Connecting Residual Depressive Symptoms to Self-reported Executive Functioning: A Network Analytical Approach

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Abstract

Persisting executive functioning (EF) impairments following remission from depression form an important source of disability in daily life. However, little is known regarding how specific aspects of EF relate to residual depressive symptomatology. Using network analysis, the current study investigates unique associations between cognitive-, affective-, and somatic depressive symptoms (Beck Depression Inventory 2nd edition, BDI-II) and self-reported EF (Behavior Rating Inventory of Executive Function – Adult version, BRIEF-A) in a sample of 161 remitted depressed individuals. We identified three clusters of closely connected nodes, corresponding with the Metacognition- and Behavioral Regulation Index of the BRIEF-A, and one cluster consisting of cognitive, affective-, and somatic depressive symptomatology. Among the clusters consisting of EF domains, working memory and shifting difficulties emerged as bridging nodes. Depressive cognition most strongly connected the cluster of depressive symptoms with the EF clusters. Depressive symptom dimensions demonstrated both shared and unique associations with EF domains. Each depressive symptom dimension was directly related to emotional control impairments. In addition, multiple associations were observed between depressive symptomatology and complaints at the level of working memory, shifting, and planning/organizing. Depressive affect was uniquely related to difficulties initiating activity. The current findings provide insights into the relationship between perceived difficulties in EF and residual depressive symptomatology. EF domains were differentially related to depressive symptom dimensions, suggesting the need for further research into the role of EF following remission from depression.

Keywords: executive functioning; remission from depression; depression vulnerability; residual symptoms; network analysis

Highlights

- Executive functioning (EF) complaints persist following remission from depression
- Working memory and shifting impairments played a bridging role within the EF cluster
- Depressive cognition played a bridging role among the depressive symptom cluster
- Depressive symptom dimensions showed both unique and shared connections with EF
- Impaired initiation of behavior was uniquely related to depressive affect

Introduction

Cognitive impairments are frequently reported in the context of depression and are associated with poor psychosocial functioning (Knight et al., 2018). In particular, meta-analytical findings suggest significant executive functioning (EF) impairments – referring to higher order self-regulatory cognitive processes, such as inhibition, updating of information in working memory, and shifting, that allow adaptation of one's thoughts and behavior as a function of one's goals (Braver et al., 2002; Cohen, 2017; Miyake and Friedman, 2012) – in patients diagnosed with major depressive disorder (MDD; Rock et al., 2014; Semkovska et al., 2019; Sinha et al., 2022; Snyder, 2013). Importantly, impaired EF is known to persist following symptom improvement and remission of depression (Rock et al., 2014; Semkovska et al., 2019), where it has been identified as a major contributor to experienced disability in daily life (Knight et al., 2018). In this context, it has been suggested that strategies aimed at preventing recurrence of depression would likely benefit from targeting executive processes (Semkovska et al., 2019; De Raedt and Koster, 2010).

Indeed, recent findings suggest that interventions aimed at remediating executive dysfunction are effective in reducing residual depressive symptomatology in remitted depressed (RMD) individuals. For instance, top-down interventions such as Goal Management Training, which target executive dysfunction via the use of metacognitive- and problem-solving strategies, have shown promise in impacting EF complaints, emotion regulation processes, and depressive symptoms (Hagen et al., 2020; Stubberud et al., 2021). Previous studies also report beneficial effects of bottom-up interventions targeting EF, among which cognitive control training procedures where participants repeatedly conduct computerized tasks such as the adaptive Paced Auditory Serial Addition Task (Siegle et al., 2007). For instance, in line with prior observed effects of cognitive control training on indicators of functioning in clinically depressed patients (Siegle et al., 2007), Hoorelbeke and Koster (2017) observed beneficial effects of cognitive control training on indicators of EF, emotion regulation, and residual depressive complaints in RMD individuals. Moreover, recent findings suggest cognitive control training to reduce the risk for recurrence of depression (Hoorelbeke et al., 2021). At the same time, however, such interventions have shown considerable heterogeneity in treatment effects. For instance, while Vervaeke and colleagues (2021) observed improvements in cognitive functioning in RMD individuals following cognitive control training, this only showed limited effects on broader indicators of functioning (for recent reviews on cognitive control training, we refer to Edwards et al., 2022; Koster et al., 2017; for a meta-analysis of effects of Goal Management Training, see Stamenova and Levine, 2019). One potential explanation for these mixed findings is that, while these interventions often target a specific (set of) EF(s), depending on the type and level of residual depressive symptomatology experienced by the participant, multiple EF domains may be differentially involved.

Evidence for this notion can be found in prior literature investigating the relation between specific depressive symptom dimensions and EF domains in individuals at-risk for depression. For instance, in a recent cross-sectional study Davidovich and colleagues (2016) investigated the association between two central components of EF and type of depressive symptomatology in offspring of MDD patients (N=288). Relying on behavioral measures of EF, Davidovich et al. (2016) found higher levels of inhibitory control and mental flexibility to demonstrate a buffering effect for the occurrence of depressive symptomatology within this at-risk population. Importantly, this effect was moderated by type of depressive symptomatology (i.e., depressive symptom dimension), which was assessed using the Beck Depression Inventory 2nd edition (BDI-II; Beck et al., 1996). The BDI-II allows to differentiate between level of cognitive- and affective symptoms of depression, including symptoms such as self-criticalness and sadness, and level of somatic depressive symptoms (e.g., loss of energy; Steer et al., 1999; Storch et al., 2004; Whisman et al., 2000). Similar multi-factor models of depressive

symptomatology have also been obtained using other widely used screeners for depression (e.g., Elhai et al., 2012; Krause et al., 2010). In this context, Davidovich et al. (2016) observed that both inhibitory control and mental flexibility were negatively related to level of cognitive-affective symptoms of depression in offspring of MDD patients. In contrast, only inhibitory control seemed to exert a buffering effect for somatic symptoms of depression. Similar findings have been obtained in other at-risk samples for depression, where cognitive processes, including EF, have shown to be differentially related to depressive symptom dimensions (e.g., Bridwell et al., 2015; Castellon et al., 2006; Hawkins et al., 2015; Szymkowicz et al., 2018).

