

**Exploring the Relationship between Cognitive Functioning and Psychopathology**

**Symptoms: A Transdiagnostic Network Approach**

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Master Thesis Clinical Psychology

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July 2023

### Abstract

**Introduction:** Impairments in cognitive functioning are common in psychopathology. Even though extensive research has been done, the exact relationship and mechanism remains unclear. This study aims to provide a more nuanced understanding of this complex relationship in a young high-risk sample, by using a network approach to link cognitive domains with individual psychopathology symptoms.

**Method:** Cross-sectional data were collected from 490 offspring (13 to 25 years) of parents treated for depression/anxiety. 8 Cognitive domains were assessed with the *Amsterdamse Neuropsychologische Taken*, and 55 depression/anxiety symptoms were assessed with the DSM-IV questionnaire. First, a Spearman correlation was used to examine the relationship between the cognitive domains and psychopathology severity. Then the network was estimated with all 8 cognitive domains and 55 psychopathology symptoms, providing information about the direct and indirect links.

**Results:** No-to-weak correlations were found between the cognitive domains and psychopathology severity scores, with significant correlations ranging from .09 to .11. Moreover, the network analysis detected two clusters representing cognitive domains and psychopathology symptoms, indicating that both are separate entities. The correlations found between cognitive domains and psychopathology severity were found to be driven/maintained by only one symptom.

**Discussion:** Even though, NCF did not seem to play a prominent role in this young high-risk population, the importance of the network and symptom-specific approach has been emphasized. This current approach has provided a better nuance compared to traditional analysis, by highlighting relationships between variables that may not be found with traditional approaches.

*Keywords:* Neurocognitive functioning, psychopathology severity, network analysis

## **Exploring the Relationship between Cognitive Functioning and Psychopathology Symptoms: A Transdiagnostic Network Approach**

Neurocognitive functioning (NCF) plays a crucial role in various aspects of daily life, including behavioral functioning (Batty et al., 2008), academic achievement (Duckworth et al., 2019), and psychopathologies such as depression and anxiety (Gale et al., 2008). NCF includes various cognitive processes, Table 1 provides an overview of the main NCF domains. It has been well-established that individuals with major depressive disorder display impairments in working memory (Chai et al., 2018), attentional flexibility (Snyder et al., 2013), response inhibition (Stefanopoulou et al., 2009), psychomotor speed (Zakzanis et al., 1998), sustained attention (Piani et al., 2022), and facial emotion recognition (Krause et al., 2021). However, knowledge regarding the relationship between NCF and anxiety disorders is limited. Studies observed deficits in attentional flexibility (Cohen et al., 1996) and emotion recognition (Demenescu et al., 2010) in generalized anxiety disorder, and impairments in attention shifting (Airaksinen et al., 2005), cognitive flexibility (Zhou and Ni, 2017; Castillo et al., 2010), and working memory (Micco et al., 2009) in panic disorder. While others find no such impairments (Purcell et al., 2013; Airaksinen et al., 2005). On the other hand, obsessive-compulsive disorder consistently shows impairments in inhibition (Chamberlain et al., 2007; Menzies et al., 2007), working memory, verbal fluency, and motor speed (Ozcan et al., 2016). Although research on this topic is abundant, there is consistent evidence that individuals with depression and obsessive-compulsive disorder exhibit impairments in various NCFs, while the relationship between other anxiety disorders and NCFs remains unclear.

**Table 1**

*Neuropsychological domains*

<b>Cognitive domain</b>	<b>Explanation</b>
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Working memory	A system for actively maintaining, storing, and manipulating information in the short term. It is important in the control of attention. Working memory can be divided into a visuospatial (e.g., patterns and shapes) and a verbal (e.g., words and letters) component.
Response inhibition	The ability to suppress or avoid a prepotent response to make a less automatic but task-relevant response.
Sustained attention	The ability to maintain concentrated attention over prolonged periods of time.
Attentional flexibility	The ability to mentally switch between two response sets or the readiness for a person to change their mindset in response to an external stimulus.
Psychomotor speed	The time it takes to process incoming information and respond accordingly to a motor task. Reaction time tasks are often used to determine processing and response speed.
Fine motor control	The coordination of muscles, bones, and nerves to produce small, precise movements.
Facial emotion recognition	Recognition of emotions as expressed by the face. An important nonverbal cue in daily life, as it is needed to properly function in social contexts.

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### **Bidirectional Relationship of Neurocognitive Impairments with Depression and Anxiety**

The relationship between NCF and psychopathology is complex and bidirectional (Schweizer and Hankin, 2018). *Vulnerability theories* propose that impaired NCF plays a role in the emergence of psychiatric symptoms and disorders (Bessette et al., 2020). For example, poor executive functioning was found to be a precursor of later heightened depression and anxiety (Zainal & Newman, 2018). Additionally, individuals who are already at risk for developing depression/anxiety often exhibit early neurocognitive deficits; for instance, offspring of mothers with major depressive disorder already present attentional and motor function problems at a young age (Klimes-Dougan, 2006). In contrast, according to *scar theories*, a rise in psychiatric symptoms may precede and predict impairments in NCF (Zainal

and Newman, 2022). For instance, chronic depression and anxiety have been suggested to cause a deterioration in executive functioning (Galecki et al., 2015). Additionally, psychiatric disorders can create long-term impairments even after remission (Semkovska et al., 2019) Clayton et al., 2021; Zainal & Newman, 2022). Thus, the relationship between NCF and psychopathology is complex and multifaceted.

