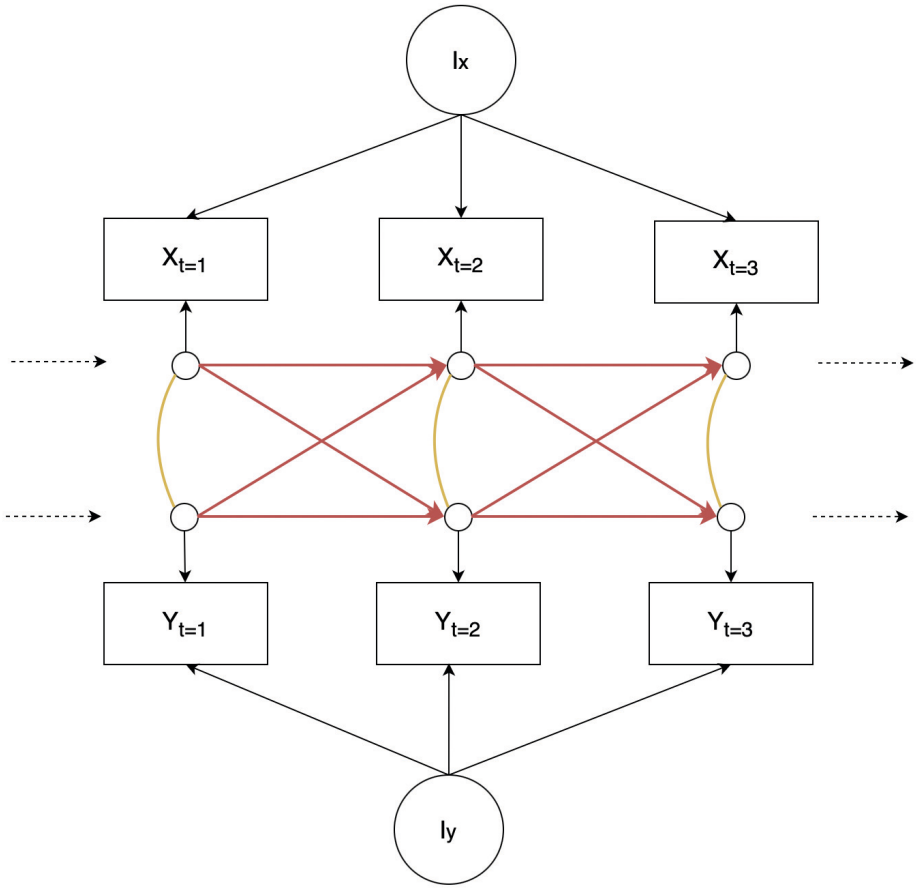


Note. Squares represent observed variables. Large circles represent random intercepts. Small circles represent innovation terms. t refers to the measurement time point. Directed curved arrows represent marginal covariances. The marginal covariances at t=1 have been colored in purple as it is distinct from the covariances at other time points. The random intercepts are intercorrelated. Network visualizations typically visualize the residual co-variances in an undirected contemporaneous network (see yellow circular arrows) and the autoregressive and cross-lagged estimates (see red arrows) in a directed temporal network.

(4) **Panel Graphical Vector Autoregression Model (panel GVAR)**

Panel GVAR models (Epskamp, Waldorp, et al., 2018; Epskamp, 2020) can be used to identify partial within-person temporal effects based on at least three time points of data. Every variable is predicted by itself and the past values of all other variables (cross-lagged effects) as well as the person-wise mean for that variable over time. The panel GVAR model is structurally similar to the RI-CLPM (see Figure 4) with three important differences. First, the within- and between-person covariance structures are modeled through Gaussian Graphical Models (GGMs), leading to a (within-person) contemporaneous network based on the covariance structure of the innovations, and a between-person network based on the covariance structure of the random intercepts/means (Epskamp, 2020). Second, model parameters, including the intercepts and network parameters, are treated as stationary and invariant over the entire period of measurement. This stationarity assumption poses that the same variance-covariance structure holds for each time point. Finally, as a result of the strong stationarity incorporated in the model, the first time point is treated differently in the panel GVAR compared to the RI-CLPM. In panel GVAR, the first time point is treated as endogenous (i.e., does not have its own variances and covariances): the variance-covariance structure is based on the (stationary) temporal and contemporaneous structures (Epskamp, 2020). This model specification implies that the first time point may be influenced by other preceding time points (i.e., a stationary VAR model underlying it). As a result of these differences, panel GVAR is less flexible than the RI-CLPM to handle non-stationarity, but at the same time it also leads to more generalizable results outside the period of study. To facilitate stationarity, the data can be detrended for possible linear and non-linear trends (Freichel et al., 2023; Speyer, Ushakova, et al., 2022). This involves regressing out the effect of time on all variables, or alternatively standardizing variables at all waves of data. It should be noted, however, that doing so might artificially inflate model fit, as stationarity is part of the panel GVAR model and not an assumption. Before estimating the model, researchers should first inspect trends in means and variances across time points for each variable. Moreover, estimating cross-sectional (i.e., between-person) networks/ GGMs at each measurement time point separately may provide a snapshot for judging the assumption of stationary co-variances. When estimating panel GVAR models, it is important to consider different model selection procedures (see Blanken et al., 2022 for a complete overview). After estimating an unconstrained saturated model (including all possible edges), researchers may use thresholding (i.e., hiding non-significant edges) or pruning (i.e., fixing non-significant edges to zero and re-estimating the model) procedures. A detailed description of the (multi-level) GVAR model can be found elsewhere (Epskamp, van Borkulo, et al., 2018; Epskamp, Waldorp, et al., 2018). The panel GVAR model can be estimated using the psychonetrics R package (Epskamp, 2021). We provide relevant code to estimate a panel GVAR model with default specifications in the appendix.

Figure 4. Panel GVAR Model Path Diagram.



Note. Squares represent observed variables. Large circles represent random intercepts. Small circles represent innovation terms. t refers to the measurement time point. Undirected curved arrows represent partial covariances. Directed curved arrows represent marginal covariances. The dashed arrows indicate a continuous and stationary VAR model. Network visualizations typically visualize the innovations in an undirected contemporaneous network (see yellow lines) and the autoregressive and cross-lagged estimates (see red arrows) in a directed temporal network.

The four models described above differ with respect to various characteristics, with each model possessing distinct advantages and limitations. Table 1 provides an overview of these characteristics, the estimated network structures, and relevant software for estimating the models.

Table 1. Different Structural Equation and Network Panel Data Models.

Model	Estimated network structures	Separates within and between	Required time points	Advantages (+) and Constraints (-)	Software
Cross-lagged panel model (CLPM)	* Temporal * Contemporaneous	No	≥2	- No separation of within- and between-person effects + Simplicity and ease of interpretation + Easily accommodates time-invariant, exogenous covariates	Any SEM software (e.g., lavaan in R, Mplus)
Cross-lagged panel network model (CLPN)	* Temporal	No	≥2	- No separation of within- and between-person effects + Easily accommodates time-invariant, exogenous covariates + Simplicity and ease of interpretation	R (glmnet)
Random-intercept cross-lagged panel model (RI-CLPM)	* Temporal * Contemporaneous * Between	Yes	≥3	- Computationally demanding with many variables + Separates within- and between-person effects + Can account for patterns of change over time + Allows for manual specification of model constraints (e.g., constrained means or cross-lagged effects)	Any SEM software (e.g., lavaan in R, Mplus)
Panel graphical vector autoregression model (panel GVAR)	* Temporal * Contemporaneous * Between	Yes	≥3	- Includes stationarity in model specification - Computationally demanding with many variables + Separates within- and between-person effects	R (psychonetrics)

Note. The panel GVAR model yields partial contemporaneous, and between-person effects. The temporal effects derived from the CLPN, CLPM, RI-CLPM, and panel GVAR models can be considered partial. The contemporaneous and between-person estimates from the CLPM and RI-CLPM represent marginal, not partial associations.

In addition to the constraints outlined in Table 1, several limitations apply to all models. These SEM and network panel data models estimate linear temporal effects and capture lag-1 processes (i.e., $X_{t=1} \rightarrow Y_{t=2}$, but not $X_{t=1} \rightarrow Y_{t=3}$). Thus, the models cannot accommodate non-linear effects or processes that occur at other time lags. All models (assuming at least three time points and constrained time-invariant cross-lagged effects) assume 'equidistance,' that is, equally spaced time points, and deviations from this assumption can lead to biased estimates (Kuiper & Ryan, 2018).[1] With respect to missing data, the CLPM, RI-CLPM, and panel GVAR models allow for missing time points and the use of different estimators, such as Full-Information-Maximum-Likelihood (FIML) to account for missingness.

An empirical example

Thus far, we have described different panel data models, and their unique characteristics, advantages, and constraints. To illustrate how these different models may yield different temporal associations and substantial interpretations, we showcase their use in an easily accessible dataset. Data stems from the NSPN 2400 cohort study (Kiddle et al., 2018), a longitudinal panel dataset that contains responses from over 2,000 participants who have been assessed at three time points that were each approximately 12-13 months apart. Participants were adolescents and young adults (aged 14-24) living in Cambridgeshire and Greater London (United Kingdom). The project received ethical approval by the National Health Service Research Ethics Committee (#97546). A description of the sample characteristics, cohort profile, and study procedures appears elsewhere (Kiddle et al., 2018; Polek et al., 2018; Wiedemann et al., 2023).

Research question and methodology. Our illustrative research question focused on examining the links between impulsiveness (i.e., comprised of different facets, such as attentional impulsivity or self-control) and symptoms of depression and anxiety. High impulsiveness was shown to be a risk factor for the development of anxiety and depression (Granö et al., 2007; Zhou et al., 2014). Research points to evidence that impulsivity predicts depressive/anxiety disorder symptoms (Boschloo et al., 2013). However, the reverse direction (anxiety and, to a lesser extent, depression predicting impulsivity) has also been found in young people (Moustafa et al., 2017). The present illustration aimed to identify temporal associations between impulsiveness and symptoms of anxiety and depression. Impulsiveness was assessed using the BIS-11 version of the Barrat Impulsivity scale (Patton et al., 1995). Participants rated all 30 items on a scale from rarely to always, and a total sum score was computed. Supplemental analyses were conducted using the respective six first-order subscales (i.e., sum scores). These subscales assess different facets of impulsivity, including attention, motor impulsiveness, lack of self-control, cognitive complexity, perseverance, and cognitive instability (Patton et al., 1995). Higher scores indicate greater levels of impulsiveness. Anxiety was assessed using the Revised Children's Manifest Anxiety Scales (RCMAS, (Reynolds, 1980; Reynolds & Richmond, 1978; Shahar et al., 2021). This 28-

1 Of note: SEM based approaches and the panel GVAR can relax the assumption of equidistant measurements somewhat by modeling missing waves of data as latent variables.

item self-report measure assesses different anxiety symptoms. We calculated sum scores for the three subscales (physiological anxiety, worry/oversensitivity, and social anxiety), with higher scores indicating more severe levels of anxiety. Lastly, depression symptoms were assessed using the 33-item self-report Moods and Feelings Questionnaire (MFQ, Costello & Angold, 1988). This screening tool for depression assesses the presence of depression symptoms, and a single-sum score was calculated. Higher scores indicate more severe depression symptoms. We included all measures (i.e., sum scores) as observed variables in the different panel models, and thus, the models cannot capture measurement error. To facilitate visual comparison, we only report temporal effects that are displayed in network visualizations that include cross-lagged (i.e., directed arrows in-between different nodes) and autoregressive (i.e., curved arrows on the same node) effects. We have used FIML to account for missingness in CLPM, RI-CLPM, panel GVAR, and complete case analysis for CLPN.

CLPM. The CLPM was estimated using the lavaan R package (see code in supplementary materials). It includes autoregressive paths (from each time point to the subsequent one), cross-lagged paths, and within-time covariances. The estimates are not constrained between subsequent time points, allowing each path coefficient to be freely estimated. The model fit was acceptable (RMSEA = 0.06, CFI = 0.99, TLI = 0.96). The temporal network is shown in Figure 5A, and it visualizes beta estimates for the change from t_1 to t_2. Higher levels of physiological anxiety and depressive symptoms predicted more impulsiveness over time. Both physiological and social anxiety symptoms predicted higher levels of depressive symptoms.

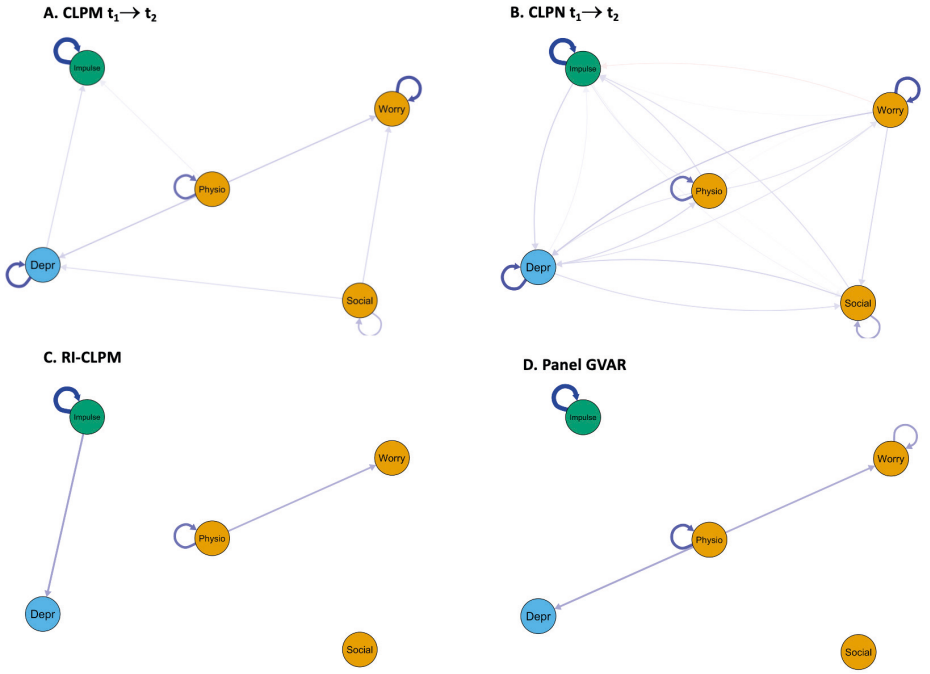
CLPN. Next, we estimated CLPN separately for both changepoints (i.e., time point 1 to time point 2, time point 2 to time point 3). We based the estimation on the initial version of the CLPN with code published in 2017 on OSF. This version of the CLPN (i.e., series of regularized regressions) has been used widely in the extant literature (e.g., see Freichel et al., 2024; Funkhouser et al., 2021; Schlechter et al., 2022). The model uses 10-fold cross-validation to select the λ-value with the lowest squared error for the LASSO regularization. The temporal network (Figure 5B) visualizes regularized beta regression coefficients for the first changepoint is shown in Figure 5B). The dense network reveals a more complex pattern of associations, with reciprocal associations between impulsiveness, physiological anxiety, and depressive symptoms. Higher levels of worry/oversensitivity predicted less impulsiveness. The CLPN emphasizes the interconnectedness of impulsiveness with both anxiety and depressive symptoms, highlighting multiple ways through which impulsiveness and symptoms are interrelated.

RI-CLPM. The RI-CLPM was estimated using the lavaan R package with default specifications, including measurement error variances constrained to zero. The means of the observed variables were modeled through the random intercepts. The model showed an excellent fit to the de-trended data (RMSEA=0.05, CFI=0.99, TLI = 0.98). The temporal network (Figure 5C) shows partial directed correlations. Impulsiveness predicted more depressive symptoms over time. However, the panel GVAR model showed no cross-construct associations between anxiety symptoms and impulsiveness.

Panel GVAR. We first fitted a saturated panel GVAR model (i.e., including all edges) to the data using default specifications, including the use of GGMs for modeling the between-

person and within-person contemporaneous networks. In an additional pruning step, we removed all non-significant edges (at an alpha level of 0.01). The model showed a good fit to the data (RMSEA=0.05, CFI=0.98, TLI=0.96), and thus, detrending (i.e., removing linear/quadratic effects of time) was not considered appropriate. The temporal network derived from the panel GVAR model depicts partial directed correlations (see Figure 5D). Similar to the RI-CLPM, there were no temporal associations between anxiety symptoms and impulsiveness. Moreover, depressive symptoms and impulsiveness were unrelated.

Figure 5. Temporal Networks Based on Panel Data Models.



Note. RI-CLPM = Random-Intercept Cross-Lagged Panel Model, Panel GVAR = Panel Graphical Vector Autoregression Model, CLPM = Cross-Lagged Panel Model, CLPN = Cross-Lagged Panel Network Model. The color of the nodes refers to the domain that the variables belong to (orange = anxiety, blue = depression, green = impulsiveness). The thickness and color of the edges describe the strength and direction of associations respectively. Nodes are colored according to the domain that they belong to. The figure visualizes partial directed correlations for the RI-CLPM and panel GVAR models, and beta-estimates for the CLPM and CLPN models. **Physio** = Physiological anxiety, **Worry** = Worry/oversensitivity, **Social** = social concerns/concentration, **Depr** = Depression symptoms, **Impulse** = Impulsiveness.

Simulation study

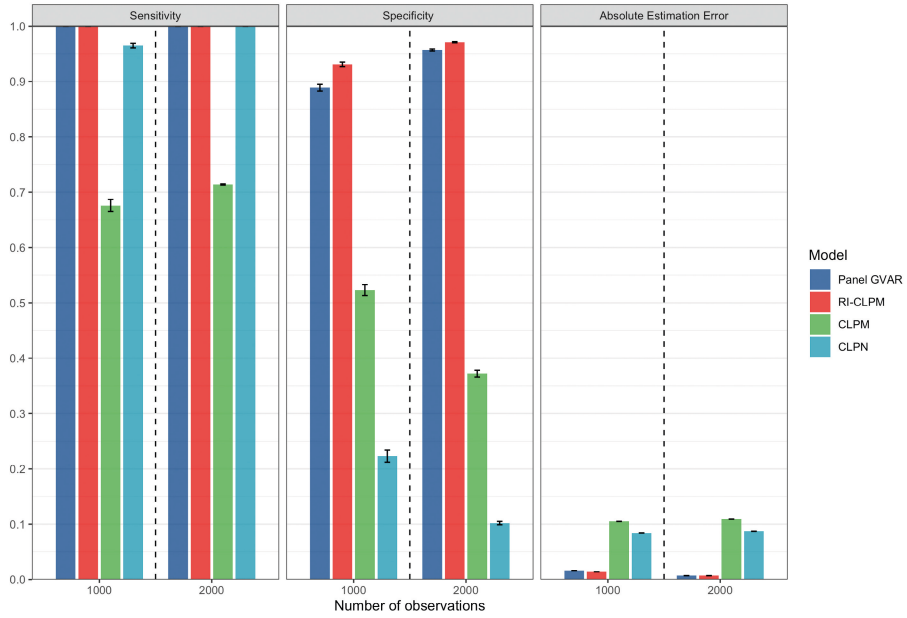
The results from our empirical illustration showed similar network structures for panel GVAR and RI-CLPM, and stark differences when comparing the temporal relations to the results obtained from CLPM and CLPN. For instance, impulsiveness and physiological anxiety symptoms showed reciprocal associations in CLPN but were unrelated in the models that

separate within- and between (RI-CLPM, panel GVAR). Researchers may thus wonder which model approximates the true within-person temporal effects most closely.

To further investigate whether these results may indeed reflect differences in the models' capacity to uncover true within-person effects, we employed a simulation approach: We simulated a true network structure based on three time points, seven nodes, and reasonable within-person edge weights (i.e., 0.4 for autocorrelations, cross-lagged effects varying in simulations between -0.2 to 0.2) in a stationary time-series. Based on these conditions, we sampled data for different number of observations (n = 2000-3000). In each iteration (out of 100), all four models were estimated based on the sampled data, and we computed measures of sensitivity, specificity, and the mean absolute estimation error. Sensitivity describes the true positive rate, indicating the proportion of existing edges in the true model that were correctly identified in the estimated model. Specificity refers to the true negative rate, indicating the proportion of absent edges in the true model that were correctly identified as such in the estimated model. The mean absolute estimation error describes the average magnitude of deviation between the estimated edge weights and the true edge weights. To the best of our knowledge, the present simulation study is the first to evaluate such diagnostic metrics for CLPN. The specifications of the different models used in the simulation study are identical to the ones used for the empirical illustration above. We used thresholding (with alpha = 0.05 for panel GVAR, CLPM, and RI-CLPM, and estimated unconstrained models (i.e., no fixed mean or cross-lagged variances) for CLPN. Results from our simulation study are shown in Figure 6. The panel GVAR and RI-CLPM recovered the true within-person temporal effects across different sample sizes to a large extent (i.e., above 95%). However, both the CLPM and CLPN analyses showed higher rates of estimation error and low rates of specificity, indicating that they identified many false positive edges (i.e., associations not present in the true model). This finding is consistent with our empirical illustration that showed more dense network structures (concerning the number of edges present) in these models. Moreover, this result is consistent with prior reports indicating that stable trait-level between-person effects may lead to spurious temporal associations when using models that cannot separate within- and between-person effects (Lucas, 2023).

Based on these simulations, we conclude that researchers interested in uncovering true within-person cross-lagged effects should use models that can separate within- and between-person effects. The traditional CLPM and the recent CLPN adaptation produce many spurious temporal effects, especially when strong between-person associations are expected. Thus, temporal effects from these models should not be interpreted as mechanistic effects occurring within individuals over time.

Figure 6. Results from our simulation comparing different models.



Note. RI-CLPM = Random-Intercept Cross-Lagged Panel Model, Panel GVAR = Panel Graphical Vector Autoregression Model, CLPM = Cross-Lagged Panel Model, CLPN = Cross-Lagged Panel Network Model. Sensitivity indicates the true positive rate; specificity indicates the true negative rate; absolute estimation error reflects the accuracy of the models (i.e., deviation of estimated and true edge weights). The error bars indicate standard errors.

Other challenges in panel data modeling

Besides distinguishing between within- and between-person effects, there are other common challenges in the estimation of panel data models. First, cross-lagged panel models identify patterns of covariance, thus, making it essential to have adequate variance in all incorporated variables for identifying temporal relations. This may create challenges when researchers are interested in studying certain phenomena that naturally show less variance in general population samples, as in the case of suicidal ideation. Second, cross-lagged effects can only be understood within the context of the 1) time scale of the individual measures (e.g., number of drinks in the past week), and 2) the time-lag (e.g., two weeks) in-between time points (Bringmann et al., 2022). Estimating cross-lagged relations between measures that occur at vastly different or substantially slower or faster time scales may thus lead to a biased interpretation of estimates.

Concluding comments

The present overview focused on four commonly used cross-lagged panel models for studying the co-development of various constructs. Needless to say, there are other panel data models that focus on describing mean changes or trajectories across time, including

survival or growth curve mixture models (Ebrahimi et al., 2023). Moreover, latent change score/latent growth curve models (Kievit et al., 2018) provide an alternative approach that estimates intercepts and latent growth parameters for every variable. It goes beyond the scope of the present article to discuss these models that focus on developmental growth, however, there are recent approaches that combine the growth curve and cross-lagged developmental perspectives. A recent extension of this model includes the integration of covariances for latent growth curve model parameters (intercepts, slopes) into GGMs (see Deserno et al., 2021). In addition, separate correlated symptom change (slope) networks (Crowe et al., 2023) have been used to visualize conditional between-person associations in symptom change over time. In these undirected between-subject person slope networks, edges describe the extent to which individuals who report greater than average change in one node also report greater than average change in other nodes.

Our methodological review, empirical illustration, and simulations highlighted the substantial heterogeneity in temporal estimates derived from different panel models. Thus, applied researchers should carefully select their analytical approach, taking into account the availability of data at hand (e.g., the number of time points) and the particular research objective. With data from three or more time points, researchers can select from the four models discussed in this paper. Three waves of data are necessary to be able to distinguish between covariance that is due to a temporal effect as opposed to stable averages. Researchers interested in mere prediction across time or identifying stable between-person risk factors may use panel data models that conflate within- and between-person effects. However, when the focus is on understanding mechanisms (i.e., processes unfolding over time) or testing within-person developmental theories (i.e., questions about how an elevated score on one variable compared to their own average predicts another variable), researchers should employ analytical approaches that separate within- and between-person effects. As described in detail by Orth et al. (2021), the conceptual meaning of cross-lagged coefficients differs between models: CLPN and CLPM focus on how individual differences/variation in X changes individual differences/variation in Y. In contrast, panel GVAR and RI-CLPM focus on how deviations from individuals' trait level in X affect deviations (from within-person average) in Y. Lastly, when the focus is on understanding partial directed correlations (i.e., the unique contribution of different nodes) as opposed to zero-order estimates, researchers should adopt recently introduced panel network approaches. When designing new panel studies aimed at investigating research questions about symptom evolution, symptom co-dependence, and tracking mechanisms of intervention and disease progress, researchers should first determine the 1) time-scale at which processes occur (ranging from minutes to decades), 2) the time-interval in-between measurement time points, and 3) the number of measurement occasions (Hamaker, 2023). These design choices can ultimately help inform the analytical strategy. If feasible, researchers may use multiple approaches simultaneously within the context of multiverse analyses to convey the degree to which specific findings depend on analytical choices. Ideally, convergent evidence from different approaches can inform the robustness of specific findings. By openly acknowledging and exploring the impact of different methodological choices, researchers can provide a more nuanced view of their data.

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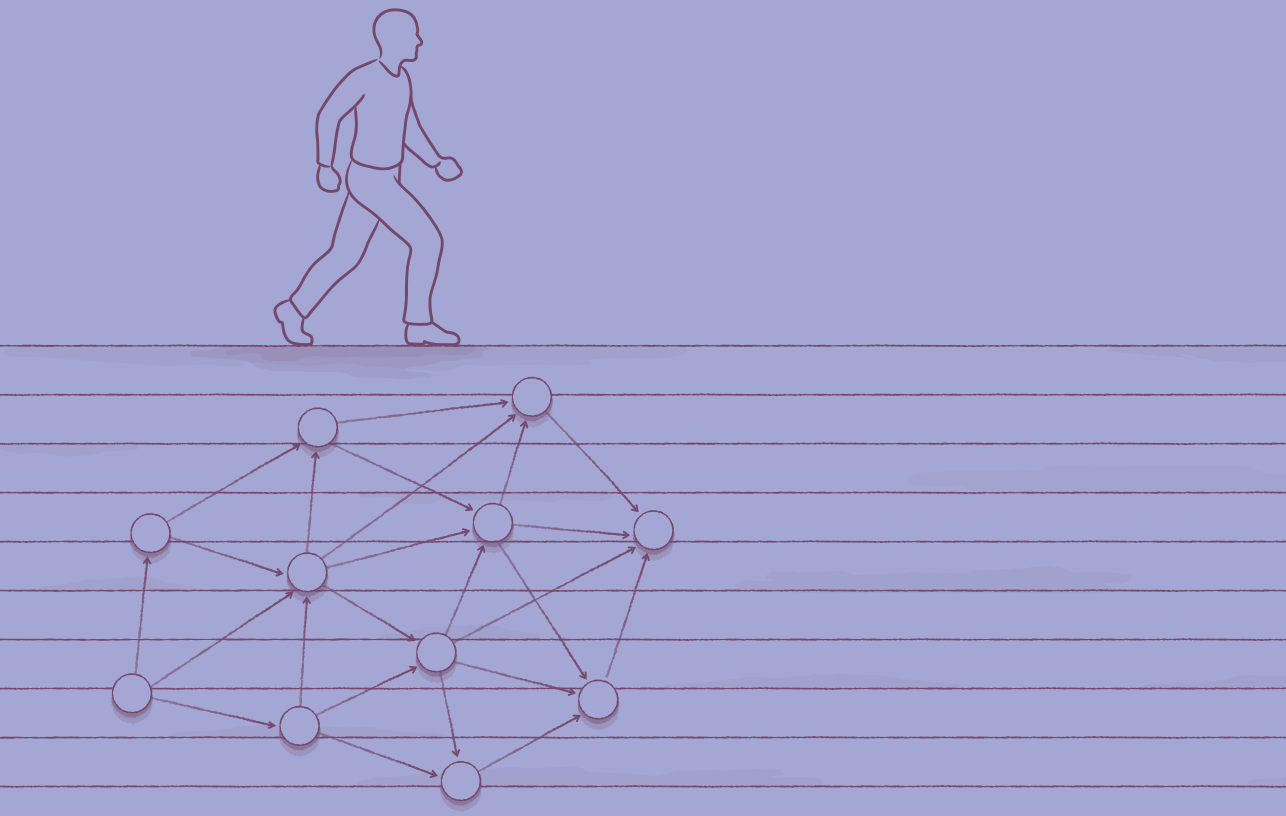

CHAPTER 10

Preregistration Guidelines for Longitudinal Network Analyses

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Abstract

Over the past decade, longitudinal network analyses have grown in popularity in psychological science. These approaches, applied to intensive time-series or panel data, require a high level of flexibility and involve numerous modeling decisions, which can introduce considerable degrees of freedom into the process. Despite their growing use in confirmatory research, a notable lack of preregistration guidelines remains. We propose a preregistration checklist tailored to longitudinal network analyses, addressing dynamic modeling choices and preprocessing decisions not captured by general preregistration frameworks. The checklist is designed to be a resource for both authors and reviewers, ensuring that all critical aspects of the preregistration process are adequately addressed. We specifically focus on four widely used models: Panel Graphical Vector Autoregression (VAR) models, cross-lagged panel network analysis (CLPN), multilevel VAR (mlVAR), and Group Iterative Multiple Model Estimation (GIMME). We offer guidance on preregistering studies using these approaches to mitigate biases and enhance transparency.

