The effects of mindfulness and positive fantasizing on rumination and depression: A network perspective

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Introduction

About one in ten people currently suffer from Major Depressive Disorder (MDD; Kessler & Bromet, 2013; Tolentino & Schmidt, 2018), and more than one in five will be affected by this debilitating mood disorder at some stage in their lives (Hasin et al., 2018). Furthermore, MDD is a recurrent disorder, meaning that individuals that have endured one episode, commonly experience further ones in the future. In fact, the likelihood of recurrence is greater than 80% after 15 years (Hardeveld et al., 2010; Mueller et al., 1999).

According to the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013), depression encompasses behavioral, emotional and cognitive symptoms (see Table 1). Cognitive symptoms that are routinely observed in depressed patients such as poor executive control, attentional biases toward negative information, and impaired working memory, are sometimes viewed as mere epiphenomena of the symptoms of the emotional symptoms. However, a meta-analysis by Rock et al. (2014) shows that cognitive impairments remain detectable even in-between low-mood episodes. Accordingly, cognitive theories highlight the importance of cognitive factors in the onset and maintenance of depression (Kircanski et al., 2012).

Rumination

A concept that commonly takes a leading role cognitive theories of depression is *rumination*. Rumination is a form of repetitive negative thinking, and as such, is characterized by a repetitive, uncontrolled stream of negatively-valenced thoughts and memories that follow a common theme (Lyubomirsky et al., 2015). In particular, rumination is focused on the past or the present, revolves around themes of loss, meaning, and lack of self-worth, and involves viewing events as certain and uncontrollable (Nolen-Hoeksema et al., 2008).

Recently, Watkins and Roberts (2020) proposed the H-EX-A-GO-N model to explain the onset and perpetuation of rumination as a consequence of the interplay of some or all of five key mechanisms. According to this model, at its core, rumination is an initially functional response to perceived goal discrepancies (GO), but over time can become a mental habit (H) and triggered by low mood. As a result, rumination ultimately becomes dysfunctional, interfering with rather than stimulating efforts to reduce perceived goal discrepancies. The acquisition of mental habits by repeated occasions of rehearsal and conditioning is a central route for the development of pathological rumination (Watkins & Nolen-Hoeksema, 2014). Crucially, however, the model hypothesizes that this route from occasional, healthy rumination (or state rumination) to excessive, pathological rumination (or trait rumination) is facilitated and made more (or less) likely through the effects of the other mechanisms.

A lack of executive control (EX) facilitates the development of pathological rumination. Individuals with poor executive control may struggle to disengage from repetitive thinking about negative life events and personal concerns. This mechanism is supported by substantial neurobiological evidence. MDD is associated with a weak prefrontal cortex (PFC), so-called "hypofrontality". The PFC's inadequate control results in a hyperactive amygdala (for example, Johnstone et al., 2007; Siegle et al., 2007). As a consequence, people suffering from MDD recover more slowly from psychological stress than non-depressed individuals. Neurologically, this manifests itself as more enduring elevated post-stress cortisol levels, which over time can damage the hippocampus (Steffens et al., 2011). This neurobiological chain reaction not only results in episodic negative changes in mood but also several cognitive deficits, among them executive control (Rock et al., 2014). Executive control may be viewed as consisting of three main components: shifting, updating, and monitoring representations in working memory and inhibition of irrelevant information (Miyake et al., 2000). As Koster et al. (2011) indicate, impairment of such executive functions may contribute to rumination in at least two ways. First, problems with monitoring and manipulating information in working memory might result in the proliferation of irrelevant, negatively-valenced information in working memory, making it evermore salient and harder to disengage from. Such poor cognitive control manifests itself as

mental "stickiness" – uncontrolled and perseverative cognition (Joormann et al., 2011). Second, as executive control is needed to instigate and maintain goal-directed behavior, the weakening of it may result in difficulties in overriding ruminative habits (H) that have formed.