Given that the processes incorporated in EF are central to a variety of day-to-day tasks, EF deficits can severely hinder independent and productive functioning in everyday life for RMD patients (Knight et al., 2018; Knight and Baune, 2018). However, to date relatively little is known regarding the manifestation of EF complaints following remission from depression, and how these relate to patterns of experienced residual depressive symptomatology. Moreover, the literature reviewed above has mostly explored the association between EF and depressive symptom dimensions using a limited set of EF domains, which were typically assessed using performance-based measures (e.g., neuropsychological tests, behavioral EF tasks). Even if performance-based measures allow to assess an individual's optimal capacity and efficiency of EF in a highly controlled setting (Toplak et al., 2012), such measures likely suffer from limited ecological validity when aiming to investigate impairments in EF domains in daily life (e.g., Barkley and Fischer, 2011; Snyder et al., 2021). This, among other things (e.g., method variance), may explain the relatively poor correspondence between performance-based measures and subjective indicators of EF (e.g., Hagen et al., 2021; Serra-Blasco et al., 2019; Snyder et al., 2021), which may more closely capture success in goal pursuit in daily life (Toplak et al., 2012). As such, it has been argued that self-report measures for EF, such as the Behavior Rating Inventory of Executive Function – Adult version (BRIEF-A; Roth et al., 2005), may better reflect experienced EF difficulties in daily life (Schmid and Hammar, 2021).

The current exploratory study set out to investigate patterns of associations between self-reported EF – relying on the BRIEF-A, a self-report measure which assesses difficulties in various aspects of EF in daily life (e.g., inhibition, shifting, initiation of behavior; Roth et al., 2005) – and residual affective-, cognitive-, and somatic depressive symptoms, as measured by the BDI-II, in RMD patients. For this purpose, we will rely on network analysis as this allows for a data-driven investigation of complex interrelations between subjective EF domains and depressive symptom dimensions (for a detailed discussion of clinical implications of the network approach to psychopathology, we refer to Hofmann et al., 2016). Specifically, our first aim is to investigate the network structure of different types of residual depressive symptomatology (cognitive-, somatic-, and affective symptoms) and EF complaints. In particular, we will investigate indices of node centrality, which provide a measure for level of connectivity of the node within the network, as well as model how indicators of residual depressive symptoms and EF cluster (i.e., are more closely related) within the obtained network model. In addition, we will explore direct associations between different types of residual depressive symptoms and self-reported EF complaints, allowing for a more fine-grained understanding of how EF is involved in functioning in RMD individuals.

Materials & Methods

Participants

The current study offers a secondary analysis of baseline data collected in the context of two cognitive training studies (Hoorelbeke and Koster, 2017; Hoorelbeke et al., 2021). These participants have in common that they needed to be aged between 23–65 years old, roughly corresponding to the working population in Belgium, reported a history of depression, and were in a state of (partial) remission, in absence of extensive substance abuse or psychosis. The investigation was carried out in accordance with the latest version of the Declaration of Helsinki. Informed consent was obtained after the nature of the procedures had been fully explained. Both studies were approved by the local ethical committee of the Faculty of Psychology and Educational Sciences of Ghent University.

Materials

Screening

During a two-step screening procedure, in- and exclusion criteria were assessed using the relevant modules of the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1989; Van Vliet and De Beurs, 2007). Given that individuals showing either full- or partial remission from depression were allowed to partake in the study, inclusion was based on no longer meeting the criteria for MDD as assessed in this clinical interview. First, participants completed a telephone interview to assess the occurrence of a past depressive episode and their current state of remission. Subsequently, they were invited for a second MINI interview that was performed by trained clinical psychologists at the Faculty of Psychology and Educational Science of Ghent University prior to the baseline assessment.

Depressive symptomatology

We used the Dutch version of the BDI-II (BDI-II-NL; Beck et al., 1996; van der Does, 2002) to measure severity of residual depressive symptomatology. This self-report measure contains 21 items, each rated on a four-point scale. The factor structure of the BDI-II-NL comprises of three subscales, reflecting affective-, cognitive-, and somatic symptoms of depression (van der Does, 2002). The Affective symptoms subscale consists of five items (range: 0–15; Cronbach's α=.73) including symptoms of Sadness, Pessimism, Loss of Pleasure, Suicidal Thoughts, and Loss of Interest. The Cognitive symptoms subscale contains seven items (range: 0–21; Cronbach's α=.84; e.g., Failure, Self-Criticalness, Indecisiveness, Worthlessness). The remaining nine items belong to the Somatic symptoms subscale (range: 0–27; Cronbach's α=.82), among which Agitation, Loss of Energy, Irritability, Change in Sleep pattern and Appetite, Difficulties Concentrating and Tiredness. For an evaluation of this three-factor structure in the current sample we refer to the Supplemental Materials.

Executive functioning complaints

We relied on the Dutch translation of the BRIEF-A (BRIEF-A-NL; Roth et al., 2005; Scholte and Noens, 2011) to assess EF complaints in daily life. The BRIEF-A consists of 75-items that are scored on a three-point scale (1="never", 2="sometimes", 3="often"). This measure contains nine clinical scales, four of which belong to the Behavioral Regulation Index (Inhibit, Shift, Emotional Control, and Self-Monitor). This index provides an estimation of the extent to which individuals are able to adequately regulate their behavior and emotional responses. The Metacognition Index consists of the remaining five clinical scales (Initiate, Working Memory, Plan/Organize, Task Monitor, Organization of Materials) and reflects the extent to which individuals can initiate activity and generate problem-solving ideas, as well as plan, organize, and monitor performance of problem-solving strategies (Roth et al., 2005; Roth

et al., 2013). Table 1 offers an overview of the nine clinical scales which are used as input for the network model, reflecting different domains of EF. For each of these measures, a higher score is indicative of more severe EF complaints.

Procedure

After assessment of eligibility for participation to the study, participants entered the baseline assessment of the training studies during which they provided informed consent and completed a set of questionnaires including the BDI-II-NL and BRIEF-A-NL. After completing the training study, participants received a debriefing and financial reimbursement. For more information on participants and procedures, we refer to Hoorelbeke and Koster (2017) and Hoorelbeke and colleagues (2021).