Keywords: longitudinal network analysis, preregistration, open science, network modeling

Introduction

The past decade of psychological science have witnessed the emergence of network psychometrics (Borsboom & Cramer, 2013; Cramer et al., 2010), a field featuring the use of network analysis in psychological research. Network analytic approaches have flourished, especially in clinical psychological science (see review by Robinaugh et al., 2020). Initial studies involved many participants whose symptoms were assessed at a single point in time (e.g., Robinaugh et al., 2014). Hence, these exploratory investigations were restricted to cross-sectional data and focused on identifying the (between-person) network structure depicting symptom co-occurrence. Recently, researchers have increasingly estimated temporal (or 'lagged') networks based on intensive longitudinal data (e.g., time-series or ecological momentary assessment [EMA], often comprising at least 20 assessment points per participant), as well as longitudinal panel data (typically three to ten assessment points per participant; Epskamp, 2020). The term 'lagged' is commonly used in the vector autoregression (VAR) literature, where it refers to the inclusion of past values of variables to predict future values. Longitudinal approaches depict symptom dynamics over time, highlighting how symptoms prospectively predict each other. Moreover, longitudinal methods enable exploration of intra-individual dynamics that reveal patterns of excitatory and inhibitory links among symptoms within a certain individual. Insights derived from such longitudinal network models promise to address clinically relevant questions about dynamic symptom interplay (McNally, 2021). For example, a recent study by Hoffart et al. (2023) showed that stronger metacognitive beliefs (i.e., those concerning the danger of thoughts and feelings) predicted elevated symptoms of anhedonia across a two-month window, highlighting a potential target for interventions.

Although most cross-sectional and longitudinal network analysis studies have been exploratory and hypothesis-generating, there is growing interest in confirmatory network modeling (Epskamp et al., 2017; Kan et al., 2020). Confirmatory modeling may refer to 1) estimating a network where certain relations are prespecified based on theory or prior evidence, analogous to a VAR model with fixed parameters set to zero for non-hypothesized connections, or 2) a situation where researchers have hypotheses about expected patterns in the network that are obtained using a data-driven method. Inspired by the open science movement, preregistration has become the norm in some areas of psychology (Nosek & Lindsay, 2018) and is increasingly acknowledged as a means of promoting replicable science (Nosek et al., 2018). Prominent journals, including Psychological Science, actively promote the practice by awarding 'preregistration' badges for studies that specify hypotheses, research questions, variable selection, and data analysis plans. This aims to separate hypothesis generation from hypothesis testing, thereby mitigating the risk of bias arising from the problematic practice of 'HARKing: Hypothesizing After the Results are Known' (Kerr, 1998). Preregistration guidelines exist for psychopathology research in general (Krypotos et al., 2019) and for specific subfields, such as event-related potentials research (Paul et al., 2021) and experience-sampling studies (Kirtley et al., 2021). The practice of preregistering network analytic studies has become increasingly common (Ebrahimi et al., 2021; Freichel

et al., 2023; Zainal & Newman, 2022). However, there are no standard guidelines for preregistering longitudinal network analysis studies. Burger et al. (2023) outlined important reporting standards for network analysis studies, with a focus on cross-sectional networks, though preregistration was only briefly addressed. The present article aims to offer actionable advice on key aspects to consider when preregistering longitudinal network analysis studies. Furthermore, to balance the inherent exploratory nature of network analysis with preregistration rigor, we recommend a tiered approach that locks in core hypotheses and preprocessing steps while defining a transparent flexibility window for modeling parameters

Should longitudinal network analysis studies be preregistered?

The question of whether to preregister longitudinal network analysis studies is complex and nuanced and often rests on the nature and objectives of the study. Importantly, preregistration is most beneficial when the analysis is hypothesis-driven and aims to test a-priori theories. In such cases, preregistration may mitigate bias and increase the credibility of the findings. For example, consider a network study designed to test the risk factor account of loneliness in depression (Cacioppo et al., 2006). According to this perspective, increases in loneliness should predict higher levels of depressive symptoms, rather than the reverse pathway suggested by the consequence account. In this scenario, researchers may be advised to preregister their predictions (e.g., loneliness nodes predicting depression nodes) and data analysis plan. Generally, there are two types of hypotheses that researchers can delineate when preregistering a longitudinal network analysis study. First, researchers might want to make predictions about specific edges of interest, for example, to hypothesize that loneliness will directly predict symptoms of depression, such as anhedonia and fatigue. In larger networks, hypotheses about specific edges may be of less interest. Instead, researchers might have hypotheses about 1) the clustering of nodes. For instance, a recent study hypothesized the presence of adolescent depressive symptom clusters that, in turn, would differentially predict treatment outcomes (Kim et al., 2024). There may also be hypotheses concerning 2) which nodes (i.e., variables) are the most prominent with respect to their ability to activate or inhibit other nodes in the network (e.g., centrality measures, such as out-strength). A study by Ma et al. (2022) hypothesized that low mood and decreased interest would represent the most central depression symptoms in the network. Lastly, hypotheses may concern 3) overall network features, such as density or global efficiency. For example, one study hypothesized that individuals with depression would exhibit a more densely connected emotion network than healthy adults.

Even in exploratory settings, preregistration may help the reader understand (i) which modeling decisions were planned, and which emerged ad-hoc, and (ii) which results emerged during analysis as opposed to being predicted in advance. There are, however, several inherent challenges with preregistering longitudinal network analysis studies: (1) Network analysis can serve as an exploratory and hypothesis-generating toolbox to uncover patterns of associations among symptoms and related factors that might play a part in mental health syndromes (Freichel, 2023). However, to harness their potential as an exploratory toolbox, researchers need to employ data-driven procedures, for example, to use model

search algorithms to find the best fitting model (e.g., model selection based on the lowest Bayesian Information Criterion during cross-validation). (2) Longitudinal network analysis methods are complex statistical approaches that require investigators to manually specify numerous modeling decisions, such as the way in which the innovation variance-covariance structure is modeled (e.g., Gaussian Graphical Model). Depending on one's data, it may be necessary to explore different modeling decisions to facilitate model identification and obtain interpretable estimates. For instance, a simulation study by Freichel and Epskamp (2024) shows that using Cholesky decomposition for modeling between-person covariances may be beneficial (i.e., yielding credible contemporaneous and temporal effects) in situations where investigators encounter problematic between-person estimates. A strict preregistration for these statistical modeling decisions may constrain the flexibility needed to navigate the data-driven exploration process. (3) The software used for network estimation based on time series and panel data is still in its early stages, possibly in beta versions, and is subject to updates with ongoing technical and methodological developments.

These important issues highlight the need for flexibility when conducting longitudinal network analyses. The inherent need for flexibility is somewhat contradictory to the purpose of preregistration – that is, to restrict the degrees of freedom. Despite these seemingly conflicting goals, a middle ground can be found. Many of the essential components pertaining to dynamic network analyses can be preregistered. Primarily and most importantly, a) researchers can pre-specify the system of variables they seek to investigate in their planned network model, and b) provide the theoretical rationale for why they are investigating this question by using these variables and accompanying practical operationalizations (Ebrahimi et al., 2021; Freichel et al., 2023). Reporting these two aspects may improve the quality of the research and the credibility of the findings, as they prevent researchers from tweaking and modifying variables and measurements after the results are known. A focus on conditional analyses (Lakens, 2024) may allow researchers to follow alternative analysis plans based on potential contingencies. A decision tree (with if-then rules) may describe the respective analyses and the corresponding assumptions. For instance, "If the model estimated on the raw data shows poor fit, then we will detrend the time series and refit the model". Understanding the preregistration as a set of plausible modeling decisions rather than a single predefined path may provide a practical solution to the required flexibility in longitudinal network modeling.

In addition, researchers can also c) preregister data processing strategies, such as specifying whether and how trends in data (e.g., linear trends), which violate the central assumption of stationarity in many network (e.g., VAR) models, are to be addressed (e.g., regressing out the linear trend by regressing on the day-count variable in daily diary studies). They may d) specify which network analytic technique they aim to use, as many are available for many dynamic network analyses (e.g., mlVAR or structural VAR techniques for N > 1 time-series data). It is possible to preregister alternative analyses (for example, "if problematic between-person estimates will be encountered, an alternative estimation using Cholesky decomposition (Freichel & Epskamp, 2024) will be used").

Investigators also can e) specify planned network inferences, such as which variables they find important with respect to their overall connectivity with other variables in the

system (e.g., strength centrality), and whether a certain node is more likely to predict (e.g., out-strength) or be predicted (e.g., in-strength) by other variables in the system. When investigators have strong hypotheses about the presence of single edges (e.g., suicidal ideation with worthlessness), these can be specified. However, it is infeasible to pre-specify all possible connections in network models. For example, a 15-node network (n = 15) has over 225 (n^2) possible temporal edges and 105 ($n * (n-1)/2$) contemporaneous or between-subject relationships. In situations where f) researchers seek to validate specific predictions derived from a longitudinal network model by using one dataset, it is sensible to preregister the replication study in a secondary dataset. Accordingly, most aspects of dynamic network models can be pre-registered. Preregistration is an important step toward open science, and the first step toward theoretically informed (and confirmatory) network models.

Overview of longitudinal network modeling approaches

The present preregistration guidelines are tailored toward longitudinal network models estimated from time series or panel data. We focus specifically on four models (see Table 1), since they represent the most common approaches for group-level network model estimation. These models differ with respect to three aspects: the data type (e.g., intensive time series or panel), the software they require (e.g., R package), and the interpretation of their relevant estimates (e.g., edge weights in the temporal network).

There are two popular approaches for panel network estimation: Panel graphical vector-autoregression (VAR) models (GVAR) and cross-lagged panel network analysis (CLPN). These models fundamentally differ in their focus on the level of analysis: the panel GVAR model estimates within-person temporal effects, while the CLPN model does not distinguish between within-person and between-person effects. A comprehensive explanation of the differences between these models and the corresponding structural equation models - namely, the random-intercept cross-lagged panel model (RI-CLPM (Hamaker et al., 2015; Mulder & Hamaker, 2021)) and the cross-lagged panel model (CLPM) - is available in Freichel et al. (2024).

(1) Panel GVAR models estimate cross-lagged (i.e., temporal associations between different variables) and autoregressive (i.e., the same variables predicting itself over time) effects. The VAR part of the model regresses every variable on a lagged version of itself and all other variables. The multi-level part of the model allows the estimates to differ across individuals – see Bringmann et al. (2013) for a detailed description of the multi-level VAR model.

The model shares structural characteristics with the RI-CLPM as it also estimates the average value for each variable for every individual (between-person effects). The resulting covariances are modeled using Gaussian Graphical Models (GGM), yielding stationary within-person temporal (i.e., effects over time), contemporaneous (i.e., within the same time window), and between-person (i.e., interrelated stable means) estimates in the network structure. More information on the model can be found elsewhere (Epskamp, 2020; Freichel et al., 2024). This approach is particularly useful when the focus is on the within-person fixed-effects structure, pertaining to research questions about how fluctuations in different constructs relate to each over extended time periods at the within-person level. The resulting estimates refer to effects

expected to be consistent across time (assuming stationarity) and similar across a group of individuals. For example, one may be interested in examining relations between more stable constructs, such as metacognitions or chronic stress, which exhibit fluctuations over longer periods, provided that no significant changes (e.g., negative life events) occur during the study window. Conversely, examining rapidly changing constructs, such as moment-to-moment feelings of sadness or periods of heightened sensitivity (e.g., during treatment), may be less optimal for this modeling approach.

(2) An alternative panel network model is the CLPN approach (see Wysocki et al., 2022), which is based on regularized regressions with cross-validation. This model yields (interindividual) temporal estimates that do not separate within- and between-person effects. Thus, the CLPN model does not assume stationarity and can be estimated with just two time points, whereas panel GVAR requires three equidistant (i.e., similar time intervals in between all waves) time points. This model may be useful when the focus is on understanding how differences between individuals in one construct relate to interindividual differences in another construct over time. For instance, it can be used to study the population-level associations between individuals' level of physical health (e.g., number of chronic diseases) and life satisfaction over time.

For estimating networks based on intensive time-series data, researchers often use two different analytical approaches:

(3) Multi-level VAR models. This approach regresses every variable at time point t on the value of that and all other variables at the previous time point (Epskamp et al., 2019). The estimates can vary among individuals (i.e., random effects), and the model estimates the average fixed-effects structure. The random effects can be estimated as correlated (with few nodes) or orthogonal (uncorrelated, with more than about 10 nodes) for both contemporaneous and temporal effects (Burger et al., 2022).

(4) GIMME. Group Iterative Multiple Model Estimation (GIMME, (Beltz & Gates, 2017; Gates & Molenaar, 2012; Lane & Gates, 2017) has been introduced for the estimation of person-specific and group-level temporal and contemporaneous networks. This model estimates idiographic networks that are in turn used to determine the group network based on an iterative step-wise search process.

Table 1. Relevant time-series/panel network models.

Model	Data type	Software (R package)	Estimates
panel GVAR	Panel	psychonetrics (Epskamp, 2021)	· Temporal: within-person deviations from the mean (group-level) · Contemporaneous (group-level) · Between-person (group-level)
CLPN	Panel	glmnet (Friedman et al., 2017; Wysocki et al., 2022)	· Temporal: linear regression estimates (group-level)
mlVAR	Time-series	mlVAR (Epskamp et al., 2019)	· Temporal: within-person deviations from the mean (group-level) · Contemporaneous (group-level) · Between-person (group-level)
GIMME	Time-series	gimme (Lane et al., 2024)	· Person-specific and group-level temporal · Person-specific and group-level contemporaneous

Note. GVAR = Graphical Vector-Autoregression; CLPN = cross-lagged panel network analysis; mlVAR = Multilevel Vector-Autoregression; GIMME = Group Iterative Multiple Model Estimation.

Preregistration guidelines for longitudinal network analysis studies

Preregistering longitudinal network analysis studies involves the specification of a range of general and analysis-specific choices. Our complete preregistration checklist is available on the Open Science Framework (https://tinyurl.com/3m9bj7uh). Researchers and reviewers of preregistrations may use it to confirm that the preregistration addresses the necessary details by marking the boxes 'Yes', 'No', or 'Not applicable' next to each item on the checklist. The next sections describe important guidelines for different components of the preregistration.

1. Conceptual background and theoretical rationale

As with the preregistration process for any research study, outlining the conceptual foundation and theoretical rationale of the study is crucial. Therefore, researchers should open by articulating their central research questions (RQs), followed by the theoretical rationale and background for selecting the variables they intend to include in the network estimation. Notably, network theory is a systems-based approach inspired by the belief that relevant phenomena are emergent properties arising from the interactions among components of the network (e.g., a disorder arises from a self-perpetuating system of interacting symptoms; Borsboom et al., 2022; Ebrahimi, 2023). Accordingly, an important task for investigators is to specify which group of variables play key roles in the system. For example, a researcher investigating maintaining mechanisms of depression may provide a rationale for the specific constructs (e.g., rumination, learned helplessness) selected for the system (e.g., Ebrahimi et al., 2021).

2. Network inference

Investigators may subsequently specify the level at which they want to interpret their network. This may be at the micro-level and include hypotheses concerning central edges in the network, including the magnitude and polarity (negative/positive) of edges, or both. At the meso-level, hypotheses may concern the clustering of certain nodes to specific communities (e.g., the formation of an anxiety symptom cluster). Finally, there may be hypotheses at the macro-level concerning specific centrality measures (e.g., 'worrying as the most connected symptom') or global measures, such as network connectivity. We recommend assigning unique numbers (e.g., H1a, H1b) to specific hypotheses to facilitate subsequent reference to them. When stating their hypotheses, authors should explicitly specify (1) to which estimates the hypotheses relate (e.g., specific edges, centrality indices, or aggregate descriptive measures of the network, such as density and sparsity); (2) the network structure that the hypothesis refers to (temporal or contemporaneous or both combined, between- or within-person). Finally, it is important to distinguish between confirmatory (a-priori specified) and exploratory research questions and analyses.

Table 2. Important preregistration considerations concerning the conceptual background and theoretical rationale for the investigation.

Yes	No	Not applicable	I have specified ...
☐	☐	☐	Research questions (RQs)
☐	☐	☐	Theoretical rationale for variables investigated in the network
☐	☐	☐	Network inferences (and corresponding estimates): - Micro-level: specific edges - Meso-level: clustering - Macro-level: density, sparsity, centrality measures
☐	☐	☐	Network structures that RQs and hypotheses refer to: - temporal - contemporaneous - between-person - group level or individual estimates
☐	☐	☐	Exploratory RQs and analyses

3. Data and variable selection

After describing the rationale and conceptual background of the study, we recommend that investigators provide more details regarding the data and variable selection. If data collection has not been completed, investigators may provide more information on the (planned) data collection procedures: participant recruitment, eligibility criteria, planned sample size, and relevant procedures and materials. When working with existing secondary datasets, it is important to briefly describe the dataset and any prior work on similar research questions using the same data. Investigators may point out key similarities and differences to their current preregistration. The researchers should further describe whether they have accessed or previously examined the set of variables to be used in the study. Further, it is

important to describe all variables of interest and their measurement (e.g., sum scores or collapsing of answer categories). Multicollinearity (i.e., high correlation among nodes) can be a challenge in network estimation. This further relates to the topic of topological overlap, or whether two nodes (e.g., feeling "sad" and feeling "down") represent meaningfully different constructs. Thus, researchers should carefully select nodes beforehand with the aim of avoiding topological overlap (e.g., Fried & Cramer, 2017). In many cases, highly overlapping variables are being combined or removed based on conceptual/theoretical criteria. Common approaches focus on examining pairwise correlations and using measures, such as variance inflation factors (Shrestha, 2020). Another approach is to remove redundancies through unique variable analysis within the Exploratory Graph Analysis (EGA) framework (Golino & Epskamp, 2017) using the EGAnet R package (Golino & Christensen, 2024). Any such decisions or methods should be part of the preregistration. In some cases, the preregistered analyses may focus on only a subset of data, and thus, it may be important to describe the use of a relevant subset (e.g., training and/or test sets). When choosing a subsample of individuals based on specific criteria (such as those with scores above a certain threshold) that also appear in the network model, there is a well-documented risk of Berkson's bias (de Ron et al., 2021). Therefore, researchers should discuss potential solutions to mitigate this issue.

Table 3. Important preregistration considerations concerning data and variable selection.

Yes	No	Not applicable	I have specified ...
☐	☐	☐	Study design (recruitment, procedures, eligibility criteria, materials) or name of dataset (when using existing data)
☐	☐	☐	Prior access to data to the data by investigators
☐	☐	☐	Existing publications on relevant variables (explain similarities and differences)
☐	☐	☐	Variables and measurement
☐	☐	☐	Training and/or testing sets

4. Data preprocessing

In line with standard preregistration practices, it is crucial to detail the procedures involved in data preparation. This includes criteria for outlier removal, standardization, or other transformations. Procedures for identifying low variances and high correlations between variables should be outlined, along with any methods used to address these issues, such as removing or combining variables if necessary. Moreover, strategies for handling attrition or missing data (e.g., due to skip-structures in the item order) should be described. Frequently used techniques for addressing missing data include Full-Information Maximum Likelihood Estimation (FIML; Enders & and Bandalos, 2001), multiple imputation (Rubin, 1996), and opting for analyses based solely on complete cases.

Another important consideration is handling equidistance (i.e., evenly spaced intervals). This may concern the time intervals in-between assessment waves (in panel data) or the overnight periods (in EMA). A common approach is to treat these periods as missing data through insertion of dummy (i.e., empty) waves (Freichel et al., 2023). Others may choose to

ignore these longer time gaps entirely or apply interpolation techniques, such a cubic-spine interpolation (Fisher et al., 2019) to the time-series.

More specific to preregistrations of longitudinal network analyses is the handling of trends in the data. For instance, GIMME, mlVAR, and panel GVAR models assume stationarity (i.e., that the variables' means and variances are stable across time) and thus, the process of detrending (i.e., removing trends) is a common practice in cases where the stationarity assumption is violated (Freichel et al., 2023). Detrending refers to the process of removing the effect of time from the data. Thus, the network is estimated on the residual variance. It is important to note that such procedures may artificially inflate the model fit and impact the interpretation of the resulting estimates.

Researchers have used different procedures to detect trends. For instance, Speyer et al. (2022) used regression models to identify linear, quadratic, and cubic effects of time. Beyond linear and curvilinear trends, Ebrahimi and colleagues (2021) detrended for weekend effects on mood by regressing out on a binary (weekday versus weekend) variable. Another study using individual time-series data (McGowan et al., 2023) used Kwiatkowski-Phillips-Schmidt-Shin tests for every variable and individual to test for stationarity.

Although researchers cannot ascertain the type of trend prior to data collection and, therefore, the appropriate detrending procedure, they can specify whether they intend to inspect for trends (e.g., plotting of raw data over time) and detrend the data. In the manuscript, they need to identify the type of trend in the data (e.g., linear or quadratic trends) and the method for correcting it. For the sake of consistency, investigators sometimes detrend all variables even if only a subset exhibits trends (e.g., Ebrahimi et al., 2024; Epskamp, van Borkulo, et al., 2018), Simulation studies indicated no difference between detrending all variables and detrending only those with significant trends (Epskamp, van Borkulo, et al., 2018). However, such procedures should be conducted with caution and ideally include sensitivity analyses that account for the trend.

Table 4. Important preregistration considerations concerning data preprocessing.

Yes	No	Not applicable	I have specified ...
☐	☐	☐	Outlier definition and handling
☐	☐	☐	Standardization
☐	☐	☐	Missing data handling - complete case analysis - multiple imputation - ML / FIML
☐	☐	☐	Handling time interval/equidistance
☐	☐	☐	Inspection of trends in data (checks for stationarity assumption) and procedures to remove trends

Note. ML = Maximum Likelihood Estimation, FIML = Full-Information Maximum Likelihood Estimation

5. Statistical modeling

Several important aspects of data analysis should be addressed here: (1) type of statistical modeling approach; (2) software tools; (3) method for integrating variables in the network; (4) model fit criteria; (5) comparisons between different networks; (6) model stability; (7) sensitivity analyses, and (8) alternative strategies in case of bad model fit or non-convergence.

(1) The type of statistical modeling should be elaborated (in connection with specific RQs and variables), (2) including the respective software (e.g., R package) used to estimate the model. Given the rapid developments in the fields, investigators may consider specifying the R package version they intend to use and subsequently document any deviations transparently during the data collection or analysis. (3) investigators should clarify how variables will be integrated within the model – whether each one will be treated as a single observed variable or combined into composite variables (e.g., sum, component, or latent variable scores; see Epskamp, 2020). (4) Investigators may specify relevant model fit criteria (if applicable) they intend to use to evaluate the adequacy of the model (e.g., CFI > 0.9). Articles outlining the appropriateness and cut-offs for different types of fit indices in network models can be cited (e.g., Du et al., 2024). Traditional indices from structural equation modeling (e.g., RMSEA, TLI, CFI) perform well for confirmatory panel (e.g., panel-GVAR) network models (Du et al., 2025); (5) Comparisons of different networks, if applicable, should be described in terms of the methodological approach, whether it involves visual inspection of network structures, imposing equality constraints, or formal comparison tests. The Network Comparison Test (NCT) is a commonly used permutation-based test to evaluate differences between networks with respect to the network structure, edge strength and global strength (van Borkulo et al., 2022). Relevant parameters to preregister include the number of iterations/permutations and the type of multiple comparison correction (e.g., Bonferroni correction) that will be applied, if any. Another method for comparing idiographic network structures is the Individual Network Invariance Test (INIT, Hoekstra et al., 2024) that requires investigators to specify a relevant alpha level (e.g., 0.05) for pruning edges from the network. Lastly, Haslbeck et al. (2023) developed parametric and non-parametric comparison tests for testing group differences in multilevel VAR models. If of interest, the data analysis section should outline the method for comparing groups, and the specific parameters that will be used in the respective tests. (6) To evaluate model accuracy (i.e., confidence intervals around edge weights) and stability (i.e., sampling variation), investigators may choose to describe bootstrapping methods (including the number of bootstraps) and the metrics (e.g., edge retention in > 50% of bootstraps). (7) Lastly, it may be necessary to describe robustness/ sensitivity analyses, such as repeating the analysis in different conditions or groups (e.g., replication across different comorbidities). The preregistration may detail the scope and nature of such sensitivity analyses, providing a plan for assessing the robustness of findings. For instance, researchers may showcase the results from the models estimated on the detrended data and the data without detrending. (8) Longitudinal network models may show poor model fit, non-convergence or potential estimation problems with (negative) variances. The preregistration may mitigate these challenges by specifying necessary criteria for model fit, such as RMSEA, CFI, and TLI thresholds, and outlining steps to be taken if the model does not meet these criteria. This

may include re-specifying the model by selecting different assessment waves, variables, or simplifications, such as using sum/mean scores of variables instead of specifying latent constructs. The preregistration may preemptively describe such steps to reduce model complexity and facilitate model identifiability. The results section of the manuscript should ultimately describe such estimation problems, including for example the number of non-converging idiographic models (in the case of GIMME).

Table 5. Important preregistration considerations concerning statistical-computational modeling.

Yes	No	Not applicable	I have specified ...
☐	☐	☐	Specific model, e.g.: - Panel GVAR - CLPN - mlVAR - GIMME
☐	☐	☐	Variables modelled as - Observed variables (e.g., single items, sum-scores) - Latent variables
☐	☐	☐	Software
☐	☐	☐	Criteria for model fit, e.g.: - RMSEA - CFI, TLI - Chi square
☐	☐	☐	Type of group comparison - Visual inspection - Network Comparison Test (NCT) - (Individual) Network Invariance Test (INIT) - Parametric and nonparametric comparison tests - Model (equality) constraints
☐	☐	☐	Model stability inspection procedures/ bootstrapping
			Robustness/ sensitivity analyses

6. Model-specific preregistration guidelines

Thus far, we have described general guidelines that apply to different analytical approaches. There are, however, model-specific parameters and criteria potentially important to specify (see Box 1 for important model-specific guidelines).

Box 1. Model-specific preregistration guidelines.

Panel GVAR

Model selection. There are multiple model selection algorithms that can be used to obtain an interpretable, sparse network structure. For a complete description see the chapter by Blanken et al. (2022).

Thresholding. To hide non-significant edges (based on p-values) from the network structure, thresholding is commonly used. Non-significant edges are then hidden from the network visualization.

Pruning. In pruning, non-significant edges are being removed and the model is re-estimated with these parameters fixed to zero. The contemporaneous and between-person networks have two p-values associated with each edge (for the regressions A predicting B as well as B predicting A), and thus, researchers may decide to threshold/prune based on both p-values ('AND-rule': only edges with two significant p-values retained) or just one p-value ('OR-rule': edges with at least one significant p-value retained) (Epskamp, 2017). We recommend researcher specify the corresponding alpha level (e.g., alpha = 0.05) and the type of rule (AND-; OR-rule) used for thresholding/pruning.

Model search algorithms. In addition to thresholding and pruning, there are various other model algorithms that can be used to identify the best-fitting model using indices, such as BIC. It is beyond the scope of this article to describe all model search algorithms and a complete description of relevant procedures can be found elsewhere (Blanken et al., 2022). The two most used model search algorithms include stepup estimation and modelsearch.

Stepup estimation. This function continuously adds edges (based on the strongest modification index) to optimize some criterion (e.g., BIC).

Model search algorithm. This process searches the entire model space through stepwise adding and removing edges to identify an optimal model.

It is important to describe which model selection procedures (e.g., a combination of pruning and step-up estimation) will be used to obtain the final network structure.

2. **Type of within- and between-person latent model.** By default, the within-person latent contemporaneous and between-person latent models are modelled as Gaussian Graphical Models (GGMs). In certain situations (e.g., when obtaining unreasonable between-person estimates), it may be useful to use other estimation methods, such as Cholesky decomposition (Freichel & Epskamp, 2024). The researcher could specify which primary model they aim to use, while specifying which model/algorithm/procedure would be used upon obtainment of unrealistic estimates or bugs (e.g., Cholesky decomposition; Freichel and Epskamp, 2024).

3. **Confirmatory model structure or group comparison.** For confirmatory network modeling, investigators may describe what parameters (i.e., edges) are constrained to zero or to be freely estimated (e.g., in the omega_zeta_within object in psychonetrics). Similarly, for group comparison using equality constraints, it may be important to describe for which parameters these constraints apply to.

CLPN	**Method for lambda selection.** The default procedure for selecting the tuning parameter lambda (for determining degree of penalization) is cross-validation (with a certain number of folds). However, it is also possible to use model selection with the Extended Bayesian Information Criterion (EBIC) with an arbitrarily set hyperparameter γ for selecting lambda. Considering these methods differ with respect to how conservative/liberal they are (Haslbeck, 2022), we advise researchers to name the method for selecting the lambda parameter **Covariates.** CLPN allows investigators to include covariates in the model estimation. These are variables used for predicting the subsequent cross-lagged values, however, they are not themselves being predicted by any other variable. **Additional pruning steps.** CLPN may also include an additional pruning step in which the zero-paths obtained from the regularized model are fixed to zero and the model is being re-estimated. This allows researchers to test for cross-time constraints. We recommend preregistering such additional approaches, and the series of nested models that will be tested.
mlVAR	**Contemporaneous and temporal model estimation.** Contemporaneous and temporal estimates can be modelled as correlated (correlated random effects), orthogonal (uncorrelated random effects), fixed (all residuals for all subjects, or unique (per subject). Orthogonal estimation has been recommended for networks that include more than 6 nodes (Epskamp, Waldorp, et al., 2018).
GIMME	**Type of exogenous variables.** GIMME allows investigators to adjust for other exogeneous variables. These variables may predict the outcome variables but cannot be directly predicted by them. We recommend reporting any exogeneous variables and the rationale for including them. **Cut-offs for group- and subgroup-level paths.** The proportion (e.g., 75%) of edge presence in individual models to be included in (sub-) group models. **Contemporaneous model estimation.** Contemporaneous estimates can be modeled as correlated (VAR modeling), directed (structural VAR), or both (hybrid-VAR). **Subgroup-specific edge identification.** Options exist for identifying edges common in either a priori (confirmatory) subgroups or subgroups derived from unsupervised classification. **Latent variable modeling.** If latent variables will be included, researchers should specify how they will be estimated: model implied instrumental variables (MIIV), pseudo-maximum likelihood, or singular value decomposition. MIIV estimation has been recommended (Gates et al., 2020).