### **Symptom Heterogeneity**

Recent studies convincingly demonstrated that depression and anxiety exhibit a high degree of heterogeneity, encompassing a broad array of subtypes, each exhibiting unique features regarding symptomatology, neurobiology, and physiological as well as endocrine functioning (Rush et al., 2007). Regarding symptom heterogeneity within diagnoses, a study by Fried et al. (2015) in 3,703 patients with major depressive disorder revealed 1,030 symptom profiles based on unique combinations of symptoms. Almost half of the participants displayed unique symptom profiles not shared with any other individual in the study. Regarding anxiety disorders, an individual must experience 3 out of 6 symptoms as described in the DSM-4. This indicates that there are also numerous symptom combinations possible. This highlights the heterogeneity of symptoms within different diagnoses. Additionally, boundaries between diagnoses are unclear, as depression and anxiety rarely exist in isolation (Kaufman & Charney) and high rates of comorbidity are observed (Zbozink et al., 2012; Lamers et al., 2011). For instance, over half of the patients with generalized anxiety disorder meet the criteria for major depressive disorder or exhibit (sub)clinical depressive symptoms, as reported by Zhou et al. (2017). These findings underline the importance of a focus on individual symptoms, both within and across diagnoses, to capture the complexity of depression and anxiety.

### **The Network Approach**

The network approach to psychopathology is a framework that emphasizes the

importance of individual symptoms and their connections with one another, providing information on the complex relations between symptoms (Borsboom., 2017). Numerous studies investigated the network structure between depressive and anxiety disorders revealing that not all symptoms of depression and anxiety were found to be interconnected. Each symptom was associated with a unique set of other symptoms, between and within its own diagnosis. Certain symptoms showed stronger connections with each other than with other symptoms (Bekhuis et al., 2016). This further reinforces the concept of the heterogenous nature within the diagnosis. Additionally, symptoms within the same diagnosis generally exhibit stronger connections than between diagnoses (Beard et al. 2016). However, also many overlapping symptoms and cross-connections were observed (Boschloo et al., 2016; Curtis et al., 2016). Suggesting that both are not distinct disorders but may underlie the same mechanism. Taken together, these findings imply that the network approach can do justice to the complexity of depression and anxiety.

### **Network Approach in Cognitive Neuroscience**

This network approach is also a growing concept in cognitive science and provides a quantitative approach to representing cognitive systems. A recent review reported that networks in neuroscience provide two important assumptions about cognition. First, cognitive functions are interrelated. Königs et al (2021) used a network approach in a healthy sample to investigate neurocognitive network organization and reported higher connectivity within cognitive functions that were more closely related to each other. Additionally, verbal memory was found to be the most influential function in the healthy population. Second, cognitive rearrangement can occur in neurological disorders. For example, it has been found that processing speed and fluency are the most influential variable in Alzheimer's disease while in mild cognitive impairment, episodic memory variables were most central (Ferguson, 2021).

Moreover, this highlights the complexity of cognitive functions and their ability to change in specific disorders.

### **The potential of the Network Approach in Linking Neurocognitive Impairments to Symptoms of Depression and Anxiety**

Given that impaired NCF is recognized as a transdiagnostic factor that spans different disorders (Chavez-Baldini et al., 2023), it would be pertinent to investigate the association of NCF with individual symptoms of various diagnoses. The study by Chavez-Baldini et al. (2023) was the only one performing network analyses on cognitive domains and psychopathology diagnosis. The analysis detected three clusters: cognition, general psychopathology, and substance use. Multiple (weak) links were found between cognition and general psychopathology. Interesting patterns were observed. Psychopathology nodes were consistently related to cognitive domains. Specifically, depression was consistently correlated with poor NCF across domains. However, cognitive domain nodes showed mixed relationships with psychopathology nodes. For example, subclinical psychotic experiences were linked to impaired verbal memory, while obsessive-compulsive disorder was associated with better verbal memory. This emphasizes the complexity of the relationship between NCF and psychopathology. Therefore, it's important to delve into these relationships and underlying mechanisms. Despite the potential for cognitive domains to have distinct connections with individual symptoms, the study did not account for individual symptom networks, which motivated me to delve into this area of research. Filling this gap and shifting the focus from diagnostic categories to individual symptoms can provide a more nuanced understanding of the complex relations between NCF domains and individual psychiatric symptoms.

### **The Current Study**

This study represents the first study using network analyses to link a wide range of

cognitive domains with individual symptoms of depression and anxiety. Data will be obtained from the large-scale study, Adolescents at Risk of Depression and Anxiety: A Neurobiological and Epidemiological Approach (ARIADNE). The sample includes 523 offspring of patients who received treatment for depressive and/or anxiety disorders. Therefore, this sample can be regarded as a high-risk group for the development of psychopathology. It is important to research this population since they are at higher risk of experiencing depressive and anxiety symptoms, and they often exhibit early impairments in NCF. Investigating specific symptom patterns in this population may aid in developing personalized interventions and treatments. Firstly, the relationship between 8 cognitive domains and 8 psychopathology severity scores will be investigated. In line with other research, it is hypothesized that all cognitive domains are negatively related to different types of psychopathologies. To unravel the complex relationship between NCF and depression and anxiety severity, a network is estimated with all 8 cognitive domains and 55 depression/anxiety symptoms. The general structure of the network and the specific connections between cognitive domains and individual symptoms will be explored.