Statistical Analyses

Analyses were conducted in R, version 3.6.1. Below we provide a brief overview of the data analytical approach used. For a more detailed discussion of these steps and information on R-packages used, we refer the interested reader to the Supplemental materials.

We first used a nonparanormal transformation to improve normality, after which we estimated a Gaussian Graphical Model (GGM; Epskamp and Fried, 2018) relying on the Graphical Least Absolute Shrinkage and Selection Operator (gLASSO; Friedman et al., 2014) with Extended Bayesian Information Criterion model selection (EBIC). We implemented bootstrapping procedures described by Epskamp et al. (2018) to evaluate the accuracy and stability of the obtained GGM. We then relied on Strength – reflecting absolute strength of connectivity of a node within the model – to identify the most central nodes in the network, and computed node predictability (explained variance of each node by neighboring nodes; Haslbeck and Fried, 2017). Exploratory graph analysis was used to identify communities of more closely connected nodes (Golino and Epskamp, 2017), where we relied on Bridge Strength – referring to the extent to which an edge within a given community is directly connected to edges from other communities – to identify bridging nodes (Jones et al., 2021). The position of the nodes in the network is based on patterns of connectivity, relying on the Fruchterman-Reingold's algorithm (Fruchterman and Reingold, 1991). Edge color indicates the valence of the association (blue/full edges=positive, red/dashed edges=negative) and edge thickness reflects association strength. Finally, to investigate how depressive symptom dimensions were uniquely related to EF complaints within the obtained GGM, we relied on flow diagrams.

Results

The analyses are based on *N*=161 RMD individuals. Descriptive statistics and sample characteristics are reported in Table 2. Among this sample, based on the BDI-II, 122 participants reported experiencing minimal residual depressive symptomatology (<14), 12 participants reported mild residual symptoms (14-19), and for 21 participants residual symptoms appeared to be in the range of moderate severity (20-28). Although none of the participants met clinical criteria for depression, six participants reported experiencing severe depressive complaints (>28). Evaluation of T-scores for the BRIEF-A indicate that EF complaints, based on the Global Executive Composite score, were around one *SD* above the mean of a norm group consisting of 1600 Dutch-speaking adults between the age of 18 – 65 (Scholte & Noens, 2011; Table 2 provides an overview of T-scores for the different EF domains).

Figure 1 presents the obtained GGM (for the edge weight matrix, see Supplemental Table 1). The obtained network model showed adequate accuracy and stability (Supplemental Figures 1-5). The strongest edges in the network appeared between Initiate (B5)–Plan/Organize (B7), and Depressive Somatic (D2)–Depressive Affect (D3). Both edges were significantly stronger than 74% of the other edges identified in the network model (see Supplemental Figure 4). Overall, the abilities to Plan/Organize (B7), Working Memory (B6), Initiate (B5), and Task Monitor (B8) emerged among the most central nodes in the network in terms of Strength centrality. Organization of Materials (B9) was ranked as the least central node in the network (Figure 2). On average, 53% of variance of the nodes in the network was explained by neighboring nodes.

We identified three communities of closely connected nodes (Figure 1). The first community contained subscales of the BRIEF-A which belong to the Metacognition Index (B5:B9). The second community consisted of the BRIEF-A subscales underlying the Behavioral Regulation Index (B1:B4). The third community contained the BDI-II subscales reflecting severity of cognitive-, somatic-, and affective residual depressive symptoms (D1:D3).

Within the community of EF domains belonging to the Metacognition Index, Working Memory (B6) was ranked first in terms of Bridge Strength (Figure 3). For the community consisting of EF domains belonging to the Behavioral Regulation Index, Shifting (B2) was ranked first in terms of Bridge Strength. Finally, among the indicators of depressive symptomatology, Depressive Cognition (D1) was ranked first in terms of Bridge Strength, transferring activation of indicators of depressive symptomatology to EF domains, and vice versa.

Further investigation of unique associations between specific types of depressive symptomatology and EF domains suggests that Depressive Cognition (D1) is directly connected to self-reported impairments at the level of Shifting (B2), Emotional Control (B3), Working Memory (B6) and Plan/Organize (B7; Figure 4a). For the node representing severity of Depressive Somatic symptomatology (D2), we observed direct associations with Emotional Control (B3) and Working Memory (B6) complaints (Figure 4b). Finally, Depressive Affect (D3) was directly connected to dysfunctioning at the level of Shifting (B2), Emotional Control (B3), Initiation of behavior (B5), and difficulties Planning/Organizing (B7; Figure 4c). All aspects of depressive symptoms showed direct associations among one another (D1:D3).[1]

[1]The same direct associations between depressive symptom domains and EF complaints were observed when excluding the six RMD patients with a history of bipolar depression. In addition, excluding participants reporting severe depressive symptoms from the analysis only had a limited impact on the direct associations between EF domains and depressive symptom dimensions. All

Discussion

EF is proposed to play a key role in the pathophysiology of recurrent depression (De Raedt & Koster, 2010; Hoorelbeke et al., 2021), where RMD patients have reported EF impairments to be an important source of disability (Knight et al., 2018). In the current study, considerable variability in level of residual symptomatology was observed, where the obtained BRIEF-A scores corresponded with previously obtained levels of EF complaints in the context of depression (Hagen et al., 2021; Schmid & Hammar, 2021). Prior research in at-risk populations suggests that EF domains may be differentially involved in depression, depending on the symptom dimension under investigation. However, to date, studies on how experienced difficulties across different EF domains uniquely contribute to functioning following remission from depression are lacking. For this purpose, we relied on network analysis, a data-driven exploratory analytical strategy, to investigate patterns of connectivity between EF domains and depressive symptom dimensions in RMD individuals. Given the non-directed nature of this model, the obtained pattern of results should strictly be considered as hypothesis generating, where identified edges in the network may be indicative of causal relationships which remain to be tested in follow-up studies using appropriate prospective or experimental designs.