An empirical example of preregistering longitudinal network analysis studies

Freichel et al. (2023) preregistered a longitudinal network analysis that aimed to examine the dynamic relationship between PTSD symptoms and specific theory-derived mechanisms. The preregistration and code can be accessed online on the Open Science Framework (https://tinyurl.com/bd5b2byd). This preregistration was based on an existing secondary dataset and served the primary purpose of specifying the rationale, variable selection, and data analysis plan. The authors describe the rationale for the study and general predictions that concern specific edges of interest (e.g., "we expect threat monitoring to be closely associated with the avoidance PTSD cluster."). Some predictions concern the connectivity of the overall network structures ("expect greater number ... and stronger edges in the contemporaneous than the temporal network"). The preregistration includes a list of all variables/nodes and their respective specification (observed/latent). The authors describe that some variables "may be modeled as simpler sum-scores to foster model identification if necessary". The data analysis plan includes the specific model, criteria for good model fit, relevant software, and

thresholding procedures. At the bottom of the preregistration, there is a disclaimer describing potential deviations from the preregistered data analysis plan:

"If the model as specified in this pre-registration does not fit, for example, due to multicollinearity issues or variance problems (e.g., problems common in structural equational models), standard steps such as removal of the measures or items causing negative variances (i.e., implausible artifact) will be conducted. Any such procedure will be elaborated in the manuscript." - Freichel et al., 2023

Several important aspects are missing in this preregistration, including references to any prior publications using similar variables in the data as well as methodological details, such as procedures to test the stability/robustness of the results and the type of within- and between-person latent model used. Examples of preregistration for other dynamic network models exist in the literature (e.g., mlVAR: Ebrahimi et al., 2021; cross-lagged network analysis: Hamlett et al., 2024; GIMME: van der Tuin et al., 2021).

Concluding comments

In this article, we introduced important guidelines that researchers should consider when preregistering longitudinal network analysis studies. The comprehensive checklist, appearing in the supplementary materials, is designed to assist both authors and reviewers in ensuring the transparency and reproducibility of research findings in this evolving field. We encourage integration of this checklist into OSF and AsPredicted templates, enabling journals and reviewers to adopt it as a field-wide standard for preregistered longitudinal network studies. With this practice of specifying hypotheses, methods, and analytical strategies in advance, researchers may mitigate biases and enhance the credibility of their findings. As the field continues to evolve, these guidelines may be adapted and refined to accommodate new developments and insights.

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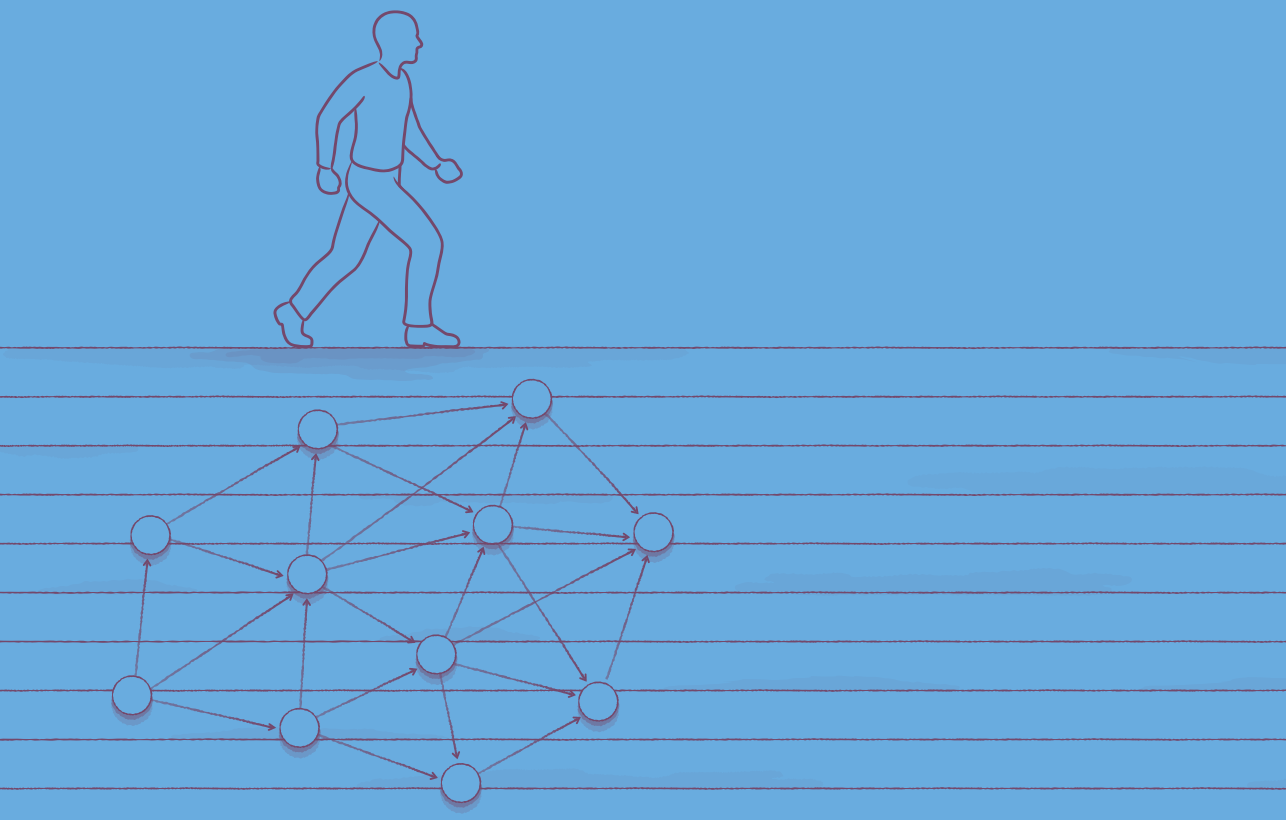


CHAPTER 11

General Discussion

Adolescence is a developmental stage at which neurocognitive development intersects with the emergence of mental health problems. This makes it an ideal window of opportunity to study the associations between cognitive control and psychopathological symptoms. This thesis adopted a developmental psychopathology framework and applied novel network analytical approaches (chapters 2-5) to examine the role of cognitive control in symptom development across time scales (ranging from hours to years), assessment modalities (self-report and behavioral tasks), and methodological lenses (from distal vulnerability factor to mediating and moderating mechanisms). The thesis also employed novel experimental paradigms to study cognitive and attentional control. Chapter 6 examined cognitive control within everyday life through the use of EMA. Chapter 7 presented a novel behavioral measure of attentional control that extended previous measures by incorporating both reward and punishment cues. The final part 3 of the thesis addressed broader methodological challenges in studying developmental psychopathology from a network analytical perspective, arguing for (1) the expansion of networks to include neural markers (chapters 8), (2) the importance of multiverse analysis and model choice (chapter 9), and (3) the relevance of preregistration and reporting guidelines (chapter 10).

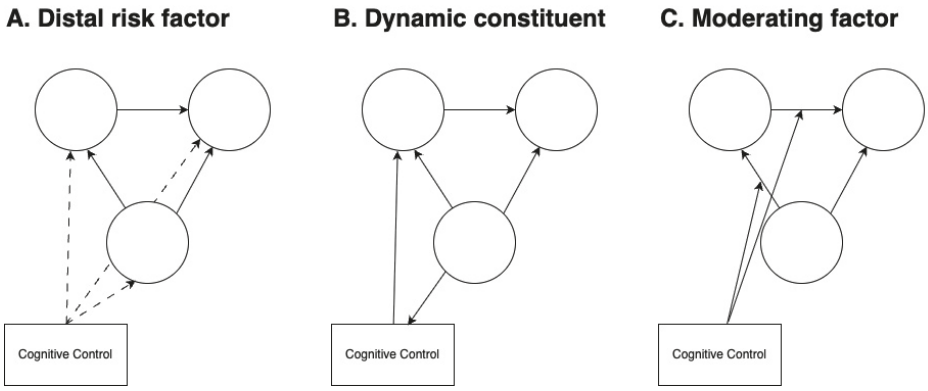
Theoretical implications

Poor cognitive control is more than just a risk factor.
A central theme of this thesis is that executive functioning was associated with broad-band symptom domains across developmental stages of adolescence. For instance, Chapter 2 showed that sustained attention deficits at age 11 were prospectively associated with internalizing and externalizing symptoms at age 13. Similarly, as shown in Chapter 3, impulsivity was concurrently related to alcohol-related problems. Better inhibitory control performance at age 10 was associated with fewer externalizing symptoms at age 13/14 (see Chapter 4). This line of work fits prior empirical evidence for the role of poor cognitive control as a risk factor (C-factor, Abramovitch et al., 2021). This reinforces the importance of cognitive control in contemporary frameworks for studying mental health, including the Research Domain Criteria (RDoC, Insel et al., 2010). However, our findings caution against the view that cognitive control deficits represent a static trait-like vulnerability. The longitudinal network analysis presented in Chapter 4 clearly showed that during early adolescence, poorer inhibitory control was a risk factor for externalizing problems that, in turn, also predicted lower working memory capacity. In middle adolescence, externalizing symptoms predicted and were predicted by poorer inhibitory control. These findings contribute to the long-standing debate in the literature that distinguishes vulnerability models of cognitive dysfunction (the 'cause' perspective) and 'scar' (the 'consequence' perspective) theories (Maasalo et al., 2021; Zainal & Newman, 2022). Our data suggests that both theoretical accounts may be true depending on the developmental stage and context. Importantly, the methodological approach used to examine associations between cognitive control and symptoms largely determines the results and the theoretical implications that follow (see Figure 1). For instance, in our analysis of the TRAILS data (see Chapter 2), we modelled cognitive control functions

as predictors and symptoms as outcomes. Similarly, in chapter 5, cognitive control at age 14 was assumed to be a distal moderator of the symptom interplay. These directional specifications implied an underlying assumption that cognitive control impairments may lead to psychopathological symptoms, rather than the reverse pathway. As showcased in several other chapters, functions of cognitive control and features of mental disorders show bidirectional associations. Thus, future work should formalize dynamic cascade models of cognitive dysfunction that incorporate feedback loops and predict outcomes for specific cognitive control functions and developmental stages.

Figure 1

Different Ways to Conceptualize and Model Cognitive Control



Note. The figure illustrates the different modeling approaches. Low cognitive control may represent a distal risk factor (see Chapter 2), a dynamic constituent (i.e., predictor and outcome, see Chapter 4), and a moderating factor (see Chapter 5).

Towards an Extended Network Theory Incorporating Cognitive Control

In line with the network theory of mental disorders (Borsboom & Cramer, 2013) and the network perspective on comorbidity (Cramer et al., 2010), our longitudinal analyses indicated a web of direct predictive associations between symptoms of different disorders. By incorporating cognitive control functions in these networks (see chapters 6-7), we found that these act as important mediating mechanisms. For instance, internalizing symptoms predicted poorer performance on a logit digit span (i.e., lower working memory). Better working memory, in turn, was associated with fewer externalizing symptoms. Given this important mediating role of cognitive control functions, temporal symptom network analyses should consider cognitive control as an active component when inferring underlying mechanisms. As shown in Chapter 8, specific neural correlates, such as the insula's cortical thickness, showed negative associations with cognitive dysfunction as a key symptom of depression. A network model that included an overall depressive symptom severity score did not show any cross-modal associations. Altogether, these findings underscore the importance of extended network models that include cognitive and biological factors that are constituents of the larger network

system (Freichel, Lenartowicz, et al., 2024; Isvoranu et al., 2021; Piazza et al., 2024; Vera et al., 2024). Extended network models may better characterize underlying mechanisms and help uncover sources of heterogeneity of mental health conditions.

Towards rigorous empirical applications of longitudinal network models

Longitudinal network models are increasingly being used in clinical and developmental science. As described in Chapter 9, different modeling approaches differ significantly in their i) underlying assumptions, ii) estimation process, and iii) interpretability. Researchers should carefully select the respective analytical approach and opt for multiverse analyses if feasible. In line with cross-sectional network estimation (Burger et al., 2023), following preregistration and reporting guidelines is essential for promoting transparency. The preregistration checklist introduced in Chapter 10 may offer practical guidance to authors, reviewers, and editors.

Clinical implications

In 1967, the American clinical psychologist Gordon Paul posed an iconic question to the field: "What treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances?"(Paul, 1967, p. 111). While the findings presented in this thesis cannot answer this question directly, they offer some hints with respect to treatment targets, mechanisms of change, and opportunities for personalizing interventions. Each chapter describes specific clinical implications, but several key themes emerge across the chapters:

First, this thesis provided support for early intervention targets. For instance, Chapter 4 showed that depressive symptoms in early adolescence predicted a range of other internalizing symptoms (i.e., panic, somatic problems, separation anxiety, general anxiety, social phobia) later on (Freichel, Pfirrmann, et al., 2024). These results fit with developmental models and clinical observations that social withdrawal and reduced activity (typical features of adolescent depression) may lead to a higher sensitivity towards anxiety-related experiences over time. Thus, targeting depressive symptoms in early adolescence (at age 11), for instance, through cognitive-behavioral therapy (CBT) approaches (Beck, 1979), such as behavioral activation and interpersonal skills training, may disrupt this developmental progression from depression to anxiety. Existing community-based prevention programs, such as Coping Cat (van Starrenburg et al., 2017) may offer relevant frameworks for addressing depressive symptoms in early adolescence.

Second, Chapter 3 tested a developmental theory according to which distal risk factors of alcohol use, such as stressful life events or personality traits, converge on proximal and specific drinking motives. Our data showed that social drinking motives and prior alcohol use represented the strongest risk factors of alcohol-related problems during adolescence (Freichel, Pfirrmann, et al., 2023). Within a given moment, impulsivity and coping with depression motives were related to alcohol-related problems. These findings fit with motivational models of alcohol use among adolescents (Cooper, 1994) and highlight the relevance of cognitive-motivational factors in forecasting problematic alcohol use. While these results were based on within-person models applied to a large panel dataset spanning many years, such information derived from longitudinal EMA assessments may

provide additional insights relevant for personalizing psychotherapy. In clinical contexts, case conceptualizations typically rely on retrospective self-reports that may be biased. Incorporating idiographic network information based on EMA assessments that include drinking motive items may help identify proximal triggers or motives that patients may not be fully aware of. Existing frameworks, such as the Prior Elicitation Module for Idiographic System Estimation (PREMISE, Burger et al., 2022) provide methods to integrate these idiographic data-driven networks into an individual's problem, context, history, and perceived relations.

Third, the results from the EMA study discussed in Chapter 6 showed that performance-based EF measures were not directly related to affect dysregulation. In contrast, subjectively perceived difficulties with executive functions were closely associated with positive and negative affect in daily life. These results highlight the relevance of perceived cognitive problems, some of which may represent core symptoms of disorders (e.g., cognitive dysfunction in depression), while others may represent relevant maintaining mechanisms (e.g., attentional control). Given these links with everyday positive and negative affect, perceived cognitive problems, in particular those that are modifiable, should be assessed as part of routine outcome monitoring, alongside traditional symptom assessment. Importantly, some of the perceived cognitive problems may directly map onto modules from established interventions. For instance, difficulty disengaging from thoughts, a common cognitive problem that co-occurred with negative affect within the same time window (see Chapter 6), may be effectively targeted through techniques, such as cognitive defusion (from Acceptance and Commitment Therapy, Hayes et al., 2011) or detached mindfulness (from Metacognitive Therapy, Wells, 2005).

As a clinical psychologist (in training) working in a hospital setting, I remain hopeful about the promise of network modeling. However, several open questions remain before (idiographic) network models can be integrated into clinical decision-making. First, it is necessary to empirically test the common intuition that idiographic networks indeed improve clinical case conceptualizations. Second, further research is needed to better understand the extent to which incorporating cognitive control impairments into idiographic symptom networks can dissect the phenotypic heterogeneity observed in clinical practice. Do two patients with similar symptoms exhibit different networks when self-reported cognitive control problems are considered in their idiographic networks? Third, randomized controlled trials are needed to determine whether network-informed psychotherapy (e.g., personalization based on specific edges or node centrality) is better than existing manualized psychotherapy.

Strengths, limitations, and future directions

A key strength of this thesis is the use of a multi-method approach for studying the association between cognitive control and symptoms, ranging from experimental studies to longitudinal panel surveys and ecological momentary assessment. This allowed us to examine the role of cognitive control at a micro- (hours) and macro-level (years). In addition to the variety of time scales, the thesis leveraged varied sampling: adolescent cohorts (e.g., TRAILS and IMAGEN study), student samples, and individuals at high risk for psychopathology (e.g., BHRCS). Several chapters used cutting-edge network approaches, including cross-lagged

panel network analysis and panel graphical vector autoregressive models. To the best of our knowledge, Chapter 2 is the first study to apply both approaches in parallel to study adolescent psychopathology. Chapter 5 introduced a moderated CLPN approach – a novel extension that may have various applications in the field of developmental psychopathology. Chapter 7 introduced a novel behavioral task paradigm to assess attentional control, specifically value-modulated attention capture, and examine its association with psychopathology. Consistent with prior work on VMAC tasks, we found stronger attentional capture for stimuli associated with high rewards as opposed to low rewards (Freichel, Mrkonja, et al., 2023). A unique contribution of this study is that we demonstrated these effects using a much shorter and more scalable task that incorporated alternating punishment blocks. Against our expectations, we found (i) no VMAC effects in the punishment context, and (ii) no direct associations between VMAC and symptom measures.

Beyond the chapter-specific limitations, several broader limitations should be noted. First, while longitudinal (network) models identify temporal ordering and may fulfil the criteria of (Granger-causal) temporal precedence, they are vulnerable to the influence of node selection bias and unmeasured confounders. Thus, while these models allow us to identify patterns of covariance, they do not permit any strong causal claims. Similarly, well-established conventions for effect sizes are not easily transferable to partial associations derived from network models. More methodological work is needed to develop criteria to judge the magnitude of specific edge weights depending on the number and type of nodes included in the model. Second, the thesis did not model the influence of important contextual factors, such as genetic liability, parenting, or sociodemographic influences. Third, the samples were primarily restricted to European, North American, and South American contexts, which may limit the generalizability of the findings. Lastly, this thesis focused on the associations between broadband symptom domains (e.g., internalizing and externalizing) and cognitive control impairments. These aggregate dimensions may have obscured associations that are present only at the symptom level. This interpretation aligns with the results presented in Chapter 8, which only found associations between neural markers and depressive symptoms, rather than with the depression sum score.

Taken together, this thesis advances our understanding of the dynamic interplay between cognitive control impairments and psychopathological symptoms across adolescence. This line of work highlights the role of cognitive control as a contributor, consequence, and moderator of symptom development. As the field moves towards the widespread use of longitudinal network analysis, this thesis provides a foundation with relevant empirical applications and methodological innovations, including moderated network models, granular brain-symptom network models, multiverse network and SEM analysis, and guidelines for preregistration.

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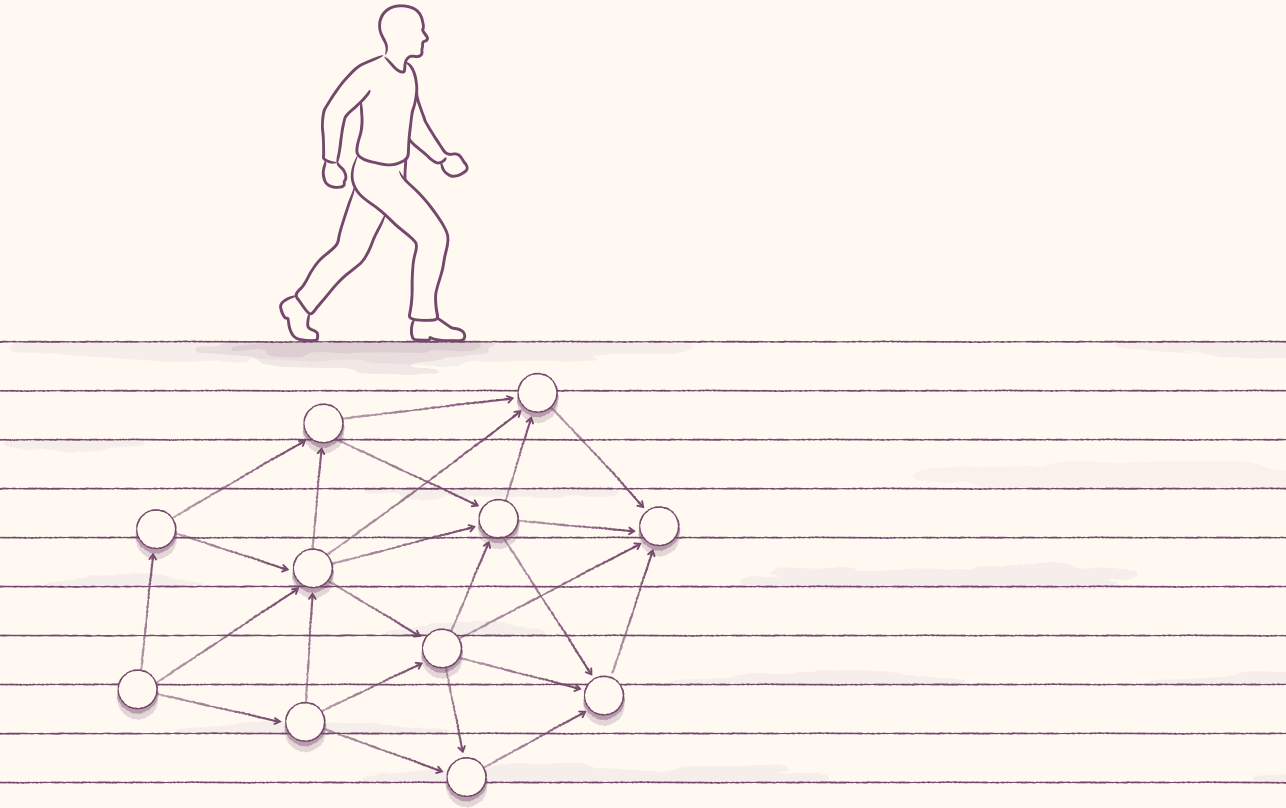


Appendices

Supplement to Chapter 2. Executive Functioning, Internalizing and Externalizing Symptoms – Understanding Developmental Dynamics Through Panel Network Approaches

Section 1: Trends Across Time

Supplement 1: To examine how symptoms change across time, we used linear mixed effects models. For every symptom measure, we estimated a separate model that accounted for the repeated measurements within individuals. Table S1 reports the main effects of time compared with the measurement during the first wave.

Table S1

Estimates from mixed effects models to test change across waves

Variable	Int (Est)	Wave 2 (Est, SE)	Wave 3 (Est, SE)
Obsessive-compulsive disorder (RCADS)	0.596	-0.256 (0.010)***	-0.308 (0.011)***
Panic disorder (RCADS)	0.426	-0.126 (0.008)***	-0.149 (0.009)***
Separation anxiety (RCADS)	0.374	-0.139 (0.007)***	-0.159 (0.008)***
Social phobia (RCADS)	0.777	-0.094 (0.010)***	-0.063 (0.011)***
General anxiety disorder (RCADS)	0.587	0.083 (0.008)***	0.093 (0.009)***
Depressive Problems (YSR)	0.291	-0.018 (0.006)*	-0.001 (0.006)
Attention deficit hyperactivity (YSR)	0.59	0.083 (0.008)***	0.093 (0.009)***
Conduct Problems (YSR)	0.235	-0.006 (0.005)	0.004 (0.005)
Oppositional-defiant Problems (YSR)	0.445	0.013 (0.009)***	0.016 (0.009)***
Somatic Problems (YSR)	0.457	-0.142 (0.008)***	-0.200 (0.008)***
Externalizing scale score (YSR)	0.271	0.016 (0.005)**	0.045 (0.005)***
Internalizing scale score (YSR)	0.363	-0.036 (0.005)***	-0.054 (0.006)***

Note. *** indicates $p < 0.001$, ** indicates $p < 0.01$, * indicates $p < 0.05$. The standard errors (SE) are given in parentheses next to the estimates (EST) for wave 2 (TF2) and wave 3 (TF3). INT = intercept. The model examines change in symptoms relative to wave 1. YSR = Youth Self-Report; RCADS = Revised Child Anxiety and Depression Scale.

Section 2: Missingness analysis

Table S2

Percentage of Missing Values for Each Measure At All Waves

Measure	Wave 1	Wave 2	Wave 3
Fluctuations RT (Sustaining)	0.81	NA	NA
Fluctuations error (Sustaining)	0.63	NA	NA
Cognitive flexibility RT (Shifting)	1.03	NA	NA
Response inhibition RT (Shifting)	0.85	NA	NA
RT working memory	0.76	NA	NA

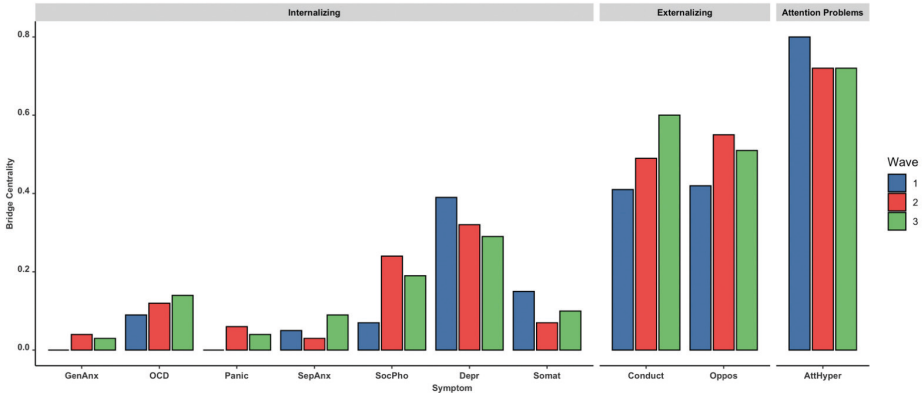
Table S2
Continued

Measure	Wave 1	Wave 2	Wave 3
Cognitive flexibility error (Shifting)	1.7	NA	NA
Response inhibition errror (Shifting)	0.9	NA	NA
Accuracy working memory	0.67	NA	NA
Internalizing scale score (YSR)	2.65	6.95	26.38
Externalizing scale score (YSR)	1.88	6.19	25.53
Depressive Problems (YSR)	1.75	6.19	25.53
Somatic Problems (YSR)	2.65	7.00	26.33
Attention deficit Hyperactivity (YSR)	1.62	6.19	25.66
Oppositional Defiant Problems (YSR)	2.11	6.33	25.75
Conduct Problems (YSR)	1.79	6.19	25.53
General Anxiety (RCADS)	0.99	6.55	25.57
Social Phobia (RCADS)	0.94	6.55	25.66
Separation Anxiety (RCADS)	0.94	6.55	25.66
Panic Disorder (RCADS)	0.99	6.55	25.57
Obsessive-compulsive Disorder (RCADS)	1.08	6.68	25.66

Note. RT = Reaction Time. The cognitive measures have only been assessed during the first wave (NA refers to non-applicable). YSR = Youth Self-Report; RCADS = Revised Child Anxiety and Depression Scale.

Section 3: Cross-Lagged Panel Network Analysis

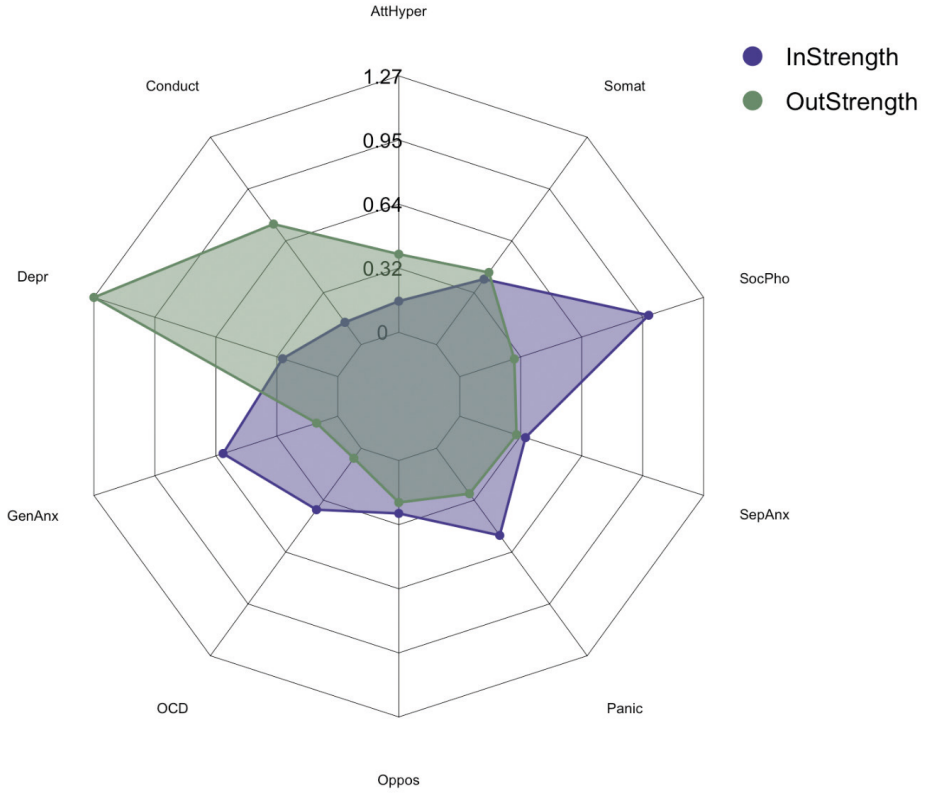
Figure S1
Bridge Centrality Measure for Contemporaneous Networks



Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems.