## **Methods**

### **Study Design and Participants**

Data were derived from ARIADNE, a prospective cohort study initiated in 2000. The objective of this study was to enhance understanding of etiological mechanisms involved in the incidence and course of depressive and anxiety disorders. The study sample consisted of 523 individuals with an age range of 13-25 years, who are offspring of 366 patients that received treatment for depressive disorders such as MDD and dysthymia, as well as anxiety disorders such as panic disorder, and obsessive-compulsive disorder. Patients (index-parents) were recruited through 16 psychiatric facilities located in the northern provinces of the Netherlands. Those who had a history of schizophrenia spectrum diagnosis, post-traumatic



stress disorder, or inadequate proficiency in the Dutch language were excluded from participating in the study. Both the index-parents and offspring underwent a psychiatric diagnostic interview, the Composite International Diagnostic Interview (CIDI), at baseline. Of the index-parents, 320 had a depressive disorder (87.4%; of which 43.1% had a pure depressive disorder and 56.9% had a comorbid anxiety disorder) and 207 had an anxiety disorder (56.6%; of which 12.1% had a pure anxiety disorder and 87.9% had a comorbid depressive disorder). No formal CIDI diagnosis was present in 5.5% of the index parents. Except for one index-parent, all of them passed the CIDI screener, indicating the presence of subclinical depressive and/or anxiety symptoms. There was no CIDI information available for one index-parent. The offspring also underwent a psychiatric diagnostic interview, assessing temperament, social support, coping, family functioning, parent-adolescent communication, and DSM-IV symptoms. Additionally, cognitive functioning was measured using the Amsterdamse Neuropsychologische Taken (ANT).

Baseline data were used in this current study and therefore consisted of a cross-sectional design. The ARIADNE study protocols were approved by the Medical Ethics Committee of the University Medical Center Groningen, Groningen, The Netherlands. In addition, informed consent was obtained.

## **Procedure**

Index parents were contacted by a local psychologist at the mental health services. The study aims and measurement procedures were presented by information letters. The research team contacted the individuals that agreed to participate in the study. Both the index parent and offspring had to sign an informed consent to participate. Withdrawing from participating was possible at any time, without any implications for their regular mental health care.

## **Instruments**

### ***Depression and anxiety***

DSM-IV Questionnaire (Hartman, 2002; Hartman et al., 2001) was used to assess depression and anxiety symptoms, which includes items that correspond to the symptoms as defined in the Diagnostic and Statistical Manual of mental disorders (DSM-IV) classification system. Questions were answered on a 4-point Likert scale ranging from 0 to 3, with higher scores reflecting a greater degree of symptom severity. Participants were asked to report to what extent descriptions of symptomatic behavior accurately described their behavior at the time of measurement. The questionnaire includes items referring to both depression and a broad range of anxiety disorders. The depression scale includes items, such as: 'I am often unhappy' and 'I am low in energy or feel tired for no reason'. On the other hand, the anxiety scale includes items such as: 'I suddenly become very anxious or panicky for no reason' and 'I often feel sick to my stomach'. The items are divided into 8 subscales: Major depressive disorder consists of 12 items ( $\alpha = .913$ ), obsessive-compulsive disorder 10 items ( $\alpha = .693$ ), panic disorder 14 items ( $\alpha = .859$ ), separation anxiety 3 items ( $\alpha = .527$ ), generalized anxiety disorder 5 items ( $\alpha = .7490$ ), social anxiety 4 items ( $\alpha = .787$ ), somatization 4 items ( $\alpha = .669$ ) and sleep problems 3 items ( $\alpha = .773$ ).

### ***Neuropsychological Functioning***

At baseline, seven cognitive domains were assessed by using tasks from the Amsterdam Neuropsychological Tasks program (ANT; Wittchen, 1994). The tasks measured reaction times, percentage of errors, and deviation for the motor control task (described below); these provided an indication of information processing capabilities. Reaction times were used to assess information processing speed, while percentages of error were used to assess accuracy. In a quiet room, participants were tested at their homes under similar conditions, behind a computer screen. For an overview of the tests used, see appendix A.

### **Data Analyses**

The data were analyzed using Statistical Package for the Social Sciences (version 28)

(SPSS; IBM Corp., 2018) and R version 3.0.2 (R core team, 2022). Cognitive performance was measured using Z-scores. Scores lower than -4 and greater than or equal to 4 were considered outliers and therefore removed. Descriptive statistics were used to summarize the baseline characteristics.

### **Main Analysis**

First, a correlation analysis was used to examine the hypothesis that all cognitive domains (independent variables) are negatively related to different types of psychopathology severity (dependent variables). Cognitive functioning is divided into 8 domains: sustained attention, visuomotor speed, working memory, emotion recognition, response inhibition, attentional flexibility, controlled visuospatial working memory (WM), and automatic visuospatial WM. The psychopathology severity scores consist of major depressive disorder (MDD), generalized anxiety disorder (GAD), separation anxiety (SEP), sleeping problems (SLE), obsessive-compulsive disorder (OCD), panic disorder (PAN), social anxiety disorder (SOC) and somatization (SOM). Before performing the correlation analysis, the following assumptions were tested: linearity, normality, and outliers.

R was used to assess the network structure and visualization, cluster detection, and centrality of the symptom severity scores and the 8 cognitive domains. The following R packages were installed: *qgraph*, *matrix*, *igraph*, *devtools*, *NetworkComparisonTest*, *ppcor*, and *mgm*. The network was estimated via Gaussian Graphical Model (GGM) (Lauritzen, 1996). Data was converted into a matrix format and the Extended Bayesian Information Criterion (EBICglasso) was used to estimate the network structure. The model was visualized with the *qgraph* package (Eskamp et al., 2012), size, color and labels were specified. The regularization technique called "glasso" (graphical lasso) was utilized to obtain an optimal sparse estimation of the network structure to control for false positive associations. The estimation process also considered the Extended Bayesian Information Criterion (EBIC) for

model selection to strike a balance between sparsity and goodness of fit. Positive associations are depicted in green and negative associations in red. This results in a set of partial correlations (edges) between the variables (nodes). Nodes more connected are placed closer together and nodes with a higher centrality are closer to the center of the graph. The edges can be negative (red) or positive (green). Thicker edges represent a higher correlation. Layout was applied displaying cognitive domains on the right legend and psychopathology symptoms on the left legend.