In addition to the effects of poor executive functions, pathological rumination is accompanied by a preferential bias for processing negatively-valenced information (Gotlib & Joormann, 2010) as well as an engagement bias that orients attention towards negatively-valenced information and a disengagement bias that hampers the ability to shift attention away from it (Beckwé & Deroost, 2016; Donaldson et al., 2007; Whitmer & Gotlib, 2013). Such biases, in turn, increase the frequency and accessibility of negative thinking, and therefore, prolong rumination (Watkins & Roberts, 2020). Stated more generally, rumination and depression appear to be linked to a universal bias for negatively-valenced information (N; Dalgleish & Watts, 1990).

The final mechanism is particularly important to understand why rumination becomes maladaptive. Pathological rumination is characterized by an abstract processing style (A). That is, a processing style focused on generalized and decontextualized mental representations that convey the causes, consequences, and meaning of a goal or an event (the "why" aspects). Unlike the more adaptive and concrete processing style of functional rumination, however, it neglects the feasibility, mechanics, and means of goals and events (the "how" aspects; Watkins, 2008). Consequently, pathological rumination fails to alleviate goal discrepancies (GO), leading to longer and more frequent periods of rumination, further promoting habit formation (H).

As rumination becomes increasingly maladaptive, it leads to several negative consequences. Rumination has been shown to exacerbate and prolong negative affective states such as sadness, anger, and depressed mood (Lavender & Watkins, 2004; Lyubomirsky et al., 1998; Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky et al., 1999; Rimes & Watkins, 2005). Similarly, it has been demonstrated that both increased negative affect and decreased positive affect predict subsequent rumination (Hjartarson et al., 2021; Moberly & Watkins, 2008). These bidirectional effects lead to a vicious cycle between affective and cognitive symptoms, with each amplifying the other (Ciesla & Roberts, 2007). In individuals prone to depression, the mutual intensification between negative thinking (including rumination itself) and negative mood produces a forceful downward spiral, eliciting severe levels of negative affect. The vicious cycle between rumination and negative mood is only one pathway by which rumination can lead to and prolong depressive episodes, however. Ruminators are at risk for social isolation, both because they might shun social interactions themselves and because they may be perceived as unwelcome company. Indeed, rumination has been linked to a number of undesirable inter-

personal characteristics, such as dependency (Spasojević & Alloy, 2001), neediness (Spasojević & Alloy, 2001), and sociotropy (Gorski & Young, 2002; Nolen-Hoeksema & Jackson, 2001). By eroding social support, rumination increases an individual's risk of slipping into depression. Additionally, rumination interferes with sleep, both lowering sleep quality and increasing sleep latency – the time it takes somebody to fall asleep (Thomsen et al., 2003). Rumination, moreover, has been linked to increased anxiety (Kirkegaard Thomsen, 2006; Nolen-Hoeksema & Watkins, 2011). However, this relationship may be indirect, with rumination affecting levels of anxiety primarily via its effects on other negative affective symptoms, such as anger and sadness (Thomsen et al., 2003).

Given the myriad associations between rumination and a variety of undesirable outcomes, it is not surprising that rumination's sphere of influence is not restricted to MDD. To the contrary, rumination is associated with an increased risk for social phobia (Mellings & Alden, 2000), PTSD (Mayou et al., 2002), and symptoms of generalized anxiety disorder (Watkins, 2009). Furthermore, it has been strongly implicated with substance abuse, alcohol abuse, and eating disorders (Aldao et al., 2010; Caselli et al., 2010). As a result, Nolen-Hoeksema and Watkins (2011) have proposed a transdiagnostic model featuring rumination as a primary risk factor for multiple disorders. The precise psychopathology that ultimately manifests is determined by specific moderators (e.g., environmental context factors or biological characteristics). Instead of improving our understanding "only" of depression, consequently, rumination may be a cornerstone in an improved conceptualization of a range of mental disorders. However, because it is such a complex construct, tying together affective and cognitive factors, a holistic understanding of rumination requires researchers to adopt a wider lens, investigating not the relationships it forms with individual symptoms, but its role in a complex network of interdependent variables. A more comprehensive and integrated understanding of rumination may have major clinical implications, increasing our ability to predict outcomes such as the course of illness, probability of relapse, and treatment response.