Figure S2

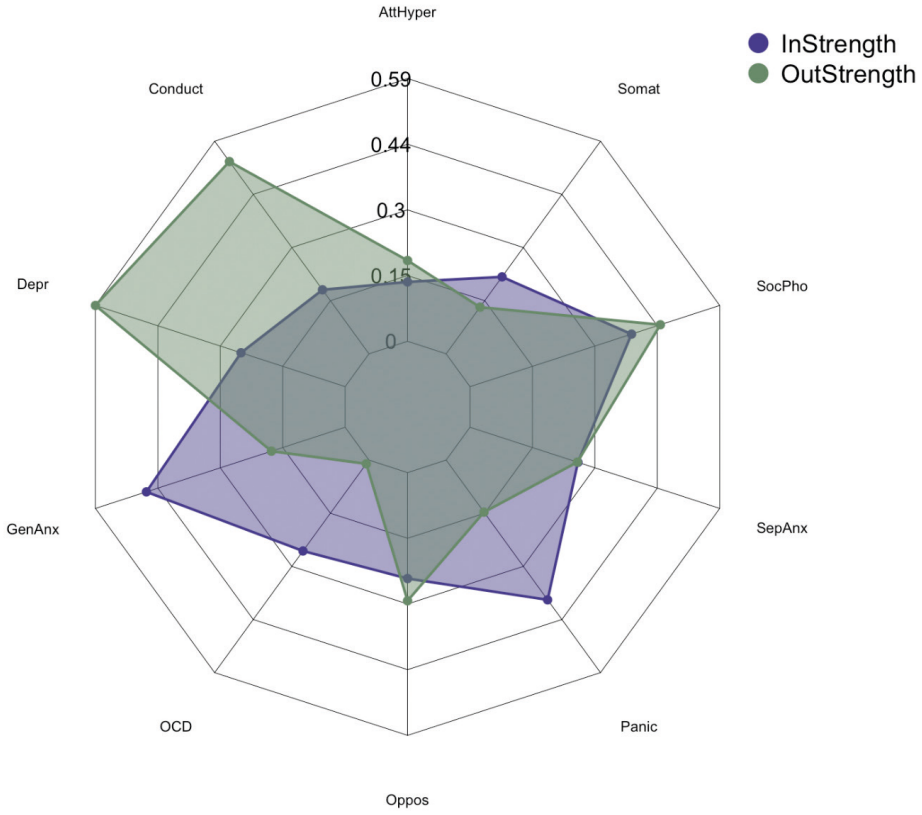
In- and Out-strength Centrality for Temporal Network From Wave 1 to Wave 2 (CLPN Model)



Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, CLPN = Cross-Lagged Panel Network Analysis.

Figure S3

In- and Out-strength Centrality for Temporal Network From Wave 2 to Wave 3 (CLPN Model)

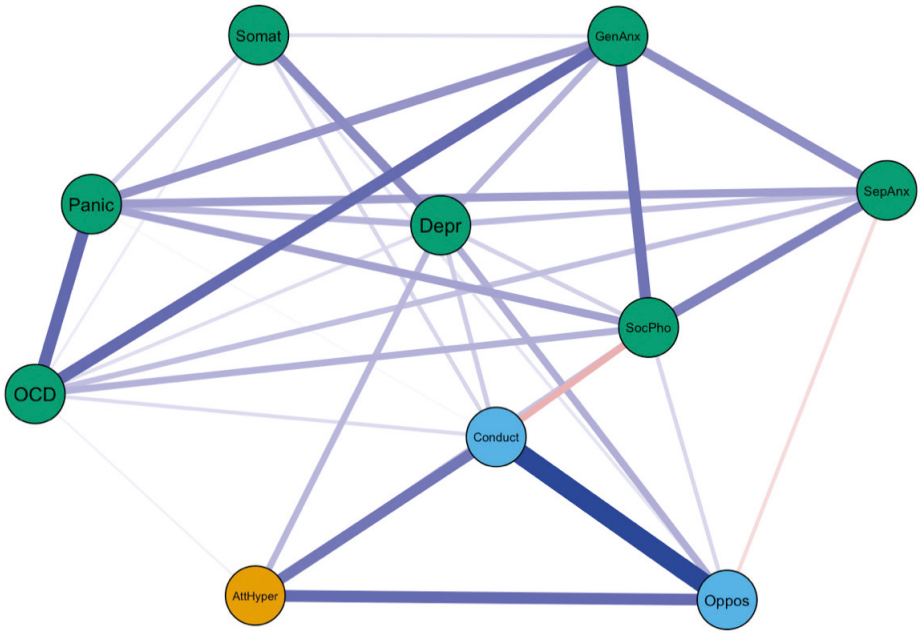


Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, CLPN = Cross-Lagged Panel Network Analysis.

Section 4: Panel GVAR Network Analysis

Figure S4

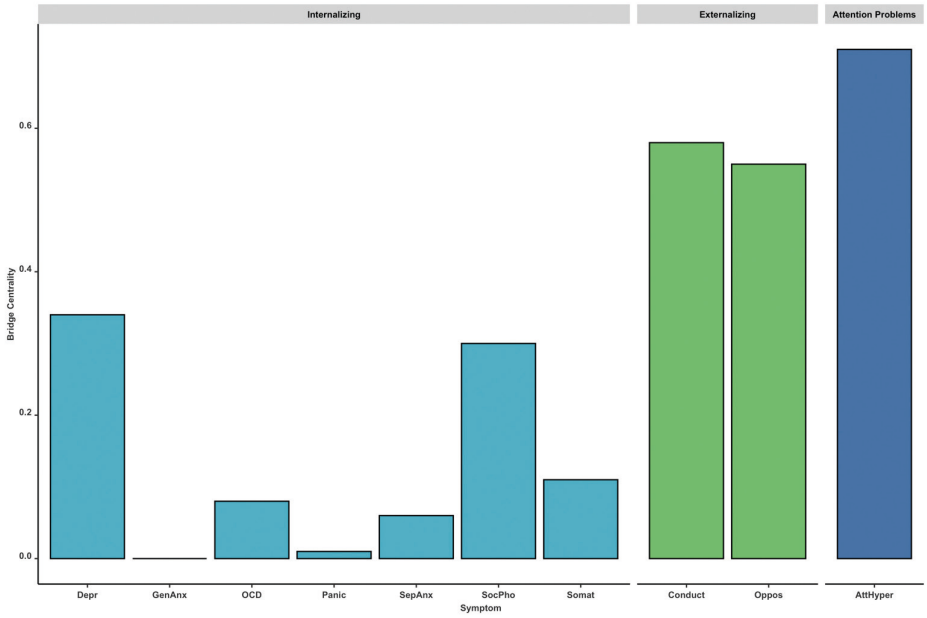
Pruned Contemporaneous Network (Panel GVAR Model)



Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, Panel GVAR = panel Graphical Vector-Autoregression Model.

Figure S5

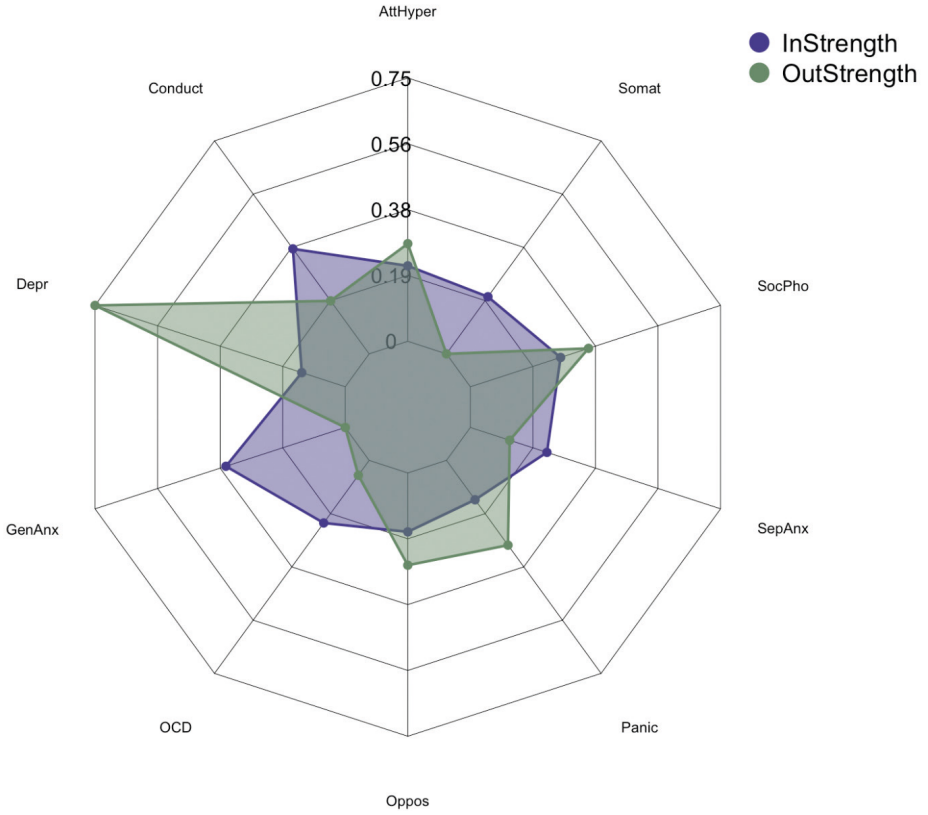
Bridge Centrality for Pruned Contemporaneous Network (Panel GVAR Model)



Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, Panel GVAR = panel Graphical Vector-Autoregression Model.

Figure S6

In- and Out-Strength Centrality for Pruned Temporal Network (Panel GVAR Model)



Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, Panel GVAR = panel Graphical Vector-Autoregression Model.

Section 5: Sensitivity Analysis (Including YSR Anxiety Subscale)

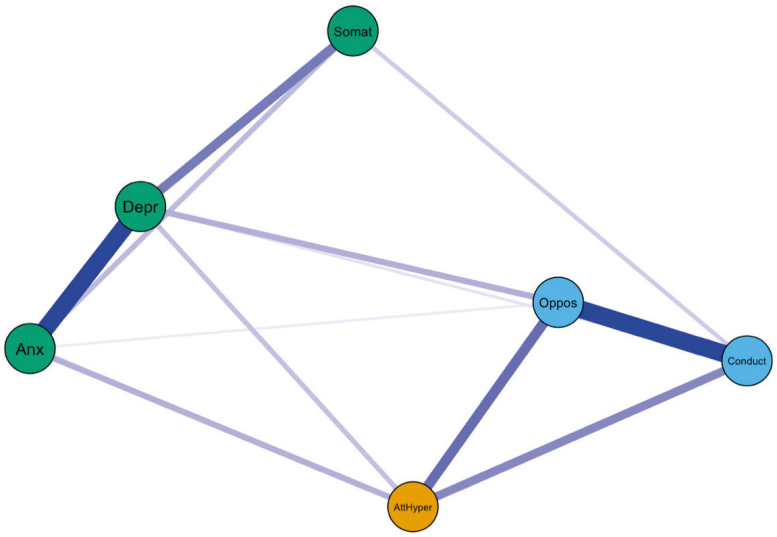
Supplement 2: The models presented in the main text contain the five separate RCADS anxiety subscales. While this approach allows us to parse heterogeneity underlying different anxiety disorders, there may be shared method variance that could explain the greater within-measure associations found. To address this concern, we repeated the same set of analyses using the YSR anxiety subscale instead of the five separate RCADS anxiety subscales. The model and analysis specifications are the same as the ones presented in the manuscript:

Panel GVAR model.

The panel GVAR model showed excellent fit to the data (RMSEA = 0.042, CFI = 0.96, TLI = 0.96). The temporal and contemporaneous networks are shown in Figures S7 and S8 respectively.

Figure S7

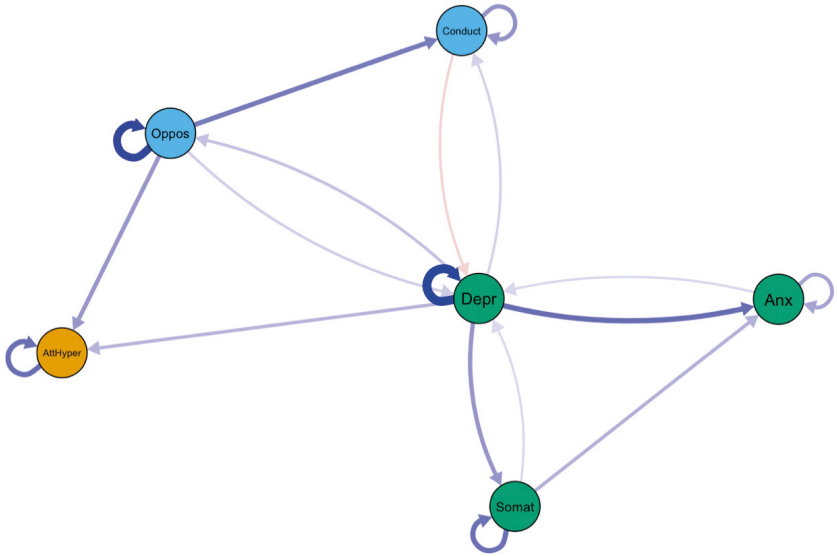
Pruned Contemporaneous Network (Panel GVAR Model) With YSR Anxiety Subscale



Pruned Contemporaneous Network (Panel GVAR Model) With YSR Anxiety SubscaleNote. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, Panel GVAR = panel Graphical Vector-Autoregression Model.

Figure S8

Pruned Temporal Network (Panel GVAR Model) With YSR Anxiety Subscale

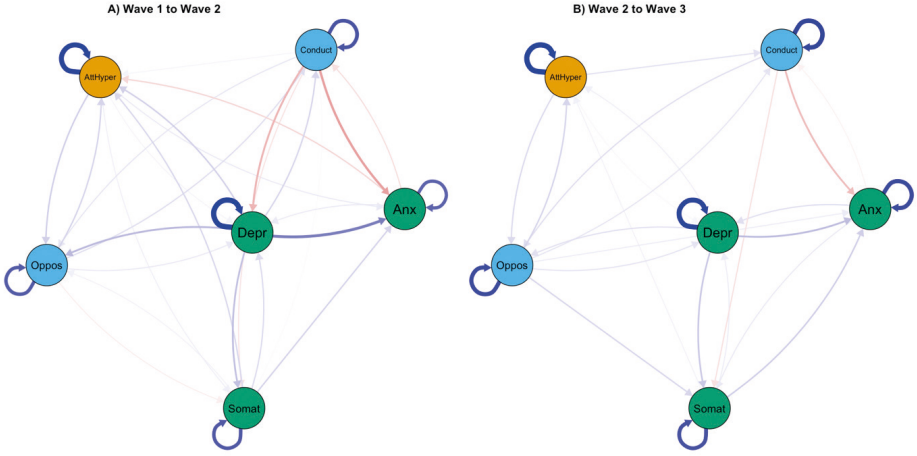


Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, Panel GVAR = panel Graphical Vector-Autoregression Model.

Cross-lagged network analysis.

Figure S9

Temporal Networks (CLPN Model) With YSR Anxiety Subscale

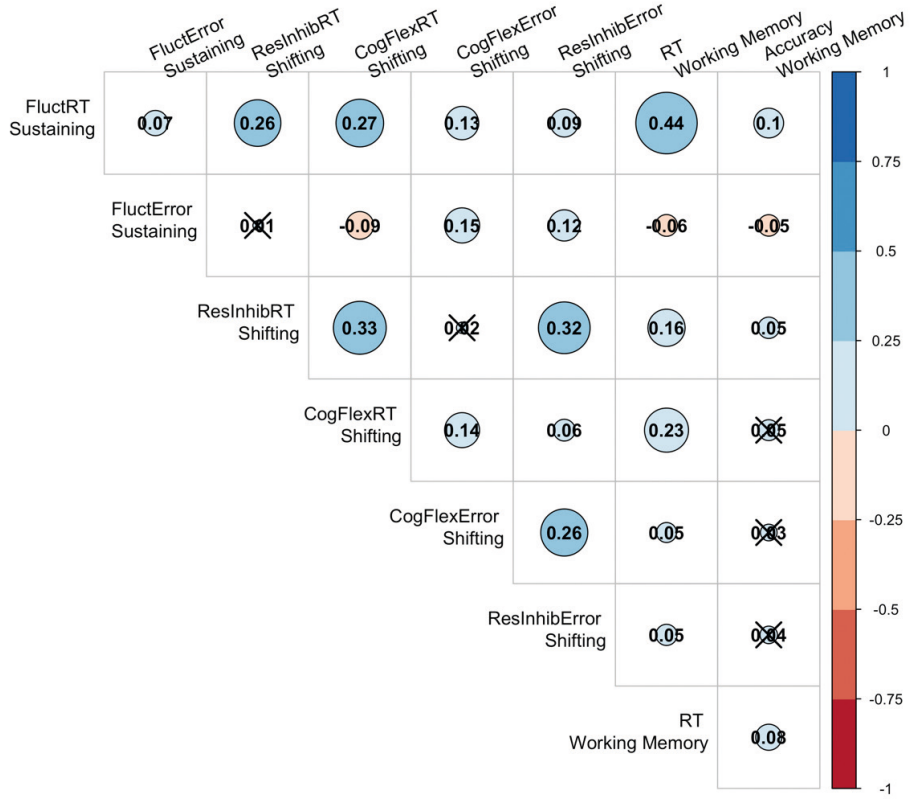


Note. GenAnx = General Anxiety, OCD = Obsessive-compulsive Disorder, Panic = Panic Disorder, SepAnx = Separation Anxiety, SocPho = Social Phobia, AttHyper = Attention Deficit Hyperactivity Problems, Depr = Depressive Problems, Conduct = Conduct Problems, Oppos = Oppositional Defiant Problems, Somat = Somatic Problems, CLPN = Cross-Lagged Panel Network Analysis.

Section 6: Executive functioning task regression models

Figure S10

Correlations between all EF measures



Note. Non-significant correlations (p > 0.05) are crossed out. RT = Reaction Times, ResInhib = Response inhibition, CogFlex = Cognitive flexibility, Fluct = Fluctuation in tempo, EF = Executive Functioning.

Table S3

Regression Results (Step 1) for Internalizing Symptoms at Wave 2

Predictor	Estimate	SE	z	p
Sex	-0,43	0,04	-10,9	<0.01
Internalizing symptoms at wave 1	0,49	0,02	21,31	<0.01
Externalizing symptoms at wave 1	-0,01	0,02	-0,58	0,562

Note. SE = Standard Error, Total R^2 = 0.3.

Table S4

Regression Results (Step 2) for Internalizing Symptoms at Wave 2

Predictor	Estimate	SE	z	p
Sex	-0,44	0,04	-10,67	<0.01
Internalizing symptoms at wave 1	0,48	0,02	20,84	<0.01
Externalizing symptoms at wave 1	-0,02	0,02	-0,76	0,447
Fluctuation in tempo (sustaining)	0,05	0,02	1,99	0,046
Errors (sustaining)	<0.001	0,02	0,23	0,822
Response inhibition RT (shifting)	0,01	0,02	0,57	0,569
Cognitive flexibility RT (shifting)	<0.001	0,02	0,04	0,965
Cognitive flexibility errors (shifting)	-0,02	0,02	-0,94	0,347
Response inhibition errors (shifting)	-0,01	0,02	-0,37	0,711
RT working memory	-0,01	0,02	-0,6	0,548
Accuracy/errors working memory	-0,01	0,02	-0,44	0,662

Note. SE = Standard Error, Total R^2 = 0.3.

Table S5

Regression Results (Step 1) for Internalizing Symptoms at Wave 3

Predictor	Estimate	SE	z	p
Sex	-0,38	0,04	-9,16	<0.01
Internalizing symptoms at wave 2	0,52	0,02	22,44	<0.01
Externalizing symptoms at wave 2	0,05	0,02	1,97	0,049

Note. SE = Standard Error, Total R^2 = 0.38.

Table S6

Regression Results (Step 2) for Internalizing Symptoms at Wave 3

Predictor	Estimate	SE	z	p
Sex	-0,38	0,04	-8,67	<0.01
Internalizing symptoms at wave 2	0,53	0,02	22,53	<0.01
Externalizing symptoms at wave 2	0,05	0,02	1,91	0,056
Fluctuation in tempo (sustaining)	0,03	0,03	1,18	0,237
Errors (sustaining)	-0,04	0,02	-1,72	0,086
Response inhibition RT (shifting)	0,06	0,03	2,56	0,01
Cognitive flexibility RT (shifting)	-0,01	0,02	-0,48	0,629
Cognitive flexibility errors (shifting)	0,04	0,03	1,66	0,097
Response inhibition errors (shifting)	-0,01	0,02	-0,22	0,827

Table S6

Continued

Predictor	Estimate	SE	z	p
RT working memory	-0,02	0,02	-0,76	0,448
Accuracy/errors working memory	0,01	0,02	0,41	0,683

Note. RT = Reaction Times, SE = Standard Error, Total R^2 = 0.39.

Table S7

Regression Results (Step 1) for Externalizing Symptoms at Wave 2

Predictor	Estimate	SE	z	p
Sex	-0,02	0,04	-0,51	0,611
Internalizing symptoms at wave 1	0,06	0,02	2,44	0,015
Externalizing symptoms at wave 1	0,43	0,02	17,7	<0.01

Note. SE = Standard Error, Total R^2 = 0.22.

Table S8

Regression Results (Step 2) for Externalizing Symptoms at Wave 2

Predictor	Estimate	SE	z	p
Sex	-0,03	0,04	-0,71	0,48
Internalizing symptoms at wave 1	0,05	0,02	2,21	0,027
Externalizing symptoms at wave 1	0,42	0,02	16,9	<0.01
Fluctuation in tempo (sustaining)	0,05	0,03	2,02	0,043
Errors (sustaining)	0,04	0,02	1,89	0,059
Response inhibition RT (shifting)	0,03	0,02	1,28	0,202
Cognitive flexibility RT (shifting)	-0,02	0,02	-1,02	0,309
Cognitive flexibility errors (shifting)	0,01	0,02	0,29	0,77
Response inhibition errors (shifting)	-0,02	0,02	-0,83	0,409
RT working memory	-0,01	0,02	-0,57	0,569
Accuracy/errors working memory	0,03	0,02	1,25	0,211

Note. RT = Reaction Times, SE = Standard Error, Total R^2 = 0.21.

Table S9

Regression Results (Step 1) for Externalizing Symptoms at Wave 3

Predictor	Estimate	SE	z	p
Sex	0,09	0,04	2,13	0,034
Internalizing symptoms at wave 2	0,01	0,02	0,48	0,634
Externalizing symptoms at wave 2	0,57	0,02	23,69	<0.01

Note. SE = Standard Error, Total R^2 = 0.33.

Table S10

Regression Results (Step 2) for Externalizing Symptoms at Wave 3

Predictor	Estimate	SE	z	p
Sex	0,09	0,05	1,98	0,047
Internalizing symptoms at wave 2	0,01	0,02	0,24	0,812
Externalizing symptoms at wave 2	0,58	0,02	23,47	<0.01
Fluctuation in tempo (sustaining)	0,05	0,03	1,82	0,068
Errors (sustaining)	0,01	0,02	0,54	0,586
Response inhibition RT (shifting)	0,02	0,03	0,91	0,361
Cognitive flexibility RT (shifting)	0,01	0,03	0,24	0,811
Cognitive flexibility errors (shifting)	<0.001	0,03	-0,01	0,996
Response inhibition errors (shifting)	0,01	0,03	0,38	0,702
RT working memory	-0,02	0,03	-0,75	0,453
Accuracy/errors working memory	0,03	0,02	1,26	0,208

Note. RT = Reaction Times, SE = Standard Error, Total R^2 = 0.34.

Section 7: Bootstrapping stability analysis

Figure S11

Bootstrapping Results for Cross-Sectional Network at Wave 1

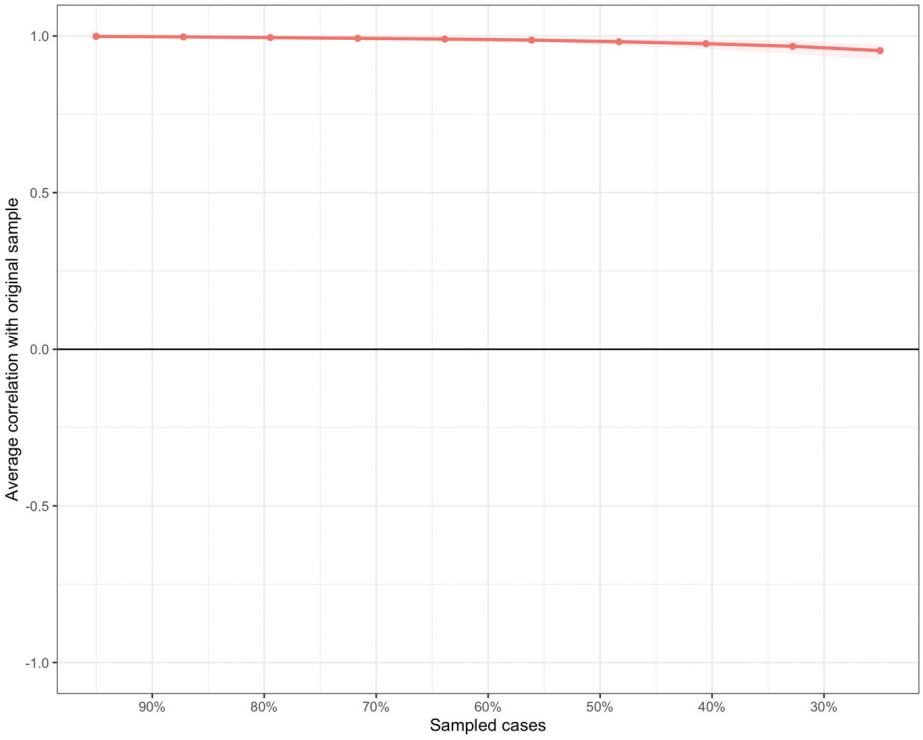


Figure S12

Bootstrapping Results for Cross-Sectional Network at Wave 2

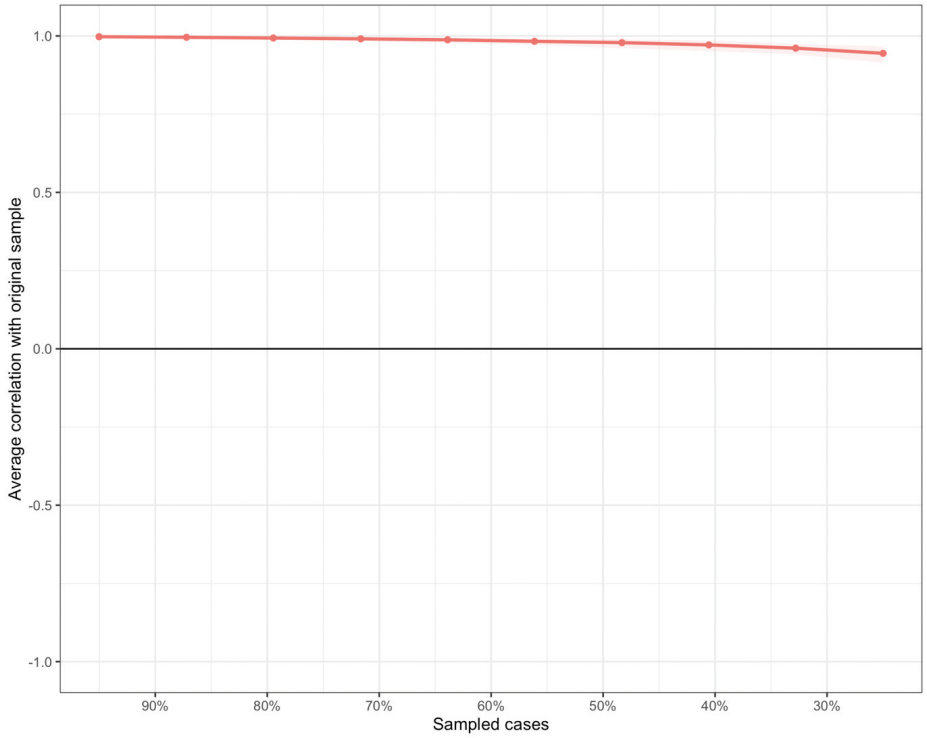
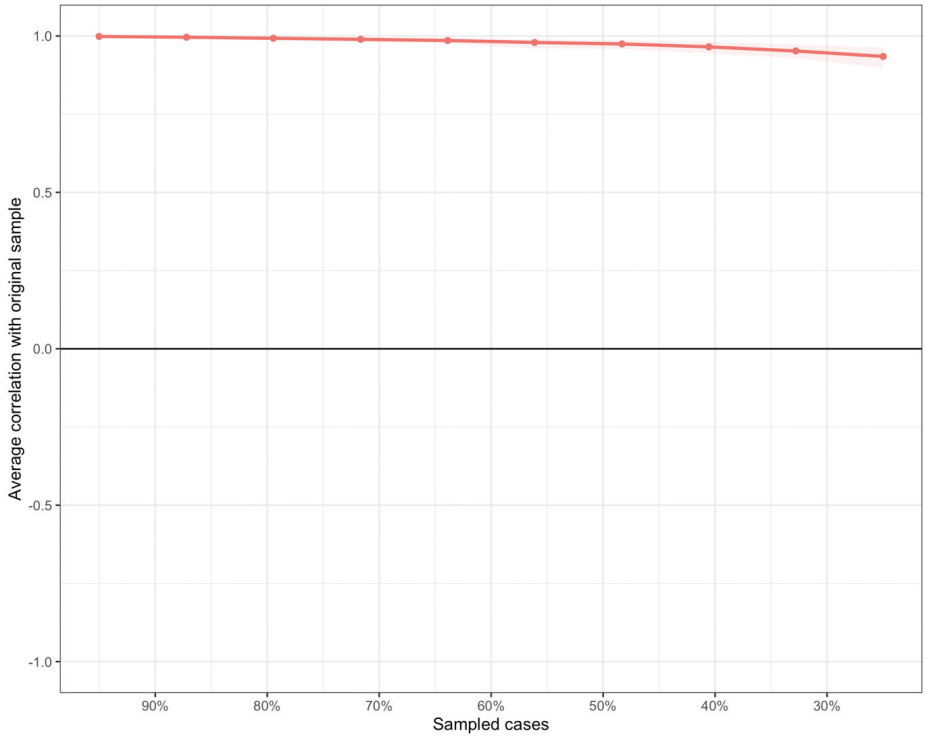


Figure S13

Bootstrapping Results for Cross-Sectional Network at Wave 3



Supplement to Chapter 3. Drinking Motives, Personality Traits, Life Stressors - Identifying Pathways to Harmful Alcohol Use in Adolescence Using a Panel Network Approach

Supplemental Materials Section 1: Materials

Table S1

Subscales and Corresponding Items for Drinking Motives Questionnaire

Subscale	Items
Coping depression	To forget your worries?
	To cheer up when you're in a bad mood?
	Because it helps you when you are feeling depressed?
	To numb your pain?
	To stop you dwelling on things?
	To turn off negative thoughts about yourself?
	To help you feel more positive about things in your life?
	To stop you from feeling so hopeless about the future?
	To forget painful memories?
Conformity	So that others won't kid you about not drinking?
	You drink to fit in with a group you like?
	To be liked?
	So you won't feel left out?
	Because your friends pressure you to drink?
Social	To be sociable?
	Because it makes social gatherings more fun?
	Because it improves parties and celebrations?
	To celebrate a special occasion with friends?
	Because it is what most of your friends do when you get together?
Coping anxiety	Because it helps you when you are feeling nervous?
	Because you feel more self-confident or sure of yourself?
	To relax?
	To reduce your anxiety?
Enhancement	Because you like the feeling?
	Because it's exciting?
	To get high?
	Because it gives you a pleasant feeling?
	Because it's fun?