We identified three communities of closely connected nodes, representing: (a) self-reported difficulties generating, initiating, planning/organizing and monitoring performance of problem-solving strategies (Metacognition Index), (b) self-reported difficulties with aspects of regulation of one's behavior and emotional responses (Behavioral Regulation Index), and (c) self-reported cognitive-, affective-, and somatic depressive symptoms. Based on level of Strength Centrality, the community containing the subscales of the BRIEF-A Metacognition Index held an influential role in the obtained network model. The current finding reaffirms the idea that such abilities are closely associated to other EF complaints and depressive symptomatology following remission from depression, where activation of these nodes is likely to further initiate the dysfunctional network of residual EF- and depressive complaints. This makes these EF domains valuable targets for top-down (e.g., Goal Management Training) and bottom-up (e.g., cognitive control training) clinical interventions targeting depression vulnerability (but see Bringmann et al., 2019).

Within each of their respective clusters, self-reported Depressive Cognition (D1), Working Memory (B6), and Shift (B2) were ranked first in terms of Bridge Strength. This indicates that activation of the cluster consisting of residual depressive symptoms was most likely to spread into activation of the EF complaints clusters via Depressive Cognition (D1), pointing towards the role of depressogenic thought content – including a focus on past Failure, Guilt, Punishment, Self-Dislike, Self-Criticalness, Indecisiveness, and Worthlessness – in linking affective- and somatic depressive symptoms with EF complaints. In addition, activation of the cluster of metacognition is most likely to result in activation of residual depressive symptoms or impairments at the level of regulation of one's emotions and responses, via experienced difficulties holding task-relevant information in working memory. Given the undirected nature of the network model, the cluster of metacognition is also most likely to be further activated by nodes belonging to external clusters via Working Memory (B6) complaints. Similarly, experienced difficulties to flexibly shift or divert attention from one activity/task or situation to another (B2) are most likely to transfer activation from the Behavioral Regulation cluster of the BRIEF-A to the clusters consisting of residual depressive and metacognitive EF complaints, and vice versa. These findings are informative to clinicians who are confronted with RMD individuals showing

observed edges remained present, except for the edge between Depressive Affect (D3) and Plan Organize (B7).

specific profiles of executive dysfunction, where one would like to prevent further activation of the network to limit risk for recurrence of depressive symptoms.

Of particular interest, both from a theoretical and clinical perspective, is the observation that depressive symptom dimensions show shared and unique associations with EF domains in RMD patients. That is, investigation of unique associations between depressive symptom dimensions and EF domains revealed that experienced difficulties in Emotional Control (B3) were directly linked to all depressive symptom dimensions (D1:D3). This suggests a key role for the ability to regulate one's emotional response for cognitive-, affective- and somatic depressive symptoms. This finding corresponds to previous work indicating emotion regulation to be an important transdiagnostic marker for the development and maintenance of psychopathology such as depressive symptomatology (Sloan et al., 2017).

Overall, Depressive Somatic (D2) showed the least amount of direct associations with EF complaints. This is noteworthy given that somatic depressive symptoms include experienced tiredness and difficulties concentrating. In this context, a recent study in which a selection of somatic depressive symptoms was included, provided support for a direct association between performance-based indicators of EF (including updating), and some of the somatic depressive items – among which fatigue or loss of energy – but not for others (e.g., changes in sleep pattern, agitation, and experienced difficulties concentrating; Kraft et al., 2022). In line with this, in addition to the connection with Emotional Control (B3), we observed a direct association between Working Memory (B6) complaints and level of Depressive Somatic (D2) symptoms. Working Memory (B6) was also directly associated with level of Depressive Cognition (D1). These findings are consistent with the observed bridging role of working memory complaints, and prior experimental research suggesting working memory processes to be causally involved in depression vulnerability (Koster et al., 2017).

In addition to a shared association with self-reported Emotional Control (B3), Depressive Cognition (D1) and Depressive Affect (D3) each showed a direct association with Shifting (B2). These associations point towards the particular importance of emotion regulation and the ability to flexibly shift attention for symptoms reflecting the presence of depressogenic thought content and -affect. This is in line with prior work in which performance-based measures were used suggesting that within individuals with a history of depression, poor EF places one at risk to elaborate on negative thought content, activating self-reinforcing cycles of (sustained) negative affect and (re)activation of negative schemata, further impacting EF and risk for recurrent depression (De Raedt and Koster, 2010). Indeed, prospective studies in RMD individuals show that deficits at the level of shifting predict a further increase in depressive symptomatology, a relation which has shown to be mediated by perseveration of negative thought content (Demeyer et al., 2012).

An interesting unique association was observed between self-reported Depressive Affect (D3) and Initiate (B5), which emerged as one of the strongest edges linking EF to Depressive Affect. Initiate (B5) was also closely related to the ability to Plan/Organize (B7), the latter showing direct connections to both Depressive Affect (D3) and -Cognition (D1). These findings allow integration of EF in reinforcement deprivation accounts of depression (Ferster, 1973; Kanter et al., 2005; Lewinsohn, 1975), where experienced difficulties at the level of initiation of behavior is likely to result in reduced exposure to reinforcing stimuli. In time, this may result in the development or maintenance of affective depressive symptoms, among which loss of pleasure and interest, and vice versa. This notion has formed the basis for behavioral interventions for depression, such as behavioral activation, which have proven to be effective in decreasing depressive symptomatology (Ekers et al., 2014). In addition, this finding fits well with more recent motivational perspectives on the role of EF impairments in depression (for a review, see Grahek et al., 2019), pointing towards the importance of research into

the role of reward probability expectancy, learned action-outcome contingencies, and effort needed to allocate executive control and/or initiate behavior. Finally, in contrast to prior work suggesting an important role of inhibition in the context of depression (Joormann, 2010), after controlling for the role of EF domains described above, our current findings suggest no direct association between self-reported inhibitory complaints (B1) and depressive symptom dimensions. This unexpected finding may potentially be explained by the relatively strong focus on behavioral inhibition in the BRIEF-A, whereas inhibition of task-irrelevant thoughts and feelings may be more relevant in the context of depression. Similarly, the EF domains Self-Monitor (B4), Task Monitor (B8), and Organization of Materials (B9) were only indirectly related to depressive symptomatology.