Network Analysis

Network analysis is an analytical framework that provides the tools necessary to explore the complex, interdependent interactions of multiple symptoms as one integrated system. It has entered the field of psychopathology as a result of the perceived shortcomings of the current diagnostic tools such as the DSM-5 (for a discussion, see Borsboom & Cramer, 2013). Instead of viewing mental disorders as being the result of an underlying root cause (analogous to Western medicine), it conceives of them as a network of interacting elements that do not need to share a root cause. Given that many mental disorders share symptoms, the established

Table 1

*Criteria for an MDD diagnosis. In addition to the **required criteria**, at least five of the **depressive symptoms** must be present for at least two weeks. Furthermore, at least one of the symptoms marked with * has to be observed.*

Depressive symptoms	Explanation
Depressive mood*	Most of the day; near-daily
Loss of interest / pleasure*	Most of the day; near-daily
Weight loss or gain	Change of >5% body weight in a month
Insomnia or hypersomnia	near-daily
Psychomotor agitation or retardation	near-daily; observable by others
Fatigue	near daily
Feelings of worthlessness/guilt	near-daily; guilt may be delusional
Lack of concentration	near daily; observable by others or merely subjective
Thoughts of death or suicide	not just fear of dying; suicidal ideation without a specific plan

Required criteria
Symptoms cause significant distress or impairment in social, occupational, or other important areas of functioning.
Episode not attributable to physiological effects of a substance or another medical condition.
Episode not better explained by, for example, schizophrenia, delusional disorder, or other psychotic disorders.
No history of manic or hypomanic episode.

boundaries between them might need to be redrawn. Network analysis might lead to greater precision in the delineation of mental disorders and a more realistic understanding of comorbidities in terms of bi-directional symptom-to-symptom connections (Cramer et al., 2010). Rumination may turn out to feature prominently not just in a network of depression but in a grander network structure explaining a number of mental disorders conventionally regarded as (entirely) separate entities.

In general, network analysis is concerned with the measurement, description, and visualization of relational structures (Furht, 2010). The end product of such an analysis is typically a graph consisting of both nodes and edges that join them. While each node represents some entity moving within the network, each edge represents a direct connection between two entities. Some connections are likely stronger than others. This is commonly accounted for by varying the thickness and/or color of edges according to – for example – the number of times two entities are linked with each other. This is referred to as the weight of an edge. Additionally, edges can be distinguished by whether they are directed or undirected, i. e. whether a connection between two nodes always runs from entity A to entity B or can go both ways. In an organization, a directed edge could indicate a well-defined chain-of-command, while in a network of symptoms it would indicate that there is a causal effect from symptom A to symptom B. These connections, then, must be explained by some underlying biological or psychological process. A connection between insomnia and fatigue, for example, can reasonably be explained by some underlying biological process. By comparison, a connection between depressed mood and feelings of worthlessness is likely of a psychological nature.

Previous research

The network perspective on psychopathology resonates with many researchers. As a result, several studies have already used network analysis to study depression. Recently, Malgaroli et al. (2021) have systematically reviewed such studies. They attempted to combine the networks created by the 23 reviewed studies into one, specifying the most recurrent centrality and edge weight indices.

Depressed mood and fatigue were the symptoms with the most robust connections across all reviewed studies. Consequently, both of these symptoms appear to be central to MDD. Neither of these findings should be surprising, given that depressed mood is the hallmark symptom of MDD and fatigue has previously been shown to be one of the main protagonists in MDD (Cramer et al., 2010) and may even be an early sign of its onset (Contreras et al., 2019). Interestingly, at least one of the two symptoms is necessary to diagnose a depressive episode with ICD-10's MDD diagnostic algorithm (World Health Organization, 1993), underscoring their pivotal roles in the disorder.