Table S2

Overview of Adapted Item Wordings of the Drinking Motives Questionnaire

Subscale	Adapted version	Original version (Grant et al., 2007)
Conformity	So that others won't kid you about not drinking?	So that others won't kid me about not using
Conformity	Because your friends won't pressure you to drink?	Because my friends pressure me to use
Social	Because it makes social gatherings more fun?	Because it makes a social gathering more enjoyable
Social	Because it improves parties and celebrations?	As a way to celebrate
Social	To celebrate a special occasion with friends?	Because it is customary on special occasions
Enhancement	Because it gives you a pleasant feeling?	Because it makes me feel good

Table S3

Cronbach Alpha For All Measures at All Waves

	Wave 2	Wave 3	Wave 4
AUDIT quantity and frequency	0.75	0.74	0.64
AUDIT related problems	0.58	0.69	0.72
DMQ coping anxiety	0.75	0.79	0.78
DMQ coping depression	0.92	0.91	0.92
DMQ conformity	0.85	0.84	0.78
DMQ social	0.87	0.85	0.84
DMQ enhancement	0.89	0.86	0.85
SURPS impulsivity	0.61	0.62	0.62
SURPS sensation-seeking	0.6	0.65	0.66
NEO-FFI neuroticism	0.85	0.87	0.88
NEO-FFI extraversion	0.75	0.75	0.79
NEO-FFI openness	0.7	0.74	0.75
NEO-FFI agreeableness	0.71	0.77	0.74
NEO-FFI conscientiousness	0.85	0.86	0.86

Note. AUDIT = Alcohol Use Disorders Identification Test, DMQ = Drinking Motives Questionnaire, LEQ = Life Events Questionnaire, NEO-FFI = Neuroticism-Extraversion-Openness Five Factor Inventory, SURPS = Substance Use Risk Profile Scale.

Supplemental Materials Section 2: Missingness Analysis

We observed attrition of participants throughout all waves of the study, with 1630 (89.12%) eligible participants providing data at wave 2, followed by a decrease to 1471 (80.43%) at wave 3, and a further decline to 1333 (72.89%) participants at wave 4. An overview of missing values for each measure is provided in Table S4.

We used Full-Information-Maximum-Likelihood Estimation (1) in the panel GVAR model to account for missingness. This estimation method uses all available data, including incomplete responses. In FIML, the likelihood function is maximized based on existing responses.

Assuming data missing at random, FIML and multiple imputation lead to similar estimates (2). FIML offers advantages over complete case analysis when and is commonly used in panel GVAR applications (3,4).

We used Little's missing completely at random (MCAR) test (5) to test whether MCAR is present. The significant test ($p < 0.01$) indicates that data were not missing completely at random; thus, complete case analysis is not advisable.

Table S4

Percentage of Missing Values for Each Measure at Waves 2, 3, and 4

	Wave 2 Missing (%)	Wave 3 Missing (%)	Wave 4 Missing (%)
AUDIT quantity and frequency	11.1	19.63	27.17
AUDIT alcohol-related problems	11.1	19.63	27.17
DMQ anxiety	22.85	26.95	31.82
DMQ depression	22.85	26.95	31.82
DMQ conformity	22.85	26.95	31.82
DMQ social	22.85	26.95	31.82
DMQ enhancement	22.85	26.95	31.82
SURPS impulsivity	12.58	20.1	28.21
SURPS sensation-seeking	12.58	20.1	28.21
NEO-FFI neuroticism	12.41	20.01	27.99
NEO-FFI extraversion	12.41	20.01	27.99
NEO-FFI openness	12.41	20.01	27.99
NEO-FFI agreeableness	12.41	20.01	27.99
NEO-FFI conscientiousness	12.41	20.01	27.99
LEQ stressful life events	16.46	21.49	28.65

Note. Percentages of missing value for each measure were calculated based on data from 1829 unique individuals that drank at least once on waves 2, 3, or 4. AUDIT = Alcohol Use Disorders Identification Test, DMQ = Drinking Motives Questionnaire, LEQ = Life Events Questionnaire, NEO-FFI = Neuroticism-Extraversion-Openness Five Factor Inventory, SURPS = Substance Use Risk Profile Scale.

Supplemental Materials Section 3: Sample Characteristics

Table S5

Descriptive Sample Characteristics at All Waves

	Wave 2 M (SD)	Wave 3 M (SD)	Wave 4 M (SD)
Age (in full years)	16.1 (0.73)	18.5 (0.68)	22.1 (0.70)
% female	52.36 %	52.20 %	53.60 %
N	1630	1471	1333
AUDIT quantity and frequency	2.98 (2.29)	4.37 (2.48)	4.25 (2.19)
AUDIT alcohol-related problems	1.08 (2.09)	1.71 (2.82)	2.00 (3.01)
DMQ anxiety	1.65 (0.77)	1.84 (0.83)	1.84 (0.83)
DMQ depression	1.32 (0.57)	1.36 (0.57)	1.34 (0.57)
DMQ conformity	1.28 (0.54)	1.34 (0.55)	1.33 (0.51)
DMQ social	2.46 (1.04)	2.80 (0.97)	2.76 (0.97)
DMQ enhancement	2.23(1.05)	2.45 (0.99)	2.33 (0.96)
SURPS impulsivity	11.4 (2.15)	11.1 (2.15)	10.7 (2.19)
SURPS sensation-seeking	16.3 (3.03)	16.5 (3.19)	16.5 (3.39)
NEO-FFI neuroticism	34.5(7.87)	33.1 (8.28)	33.1 (8.76)
NEO-FFI extraversion	41.5 (5.73)	41.5 (5.81)	40.8(6.33)
NEO-FFI openness	39.3 (6.20)	40.9 (6.49)	42.1 (6.54)
NEO-FFI agreeableness	41.7 (5.43)	43.6 (5.77)	44.9 (5.61)
NEO-FFI conscientiousness	40.2 (6.89)	41.8 (7.22)	43.4 (7.09)
LEQ stressful life events	4.81 (2.58)	3.15 (2.12)	2.27 (1.96)

4. Note. N refers to the number of participants at the respective time points with available data for at least one of the variables of interest. AUDIT = Alcohol Use Disorders Identification Test, DMQ = Drinking Motives Questionnaire, LEQ = Life Events Questionnaire, NEO-FFI = Neuroticism-Extraversion-Openness Five Factor Inventory, SURPS = Substance Use Risk Profile Scale. M represents the mean and SD the corresponding standard deviation in parentheses.

Supplementary Materials Section 4: Temporal and Contemporaneous Estimates

Table S6

Edge Weights for Temporal Associations (Pruned Temporal Network)

	freq	rprob	neg	neur	extr	open	agre	consc	copa	copd	conf	soc	enh	imp	sens
freq	0.29	0.08	0	0	0	0	0	0	0	0	-0.1	0	0	0	0.09
rprob	0	0.26	0	0	0	0	0	0	0	0	0	0	0	0	0
neg	0	0	0.22	0	0	0	0	0	0	0	0	0	0	0	0
neur	0	0	0.12	0.46	0	0	0	0	0	0.05	0	0	0	0.12	0
extr	0	0	0	0	0.45	0	0	0	-0.05	0	0	0	0	0.19	0.08
open	0	0.07	0	0	0	0.36	0	0	0.06	0	0	0	0	-0.08	0
agre	0	-0.05	0	0	0.06	0	0.5	0	0	-0.05	0	0	0	-0.16	0
consc	0	0	0	0	0.03	-0.07	0	0.5	0	0	0	0	0	-0.11	0
copa	0	0	0	0	0	0	0	0	0.28	0.07	0.09	0.08	0	0	0
copd	0	0	0	0	0	0	0	0	0	0.23	0	-0.07	0	0	0
conf	0	0	0	0	0	0	0	0	0	0	0.11	0	0	0	0
soc	0.12	0.07	0	0	0	0	0	0	0.07	0	0.1	0.3	0.13	0	0
enh	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0
imp	0	0	0	0	0	-0.08	0	0	0	0	0	0	0	0.2	0
sens	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.34

Note. The lower triangular refers to outgoing associations from nodes in the rows to nodes in the respective columns. The upper triangular refers to associations outgoing from the nodes in the columns to the respective rows. freq = alcohol use quantity and frequency, rprob = alcohol-related problems, neg = stressful life events, neur = neuroticism, extr = extraversion, open = openness, agre = agreeableness, consc = conscientiousness, copa = coping anxiety motive, copd = coping depression motive, conf = conformity, soc = social, enh = enhancement, imp = impulsivity, sens = sensation seeking.

Table S7

Edge Weights for Contemporaneous Network (Pruned Contemporaneous Network)

	freq	rprob	neg	neur	extr	open	agre	consc	copa	copd	conf	soc	enh	imp	sens
freq	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
rprob	0.3	-	-	-	-	-	-	-	-	-	-	-	-	-	-
neg	0	0	-	-	-	-	-	-	-	-	-	-	-	-	-
neur	-0.13	0.04	0.18	-	-	-	-	-	-	-	-	-	-	-	-
extr	0.07	0	0	-0.29	-	-	-	-	-	-	-	-	-	-	-
open	0	0.04	0	0.08	0	-	-	-	-	-	-	-	-	-	-
agre	-0.07	0	0	-0.09	0.2	0.11	-	-	-	-	-	-	-	-	-
consc	-0.12	0	0	-0.18	0.11	0	0.07	-	-	-	-	-	-	-	-
copa	0	0	0	0.11	0	0	0	0	-	-	-	-	-	-	-

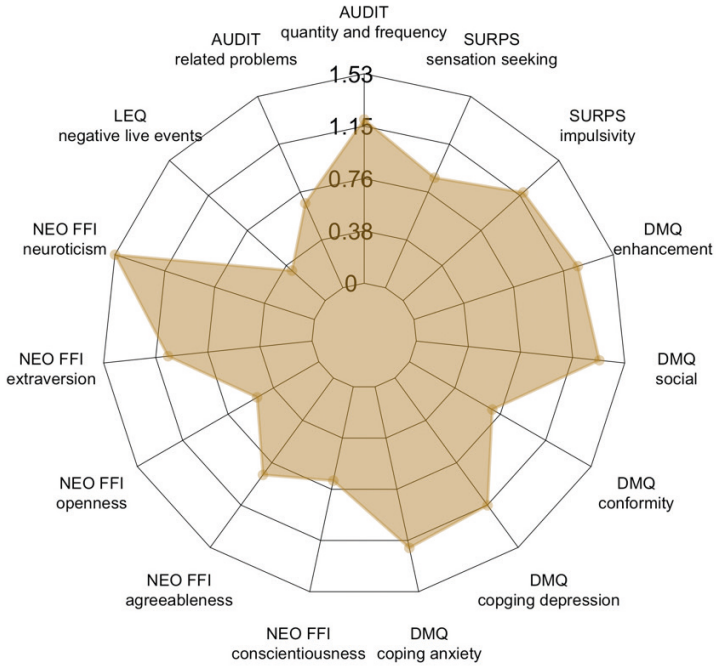
Table S7
Continued

	freq	rprob	neg	neur	extr	open	agre	consc	copa	copd	conf	soc	enh	imp	sens
copd	0.08	0.13	0.07	0.16	0	0	0	0	0.41	-	-	-	-	-	-
conf	-0.11	0	0	0	0	0	0	0	0.22	0.08	-	-	-	-	-
soc	0.17	0	0	0	0	0	0	0	0.29	-0.14	0.23	-	-	-	-
enh	0.15	0.06	0	0	0.09	0	0	0	0.18	0.09	-0.06	0.52	-	-	-
imp	0	0.09	0	0.14	0.18	-0.1	-0.25	-0.22	0	0	0	0	0	-	-
sens	0	0	0.08	-0.12	0.12	0.18	-0.08	0	0	0	0	0	0.11	0.2	-

Note. The contemporaneous network is undirected (symmetrical) so the upper triangular is included merely for completeness. freq = alcohol use quantity and frequency, rprob = alcohol-related problems, neg = stressful life events, neur = neuroticism, extr = extraversion, open = openness, agre = agreeableness, consc = conscientiousness, copa = coping anxiety motive , copd = coping depression motive, conf = conformity, soc = social, enh = enhancement, imp = impulsivity, sens = sensation seeking.

Supplemental Materials Section 5: Strength Centrality and Zero-Order Correlations

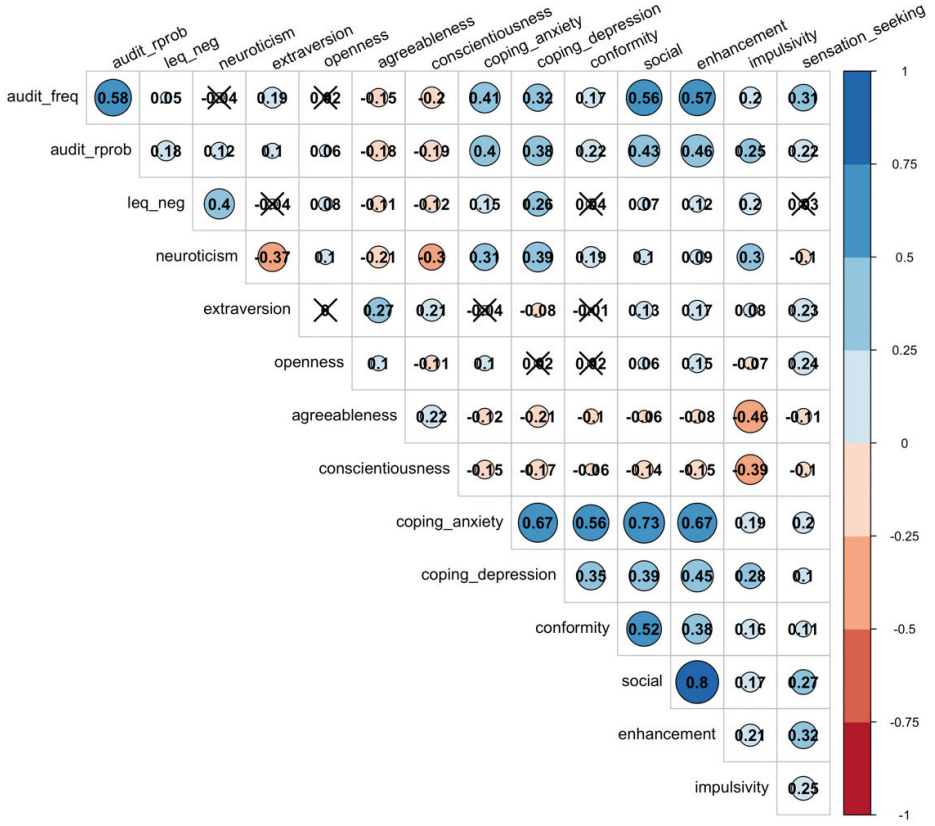
Figure S1
Strength Centrality of Pruned Contemporaneous Network



Note: AUDIT = Alcohol Use Disorders Identification Test, DMQ = Drinking Motives Questionnaire, LEQ = Life Events Questionnaire, NEO-FFI = Neuroticism-Extraversion-Openness Five Factor Inventory, SURPS = Substance Use Risk Profile Scale.

Figure S2

Zero-Order Correlation Between All Variables across Waves



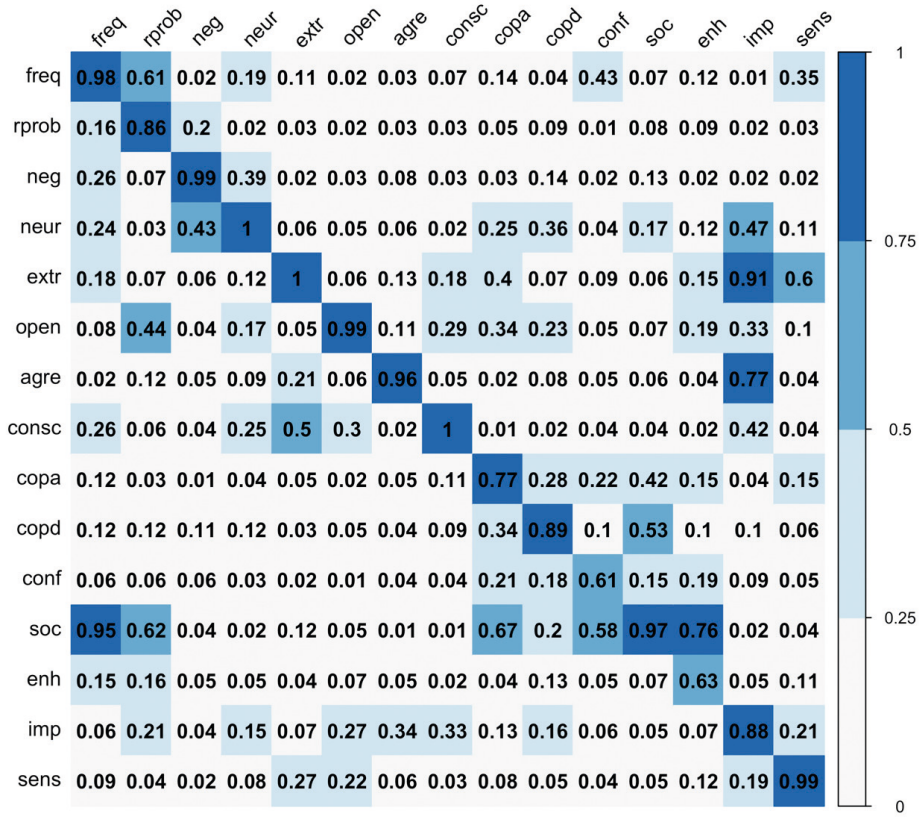
Note: The size and color of the circles represent the strength and direction of the correlations respectively. Non-significant correlations ($p \geq 0.05$) are crossed out in the figure.

Supplemental Materials Section 6: Network Stability - Bootstrapping analysis

Figures S3 and S4 describe the proportion of edges present in the pruned models (n = 1000) derived from the bootstrapping analysis (using 75% of the full sample) for the full sample networks.

Figure S3

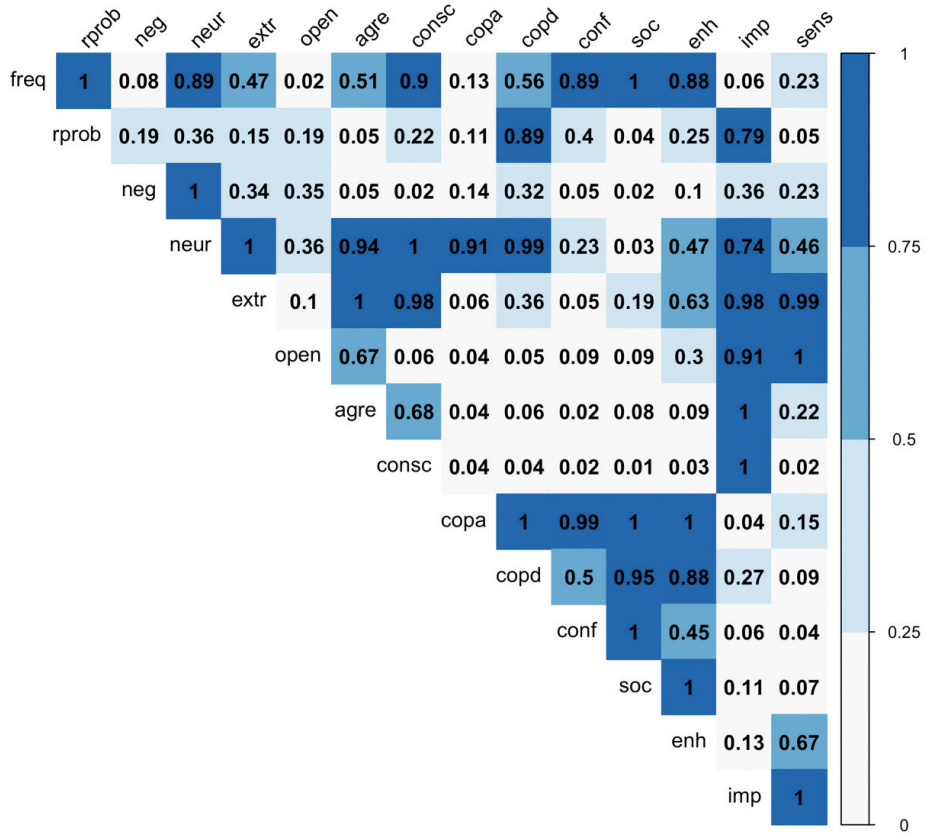
Bootstrapping Results for Temporal Network



Note. The lower triangular refers to outgoing associations from nodes in the rows to nodes in the respective columns. The upper triangular refers to associations outgoing from the nodes in the columns to the respective rows.

Figure S4

Bootstrapping Results for Contemporaneous Network



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Supplement to Chapter 4. Investigating risk factor and consequence accounts of executive functioning impairments in psychopathology: an 8-year study of at-risk individuals in Brazil

Supplementary Materials Section 1: Missingness

Percentage of missingness. The percentage of missing values for each measure at the different waves is shown in Table S5.

Table S1

Percentage of Missing Values for Each Measure at Waves 2 and 3

	Wave 2	Wave 3
Internalizing sum score	18.02	27.76
Externalizing sum score	18.02	27.76
Anxiety subscale	18.02	28.31
Withdrawn-depressed subscale	18.02	28.11
Somatic problems subscale	18.07	28.16
Thought problems subscale	18.02	28.11
Attention problems subscale	18.02	28.11
Rule-breaking problems subscale	18.02	28.41
Aggressive problems subscale	18.02	28.06
Mean RT Go	34.29	42.77
Percentage commission errors	29.47	35.79
Digit span task	26.56	34.14

Note. The percentages of missing values for each measure were calculated based on data from 1992 individuals with no missing data in wave 1. RT = Reaction Times.

Predictors of missingness. We investigated whether these symptom and cognitive measures at the first wave could predict dropout at any point during the study. Of all participants, 1,292 had available symptom data across all waves, while 700 individuals dropped out at some point. A logistic regression model was constructed, incorporating gender, age, internalizing and externalizing symptoms (measured at wave 1), mean reaction time in Go trials, percentage of commission errors, and digit span (all measured at wave 1) as predictors. Table S2 presents the results from the regression model.

Table S2

Regression model predicting drop-out during the study using wave 1 measures

Predictors	Estimates
Intercept	0.22 **
Age at wave 1	0.01
Gender (female)	0.01
Internalizing sum score	-0.03 *
Externalizing sum score	0.01
GoNoGo mean RT correct Go	0.00
GoNoGo percentage commission errors	0.01
Working memory digit span	0.00

Note. * p<0.05 ** p<0.01

Table S3

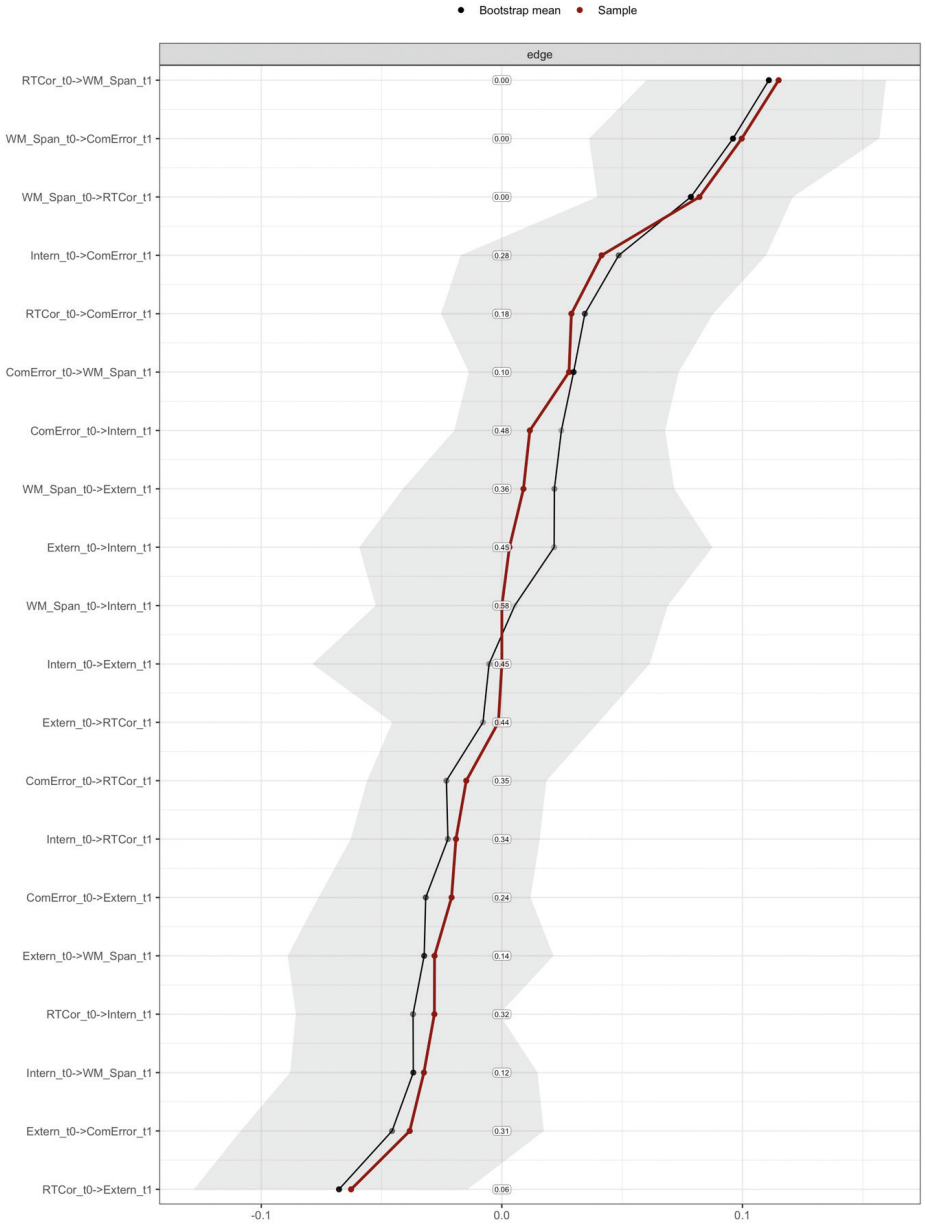
	Wave 1		Wave 2		Wave 3	
	t/ χ^2 statistic	p	t/ χ^2 statistic	p	t/ χ^2 statistic	p
Sex: % Female	2.279	0.131	0.83	0.362	1.245	0.264
Age in Years	3.277	0.001	2.618	0.009	2.652	0.008
Internalizing sum score	6.472	< 0.001	4.888	< 0.001	2.136	0.033
Externalizing sum score	6.542	< 0.001	4.692	< 0.001	2.994	0.003
Anxiety subscale	5.935	< 0.001	3.954	< 0.001	1.468	0.143
Withdrawn-depressed subscale	5.699	< 0.001	5.1	< 0.001	2.112	0.035
Somatic problems subscale	4.882	< 0.001	3.434	0.001	1.962	0.050
Thought problems subscale	5.228	< 0.001	3.223	0.001	3.552	< 0.001
Attention problems subscale	8.005	< 0.001	4.752	< 0.001	3.978	< 0.001
Rule-breaking problems subscale	4.502	< 0.001	3.813	< 0.001	2.736	0.006
Aggressive problems subscale	6.969	< 0.001	4.702	< 0.001	2.799	0.005
GoNoGo mean RT correct Go	0.916	0.360	1.605	0.109	3.219	0.001
GoNoGo percentage commission errors	1.16	0.246	2.152	0.032	0.878	0.380
WM Digit Span Task regular	-0.169	0.866	-1.056	0.291	-0.994	0.320
WM Digit Span Task corsi	-0.891	0.373	-0.551	0.581	-2.289	0.022

Test statistics for comparisons of demographic-clinical characteristics

Note. This table refers to the comparisons between the high-risk and randomly selected community samples at each wave. χ^2 refers to the comparisons with respect to gender (male/female). Chi-squared tests were used to compare gender distributions, while two-sided t-tests were used to assess differences in all symptom and cognitive measures.