## Results

### Sample Characteristics

In total 528 participants participated in this study. However, 38 patients were excluded due to missing data on key variables (10= with missing data on cognitive domains; 28= with missing data on symptomatology), resulting in a total sample of 490 participants (57.1% female, mean age=18.1,  $SD=3.21$  years). Table 2 provides an overview of the mean and standard deviations of psychopathology severity scores and cognitive domain scores.

**Table 2**

*Means, standard deviations, and range for psychopathology severity scores and cognitive domains (N = 490)*

Variabele	<i>M</i>	<i>SD</i>	Range
Psychopathology severity scores			
MDD	18.00	6.39	12.00 – 45.00
OCD	13.69	3.12	10.00 - 29.00
PAN	18.13	4.91	14.00 - 45.00
SEP	4.21	1.38	3.00 - 9.00
GAD	8.53	2.85	5.00 - 20.00
SOC	7.53	2.57	4.00 - 16.00
SOM	5.89	2.06	4.00 - 14.00
SLE	4.35	1.89	3.00 - 12.00
Cognitive domains			

Sustained attention	-.013	.97	-1.29 - 5.96
Visuo-motor speed	.009	1.00	-1.53 - 4.49
Working memory	.001	1.00	-3.36 - 4.88
Emotion recognition	.017	1.00	-2.35 - 4.89
Inhibition	-.006	-.269	-1.69 - 4.60
Attentional flexibility	.007	-.171	-2.08 - 6.27
Controlled visuospatial WM	.013	-.141	-2.28 - 5.86
Automatic visuospatial WM	.006	-.170	-6.46 - 5.14

### Correlation of cognitive domains and psychopathology severity

The Shapiro-Wilk test for normality was violated, therefore Spearman correlations were computed to assess the relationship between all cognitive domains and psychopathology severity scores. Table 3 in Appendix B provides an overview of the correlations. Weak to high correlations were found within the cognitive domains (range: -0.11 to 0.53) and within psychopathology severity scores (range: 0.17 to 0.77). In contrast, the correlations between the cognitive domains and the psychopathology severity scores were all (very) low.

In contrast to the hypothesis, no correlation was found between sustained attention, inhibition, and attentional flexibility and the psychopathology severity scores. However, in line with our hypothesis, emotional recognition was significantly and negatively related to 2 severity scores (PAN  $r = -.11$ ,  $p < .005$ ; SOM  $r = -.12$ ,  $p < .005$  respectively). This indicates that faster emotion recognition is related to more severe symptoms. In contrast to our hypothesis, working memory was positively related to the SOC severity score (SOC:  $r = .09$ ,  $p < .005$ ). Visuomotor speed and controlled visuospatial WM were both significantly and positively related to SEP severity ( $r = .09$ ,  $p < .005$ ;  $r = .09$ ,  $p < .005$ ). Lastly, automatic visuospatial WM was significantly and positively correlated with SLE severity ( $r = .11$ ,  $p < .005$ ). This indicates that a higher score on working memory, visuomotor speed, and visuospatial working memory, both controlled and automatic, is associated with higher symptoms severity.