In terms of associations between variables, depressed mood and anhedonia exhibited the strongest connection. This result aligns neatly with the scientific literature, suggesting this link to be a consequence of the dopaminergic pathway and its role in initiating MDD (Stein, 2008). Feelings of worthlessness exhibit robust connections with other affective symptoms too. Malgaroli et al. (2021) consider this to be consistent with MDD's high comorbidity rates with conditions involving such experiences as guilt, particularly post-traumatic (Lee et al., 2001), obsessive-compulsive (Shafran

et al., 1996), and alcohol/substance use disorders (Treeby & Bruno, 2012). Their review, however, was limited in that it only included cross-sectional studies. As such, their network reveals nothing about the causal directions between two symptoms, limiting the ability to make inferences on the dynamics of MDD. It is also noteworthy that the studies reviewed by Malgaroli et al. (2021) – even the ones they excluded from their review – rely on self-reported measures. Of course, many symptoms are not readily measured by objective means. However, cognitive symptoms like lack of concentration would permit objective measurement, potentially increasing a network's accuracy.

Other studies used longitudinal data but with large intervals between repeated measurements (for example, two years; Savelieva et al., 2021). Important information about the short-term dynamics of a network is necessarily lost at this time scale. Even (near-)daily measurements (for example, Groen et al., 2019; Lydon-Staley et al., 2019) may be too infrequent to detect some information of clinical relevance. MDD network studies that collected measurements multiple times per day using the Experience Sampling Method (ESM) are rather rare. An exception is the study by Hoorelbeke et al. (2019) who collected self-reports six times a day for one week from 85 remitted depressed patients. They looked at key vulnerability and protective factors in a network of symptoms with a focus on indicators of emotional (dys-)regulation (mainly rumination and positive appraisal), cognitive functioning, and resilience. One of their main findings was that resilience is an important factor in obtaining stable remission from depression, predicting weaker depressive symptomatology and lower levels of rumination. Like most current network studies, the study by Hoorelbeke et al. (2019) did not include a healthy control group, limiting their ability to draw conclusions based on their results.

The present study

The present study builds on and extends previous network analysis studies of depression. Our literature review divulged two gaps that we want to address in the current study. First, rumination has not been studied in detail from a network perspective. Even though Hoorelbeke et al. (2019) included rumination as a node in their network analysis, they assessed it using only two items ("focused on feelings", "focused on problems"). In our opinion, these two items are insufficient to characterize a participant's extent of rumination as they do not reveal the perceived unpleasantness or stickiness of current thoughts, and do not clearly distinguish rumination from other forms of repetitive negative thinking, such as worry. Even in-between depressive episodes remitted patients report higher levels of repetitive negative thinking such as rumination than healthy individuals (for example, McMurrich & Johnson, 2008), and typically experience residual depressive symptoms (for example, Nierenberg et al., 2010). Our

first research question, therefore, aims at investigating these findings from a network perspective:

Q1: What is the role of rumination in a complex network of symptoms of MDD and how does it differ between remitted MDD patients and healthy controls?

In an initial step, thus, we create a temporal network of depressive symptoms for remitted MDD patients with a focus on rumination using the experience sampling method (ESM; Kahneman et al., 2004). To improve upon a common limitation of current network studies, our study includes a control group of healthy individuals. The second gap we identified concerns the effects of common interventions on rumination and depression from a network perspective. Network studies that investigate the effects of common interventions with ESM data are rare. And so far, no study has investigated the effects of mindfulness and positive fantasizing from a network perspective. Both interventions have proven successful at alleviating depressive symptoms in more conventional studies. However, the combination of ESM and network analysis provides an especially potent lens for exploring how the effects of an intervention unfold. Consequently, the second research question may be stated as follows:

Q2: What are the effects of mindfulness and positive fantasizing on the network of symptoms of MDD in general and rumination in particular?

While mindfulness focuses on accepting and letting go of negative thoughts, positive fantasizing focuses more on stimulating positive thoughts. Mindfulness-based therapy has received ample attention and its efficacy in mitigating depressive symptoms has been corroborated by multiple meta-analyses (see for example, Hofmann et al., 2010). It has been suggested that mindfulness exerts its mitigating effects on depressive symptoms by reducing dysfuntional emotion regulation strategies, such as rumination (Guendelman et al., 2017). Returning to Watkins and Roberts (2020)'s HEXAGON model of rumination, we can identify a likely route this effect might take. As pointed out earlier, rumination is suggested to be a maladaptive habit (H), the formation of which is aided by poor executive control (EX). Teper et al. (2013) posit that the awareness and nonjudgmental acceptance cultivated by mindfulness training promote executive control because they heighten the meditator's sensitivity to affective cues in their experiential field. This greater awareness of subtle changes in affective states fosters executive control because it improves the meditator's ability to respond and intervene when such changes are detected. And indeed, a meta-review by Cásedas et al. (2020) supports moderate effects of mindfulness on executive control. This in turn enhances functional emotion regulation. As a result of the improved emotion regulation, the reactivity bias (N)