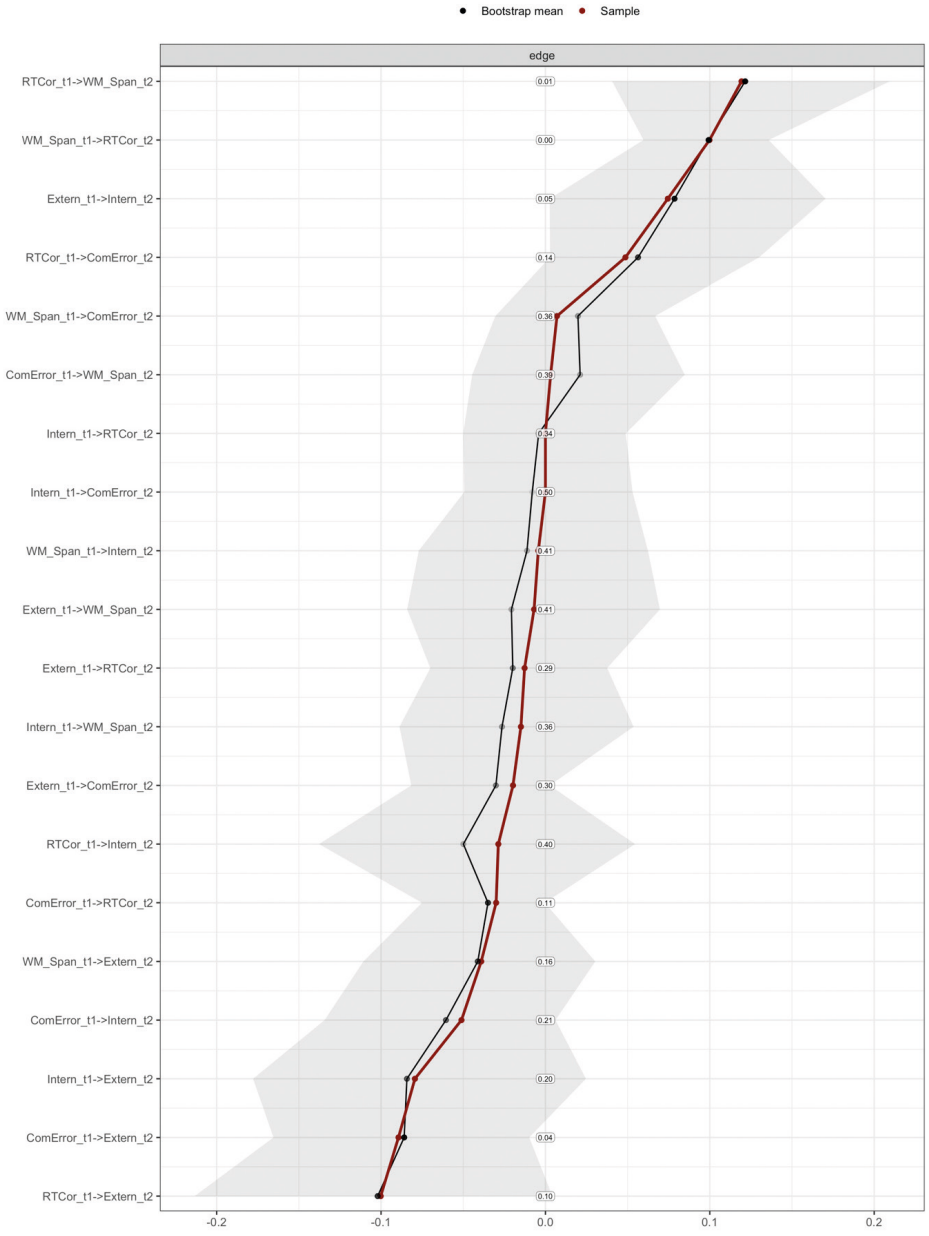
Supplementary Materials Section 2: Network stability

Figure S1. Non-parametric Bootstrapping Results for Waves 1 → 2 Network



Note. t0 = wave 1, t1 = wave 2. The grey area indicates the 95% bootstrapped confidence interval of the estimated edge weights (standardized estimates) around the sampled values (in red). The middle bar denotes the proportion of estimates being zero.

Figure S2. Non-parametric Bootstrapping Results for Waves 2 → 3 Network.



Note. t1 = wave 1, t2 = wave 2. The grey area indicates the 95% bootstrapped confidence interval of the estimated edge weights (standardized estimates) around the sampled values (in red). The middle bar denotes the proportion of estimates being zero.

Supplementary Materials Section 3: Sensitivity analysis including gender as a covariate

Figure S3.

Waves 1 → 2 Network Including Gender as a Covariate

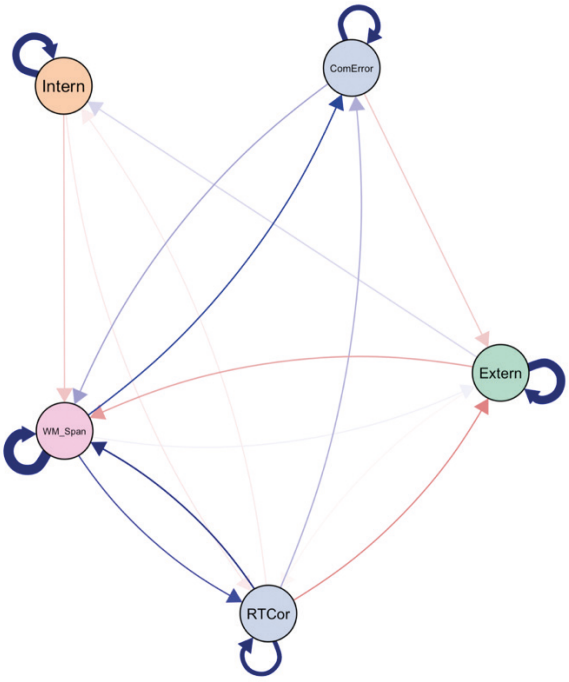
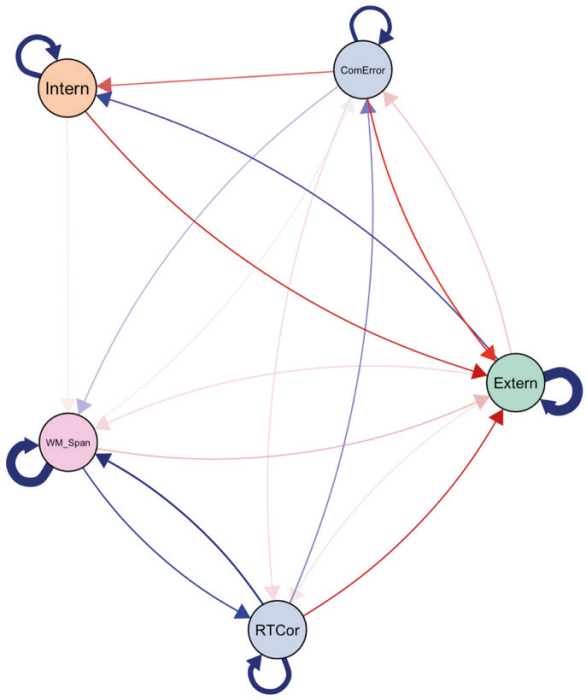


Figure S4.

Waves 2 → 3 Network Including Gender as a Covariate



Supplement to Chapter 5. Impaired Working Memory and Risk-Taking Predict Detrimental Symptom Dynamics in Adolescence – A Moderated Cross-Lagged Panel Network Approach

Supplementary Materials Section 1: Strengths and Difficulties Questionnaire

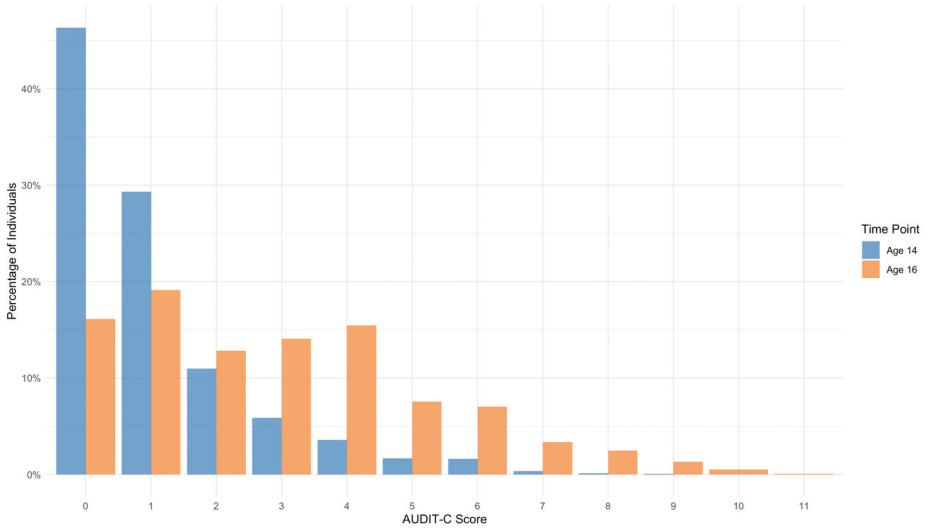
Table S1

Strengths and Difficulties Questionnaire Items and the Corresponding Scale

Item No.	Item	Scale
1	Considerate of other people"s feelings	Prosocial
2	Restless, overactive, cannot stay still for long	Hyperactivity/inattention
3	Often complains of headaches, stomach-aches, or sickness	Emotional Symptoms
4	Shares readily with other young people, for example CDs, games, food	Prosocial
5	Often loses temper	Conduct Problem
6	Would rather be alone than with other young people	Peer Problem
7	Generally well behaved, usually does what adults request	Conduct Problem
8	Many worries or often seems worried	Emotional Symptoms
9	Helpful if someone is hurt, upset or feeling ill	Prosocial
10	Constantly fidgeting or squirming	Hyperactivity/inattention
11	Has at least one good friend	Peer Problem
12	Often fights with other young people or bullies them	Conduct Problem
13	Often unhappy, depressed or tearful	Emotional Symptoms
14	Generally liked by other young people	Peer Problem
15	Easily distracted, concentration wanders	Hyperactivity/ inattention
16	Nervous in new situations, easily loses confidence	Emotional Symptoms
17	Kind to younger children	Prosocial
18	Often lies or cheats	Conduct Problem
19	Picked on or bullied by other young people	Peer Problem
20	Often volunteers to help others (parents, teachers, children)	Prosocial
21	Thinks things out before acting	Hyperactivity/inattention
22	Steals from home, school or elsewhere	Conduct Problem
23	Gets along better with adults than with other young people	Peer Problem
24	Many fears, easily scared	Emotional Symptoms
25	Good attention span, sees chores or homework through to the end	Hyperactivity/inattention

Figure S1

Distribution of the AUDIT-C score at both assessment points.



Note. AUDIT = Alcohol Use Disorders Identification Test. The AUDIT-C consists of the first three items of the AUDIT and assesses frequency of drinking. The y-axis indicates the frequency (percentage of individuals) relative to the total number within each wave.

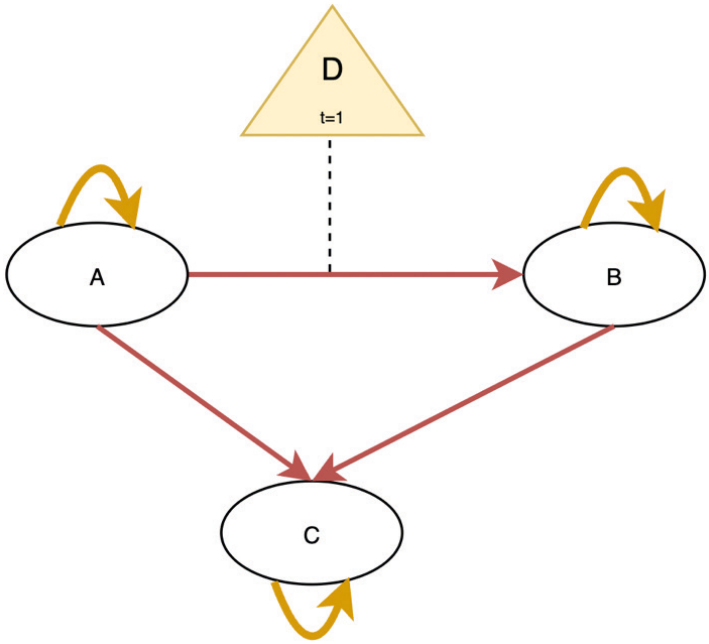
Supplementary Materials Section 2: Preliminary Simulation Study

Simulation study setup

To examine the performance of this moderated CLPN approach, we conducted a preliminary simulation study. We assessed the effectiveness of the moderated CLPN approach (i.e., sensitivity, specificity, estimation error) at varying sample sizes (n = 100-1,000). The main objective was to assess the ability of the moderated CLPN to uncover the true underlying interaction effects. Following previous simulation studies on the CLPN and related network models (Freichel, Veer, et al., 2024; Freichel & Epskamp, 2024), we computed relevant evaluation metrics (e.g., specificity) across 1,000 iterations. First, we defined specific characteristics of the true, data generating model based on prior empirical results: We set autocorrelations to 0.4, cross-lagged effects to 0.2, and moderation effects to 0.1. The value for the moderation effect was chosen based on intuition, as higher-order interaction effects are expected to be smaller than the cross-lagged effects. To explore how the moderated CLPN's ability to detect moderation effects is influenced by sample size (n), we generated datasets with 100, 500, and 1,000 observations. The true model included three variables (a, b, c) that were assessed at two time points. An additional variable d, only assessed at t1, showed a significant moderation effect on the temporal association between a and b. See Figure S2 for an illustration of the true model used for the simulation study.

Figure S2

True network model structure used for simulation study.



Note. The network indicates the true network structure (with three nodes A, B, and C) used for the simulation study. The visualization follows the approach introduced in Figure 1 in the main text. The triangle indicates an interaction effect for the cross-lagged temporal association (A → B).

In each iteration, data was sampled from the true network structure and the moderated CLPN was estimated following the specifications described above. We compared the estimated network structure to the true network and computed relevant evaluation metrics (separately for direct and moderation effects), including specificity, sensitivity, and the mean absolute estimation error following Freichel and Epskamp (2024). Sensitivity describes the proportion of edges that are correctly identified as present; specificity indicates the proportion of edges that are correctly identified as absent. The estimation error refers to the average difference between the estimated and true non-zero edge weights. Both sensitivity and specificity are important when evaluating the entire network structure, while estimation error is important when considering the strongest edges in the network.

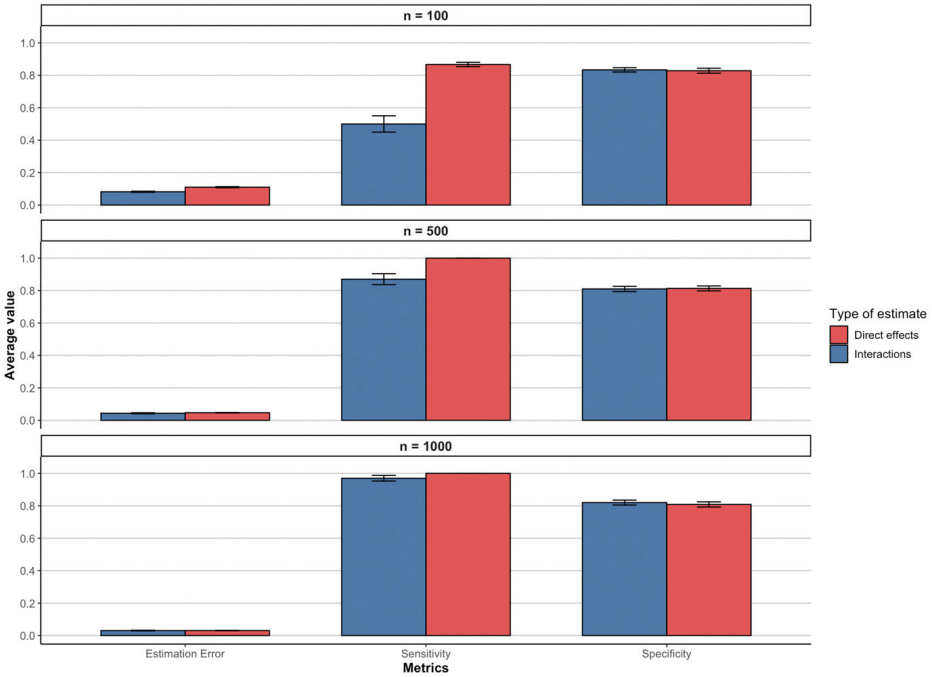
Performance of the moderated CLPN at varying sample sizes

Figure S3 visualizes the simulation results for 100, 500, and 1000 observations. We observed overall high levels of sensitivity and moderate/low levels of specificity, which aligns with prior simulation studies that evaluated the CLPN (Freichel, Veer, et al., 2024). This suggests that the probability of detecting false positive edges is relatively high while the model is reliable in identifying actual edges.

As expected, increasing the sample size led to improvements in the model performance (i.e., lower estimation error and higher sensitivity and specificity). Importantly, at large sample sizes (n = 1,000), the model showed a similar performance in detecting both moderation and direct effects. However, at small and moderate sample sizes (n = 100-500 the sensitivity for correctly including moderation effects was substantially lower (0.51) compared to direct effects (0.89). This suggests the model had greater difficulty detecting true moderation effects with less statistical power.

Figure S3

Simulations results for moderated CLPN.



Note. n = number of observations; estimation error = mean absolute difference between trye and estimated non-zero edge weights; sensitivity = proportion of correctly identified present edges; specificity = proportion of correctly identified absent edges. Error bars represent the standard error of the mean across the 1,000 iterations for each sample size and metric.

Supplementary Materials Section 2: mCLPN results

Figure S4

Bootstrapping results for moderated CLPN estimates

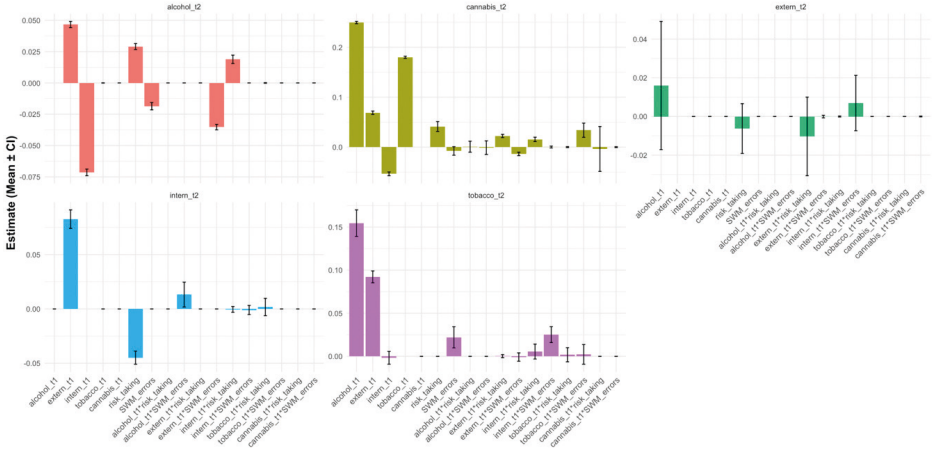


Table S2

Moderated CLPN estimates

	alcohol_t2	extern_t2	intern_t2	tobacco_t2	cannabis_t2
alcohol_t1	0.6253	0	0	0.1475	0.2489
extern_t1	0.0476	0.5239	0.0821	0.0894	0.0681
intern_t1	-0.0725	0	0.5256	0	-0.0528
tobacco_t1	0	0	0	0.5914	0.1799
cannabis_t1	0	0	0	0	0.3925
SWM_errors	-0.0197	0	0	0.0176	0
risk_taking	0.03	0	-0.045	0	0.0417
alcohol_t1*SWM_errors	0	0	0.0125	0	0
extern_t1*SWM_errors	-0.0361	0	0	0	-0.0129
intern_t1*SWM_errors	0	0	0	0.0212	0
tobacco_t1*SWM_errors	0	0	0	0	0.0315
cannabis_t1*SWM_errors	0	0	0	0	0
alcohol_t1*risk_taking	0	0	0	0	0
extern_t1*risk_taking	0	0	0	0	0.0217
intern_t1*risk_taking	0.0202	0	0	0	0.0144
tobacco_t1*risk_taking	0	0	0	0	0

Supplement to Chapter 7. Value-modulated attentional capture in reward and punishment contexts, attentional control, and their relationship with psychopathology

Supplementary Materials Section 1: Convergent Validity Analysis Study 1

Following the preregistration, we also assessed the convergent validity of our novel VMAC task using two behavioral questionnaires that measure sensitivity to reward and punishment, specifically the Behavioral Inhibition System and Behavioral Activation System Questionnaire (BIS/BAS; Carver & White, 1994), and Reward and Punishment Responsivity and Motivation Questionnaire (RPRM-Q; (Jonker et al., 2022). The BIS/BAS consists of 24 self-reported items on a Likert scale (1 = very false for me to 4 = very true for me) and is often used in studies on reward-related attention (e.g., Hickey & Peelen, 2017). The RPRM-Q consists of 18 items on a Likert scale (1 = does not apply to me at all to 5 = applies to me completely) and measures conceptually similar items.

We hypothesized that higher reward sensitivity will be associated with greater attentional capture in the reward context, and punishment sensitivity with greater capture in the punishment context.

Bivariate correlations between VMAC-Reward score and the BAS-scale/Reward Sensitivity (RPRM-Q) outcome score and between the VMAC-Punishment score and the separate BIS-scale/Punishment Sensitivity outcome score were not significant. Additional exploratory correlation analyses were conducted between the RPRM-Q and BIS/BAS questionnaire scores. Reward Sensitivity was significantly related to BAS-scale outcome, and Punishment Sensitivity was significantly related to BIS-scale outcome.

Table S1

Correlation Matrix for VMAC Scores and Reward/Punishment Sensitiviy for Study 1

N = 68	VMAC Reward Score	BAS	Reward Sensitivity	VMAC Punishment Score	BIS	Punishment Sensitivity
VMAC Reward Score	1	0.013	-0.028	0.004	-0.064	-0.119
BAS	0.013	1	0.753 **	-0.016	-0.156	-0.009
Reward Sensitivity	-0.028	0.753 **	1	-0.099	-0.152	0.067
VMAC Punishment Score	0.004	-0.016	-0.099	1	0.002	0.013
BIS	-0.064	-0.156	-0.152	0.002	1	0.703 **
Punishment Sensitivity	-0.119	-0.009	0.067	0.013	0.703 **	1

Note. ** denotes a significant correlation at $p < 0.001$.

Supplementary Materials Section 2: VMAC Analysis in Study 1

ANOVAs for VMAC RTs at separate sessions

Table S2 summarizes all relevant effects for the separate ANOVAs at both sessions. The separate analysis for the punishment VMAC task showed significant effects of both Block and Distractor Type at session 2, indicating that participants both got better at the task with each block, and took longer to respond to the high-punishment distractor (M = 598.65, SD = 64.11), compared to the low-punishment distractor (M = 588.08, SD = 67.36). These unexpected session 2 punishment effects could be a result of attrition bias. However, when restricting analyses of VMAC Punishment trials at session 1 to participants who finished both sessions, the Distractor Type main effect remained non-significant, making selective attrition an unlikely explanation.

Table S2

ANOVA Results for VMAC Reaction Time Effects for Study 1

Analysis	***F***	***p***	***DF***	η^2
Study 1 – Session 1 (n = 72)				
Reward VMAC				
Block	49.634	< 0.001 **	2.45, 174.22	0.411
Distractor Type	19.924	< 0.001 **	1, 71	0.219
Block * Distractor Type	4.132	0.007 *	3, 213	0.055
Punishment VMAC				
Block	39.517	< 0.001 **	2.64, 187.44	0.358
Distractor Type	0.341	0.561	1, 71	0.005
Block * Distractor Type	2.003	0.129	2.37, 168.29	0.027
Study 1 – Session 2 (n = 43)				
Reward VMAC				
Block	10.45	< 0.001 **	3, 126	0.199
Distractor Type	14.924	< 0.001 **	1, 42	0.262
Block * Distractor Type	2.163	0.096	3, 126	0.049
Punishment VMAC				
Block	4.489	0.005 *	3, 126	0.097
Distractor Type	10.289	0.003 *	1, 42	0.197
Block * Distractor Type	1.293	0.28	3, 126	0.03

Note. Degrees of freedom (DF) reported are corrected for sphericity. Effect sizes reported are partial eta squared (η^2). ** denotes p-values below 0.001, and * denotes p-values below 0.01.

Supplementary Materials Section 2: VMAC Accuracy Results Study 1 and 2

ANOVA for VMAC accuracy (including session as factor)

Table S3 summarizes all relevant effects for the ANOVA that included the session as a factor.

Table S3

ANOVA Results for VMAC Accuracy Effects At Both Sessions of Study 1

Analysis	*F*	*p*	*DF*	η^2
Reward VMAC				
Block	20.33	< 0.001**	2.35, 98.63	0.326
Distractor Type	0.848	0.362	1, 42	0.02
Session	42.88	< 0.001**	1, 42	0.505
Block * Distractor Type	0.098	0.961	3, 126	0.002
Block * Session	3.395	0.02*	3, 126	0.075
Distractor Type * Session	0.332	0.567	1, 42	0.008
Block * Distractor Type * Session	0.621	0.603	3, 126	0.015
Punishment VMAC				
Block	4.987	0.003*	3, 126	0.106
Distractor Type	6.627	0.014*	1, 42	0.136
Session	27.38	< 0.001**	1, 42	0.395
Block * Distractor Type	0.524	0.63	2.44, 102.4	0.012
Block * Session	1.214	0.307	3, 126	0.028
Distractor Type * Session	0.002	0.965	1, 42	< 0.001
Block * Distractor Type * Session	1.312	0.274	3, 126	0.03

Note. Degrees of freedom (DF) reported are corrected for sphericity. Effect sizes reported are partial eta squared (η^2). ** denotes p-values below 0.001, and * denotes p-values below 0.05.

ANOVA for VMAC accuracy (separately for two sessions)

We preregistered separate analyses for the two sessions. See Table S4 for the analysis of accuracy effects separately for sessions 1 and 2.

Table S4

ANOVA Results for VMAC Accuracy Effects for Study 1

Predictor	***F***	***P***
Study 1 – Session 1 (n = 72)		
Reward VMAC		
Block	27.965	< 0.001 *
Distractor Type	0.186	0.668
Block * Distractor Type	1.055	0.369
Punishment VMAC		
Block	5.476	0.002 *
Distractor Type	3.561	0.063
Block * Distractor Type	0.403	0.751
Study 1 – Session 2 (n = 43)		
Reward VMAC		
Block	4.1	0.008 *
Distractor Type	1.514	0.225
Block * Distractor Type	0.661	0.578
Punishment VMAC		
Block	5.442	0.001 *
Distractor Type	3.98	0.053
Block * Distractor Type	0.569	0.605

Table S5

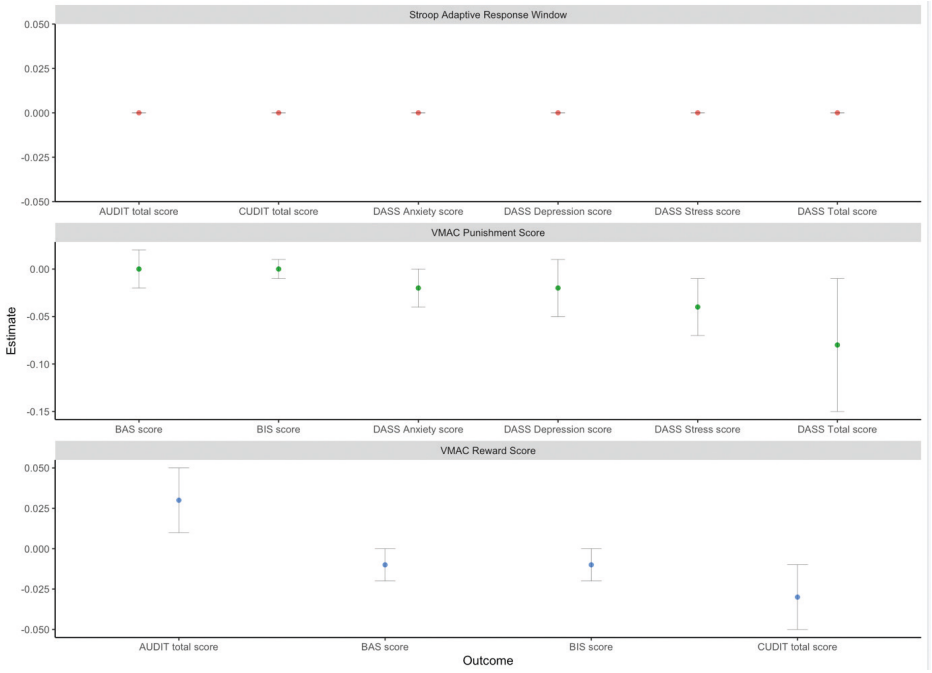
ANOVA Results for VMAC Accuracy Effects for Study 2

Predictor	***F***	***P***
Reward VMAC		
Block	12.536	< 0.001
Distractor Type	1.712	0.194
Block Order	0.777	0.38
Block * Distractor Type	0.186	0.959
Block * Block Order	5.431	< 0.001
Distractor Type * Block Order	0.246	0.621
Block * Distractor Type * Block Order	0.475	0.778

Supplementary Materials Section 3: Associations with Clinical Measures Study 1

Figure S1

Regression Results for Associations Between VMAC Scores, Stroop Deadline Response Window, and Clinical Measures (Study 1)



Note. The vertical bars represent standard errors.