The current study is the first to explore the complex associations between depressive symptom dimensions and experienced difficulties in a comprehensive set of EF domains in RMD patients using network analysis. This provides novel insights on how use of EF in goal pursuit in daily life is hindered in RMD individuals and potentially holds important implications for treatment of cognitive vulnerability for (recurrent) depression given that executive dysfunction has been related to poor long-term outcomes (e.g., Demeyer et al., 2012). In particular, although multiple studies using performance-based measures suggest the presence of working memory and shifting deficits in the context of depression (Gotlib and Joormann, 2010; Joormann and Vanderlind, 2014), our study is the first to demonstrate the bridging role of self-reported working memory and shifting impairments in connecting other EF domains with depressive symptomatology following remission from depression.

In addition, we observed both shared and unique associations between specific EF domains and depressive symptom dimensions. This allows for a more fine-grained understanding of how different cognitive processes may be involved in functioning in daily life in RMD individuals, which is crucial as prior work suggests EF domains to be differentially related to psychosocial functioning across different stages of depression (Knight and Baune, 2018). These results point towards the need of further investigation into EF complaints, among which working memory and shifting impairments, and how these relate to functioning in RMD individuals. Particularly, this is of relevance provided the encouraging findings of targeted EF training procedures among which cognitive control training (Hoorelbeke et al., 2021; Launder et al., 2021; Siegle et al., 2014), where the current study may help shed light on the working mechanisms underlying training effects by providing a model for how change in one EF domain may potentially impact other EF domains and depressive symptom dimensions. Vice versa, given the undirected nature of the network, the model allows formulating hypotheses on how presence of depressive thought patterns, feelings, and behavior may impact EF.

The current study also adds to the increased importance that is allocated to *self-reported* EF, where most previous studies have used performance-based measures. Previous work has reported that self-regulation assessments through questionnaires and performance-based measures are not strongly associated, allowing for separability (Friedman and Banich, 2019), where self-report measures may better reflect day-to-day EF (Snyder et al., 2021). Given the pattern of findings obtained in the current study, as a next step it would be interesting to rely on a more integrative approach, investigating performance-based and self-report measures of EF in relation to functioning. In addition, given the cross-sectional nature of the data and the use of undirected network models, one important limitation of the current study is that, while being hypothesis generating, it does not permit to make causal inferences. Building on the current findings, this warrants further prospective and experimental research into the relation between executive dysfunction and depressive symptom dimensions. Moreover, although self-report measures allow to capture day-to-day EF complaints, they are also susceptible to several biases, which may have resulted in over- or underreporting of symptoms. Furthermore, given that participants were recruited from cognitive training studies, this sample

potentially represents a group of RMD patients with relatively high symptom awareness. In addition, although we included RMD individuals using a broad age range, the obtained sample predominantly exists of RMD individuals who were in late middle adulthood (age 45–64; N = 89), relative to early- (age <35; N = 31), early middle- (age 35–44; N = 37) and late adulthood (age >64; N = 4). In this context, a recent meta-analysis suggests a moderating role for age, where a stronger association between depression and EF has been observed in later stages of the lifespan (Dotson et al., 2020). As such, future studies investigating differential associations between EF domains and depressive symptom dimensions would benefit from taking age and age of onset (cf. Semkovska et al., 2019) into account. Importantly, although age was directly related to higher levels of depressive somatic complaints in the current study, sensitivity analysis indicates that controlling for age did not affect the observed pattern of differential associations between depressive symptom dimensions and EF domains (supplemental Figures 5a:5c). In addition, given that EF seems to worsen with repeated episodes (Semkovska et al., 2019), it would also be interesting for future studies to investigate whether associations between EF domains and depressive symptom dimensions are impacted by other clinical characteristics, such as number of prior depressive episodes.

Finally, these findings are based on the Dutch versions of the BRIEF-A and BDI-II. This might limit the generalizability of our findings to other samples in which other questionnaires or measures of EF are used (e.g., performance-based measures). In this context we should note that the factor structure of the BDI-II is known to differ between different populations (van der Does, 2002). Importantly, several goodness of fit indices suggested adequate fit for the three-factor structure (see Supplemental Materials).

Conclusion

Based on previous work suggesting differential effects of EF on psychosocial functioning depending on stage of depression and depression symptom dimension under investigation, the current study set out to model patterns of associations between self-reported EF and depressive symptom dimensions in RMD patients. Our findings point towards the importance of impairments at the level of Emotional Control, Working Memory, Shifting, and Plan/Organize, which each showed unique associations with multiple depressive symptom dimensions. In addition, the ability to initiate tasks or activity was uniquely related to Depressive Affect. These findings suggest EF domains to be a relevant target in treatment of residual depressive symptoms, for instance through cognitive training procedures.

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CRediT Authorship Contribution Statement

KH, YVZ and EHWK developed the study concept. All authors contributed to the study design. Testing and data collection were performed by KH, who also performed the data analysis and interpretation. KH and YVZ drafted the paper, and all other authors provided critical revisions. All authors approved the final version of the paper for submission.

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Declaration of Conflicting Interests

The authors declare that there are no conflicts of interest with respect to the authorship or the publication of this article.