associated with rumination may decrease (see for example, Düsing et al., 2021). Given that functional emotion regulation is associated with a concrete, rather than an abstract (A), processing style that should ultimately lead to a better alleviation of goal discrepancies (GO) over time, mindfulness training can reasonably be expected to impact – directly or indirectly – all key mechanisms of rumination as postulated by the HEXAGON model.

Positive fantasizing is a main constituent of Preventive Cognitive Therapy – a therapeutic approach that has been shown to prevent the recurrence of depressive episodes and mitigate depressive symptoms (Bockting et al., 2009). In contrast to mindfulness, positive fantasizing may exert its effects by increasing positive affect (van Tol et al, 2021; PREPRINT). The increase in positive affect might lead to a decrease in negative affect and lower mood reactivity – changes in mood following mood provocation – in general (van Rijsbergen et al., 2013). Therefore, positive fantasizing recruits a slightly different pathway than mindfulness in combating depression, potentially operating more directly on positive and negative affect. However, the positive fantasizing intervention involves identifying dysfunctional attitudes and schemas – and challenging these using positive affect and fantasy techniques – and therefore should also impact repetitive negative thinking directly.

To assess whether mindfulness and positive fantasizing really exert their effects via different routes, we utilize a sustained attention to response task (SART; Robertson et al., 1997, collected once per day). As the name suggests, the SART was devised as a measure of sustained attention. However, given that it requires participants to inhibit automatic responses, it has been suggested to be a better indicator of executive control than sustained attention (Head & Helton, 2014; Helton et al., 2009). As a consequence, it may help us to determine whether mindfulness training truly improves executive control and whether it does so more than positive fantasizing. At the same time, including the SART gives us the opportunity to investigate how this objective measure of executive control relates to momentary subjective measures of related variables and rumination – which to our knowledge has not yet been studied.

Network analysis, as implemented today, is mainly an exploratory tool (Borsboom et al., 2021). Strong conclusions should not be drawn on the mere basis of network analysis, but need to be supported by other statistical analyses and substantiated theoretical reasoning (Bringmann et al., 2022). For that reason, we designed a tentative "hypothesis network" for remitted MDD patients prior to having received an intervention based on the evidence reviewed in previous sections. Figure 1 shows the hypothesis network. It was reduced to some of the (likely) most central nodes. Moreover, some nodes that may be analyzed separately were placed into categories (e.g., positive affect, negative affect). The
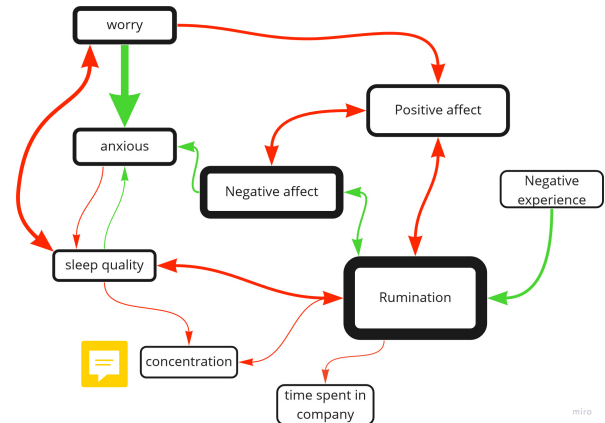

Figure 1
A simplified network of some of the main hypothesized effects. Thicker borders indicate greater centrality. Red arrows indicate a diminishing influence, green arrows an increasing one. The thickness of the arrow gives an indication of the expected effect size.

network indicates that rumination (or a combination of features akin to rumination) is expected to feature prominently in the network. Negative affect, and to a lesser degree positive affect (or at least some of their constituent parts), are also hypothesized to be highly central to the network. Strong bi-directional effects are expected between rumination and sleep as well as positive and negative affect. Worry, a form of repetitive negative thinking that is directed towards future concerns, is likely to be highly predictive of anxiety.