## Network Structure

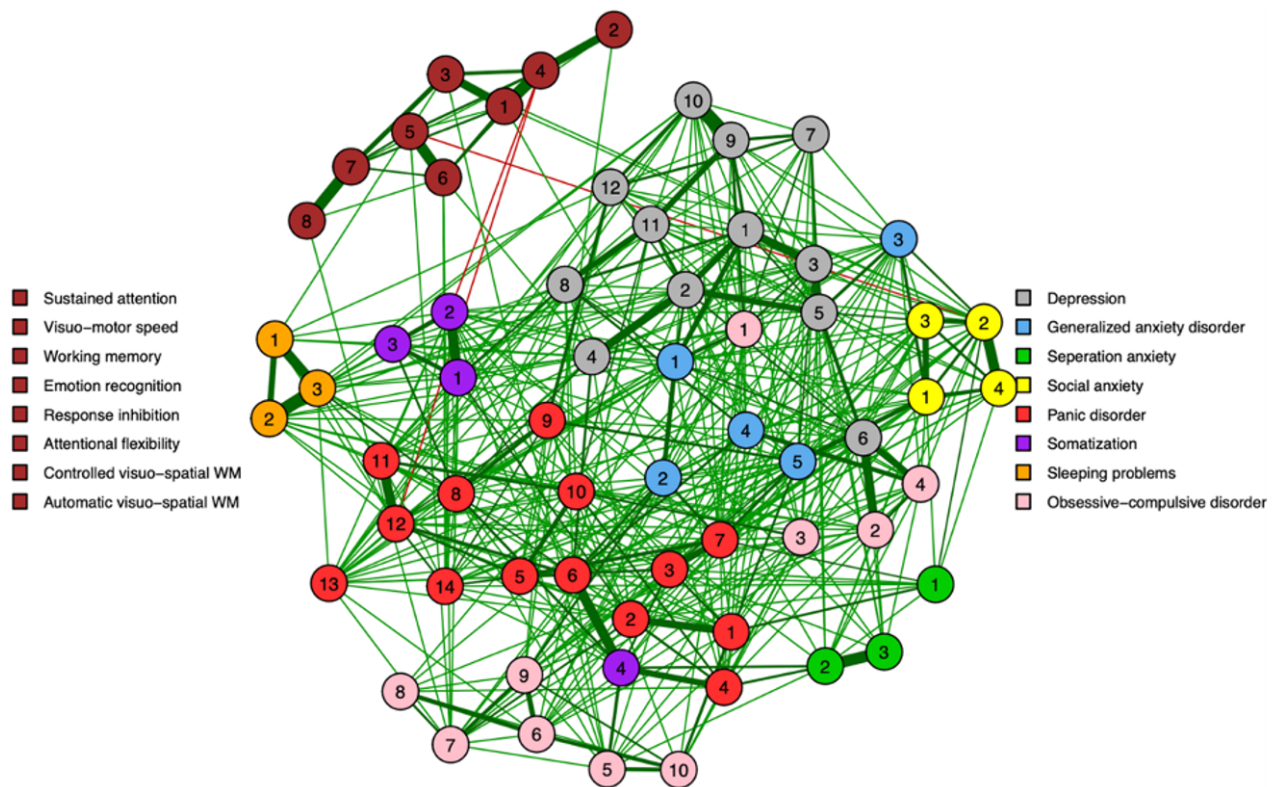
The network structure is presented in Figure 1, which is based on the estimated connections between all cognitive domains and psychopathology severity scores. Table 4 in Appendix C and Table 5 in Appendix D provide an overview of nodes with their labels, means, and standard deviations. The network comprises 1505 of  $(63 * (63 - 1)) / 2 = 1953$  potential connections, resulting in a network density of 77.06 %. All symptoms were directly or indirectly connected, and connections ranged from weak to moderate. Two clusters were detected: one cluster including all cognitive domains, and another cluster including all psychopathology severity scores. The total strength of edges within the clusters (cognitive domain: 4.92; psychopathology severity score: 47.73) was higher than between the clusters (0.08). Within the psychopathology cluster, some symptoms formed their own cluster representing psychopathology types. Intriguingly, some symptoms showed more connections with symptoms outside their psychopathology. For example, SOM4 was more connected to symptoms of PAN and GAD symptoms and GAD3 showed more connections with SOC and DEP symptoms.

However, the connections between the two clusters are limited. The cross-cluster connections between cognitive domains and symptom severity showed positive and negative edges. The between-domain connectedness ranged from 0 (controlled visuospatial WM) to 3 (working memory and attentional flexibility). Positive edges were found between working memory and the severity of social discomfort (SOC2), sleeplessness (SLE1), and obsessions (OCD5). Attentional flexibility showed edges with unexplained crying (DEP4), sweating episodes (PAN14), and compulsions (OCD7) severity. Sustained attention showed edges with fearful anticipation (SEP2) and social discomfort (SOC2) severity. Visuomotor speed showed an edge with compulsivity (OCD9) severity and automatic visuospatial working memory with compulsions (OCD7). Interestingly, the correlation found between all the cognitive domains

and severity scores seems to be driven by only one symptom in the network. For example, the relationship between emotion recognition and PAN severity was driven only by the symptom ‘palpitations’.

**Figure 1**

*Network Estimation of Cognitive Domains and Psychopathology Symptoms*



*Note.* Transdiagnostic network of cognitive domains and psychopathology symptoms (N = 490). Nodes represent the variables and edges indicate an association between two nodes. Green edges represent positive associations and red edges represent negative associations, and the thickness represents the strength of an association.

## Discussion

### Principal Findings

This study aimed to examine the precise associations of several cognitive domains with psychopathology severity scores in a large sample of offspring of patients with psychopathology. In contrast to our expectations, sustained attention, inhibition, and attentional flexibility did not correlate with any of the severity scores. However, visuomotor

speed, working memory, as well as both controlled and automatic visuospatial working memory, each showed weak correlations with one severity score. Lastly, emotion recognition showed a weak correlation with two severity scores. The network analysis resulted in a highly connected network with two clusters showing relatively weak edges between the two. When inspecting both the correlation analysis and the network analysis, it appears that the relationship between cognitive domains and severity scores is predominantly driven by the connection with only one individual symptom. For example, emotion recognition showed a correlation with PAN severity. When looking at the network analysis, it is evident that this relationship is driven by the connection with the PAN symptom 'palpitations'. This suggests that the relationship between NCF and psychopathology severity is maintained by only one symptom.

### **Relations of Cognitive Domains and Psychopathology types**

Previous literature has shown that impairments in NCF are a core characteristic of depression (Rock et al., 2013) and anxiety disorders (McDermott & Ebmeier., 2009). However, in contrast to these studies and to our hypothesis, we found no-to-weak correlations between NCF and psychopathology severity. Studies reporting such relationships mostly found a moderate correlation. For example, McDermott & Ebmeier found that correlations ranged between -.11 to -.32. However, most of these studies compared patients with severe depression or other psychopathology diagnoses with a healthy control group (Austin et al., 2001). In contrast, this current study focused on a high-risk population, diagnosed with psychopathology, or experiencing subclinical symptoms. Due to the limited number of participants without symptoms, there was no control group of high-risk participants. This study, therefore, lacked a clear reference point which in turn may have hindered the identification of possible significant effects.



Secondly, most literature consisted of patients with a broad age range between 15 to 80 years, and the effects found were carried by the more adult population (Ferreri et al., 2011). While in contrast, this current study consisted of young individuals aged 13 to 25 years. Additionally, depression occurring later in life has been associated with more consistent impairments in NCF, and later age of onset was associated with more severe impairments (Ferreri et al., 2011). A possible explanation can be linked to the *scar* theory, in which psychiatric symptoms may over time lead to impairments in NCF. For example, in line with the *scar* theory, Zainal and Newmann (2022) found that depressed and anxious moods had the biggest impact on future NCF. It has been suggested that this is caused by the buildup of stress biomarkers throughout an individual's life which in turn can lead to neural injuries in brain areas during old age (Bessette et al., 2018).