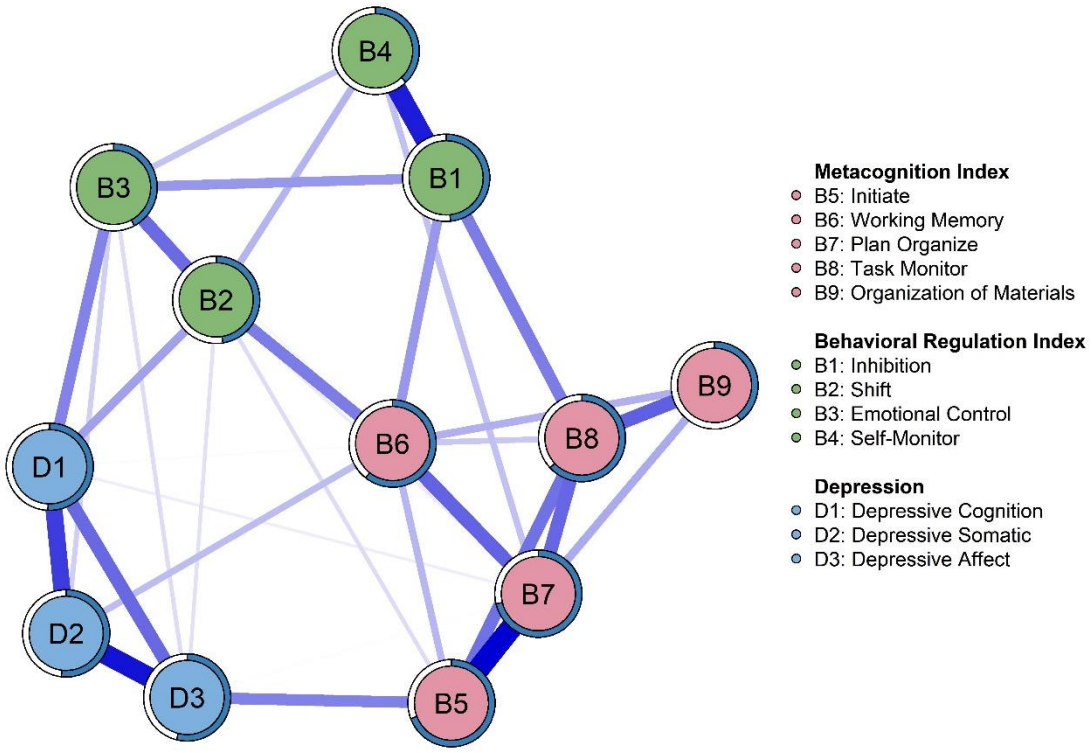

Figure 1. Regularized Partial Correlation Network

Note: Edges in this network represent unique associations between each of the constructs (nodes). Relative strength of association is reflected by edge thickness, thick edges reflect strong associations. The valence of the association is depicted by edge color: blue / full edges reflect a positive association, red / dashed edges reflect a negative association between two nodes. Nodes are positioned in the model based on the patterns of connectivity. The pie chart surrounding each node represents the amount of variance explained by neighboring nodes (node predictability). For an overview of the edge weights we refer to Supplemental Table 1.

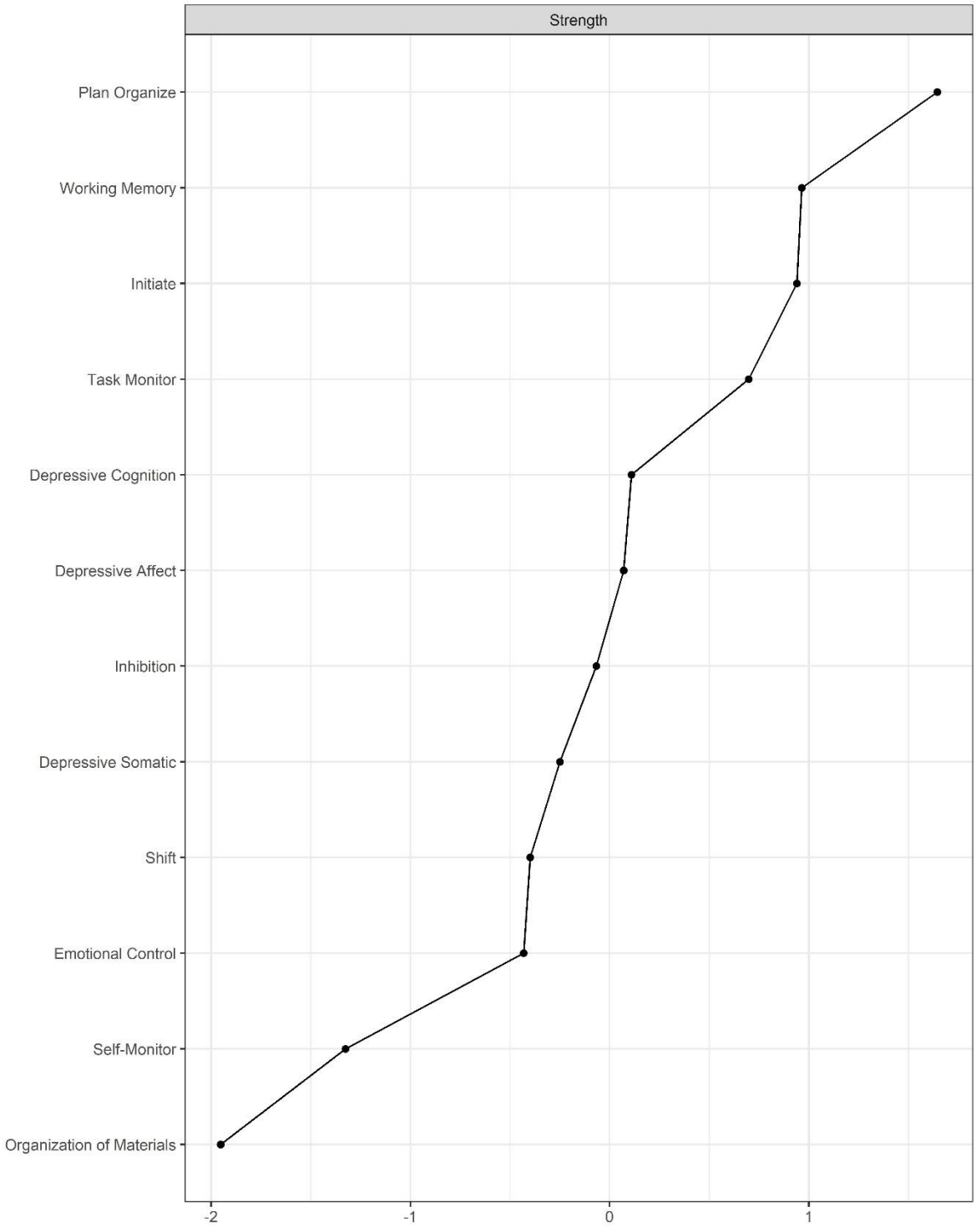

Figure 2. Strength Centrality

Note: This figure depicts how nodes within the network are ranked in terms of Strength Centrality. Strength Centrality refers to the extent to which a node is connected to other nodes within the network model, and is calculated using the sum of absolute edge weights of connected nodes. Higher levels of Strength Centrality indicate that the node plays a more central role in the network model/is more strongly connected.