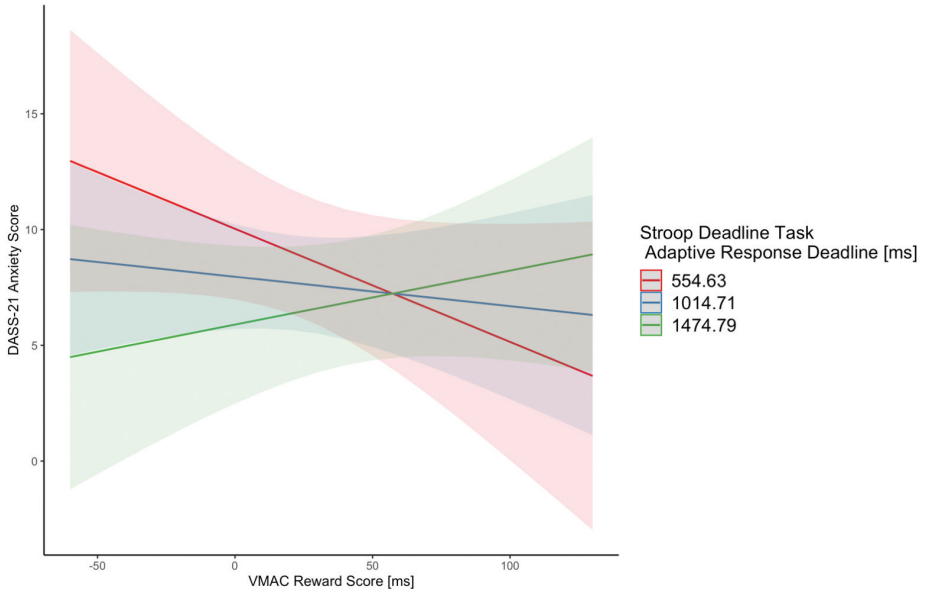
Table S6

Regression Results for Associations Between VMAC Reward/Punishment Scores and Clinical Measures (Study 1)

Effect	**Estimate**	**SE**	***p***
VMAC Reward - AUDIT			
Intercept	7.778	0.987	< 0.001
VMAC Reward Score (last 2 blocks)	0.027	0.019	0.162
Gender Male	-0.39	1.349	0.773
Gender Other	-7.86	5.664	0.17
VMAC Reward - CUDIT			
Intercept	4.466	1.109	< 0.001
VMAC Reward Score (last 2 blocks)	-0.034	0.021	0.114
Gender Male	3.37	1.517	0.03
Gender Other	-3.091	6.366	0.629
VMAC Punishment – DASS Total			
Intercept	30.69	3.094	< 0.001
VMAC Punish Score (last 2 blocks)	-0.078	0.073	0.291
Gender Male	1.101	4.679	0.815
Gender Other	25.569	19.594	0.196
VMAC Punishment – DASS Depression Subscale			
Intercept	8.361	1.415	<0.001
VMAC Punish Score (last 2 blocks)	-0.018	0.034	0.599
Gender Male	4.297	2.139	0.049
Gender Other	17.698	8.958	0.052
VMAC Punishment – DASS Anxiety Subscale			
Intercept	8.114	1.026	<0.001
VMAC Punish Score (last 2 blocks)	-0.018	0.024	0.458
Gender Male	-1.654	1.551	0.29
Gender Other	-2.054	6.494	0.753
VMAC Punishment – DASS Stress Subscale			
Intercept	14.215	1.132	<0.001
VMAC Punish Score (last 2 blocks)	-0.042	0.027	0.12
Gender Male	-1.542	1.712	0.371
Gender Other	9.925	7.169	0.171

Figure S2

Interaction Effect Between Adaptive Response Window, VMAC Reward Score, and Anxiety Symptoms

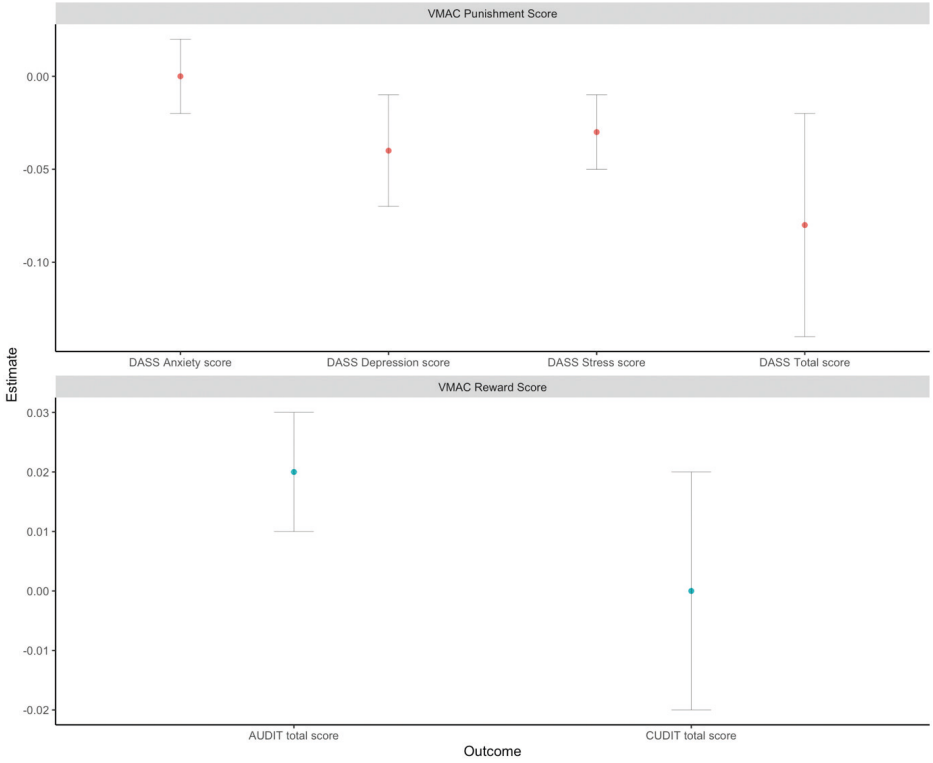


Note. The color indicates the value of the moderator (stroop adaptive response window) one standard deviation below (i.e., red) and above (i.e., green) the mean value that was used to plot the effect of the moderator following the convention suggested by Aiken et al. (1991).

Supplementary Materials Section 4: Associations with Clinical Measures Study 2

Figure S2

Regression Results for Associations between VMAC Scores and Clinical Measures (Study 2)



Note. The regression estimates stem from the regression models that include gender as covariates. The vertical bars display standard errors for the estimates.

Table S7

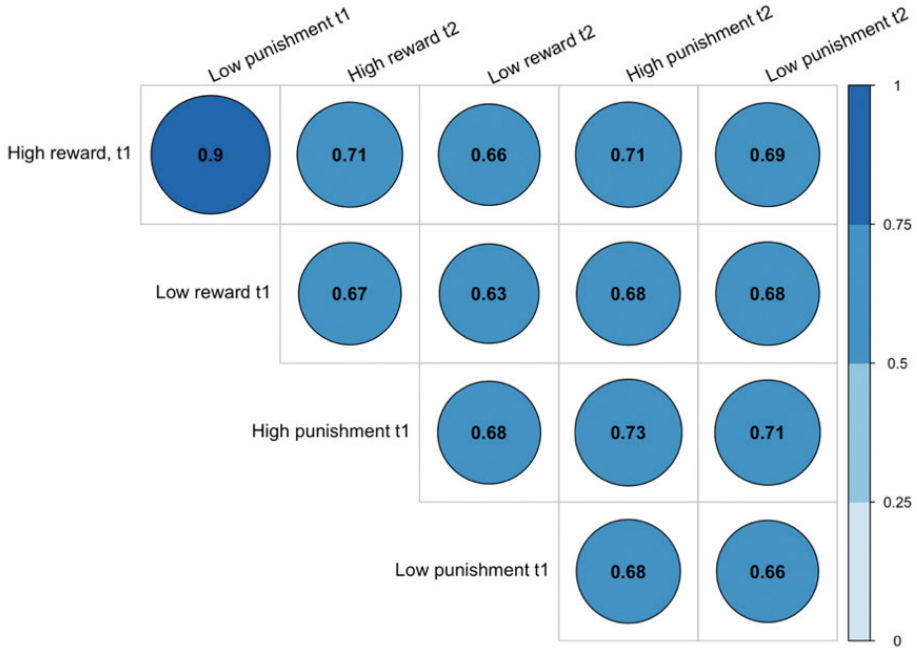
Regression Results for Associations Between VMAC Reward/Punishment Scores and Clinical Measures (Study 2)

Effect (N = 105)	**Estimate**	**SE**	***p***
VMAC Reward - AUDIT			
Intercept	6.878	0.576	< 0.001
VMAC Reward Score (last 2 blocks)	0.019	0.015	0.202
Gender Male	0.715	1.366	0.602
Gender Other	-1.884	3.082	0.542
VMAC Reward - CUDIT			
Intercept	3.378	0.656	< 0.001
VMAC Reward Score (last 2 blocks)	0.004	0.017	0.822
Gender Male	1.485	1.554	0.342
Gender Other	7.154	3.506	0.044
VMAC Punishment – DASS total			
Intercept	35.864	2.438	< 0.001
VMAC Punish Score (last 2 blocks)	-0.077	0.059	0.194
Gender Male	-1.82	5.807	0.755
Gender Other	20.365	13.146	0.124
VMAC Punishment – DASS Depression Subscale			
Intercept	11.165	1.053	<0.001
VMAC Punish Score (last 2 blocks)	-0.042	0.025	0.098
Gender Male	1.537	2.509	0.542
Gender Other	11.559	5.680	0.045
VMAC Punishment – DASS Anxiety Subscale			
Intercept	9.83	0.86	<0.001
VMAC Punish Score (last 2 blocks)	-0.003	0.021	0.881
Gender Male	-2.041	2.048	0.321
Gender Other	5.459	4.636	0.242
VMAC Punishment – DASS Stress Subscale			
Intercept	14.868	0.924	<0.001
VMAC Punish Score (last 2 blocks)	-0.032	0.022	0.161
Gender Male	-1.315	2.201	0.551
Gender Other	3.347	4.983	0.503

Supplementary Materials Section 5: Test-retest correlations

Figure S3

Associations between VMAC RT Measures in Session 1 and 2



Note. All correlations are statistically significant ($p < 0.05$).

Table S8

Cronbach's Alpha Coefficients for All Measures of Interest

Measure	Study 1 – Session 1	Study 1 – Session 2	Study 2
AUDIT total score	0.8	N/A	0.81
CUDIT total score	0.87	N/A	0.88
DASS stress subscale	0.69	0.81	0.80
DASS anxiety subscale	0.73	0.76	0.80
DASS depression subscale	0.87	0.78	0.88
BIS	0.77	0.78	0.84
BAS	0.74	0.74	0.83

Note. The N/A (Not Applicable) refers to measures that have not been assessed during the second session of study 1. AUDIT = Alcohol Use Disorders Identification Test; CUDIT-R = Cannabis Use Disorders Identification Test-Revised; DASS-21 = Depression Anxiety Stress Scales-21. BIS = Behavioral Inhibition System, BAS = Behavioral Activation System.

References

Aiken, L. S., West, S. G., & Reno, R. R. (1991). Multiple regression: Testing and interpreting interactions. Sage Publications.

Hickey, C., & Peelen, M. V. (2017). Reward selectively modulates the lingering neural representation of recently attended objects in natural scenes. The Journal of Neuroscience, 37(31), 7297–7304. https://doi.org/10.1523/JNEUROSCI.0684-17.2017

Jonker, N. C., Timmerman, M. E., & de Jong, P. J. (2022). The reward and punishment responsivity and motivation questionnaire (RPRM-Q): A stimulus-independent self-report measure of reward and punishment sensitivity that differentiates between responsivity and motivation. Frontiers in Psychology, 13. https://www.frontiersin.org/articles/10.3389/fpsyg.2022.929255

Supplement to Chapter 8. Unraveling robust brain-behavior links of depressive complaints through granular network models for understanding heterogeneity

Supplementary Material Section 1: Study and Network Descriptives

Neuroimaging

The neuroimaging procedure is described in detail elsewhere (Schumann et al., 2010; Vulser et al., 2015). Total intracranial volume and sex were used as covariates when estimating hippocampal volume following the procedure by Hilland et al. (2020). Total intracranial volume was based on the summation of gray matter, cerebrospinal fluid (CSF), and white matter volumes, and this measure was used in prior work on this dataset (Quinlan et al., 2020; Vulser et al., 2015). MR data quality-control showed that Total Intracranial Volumes (TIVs) change up to 25% in some participants from one evaluation wave to another. This suggests that there might be measurement or segmentation errors that could affect the statistical results.

ADRS depression measurement

The internal consistency of the ADRS scale in our data was good (Cronbach's alpha based on Kuder and Richardson formula for dichotomous items: 0.83). There was substantial variability in the symptom presence (see Table S1) and the aggregate depression severity score (M = 1.29, SD = 2.09).

Table S1

Proportion of ADRS Symptom Presence

ADRS Item	n symptom present	Proportion symptom present
Fati	256	19.44%
CogDys	161	12.22%
Dyst	174	13.21%
Anhend	75	5.69%
Worthless	111	8.43%
SuicIde	90	6.83%
Irrit	174	13.21%
DisCou	205	15.57%
Sleep	311	23.61%
Work	147	11.16%

Note. Fati = Fatigue, CogDys = Cognitive dysfunction, Dyst = Dysthymia, Anhend = Anhedonia, Worthless = Worthlessness, SuicIde = Suicidal ideation, Irrit = Irritation, DisCou = Discouragement, Sleep = Insomnia, Work = Work Disengagement.

Table S2

Edge Weights for Brain-Depression Sum Score Network Model (see Figure 1A)

	Depr	insula	cingulate	mOFC	Fusiform	Hippo
Depr						
insula	0					
cingulate	0	0				
mOFC	0	0.03	0.22			
Fusiform	0	0.21	0.32	0.26		
Hippo	0	0	-0.17	-0.03	-0.02	

Note. Depr = Depression ADRS sum score, Hippo = Hippocampal volume, mOFC = Medial Orbitofrontal Cortex cortical thickness, Fusiform = Fusiform Gyrus cortical thickness, Insula = Insula cortical thickness, Cingulate = Cingulate cortical thickness.

Table S3

Edge Weights for Brain-Symptom Network Model (see Figure 1B)

	Fati	CogDys	Dyst	Anhend	Worthless	Suicide	Irrit	DisCou	Sleep	Work	insula	cingulate	mOFC	Fusiform
Fati														
CogDys	0.8													
Dyst	0.11	0.17												
Anhend	0	0.11	0.32											
Worthless	0	0.5	0.14	0.44										
Suicide	0	0	0.67	0.28	0.44									
Irrit	0.37	0.16	0.38	0.37	0.21	0.26								
DisCou	0.32	0.24	0.76	0.54	0.85	0.95	0.52							
Sleep	0	0.29	0.2	0	0	0.09	0.41	0.23						
Work	0.89	0.19	0.22	0.37	0.19	0.07	0.19	0.25	0.1					
insula	0	-0.15	0	0	0.12	0	0	0	0	0				
cingulate	0	0	0	0	-0.1	0	0	0	0	0	0			
mOFC	0	0	0	-0.05	0	0	0	0	0	0	0	0.21		
Fusiform	0	0	0	0	0	0	0	0	0	0	0.19	0.32	0.26	
Hippo	0	0	0	0	0	0	0	0	0.04	0	0	-0.16	0	0

Note. Fati = Fatigue, CogDys = Cognitive dysfunction, Dyst = Dysthymia, Anhend = Anhedonia, Worthless = Worthlessness, Suicide = Suicidal ideation, Irrit = Irritation, DisCou = Discouragement, Sleep = Insomnia, Work = Work Disengagement, Hippo = Hippocampal volume, mOFC = Medial Orbitofrontal Cortex cortical thickness, Fusiform = Fusiform Gyrus cortical thickness, Insula = Insula cortical thickness, Cingulate = Cingulate cortical thickness

Supplementary Material Section 2: Network analysis and stability

Network estimation.

Before estimating the network, we checked for potential multicollinearity among all included variables. The mean zero-order correlation (among all variables included in the network) was 0.16, and the maximum zero-order correlation was 0.53. This did not indicate concerns regarding multicollinearity in the network estimation.

Cross-validation (with 10 folds) was used to select the tuning parameter parameter lambda in L1 (LASSO) regularization. In every iteration of the 10-fold cross-validation, the dataset is divided into 10 subsets. Nine of these subsets are used to train the model, and the remaining one is used to test the model. This process is repeated 10 times, with each subset serving as the validation set once. Throughout these iterations, the optimal lambda value for regularization is determined by identifying the lambda that minimizes the mean cross-validated error. This process reduces the risk of overfitting and increases the robustness of the estimation. More information on this estimation procedure can be found in the documentation of the mgm package (Haslbeck & Waldorp, 2020).

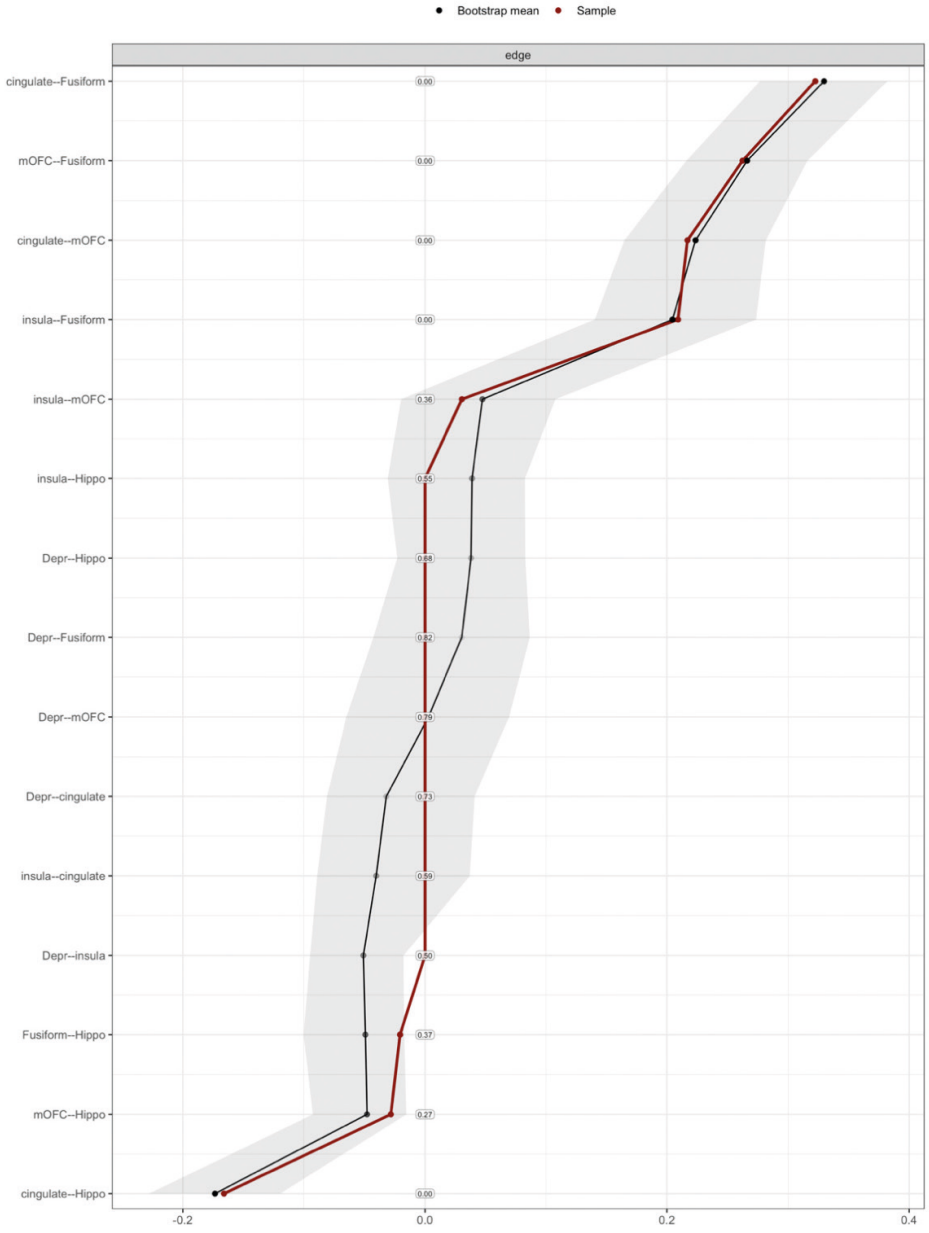
Cross-validation provides sufficient sensitivity and was considered appropriate as we were interested in identifying cross-modal links that may not be detected when using more stringent procedures for penalization, such as the Extended Bayesian Information Criterion (EBIC). We have used the Fruchterman-reingold algorithm (Fruchterman & Reingold, 1991) to determine the layout of the item-level network visualization. To facilitate visual comparison, we have used the same maximum edge strength for scaling the edge weights in both the item- and sum score networks.

Network stability.

To examine the stability of the estimated networks, we have used a non-parametric bootstrapping approach (Epskamp et al., 2018). This re-estimates the model under sampled data (with replacement) 1000 times. A bootstrapped confidence interval around the edge weights (see Figure S1) is calculated and describes the accuracy of the edge weights. See Epskamp et al. (2018) for a tutorial on non-parametric bootstrapping.

Figure S1

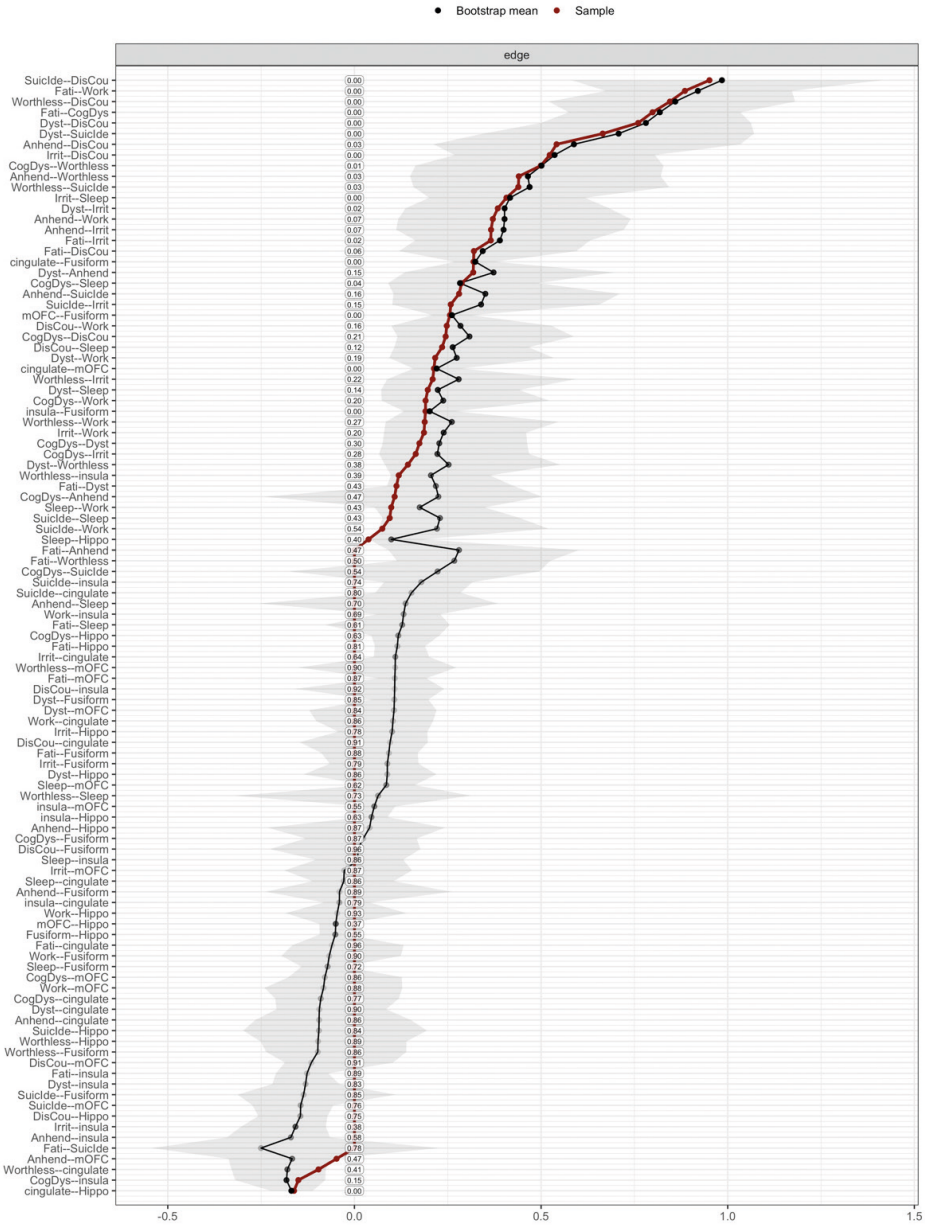
Non-Parametric Bootstrap for Brain-Depression Sum Score Network Model (see Figure 1A)



Note. The grey area describes the 95% bootstrapped confidence interval of the estimated edge weights. The sample values are shown in red. The edges are ordered according to their weight from high (top) to low (bottom). The proportion of estimates being zero is shown in the middle.

Figure S2

Non-parametric Bootstrap for Brain-Depression Symptom Network Model (see Figure 1B)

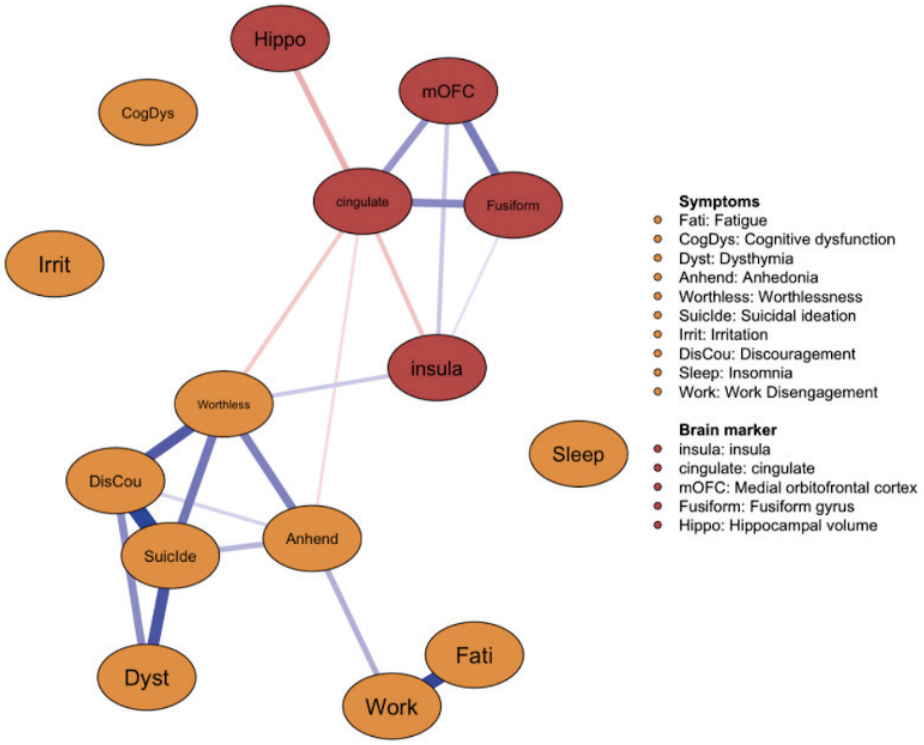


Note. The grey area describes the 95% bootstrapped confidence interval of the estimated edge weights. The sample values are shown in red. The edges are ordered according to their weight from high (top) to low (bottom). The proportion of estimates being zero is shown in the middle.

Supplementary Material Section 3: Subgroup analysis

Figure S3

Depressive Complaints – Brain Marker Network Model In Individuals With (Sub-threshold) Depression



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Supplement to Chapter 9. Cross-Lagged Panel Models for Studying Psychopathology: A Comparative Overview of Structural Equation and Panel Network Approaches

Appendix

We provide all relevant code for the model estimation and simulations on a repository on the Open Science Framework (tinyurl.com/4m7m78sm). The following code examples illustrate key parts of the panel network models:

CLPN. The following R code estimates a CLPN through regularized regressions predicting every variable k at time point 2 through all variables (and itself) at the previous time point. The code was adapted from the example script provided by Rhemtulla et al. (2017) on OSF (https://osf.io/9h5nj/).

```
for (i in 1:k){
 lassoreg <- cv.glmnet(as.matrix(data_t1_t2[,1:k]), data_t1_t2[,(k+i)],
 lambda <- lassoreg$lambda.min
 CLPN_t1_t2[1:k,i] <- coef(lassoreg, s = lambda, exact = FALSE)[2:(k+1)]
}
```

The CLPN_t1_t2 object is the adjacency matrix that contains all beta estimates. This matrix is used for the network visualization.

Panel GVAR model. The panel GVAR model (with default specifications) can be estimated using the following command code:

```
panel_gvar_model <- panelgvar(data, vars = design matrix, within_latent = “ggm”, between_latent = “ggm”, estimator = “FIML”)
```

Pruning procedures, that remove non-significant edges and re-estimate the model can be applied using the following code:

```
panel_gvar_model <- panel_gvar_model %>% runmodel() %>% prune(alpha = 0.01)
```

The model fit can be evaluated:

```
panel_gvar_model %>% fit()
```

And relevant estimates can be extracted:

```
PDC <- getmatrix(panel_gvar_model, “PDC”) # temporal
```

```
PCC <- getmatrix(panel_gvar_model, “omega_zeta_within”) # contemporaneous
PBTW <- getmatrix(panel_gvar_model, “omega_zeta_between”) # between-person
```

Empirical illustration: Results at the subscale level

Table S1. Fit indices for Models Using Subscale Data.

Model	CFI	TLI	RMSEA
CLPM	0.98	0.94	0.05
RI-CLPM	0.99	0.99	0.02
Panel GVAR	0.98	0.97	0.03

Note. RI-CLPM = Random-Intercept Cross-Lagged Panel Model, Panel GVAR = Panel Graphical Vector Autoregression Model, CLPM = Cross-Lagged Panel Model, CLPN = Cross-Lagged Panel Network Analysis.