### **Relations of Cognitive Domains and Individual Symptoms**

In line with Chavez-Baldini et al., (2023), cognition and psychopathology formed separate clusters showing relatively weak and few edges. As already described, the relationship between NCF and depression/anxiety severity was absent to weak. However, the network analysis revealed that this weak relationship was due to the connection with solely one individual symptom. Even though the found relationship is weak, it still highlights the importance of a symptoms-specific approach. This indicates that the relationship between NCF and psychopathology may not be a general pattern across psychopathology categories but instead, be driven by a specific symptom. Thus, the presence or absence of certain symptoms may, compared to other symptoms, have a stronger impact on the observed correlation.

As described by Chavez-Baldini et al., (2023), it is difficult to find clear answers about the relationship between NCF and psychopathology, since specific symptoms and impairments in cognitive functioning can vary within one diagnostic category. Additionally, conventional approaches do not consider the differentiation of NCF and psychopathology

components in testing their relationships (McNally, 2021). This current study, in contrast to more traditional approaches, focused on symptoms instead of diagnostic categories and therefore revealed a more nuanced understanding of how cognitive domains are related to psychopathology. Research has demonstrated that a symptom-specific network approach provides a clearer understanding of complex and poorly understood relationships (Vervaet et al., 2021). Emphasizing interactions of symptoms focuses on how symptoms activate and sustain each other (Roefs et al., 2022). This type of research can provide valuable insight into the mechanisms behind mental disorders, potentially informing the treatments of these disorders.

### **Network Structure Within Clusters**

Within the psychopathology cluster, symptoms that represented their own psychopathology diagnostic categories tended to cluster together, which is in line with Boschloo et al., (2015). In general, symptoms tend to exhibit stronger connections with symptoms within their own diagnosis than with symptoms of another diagnosis (Beard et al., 2016). As described by Boschloo (2018), this might be because classification systems have a categorical structure which in turn can be reflected in the network that is estimated. The instrument used in this current study represented the DSM-5 diagnosis and therefore was reflected in the estimated network. However, some symptoms showed stronger connections with symptoms outside their category, and cross-connections between the categories were present, which is in line with other studies (Cramer et al., 2010; Boschloo et al., 2016; McElroy et al., 2018). As described, symptoms are not necessarily separate disease entities, and cross-connections between two disorders can explain the high comorbidity found (Cramer et al., 2010). These overlapping symptoms can be associated with multiple disorders, especially in depression and anxiety.

### **Strengths, Weaknesses, and Suggestions for Further Research:**

This current study is the first to investigate symptom-specific relations between cognitive domains and psychopathology symptoms and therefore contributed to the limited literature on network analysis in cognitive domains and psychopathology. It included various psychopathology severity symptoms and cognitive domains within a large sample of individuals at high risk for developing psychopathology. Moreover, it served as a continuation of the study by Chavez-Baldini et al. (2023) and further emphasized the importance of network analysis and a symptom-specific focus, allowing for nuanced interpretations to be made. However, this study also has its limitations. This study chose to look at the baseline characteristics of the sample and consisted of a cross-sectional design. Therefore, no causality can be inferred, and results should be interpreted with caution (Guloksuz et al., 2017). In addition, the NCF tests took place at home, results could be influenced by external factors which in turn can influence the reliability of the scores. Finally, the neuropsychological tests used can differ between studies which can make it difficult to compare between these studies, since different results could be due to differences in tests used. To further investigate and clarify findings it is important to investigate longitudinal or via directed networks to detect patterns in a study design including a healthy control group. It is also recommended to test more cognitive domains and use multiple tests per domain.

### **Clinical Implications**

The results indicate that NCF barely plays a role in depression and anxiety in this young high-risk sample. Therefore, it can be concluded that intervening in NCF in this specific population is not necessary and may not be beneficial. Treating NCF in patients with psychopathology remains a challenge. Recently, there is increasing research on cognitive remediation therapy (CRT), which seems to have promising results in improving NCF and severe psychopathology symptoms (Kim et al., 2018). However, it is likely not sufficient as a stand-alone treatment since it only improved the symptoms in the short term. The effects were

not sustainable and after a 3-month follow-up, no significant effects were found (Legemaat et al., 2021). Moreover, research has shown that treating psychopathology symptoms instead of NCF also leads to improvements in NCF (Hallapa et al., 2018). Given these findings, it is advisable to prioritize intervening psychopathology symptoms over NCF in this high-risk population.

### **Conclusion**

In conclusion, cognitive domains, and psychopathology (symptoms) are independent but interacting dimensions. Despite the findings showing no prominent role of NCF in this high-risk population, more clarity has been gained regarding potential relationships between NCF and psychopathology. The results indicated that the relationship between NCF and psychopathology may be solely based on the association with one symptom, providing a more nuanced understanding. Thereby, this study emphasized the importance and advantages of the network approach and specifically the symptom-specific approach. Applying this approach to other populations may aid in the development of personalized interventions and treatments for patients' specific symptomatology.

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## **Appendix A: Overview of neuropsychological tests**

**Baseline speed (BS)** assesses the visuomotor reaction time. Participants were required to respond as fast as possible to a change in stimulus, by using the mouse with their dominant hand. The stimulus was a white fixation cross, displayed in the center of the screen, which changed into a white square after a random interval (ranging from 500ms to 2500ms). The task consisted of 32 experimental trials, and the mean reaction time of the dominant hand was calculated to determine the baseline speed.