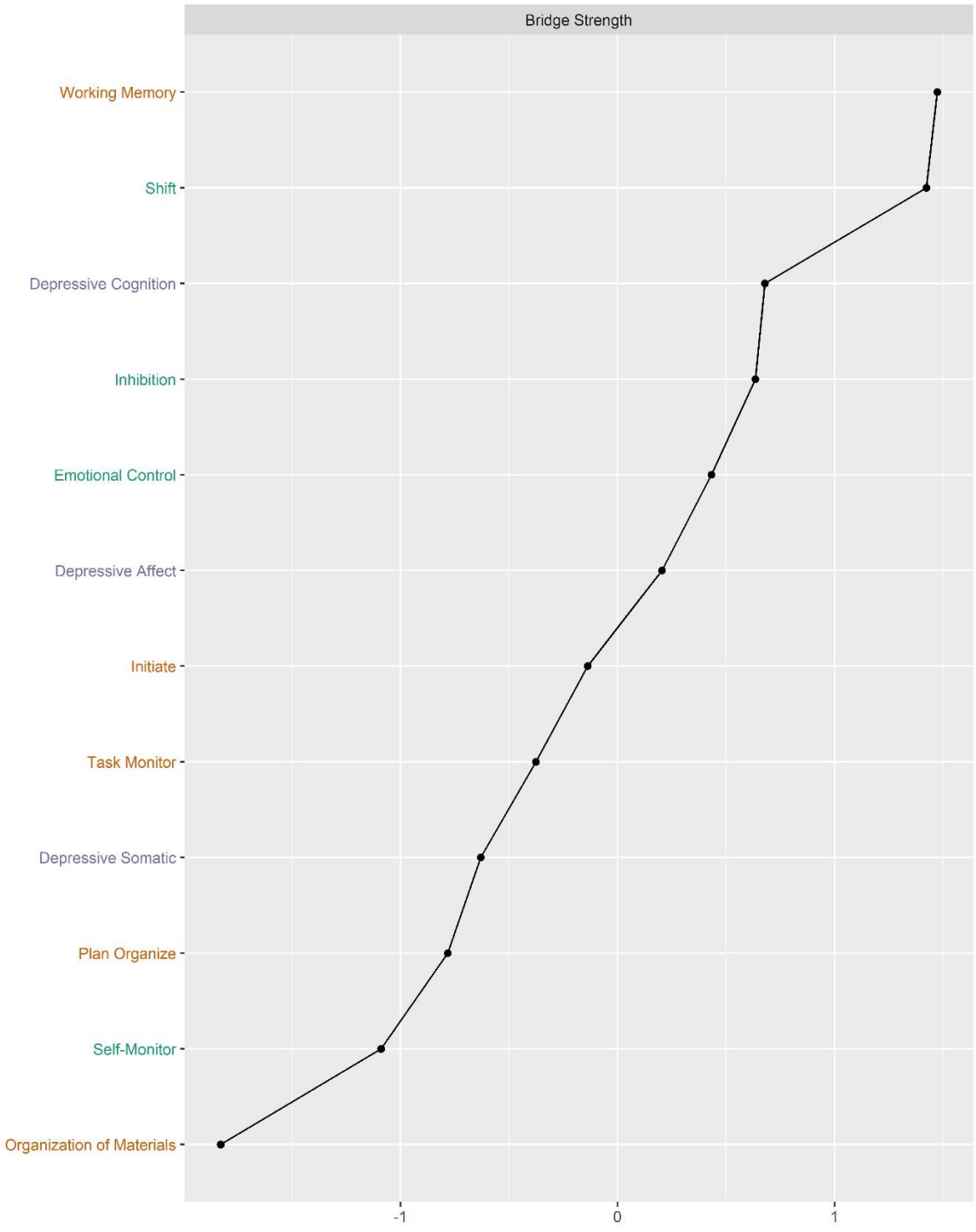

Figure 3. Bridge Strength

Note: This figure depicts how nodes within the network are ranked in terms of Bridge Strength, taking into account membership of communities consisting of more strongly connected nodes. Higher levels of Bridge Strength indicate that a node within a given community is more strongly connected to nodes from other communities.