CLPM. The temporal network is shown in Figure S1A, and it visualizes beta estimates for the change from t_1 to t_2. Social and physiological anxiety symptoms predicted more depression symptoms. Notably, there were no direct temporal associations from impulsivity towards anxiety or depression symptoms.

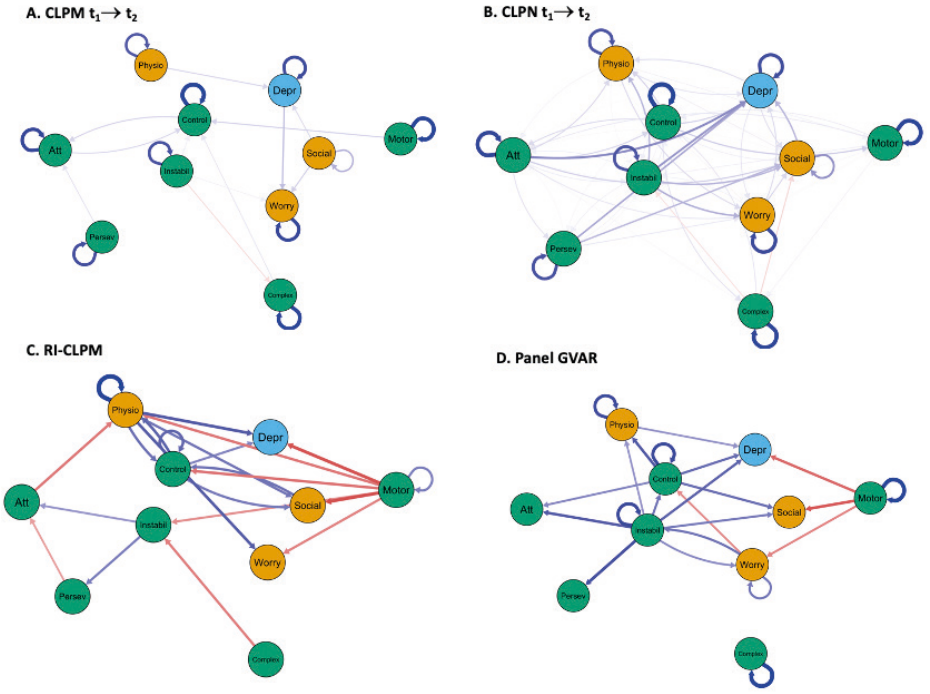
CLPN. The temporal network (Figure S1B) visualizes regularized beta regression coefficients for the first changepoint is shown in Figure 5B). The dense network shows a range of temporal effects outgoing from different facets of impulsivity (e.g., attention, cognitive instability) predicting more depression symptoms at the next wave. Different symptoms of anxiety and depression show bidirectional associations (i.e., predicting each other) over time.

RI-CLPM. The temporal network (Figure S1C) shows partial directed correlations. Several cross-construct associations emerged: motor impulsivity predicted fewer symptoms of social anxiety, worry, and depression over time. Physiological anxiety symptoms predicted more depression, social anxiety, and worry.

Panel GVAR. The temporal network derived from the panel GVAR model depicts partial directed correlations (see Figure S1D). Similar to the RI-CLPM, motor impulsivity predicted less social anxiety and worry. Moreover, lack of self-control predicted more depression, social and physiological anxiety over time -replicating three temporal relations also observed in the RI-CLPM. However, the panel GVAR temporal network is sparser than the RI-CLPM network and does not show any outgoing associations from cognitive complexity.

Figure S1

Temporal Networks Based on Subscale Data.



Note. RI-CLPM = Random-Intercept Cross-Lagged Panel Model, Panel GVAR = Panel Graphical Vector Autoregression Model, CLPM = Cross-Lagged Panel Model, CLPN = Cross-Lagged Panel Network Model. The color of the nodes refers to the domain that the variables belong to (orange = anxiety, blue = depression, green = impulsivity). The thickness and color of the edges describe the strength and direction of associations respectively. Nodes are colored according to the domain that they belong to. The figure visualizes partial directed correlations for the RI-CLPM and panel GVAR models, and beta-estimates for the CLPM and CLPN models. **Physio** = Physiological anxiety, **Worry** = Worry/oversensitivity, **Social** = social concerns/concentration, **Depr** = Depression symptoms, **Att** = attentional impulsivity, **Motor** = motor impulsivity, **Control** = lack of self-control, **Complex** = cognitive complexity (e.g., for problem-solving); **Persev** = perseverance (i.e., persistence in tasks despite difficulties or distractions), **Instabil** = cognitive instability (e.g., racing thoughts).

Supplement to Chapter 10. Preregistration Guidelines for Longitudinal Network Analyses.

Conceptual framework and research questions			
Yes	**No**	**Not applicable**	**I have specified ...**
☐	☐	☐	Research questions (RQs)
☐	☐	☐	Theoretical rationale for variables investigated in the network
☐	☐	☐	Network inferences (and corresponding estimates): - Micro-level: specific edges - Meso-level: clustering - Macro-level: density, sparsity, centrality measures
☐	☐	☐	Network structures that RQs and hypotheses refer to: - temporal - contemporaneous - between-person - group level or individual estimates
☐	☐	☐	Exploratory RQs and analyses

Data and variable selection			
Yes	**No**	**Not applicable**	**I have specified ...**
☐	☐	☐	Study design (recruitment, procedures, eligibility criteria, materials) or name of dataset (when using existing data)
☐	☐	☐	Prior access to data to the data by investigators
☐	☐	☐	Existing publications on relevant variables (explain similarities and differences)
☐	☐	☐	Variables and measurement
☐	☐	☐	Training and/or testing sets

Preprocessing			
Yes	**No**	**Not applicable**	**I have specified ...**
☐	☐	☐	Outlier definition and handling
☐	☐	☐	Standardization
☐	☐	☐	Missing data - complete case analysis - multiple imputation - ML / FIML
☐	☐	☐	Handling time interval/equidistance
☐	☐	☐	Inspection of trends in data (checks for stationarity assumption) and procedures to remove trends

Statistical modeling			
Yes	**No**	**Not applicable**	**I have specified ...**
☐	☐	☐	Specific model, e.g.: - Panel GVAR - CLPN - mlVAR - GIMME
☐	☐	☐	Variables modelled as - Observed variables (e.g., single items, sum-scores) - Latent variables
☐	☐	☐	Software
☐	☐	☐	Criteria for model fit, e.g.: - RMSEA - CFI, TLI - Chi square
☐	☐	☐	Type of group comparison - Visual inspection - Network Comparison Test (NCT) - (Individual) Network Invariance Test (INIT) - Parametric and nonparametric comparison tests - Model (equality) constraints
☐	☐	☐	Model stability inspection procedures/ bootstrapping
☐	☐	☐	Robustness/ sensitivity analyses

Model-specific preregistration guidelines			
Yes	**No**	**Not applicable**	**I have specified ...**
☐	☐	☐	Panel GVAR - Thresholding - Pruning - Model search algorithms - Type of within- and between-person latent model - Confirmatory model structure or group comparison
☐	☐	☐	CLPN - Method for lambda selection - Covariates - Additional pruning steps
☐	☐	☐	mlVAR - Contemporaneous and temporal model estimation (correlated, orthogonal, fixed, or unique)
☐	☐	☐	GIMME - Type of exogenous variables - Cut-offs for group- and subgroup-level paths - Contemporaneous model estimation - Subgroup-specific edge identification - Latent variable modeling

Key Questions for Preregistration

1. Conceptual framework, research questions, and network inference

Name your specific (confirmatory) research questions:
RQ1. ....
RQ2. ....

Name any exploratory research questions:
ERQ1. ....
ERQ2. ....
Which system (e.g., symptom, biological) are you focusing on in your investigation?

Why do you intend to use network analysis as a methodological framework?

What are your specific hypotheses?
H1a. ....
H1b. ....
What level (e.g., micro-, meso-, or macro-level) do your hypotheses refer to?

What estimates (e.g., edge weights, centrality) do your hypotheses refer to?

What network structures (e.g., temporal, contemporaneous) do your hypotheses refer to?

2. Data and variable selection

Planned or ongoing data collection: Describe the study design, including participant recruitment, eligibility criteria, planned sample size, and relevant procedures and materials.

Existing dataset: Provide the name of the study, describe its design, and list relevant publications on similar research questions using this dataset. Describe key differences and similarities to prior work. Clarify if you have previously accessed this data source or examined the study's variables.

Describe all variables of interest and their measurement (e.g., sum scores or collapsing of answer categories).

Do you anticipate multicollinearity and how will you address it? Will you intend to remove variables based on conceptual or theoretical considerations? Do you intend to use any analytical procedures (e.g., unique item analysis)?

Do you intend to use a subset of the data? How will you determine this subset? Do you anticipate any selection biases or Berskon's bias? How do you intend to mitigate these issues? Will you split the data into training and test sets?

3. Data preprocessing

How will you define outliers? Do you intend to standardize the data or apply any other transformations?

Do you intend to inspect the variables for low variance or high correlations? How do you intend to address these issues?

Do you anticipate missing data or attrition? How do you intend to manage this?

Is the data equidistant (i.e., similar time intervals between assessments)? How will you incorporate deviations from equidistance in the model? How will you model overnight missingness?

Do you anticipate (linear or non-linear) trends in the data? How will you identify trends or test for stationarity? Do you intend to remove trends, and if so, how?

4. Statistical modeling

Describe the type of model you intend to use in connection with your research questions and variables.

Describe the specific software (e.g., R packages and version) that will be used to estimate the model.

Clarify how variables will be integrated into the model (e.g., as single observed variables or combined into composite variables).

Specify relevant model fit criteria (e.g., CFI, RMSEA) you intend to use to evaluate the model's adequacy.

If applicable, describe the methodological approach for comparing different networks (e.g., visual inspection, equality constraints, formal comparison tests).

List relevant parameters for network comparison tests, such as the number of iterations/permutations and the type of multiple comparison correction (if any).

How do you intend to evaluate the stability of the model? Describe any bootstrapping methods and metrics (e.g., percentage of edge retention) you will use to evaluate model accuracy and stability.

Will you conduct any robustness or sensitivity analyses?

Do you anticipate potential model estimation issues, such as poor model fit or non-convergence? How will you address these issues?

Have you considered preemptive steps to reduce model complexity and facilitate model identifiability if necessary?

Note: Ensure all model-specific criteria (see Box 1 in main text) are covered in this preregistration.

Non-Scientific Summary

Many mental health conditions, such as anxiety, depression, and substance use disorders, begin in adolescence, a time of major change. During this sensitive period of development, young people face major changes in their physiology, in particular, rapid brain development and hormonal shifts. These changes may also affect adolescents' ability to make decisions or plans, manage emotions, or control impulses. At the same time, adolescents face various psychological and social challenges: school pressure, changing friendships, romantic relationships, questions about identity, independence, and the future.

Executive functions refer to a group of mental skills that allow us to concentrate on tasks, resist distractions, remember certain details, regulate emotions, and shift to other tasks. These skills develop as we grow, in particular during childhood and adolescence. A core that this thesis aims to answer is how changes in executive functions relate to changes in mental health problems. Do difficulties with executive functions precede mental health problems? Or inversely, do mental health problems lead to impairments in executive functions? If that is the case, which specific executive functions are involved, and which types of mental health problems are they linked to?

The first part of this thesis focuses on adolescence. We showed that problems sustaining attention in early adolescence predicted later emotional and behavioral difficulties. Moreover, symptoms of depression at age 11 predicted various other anxiety symptoms at age 13, suggesting that treating depressive symptoms in early adolescence may be relevant for preventing other mental health problems. The second study focused on adolescent alcohol use and showed that teenagers who drink for social reasons or to feel good are more likely to drink heavily and often, which may then lead to alcohol-related problems later on. Personality traits and stressful life events did not directly appear to influence the reasons why young people drink over time. To examine whether impaired executive functions represent causes or consequences of mental health problems, we used a dataset from Brazil that followed children and adolescents that were at high risk for developing mental health problems. We found that problems with inhibition and self-control predicted mental health problems in early adolescence. However, later in adolescence, the relationship became more complex, with mental health and thinking abilities influencing each other over time. In the fourth study, we developed a new method that lets us not only look at how problem develop over time but also how certain factors, such as risk-taking or executive functions change the way these problems influence each other. We termed this approach 'moderated cross-lagged panel network model'. Our analysis showed that working memory and risk-taking not only predict mental health problems, but they may also shape how other mental health problems and substance use influence each other over time.

While the first part of the thesis focused on changes across years and even decades, the second part aimed to examine changes at a much shorter time scale. We explored how young people's ability to concentrate and control their thoughts relates to changes in their mood throughout the day. We found that those individuals with worse attentional control tended to have more unstable moods, and that a brief attention task administered on a smartphone

did not help predict mood during the next days. The thesis also explored whether the way we assess cognitive control may have an impact on the associations we find with mental health problems. We explored evaluating an individual's attention when it is drawn to things associated with rewards or punishments. Individuals were more easily distracted by reward-related items, but not by punishment-related problems. While the experimental task worked well to detect reward effects, these attention patterns did not predict participants' mental health or substance use.

The third part of the thesis addressed more general methodological challenges. We used brain-symptom network models to link specific depression symptoms, such as trouble sleeping, feeling worthless, lack of interest to relevant brain markers. We found that specific symptoms were tied to specific neural markers. These links were not visible when looking at a total depression score in the model. This suggests that zooming in on specific symptoms and cognitive functions may help us better understand the many forms mental health conditions, like depression, can take. Another article in this third part of the thesis compared statistical models that are typically used to understand how mental health symptoms influence each other over time. We showed that some models mix up stable differences between people with actual changes within a person over time, potentially leading to inaccurate conclusions. Lastly, we provide a list with specific guidelines for preregistering longitudinal network analysis. These analyses involve many choices and thus preregistering helps improve transparency and reduce bias.

Nederlandse samenvatting

Veel psychische aandoeningen, zoals angst, depressie en verslavingsproblemen, ontstaan in de adolescentie, een periode van grote veranderingen. Tijdens deze gevoelige ontwikkelingsfase maken jongeren grote veranderingen door op fysiologisch gebied, met name een snelle ontwikkeling van de hersenen en hormonale veranderingen. Deze veranderingen kunnen ook van invloed zijn op het vermogen van adolescenten om beslissingen te nemen of plannen te maken, emoties te beheersen of impulsen te controleren. Tegelijkertijd worden adolescenten geconfronteerd met verschillende psychologische en sociale uitdagingen: druk op school, veranderende vriendschappen, romantische relaties, vragen over identiteit, onafhankelijkheid en de toekomst.

Cognitieve functies verwijzen naar een groep mentale vaardigheden die ons in staat stellen ons te concentreren op taken, afleidingen te weerstaan, bepaalde details te onthouden, emoties te reguleren en over te schakelen naar andere taken. Deze vaardigheden ontwikkelen zich naarmate we ouder worden, met name tijdens de kindertijd en adolescentie. Een kernvraag die deze thesis beoogt te beantwoorden, is hoe veranderingen in cognitieve functies verband houden met veranderingen in psychische gezondheidsproblemen. Gaan moeilijkheden met cognitieve functies vooraf aan psychische gezondheidsproblemen? Of juist omgekeerd: leiden psychische gezondheidsproblemen tot stoornissen in cognitieve functies? Als dat het geval is, welke specifieke cognitieve functies zijn dan betrokken en met welke soorten psychische gezondheidsproblemen houden ze verband?

Het eerste deel van deze thesis richt zich op de adolescentie. We hebben aangetoond dat problemen met het vasthouden van de aandacht in de vroege adolescentie voorspellend waren voor latere emotionele en gedragsproblemen. Bovendien waren symptomen van depressie op 11-jarige leeftijd voorspellend voor verschillende andere angstsymptomen op 13-jarige leeftijd, wat suggereert dat de behandeling van depressieve symptomen in de vroege adolescentie relevant kan zijn voor het voorkomen van andere psychische problemen. De tweede studie richtte zich op alcoholgebruik door adolescenten en toonde aan dat tieners die drinken om sociale redenen of om zich goed te voelen, vaker en meer drinken, wat later kan leiden tot alcoholgerelateerde problemen. Persoonlijkheidskenmerken en stressvolle levensgebeurtenissen leken geen directe invloed te hebben op de redenen waarom jongeren in de loop van de tijd drinken. Om te onderzoeken of verminderde cognitieve functies oorzaken of gevolgen zijn van psychische problemen, hebben we een dataset uit Brazilië gebruikt waarin kinderen en adolescenten werden gevolgd die een hoog risico liepen op het ontwikkelen van psychische problemen. We ontdekten dat problemen met remming en zelfbeheersing voorspellend waren voor psychische problemen in de vroege adolescentie. Later in de adolescentie werd de relatie echter complexer, waarbij psychische gezondheid en denkvermogen elkaar in de loop van de tijd beïnvloedden. In het vierde onderzoek hebben we een nieuwe methode ontwikkeld waarmee we niet alleen kunnen kijken naar hoe problemen zich in de loop van de tijd ontwikkelen, maar ook hoe bepaalde factoren, zoals het nemen van risico's of cognitieve functies, de manier waarop deze problemen elkaar beïnvloeden mogelijk veranderen. We noemden deze benadering 'moderated cross-lagged panel network

model'. Onze analyse toonde aan dat het werkgeheugen en het nemen van risico's niet alleen voorspellend zijn voor psychische problemen, maar ook kunnen bepalen hoe andere psychische problemen en middelengebruik elkaar in de loop van de tijd beïnvloeden.

Terwijl het eerste deel van het proefschrift zich richtte op veranderingen over een periode van jaren en zelfs decennia, was het tweede deel bedoeld om veranderingen op een veel kortere tijdschaal te onderzoeken. We onderzochten hoe het vermogen van jongeren om zich te concentreren en hun gedachten te beheersen verband houdt met veranderingen in hun stemming gedurende de dag. We ontdekten dat personen met een slechtere aandachtscontrole doorgaans een meer onstabiele stemming hadden, en dat een korte aandachtsopdracht op een smartphone niet hielp om de stemming tijdens de volgende dagen te voorspellen. In het proefschrift werd ook onderzocht of de manier waarop we cognitieve controle beoordelen van invloed kan zijn op de verbanden die we vinden met psychische gezondheidsproblemen. We onderzochten de aandacht van een persoon wanneer deze wordt getrokken naar zaken die verband houden met beloningen of straffen. Personen werden gemakkelijker afgeleid door beloningsgerelateerde zaken, maar niet door strafgerelateerde problemen. Hoewel de experimentele taak goed werkte om beloningseffecten te detecteren, waren deze aandachtspatronen geen voorspeller voor de geestelijke gezondheid of het middelengebruik van de deelnemers.

Het derde deel van het proefschrift ging over meer algemene methodologische uitdagingen. We gebruikten hersen-symptoomnetwerkmodellen om specifieke depressiesymptomen, zoals slaapstoornissen, gevoelens van waardeloosheid en gebrek aan interesse, te koppelen aan relevante hersenmarkers. We ontdekten dat specifieke symptomen gekoppeld waren aan specifieke neurale markers. Deze verbanden waren niet zichtbaar wanneer we keken naar de totale depressiescore in het model. Dit suggereert dat het inzoomen op specifieke symptomen en cognitieve functies ons kan helpen om de vele vormen die psychische aandoeningen, zoals depressie, kunnen aannemen, beter te begrijpen. Een ander artikel in dit derde deel van het proefschrift vergeleek statistische modellen die doorgaans worden gebruikt om te begrijpen hoe psychische symptomen elkaar in de loop van de tijd beïnvloeden. We toonden aan dat sommige modellen stabiele verschillen tussen mensen verwarren met daadwerkelijke veranderingen binnen een persoon in de loop van de tijd, wat mogelijk tot onjuiste conclusies leidt. Ten slotte geven we een lijst met specifieke richtlijnen voor het vooraf registreren van longitudinale netwerkanalyses. Deze analyses omvatten veel keuzes en daarom helpt vooraf registreren om de transparantie te verbeteren en vooringenomenheid te verminderen.

Authorship Contributions

Chapter 2 - Freichel, R., Pfirrmann, J., de Jong, P. J., Cousijn, J., Franken, I. H. A., Oldehinkel, A. J., Veer, I. M., & Wiers, R. W. (2023). Executive Functioning, Internalizing and Externalizing Symptoms: Understanding Developmental Dynamics Through Panel Network Approaches. JAACAP Open, 2(1), 66–77.

R.F.: Writing – original draft; Writing – review & editing; Visualization; Methodology; Formal analysis; Data curation; Conceptualization; Investigation. **J.P.:** Writing – original draft; Formal analysis; Data curation; Conceptualization; Investigation; Methodology. **P.d.J.**: Writing – review & editing; Conceptualization; Investigation; Methodology. **J.C.:** Writing – review & editing; Conceptualization; Investigation; Methodology. **I.F.:** Writing – review & editing; Conceptualization; Investigation. **A.J.O.:** Writing – review & editing; Conceptualization; Investigation; Data curation; Funding acquisition; Resources. **I.M.V.:** Writing – review & editing; Visualization; Conceptualization; Investigation; Methodology; Supervision. **R.W.W.:** Writing – review & editing; Visualization; Conceptualization; Investigation; Methodology; Funding acquisition.

Chapter 3 - Freichel, R., Pfirrmann, J., Cousijn, J., de Jong, P., Franken, I., Banaschewski, T., Bokde, A. L. W., Desrivières, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Martinot, J. L., Martinot, M. P., Artiges, E., Nees, F., Orfanos, D. P., Poustka, L., Hohmann, S., Fröhner, J. H., … IMAGEN Consortium (2023). Drinking motives, personality traits and life stressors-identifying pathways to harmful alcohol use in adolescence using a panel network approach. Addiction (Abingdon, England), 118(10), 1908–1919.

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Chapter 5 - Freichel, R., Veer, I., de Jong, P., Cousijn, J., Franken, I., Banaschewski, T., Bokde, A. L. W., Desrivieres, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Martinot, J.-L., Paillère Martinot, M.-L., Artiges, E., Nees, F., Papadopoulos Orfanos, D., Paus, T., Poustka, L., Hohmann, S., Fröhner, J. H., Smolka, M. N., Vaidya, N., Whelan, R., Schumann, G., IMAGEN Consortium, Walter, H., Blanken, T., & Wiers, R. Impaired working memory and risk-taking predict detrimental symptom dynamics in adolescence: A moderated cross-lagged panel network approach. Manuscript in revision.

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Chapter 6 - Freichel, R., Karami Motaghi, A., Ruppin, S., Hamlett, G. E., de Jong, P., Cousijn, J., Wiers, R. W., & Veer, I. M. Executive functioning and daily mood dynamics: A multi-method EMA study. Manuscript in preparation.

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Chapter 7 - Freichel R, Mrkonja L, de Jong PJ, et al. Value-modulated attentional capture in reward and punishment contexts, attentional control, and their relationship with psychopathology. Journal of Experimental Psychopathology. 2023;14(4).

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Chapter 8 - Freichel, R., Lenartowicz, A., Douw, L., Kruschwitz, J. D., Banaschewski, T., Barker, G. J., Bokde, A. L. W., Desrivières, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Brühl, R., Martinot, J. L., Martinot, M. P., Artiges, E., Nees, F., Orfanos, D. P., Paus, T., Poustka, L., ... Blanken, T. F. (2024). Unraveling robust brain-behavior links of depressive complaints through granular network models for understanding heterogeneity. Journal of Affective Disorders, 359, 140–144.
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R.F.: Conceptualization; Formal analysis; Methodology; Visualization, Writing - original draft; Writing - review & editing. **I.M.V.:** Conceptualization; Methodology; Writing - review & editing. **R.W.W.:** Conceptualization; Methodology; Writing - review & editing. **R.J.M.:** Conceptualization; Methodology; Writing - review & editing. **S.E.:** Conceptualization; Formal analysis; Methodology; Visualization; Software; Supervision; Validation; Writing - review & editing

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2026

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1. Ruggeri, K., Panin, A., …, **Freichel, R.,** …, Toscano, F. (2022). The globalizability of temporal discounting. *Nature Human Behaviour, 6*(10), 1386-1397. https://doi.org/10.1038/s41562-022-01392-w

Under-Review Articles List

8. Menendez, M.S., Stehouwer, K., Abikenari, M., Merkx, M., & **Freichel, R.** (Under Review). Waiting time from referral to intake and its clinical implications: Routine outcome monitoring data from a large Dutch outpatient clinic. Preprint available at: https://osf.io/preprints/psyarxiv/zegjt_v1
7. **Freichel, R.,** Veer, I., de Jong, P., Cousijn, J., Franken, I., Banaschewski, T., Bokde, A. L. W., Desrivieres, S., Flor, H., Grigis, A., Garavan, H., Heinz, A., Martinot, J.-L., Paillère Martinot, M.-L., Artiges, E., Nees, F., Papadopoulos Orfanos, D., Paus, T., Poustka, L., Hohmann, S., Fröhner, J. H., Smolka, M. N., Vaidya, N., Whelan, R., Schumann, G., IMAGEN Consortium, Walter, H., Blanken, T., & Wiers, R. (Under Review). Impaired working memory and risk-taking predict detrimental symptom dynamics in adolescence: A moderated cross-lagged panel network approach. Preprint available at: https://osf.io/gnakf_v1/
6. Ruppin, S., **Freichel, R.,** Wang, Q., Hamlett, G. E., Kuckertz, J. M., Falkenstein, M. J., & McNally, R. J. (Under Review). Obsessive beliefs are distinctly related to specific OCD symptoms in OCD patients: A network approach.
5. Kleijweg, J., **Freichel, R.,** Merkx, M., Wiers, R. (Under Review). A Network Approach to Somatic Symptom Disorder (Fatigue) and Depression: Mapping Symptom Interactions in a Large Clinical Sample
4. **Freichel, R.,** Isvoranu, A., Gates, K., Ebrahimi, O. V., Ruppin, S., Abikenari, M., Blanken, T., Veer, I.M., McNally, R.J., Wiers, R.W., Epskamp, S. (Under Review). Preregistration Guidelines for Longitudinal Network Analyses. Preprint available at: https://doi.org/10.31234/osf.io/zbjd9
3. **Freichel, R.,** Epskamp, S. (Under Review). Handling Problematic Between-person Estimates in Panel Network Models: A Comparative Simulation Study. Preprint available at: https://osf.io/preprints/psyarxiv/eqaux
2. **Freichel, R.,** Veer, I., Wiers, R., McNally, R. J., Epskamp, S. (Under Review). Cross-Lagged Panel Models for Studying Psychopathology: A Comparative Overview of Structural Equation and Panel Network Approaches. Preprint available at: https://osf.io/preprints/psyarxiv/b94qt
1. Kahveci, S., Reichenberger, J., Arend, A., Mansueto, A., **Freichel, R.,** Voderholzer, U., & Blechert, J. (Under Review). Bidirectional Temporal Relationships between Emotional State and Eating across Eating Disorders: A Network Approach. Preprint available at: https://doi.org/10.31234/osf.io/f7svd_v2

Short CV

René Freichel was born in Saarbrücken, Germany on April 22, 1997. After graduating from Humboldt University of Berlin with a Bachelor's degree in Psychology, he obtained two Master's degrees in Psychology (major: clinical) and Behavioral Data Science at the University of Amsterdam in the Netherlands. He started his PhD in October 2021 at the University of Amsterdam under the supervision of Prof. Reinout Wiers and Dr. Ilya Veer. Between September and November 2023, René visited Harvard University, and between January and February 2024, he visited the National University of Singapore. In October 2024, he began his postgraduate psychotherapy training in cognitive-behavioral therapy and his clinical internship at a large psychiatric inpatient and outpatient hospital.

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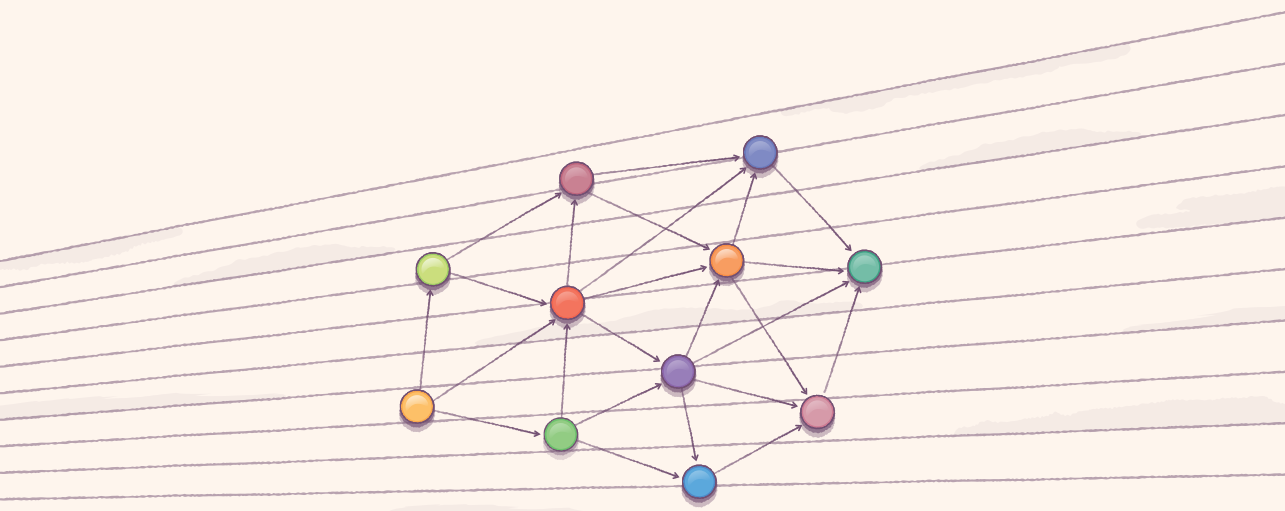

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