**Feature identification (FI)** assesses both automatic and controlled visuospatial pattern recognition. First, participants were asked to memorize a target pattern consisting of nine patches, with three red and six white. Next, they were presented with four patterns and asked to determine if the target pattern was among them. This task consisted of 80 trials, including 40 target trials that required a response (containing the target pattern) and 40 non-target trials that required no response (not containing the target pattern). Within the target and non-target trials, half of the patterns were like the target or non-target pattern ('Pattern search') and half were dissimilar ('Pattern Detection'). To compute pattern search, the mean reaction times, or percentage of errors of the non-target dissimilar trials are subtracted from the mean reaction times or error rates of the non-target similar trials. On the other hand, to compute pattern detection, the mean reaction times, or percentage of errors of non-target dissimilar trials are subtracted from the mean reaction times or percentage of errors of the target dissimilar trials.

**Memory Search letters (MS)** assess working memory capacity and consists of three parts, each with an increase in working memory load. Prior to each part, participants are instructed to memorize, respectively, one (part 1, 40 trials), two (part 2, 72 trials) or three (part 3, 96 trials) target consonants. Then a set of four consonants are displayed, where half of

the trials involve the presence of the target consonant(s) and require a 'yes' response. While the other half contains none or some of the target consonant(s) and requires a 'no' response. The working memory capacity is determined by subtracting the mean reaction time or percentage of errors in response to target trials of part 1 (memorizing and processing one consonant) from the mean reaction time or percentage of errors in response to target trials of part 3 (memorizing and processing the combination of three consonants).

**Shifting attentional set-Visual (SA)** assesses inhibition of prepotent response and attentional flexibility. In the center of the screen, a horizontal bar containing ten squares is displayed, with a colored square moving randomly across each trial. The task consists of three parts with its own set of rules. Part 1 (40 trials) requires participants to mirror the direction of the green-colored square by pressing the left mouse button for leftward movement and the right button for rightward movement (Spatially compatible response). Part 2 (40 trials) requires participants to mirror the direction of the red square by pressing the left mouse button for rightward movement and the right mouse button for leftward movement, thus inhibiting their automatic spatially compatible response. Part 3 (80 trials) requires participants to alternate between responding according to the rules of part 1 for the green square and the rules of part 2 for the red square, which demands attentional flexibility. Attentional flexibility is determined by subtracting the mean reaction time or percentage of errors for the compatible responses of part 1 from that of part 3. Response inhibition is determined by subtracting the mean reaction time or the percentage of errors for part 1 from that of part 2.

**Sustained Attentional Dots (SD)** assesses response variability and responsiveness to feedback on errors. It evaluates the extent to which a person can sustain a certain performance level as well as the adaptation of feedback after making an error. 300 dot patterns are presented in a series of 25 sets of 12 trials. Each series comprises patterns with 3, 4, or 5 dots. When a 4-dot pattern appears, participants must press a mouse button with their dominant



hand ('yes' response), while for 3- or 5-dot patterns, participants must press a mouse button with their non-dominant hand ('no' response). An auditory signal is given when participants respond inaccurately. The task parameters are (1) sustained attention ('fluctuation in tempo'), computed as the within-subject standard deviation of the mean reaction time of the 24 series; and (2) feedback responsiveness, which is the ability of a participant to adjust their response following feedback on errors, and is computed as the difference between the participant's mean reaction time of trials following an error and the mean reaction time of the remaining correct responses.

**Identification of Facial Emotions (IFE)** measures the speed and accuracy of recognizing facial expressions. The task consists of 4 parts: sad, anger fear, and disgust. Target faces with these emotions were displayed on the screen. Each negative emotion has 40 trials with 20 target and 20 non-target trials. Participants were instructed to respond with a 'yes' if a target emotion appeared on the screen, by pressing the left mouse button, and to respond with a 'no' by pressing the right mouse button if a non-target emotion appeared on the screen. The task parameter for emotion recognition was calculated as the mean reaction time or error percentage across all emotions.

**Appendix B: Correlation matrix**

**Table 3**

*Overview of the correlations between psychopathology severity and cognitive domains (N = 490)*

Variabele	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. MDD	1.000															
2. OCD	.594**	1.000														
3. PAN	.680**	.556**	1.000													
4. SEP	.350**	.406**	.425**	1.000												
5. GAD	.771**	.631**	.676**	.368**	1.000											
6. SOC	.592**	.479**	.543**	.369**	.636**	1.000										
7. SOM	.553**	.382**	.626**	.331**	.522**	.394**	1.000									
8. SLE	.437**	.255*	.543**	.168**	.360**	.235**	.394**	1.000								
9. Sustained attention	.028	-.025	-.006	0.051	.036	.088	.041	.031	1.000							
10. Visuo-motor speed	.021	-.019	.019	.104*	.054	.047	-.026	-.036	.262**	1.000						
11. Working memory	.000	-.057	-.025	.058	-.004	.093*	-.023	.060	.435**	.103**	1.000					
12. Emotion recognition	-.054	-.043	-.118*	.022	-.048	-.020	-.106*	-.020	.534**	.345**	.347**	1.000				
13. Inhibition	-.017	-.010	.009	.040	-.030	-.023	-.019	.000	.201**	.110*	.122**	.228**	1.000			

14. Attentional flexibility	-.056	-.005	.058	.103*	.044	.057	.060	.068	.308**	.120**	.211**	.322**	.460**	1.000		
15. Controlled WM	-.006	-.015	-.019	.090*	-.031	.008	.028	.060	.310**	.093*	.330**	.355*	.163**	.245**	1.000	
16. Automatic WM	.011	.037	-.009	.016	.057	.010	.062	.106*	.108*	.040	.093*	.055	.074	.145**	.469**	1.000

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*Note.* \* indicates  $p < .05$ . \*\* indicates  $p < .01$ .