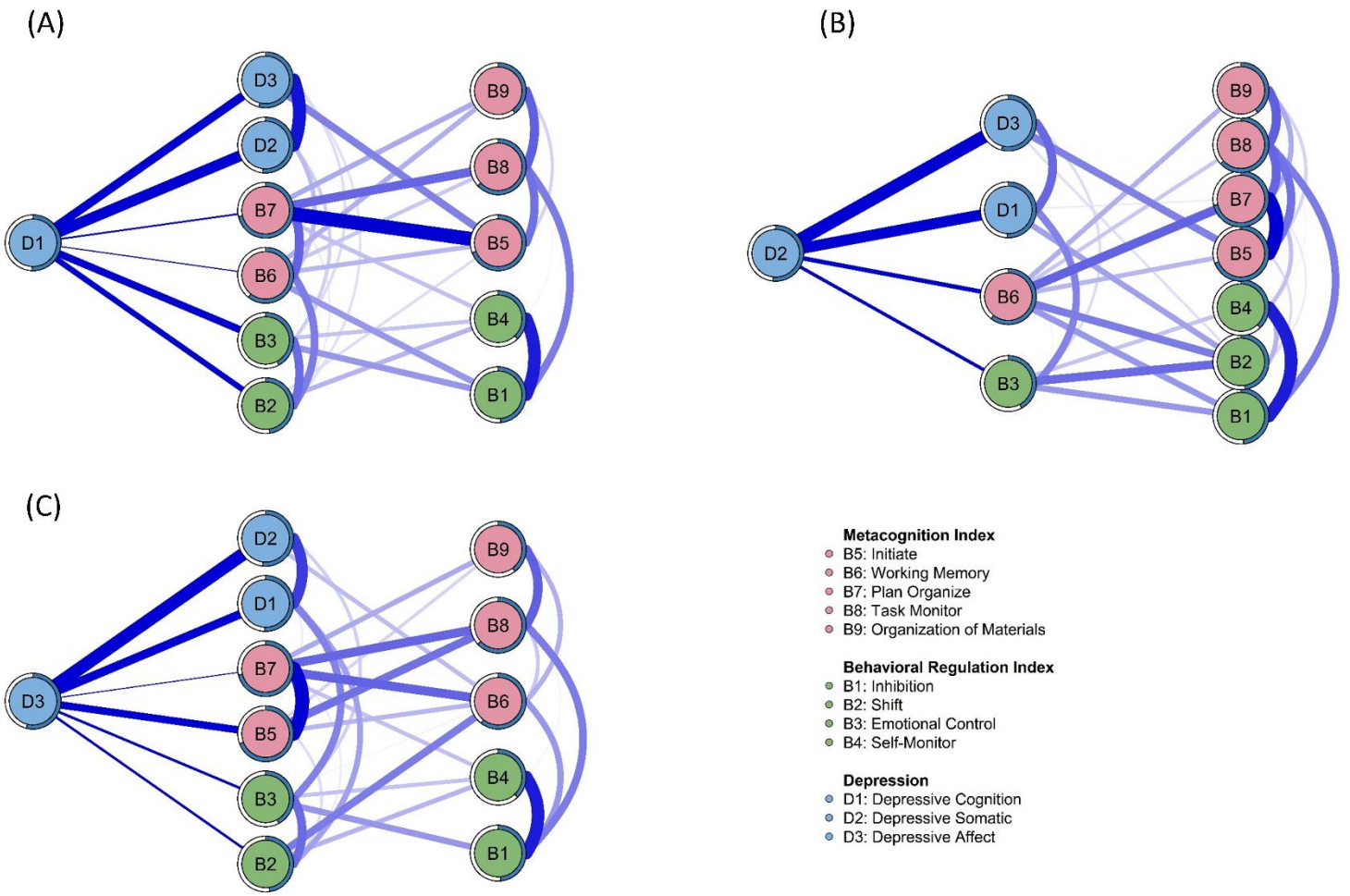

Figure 4. Flow chart of unique associations between type of residual depressive symptomatology and EF domains. *Note:* (A) Direct associations between Depressive Cognition and EF complaints; (B) Direct associations between Depressive Somatic and EF complaints; (C) Direct associations between Depressive

Affect and EF complaints. Building upon the obtained network model, these figures provide a hierarchical overview of directly and indirectly (i.e., second or third order) connected nodes for cognitive-, affective- and somatic depressive symptoms.

Table 1. BRIEF-A clinical scales

Scale	*Description*	*Items*	*Range*	*Cronbach's α*
Global Executive Composite	Total score reflecting level of EF complaints	70	70 – 210	.95
Behavioral Regulation Index		30	30 – 90	.89
Inhibit	Inhibition of thoughts and actions, impulse control E.g., "I am impulsive"	8	8 – 24	.61
Shift	Flexibility in shifting problem solving set E.g., "I have trouble changing from one activity or task to another"	6	6 – 18	.71
Emotional control	Regulation of one's emotional response E.g., "I have emotional outbursts for little reason"	10	10 – 30	.89
Self-Monitor	Monitoring of one's actions / the effects of one's behavior on others E.g., "I don't think about consequences before doing something"	6	6 – 18	.76
Metacognition Index		40	40 – 120	.94
Initiate	Ability to initiate a task or activity, independently generating ideas, responses or problem solving strategies E.g., "I have trouble getting started on tasks"	8	8 – 24	.80
Working Memory	Ability to hold information in working memory to complete a task / activity E.g., "I forget what I am doing in the middle of things"	8	8 – 24	.85
Plan/Organize	Anticipating future events, settings goals, planning and organizing problem solving approaches E.g., "I have problems organizing activities"	10	10 – 30	.83
Task Monitor	Ability to monitor success and failure in problem solving E.g., "I have trouble finishing tasks (such as chores, work)"	6	6 – 18	.72
Organization of Materials	Ability to organize one's materials and environment in an orderly manner and keep track of materials needed for the task at hand E.g., "I lose things (such as keys, money, wallet, homework, etc.)"	8	8 – 24	.87

Note: This table provides an overview of the content of the nine clinical scales derived from the BRIEF-A. These are divided over the Behavioral Regulation- and Metacognitive Index and combined in the Global Executive Composite score.

Table 2. Descriptive Statistics

Demographic and clinical features (N = 161)	*Ratio*	*M (SD)*	*M T score (SD)*
Age		46.37 (12.25)	
Gender (Male : Female)	56 : 105		
Using antidepressant medication (yes : no)	75 : 86		
Number of depressive episodes (1 : 2 : 3 : ≥4)	35 : 41 : 32 : 53		
Time in remission (months)		48.93 (62.69)	
Type of depression (unipolar : bipolar)	155 : 6		
Questionnaires			
Severity of residual depressive symptoms (BDI-II, total)		10.35 (8.70)	
Depressive Cognition		3.53 (3.72)	
Depressive Somatic		5.11 (4.24)	
Depressive Affect		1.71 (1.84)	
Global Executive Composite (BRIEF-A)		123.48 (22.20)	60.23 (10.86)
Behavioral Regulation Index (BRIEF-A)		52.16 (9.68)	57.71 (10.13)
Inhibition		12.89 (2.69)	54.74 (10.19)
Shift		11.13 (2.60)	58.61 (10.85)
Emotional Control		18.93 (4.79)	58.49 (11.14)
Self-Monitor		9.20 (2.42)	52.21 (10.86)
Metacognition Index (BRIEF-A)		71.33 (15.25)	60.92 (12.55)
Initiate		13.94 (3.53)	58.72 (12.51)
Working Memory		15.35 (3.89)	63.29 (12.84)
Plan/Organize		17.24 (4.17)	59.73 (12.52)
Task Monitor		10.76 (2.48)	57.42 (11.49)
Organization of Materials		14.03 (4.11)	56.51 (13.03)

Note: This table provides an overview of demographic variables and clinical characteristics of the sample, in addition to descriptive information for the measures of interest. T-scores for the BRIEF-A were based on a norm group consisting of 1600 Dutch-speaking adults between the ages of 18 and 65, based on Scholte and Noens (2011).