**Appendix B****Table 4***Overview of node labels and mean, standard deviations, and range of the edges (N = 490)*

Nodes	Symptom	<i>M</i>	<i>SD</i>	Range
DEP 1	'Feeling depressed'	.070	.066	.004 – .247
DEP 2	'Feeling sad'	.071	.070	.000 – .242
DEP 3	'Feeling helplessness'	.066	.070	.001 – .250
DEP 4	'Unexplained crying'	.049	.054	.003 – .242
DEP 5	'Feeling worthless'	.058	.063	.000 – .250
DEP 6	'Feeling guilt'	.064	.070	.003 – .264
DEP 7	'Feeling pessimistic'	.044	.037	.002 – .110
DEP 8	'Low energy and tired without cause'	.054	.043	.001 – .181
DEP 9	'Hardly experiencing any pleasure;	.077	.102	.003 – .395
DEP 10	'Losing interest'	.053	.102	.000 – .395
DEP 11	'Being inactive'	.053	.056	.010 – .186
DEP 12	'Thinking about suicide'	.048	.039	.000 – .122
GAD 1	'Overthinking'	.049	.045	.000 – .160
GAD 2	'Excessive worry'	.045	.033	.003 – .156
GAD 3	'Lacking confidence in performance'	.046	.040	.001 – .116
GAD 4	'Indecisiveness'	.045	.041	.001 – .154
GAD 5	'Being extremely nervous'	.040	.029	.001 – .147
SEP 1	'Finding it difficult to go somewhere without parents/caregivers'	.047	.083	.002 – .096
SEP 2	'Worrying that something will happen to parents/caregivers'	.067	.103	.016 – .330
SEP 3	'Afraid parent/caregiver will leave and never come back'	.058	.056	.002 – .330
SOC 1	'Feeling nervous while others watching.'	.052	.071	.003 – .194
SOC 2	'Social discomfort'	.048	.054	-.010 – .300
SOC 3	'Worrying about embarrassing myself around others'	.053	.084	.001 – .194
SOC 4	'Being extremely shy'	.061	.054	.008 – .300
PAN 1	'Panicking when alone outside.'	.064	.054	.005 – .229
PAN 2	'Panicking when traveling'	.049	.048	.001 – .229
PAN 3	'Panicking in crowded places'	.054	.050	.001 – .203

PAN 4	‘Moments of extreme anxiety to die’	.052	.048	.002 – .195
PAN 5	‘Extreme anxiety to lose control’	.066	.050	.005 – .154
PAN 6	‘Distress regarding bodily sensations.’	.049	.048	.000 – .289
PAN 7	‘Episodes of extreme anxiety’	.043	.063	.000 – .203
PAN 8	‘Dizziness.’	.048	.049	.001 – .125
PAN 9	‘Tremors.’	.057	.035	.001 – .154
PAN 10	‘Sensation of suffocation’	.064	.036	.001 – .154
PAN 11	‘Discomfort in chest’	.048	.044	.004 – .280
PAN 12	‘Palpitations’.	.046	.067	-.032 – .280
PAN 13	‘Numbness/tingling sensation in limbs’	.024	.065	.001 – .071
PAN 14	‘Sweating episode	.034	.019	.001 – .090
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SOM 1	‘Nausea’	.065	.027	.007 – .292
SOM 2	‘Abdominal pain’	.040	.075	-.030 – .292
SOM 3	‘Headache’	.067	.047	.016 – .150
SOM 4	‘Health concerns’	.084	.085	.003 – .289
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SLE 1	‘Insomnia’	.084	.100	.008 – .291
SLE 2	‘Poor sleep maintenance’	.053	.102	.000 – .367
SLE 3	‘Restless sleep’	.066	.106	.001 – .367
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OCD 1	‘Preoccupied by repetitive thoughts’	.045	.035	.000 – .111
OCD 2	‘High responsibility feeling’	.047	.067	.001 – .264
OCD 3	‘Concerned about bad thoughts’	.036	.026	.002 – .095
OCD 4	‘Afraid to harm others’	.042	.055	.000 – .203
OCD5	‘Obsessions’	.047	.042	.001 – .136
OCD6	‘Repetition.’	.053	.055	.001 – .167
OCD 7	‘Compulsions.’	.032	.033	.001 – .118
OCD8	‘Checking if things are in order’	.051	.052	.006 – .152
OCD9	‘Performing tasks compulsively with extreme precision’	.053	.047	.003 – .167
OCD10	‘Rituals’	.060	.051	.011 – .136

**Appendix C****Table 4**

*Overview node labels of cognitive domains and mean, standard deviation, and range of the edges (N = 490).*

Nodes	Cognitive domain	<i>M</i>	<i>SD</i>	Range
1	Sustained attention	.110	.121	.015 – .337
2	Visuo-motor speed	.096	.131	.004 – .246
3	Working memory	.093	.102	.006 – .255
4	Emotion recognition	.110	.128	-.034 – .337
5	Response inhibiton	.085	.121	-.010 – .316
6	Attentional flexibility	.086	.099	.003 – .316
7	Controlled visui-spatial WM	.134	.099	.055 – .326
8	Automatic visuo-spatial WM	.127	.172	.020 – .326