For healthy individuals we expect negative affect and especially rumination to be less central and many of the associations to be significantly weaker, possibly to the extent that they do not feature in the network at all, resulting in a sparser network (reduced global strength). Similarly, after having gone through an intervention, the networks of remitted MDD individuals might resemble that of healthy individuals more (though they will still be significantly different). Mindfulness may impact the network by increasing executive control, and consequently, its effects may start from the rumination node. By contrast, positive fantasizing might exert its influence primarily by increasing positive affect and decreasing negative affect directly, which may subsequently lead to reduced levels of rumination.

Methodology

ESM refers to the collection of self-reports regarding a participant's ongoing experience (Kahneman et al., 2004). Typically, participants are asked to report on their internal experiences and the context in which they occurred at random or set intervals while going about their regular lives (Smallwood & Schooler, 2006). While ESM is used to collect self-reports, i.e., subjective measures, SART is a method for ob-

taining objective cognitive data. Developed by Robertson et al. (1997), SART is a go/no-go paradigm that measures, as the name suggests, the ability to sustain attention. The SART involves pressing a key in response to frequently presented non-targets (Go trials) and withholding the response to infrequently presented targets (No-Go trials). Because it places very low demands on controlled processes, the SART lends itself to mind-wandering, a form of perseverative, repetitive thinking. And indeed, participants' error rates on the task are predicted by questionnaires on mind-wandering (Smallwood et al., 2006).

Following Borsboom et al. (2021), we can regard network analysis as consisting of three main phases: network structure estimation, network description, and network stability analysis (see Figure 2). The initial phase, network structure estimation, comprises two steps. First, one has to select the nodes to feature in the network. This step is primarily driven by theoretical considerations, rather than methodological ones. Second, using statistical tools, the conditional associations between nodes are estimated to determine the most important edges in the network. Towards this goal, several statistical methods can be employed. Model selection methods based on fit indices, such as regularized estimation procedures (Epskamp & Fried, 2018), null hypothesis testing procedures, and cross-validation approaches are commonly used in psychological studies. These approaches typically use conditional associations to define a network structure underlying a set of variables (Robinaugh et al., 2020). Two variables are said to be conditionally associated if they are probabilistically dependent, conditional on the remaining variables in the set. The strength of this conditional association determines the edge weight between the two variables. If the association between two variables vanishes when the other variables are controlled for, the variables, i.e., their corresponding nodes, will be disconnected in the visualized network structure.

The next phase, network description, involves describing the topology of the estimated network. We differentiate between global and local network topology. Globally, the most important topological feature of a network is the density of its connections. If the number of edges relative to the number of nodes is low, the network is said to be sparse. Conversely, a high number of edges as compared to nodes indicates a dense network. This distinction is critical because some estimation procedures are more suitable for sparse networks, some for dense ones. Another way to think about a network's density is sometimes referred to as global strength. In psychopathology networks, it's a measure of total symptom connectivity, or edge weights, and is hypothesized to be a central factor in an individual's vulnerability profile. Cramer et al. (2016), for example, simulated MDD networks, varying the strengths of connections between symptoms. They found that more strongly connected networks are more likely to be tipped into

a state of depression as compared to more loosely connected networks. This is in line with findings from other areas of science that show that strongly connected dynamic systems are more easily tipped from one state into another (e.g., Chen et al., 2012) and with the observation that successful therapeutic interventions are sometimes targeted at weakening symptom-to-symptom relations (such as exposure therapy). Global strength can be measured, for example, via the Network Comparison Test (NFT; Van Borkulo et al., 2017). The most commonly investigated local feature of a network's topology is node centrality. It measures how important a node is. "Importance" within a network can be defined in different ways, and consequently, there are many measures for this property (Das et al., 2018). The most basic of these is referred to as degree, which measures how many connections a node possesses. In psychological research, strength – the sum of all absolute edge weights a node is directly connected to – is in high demand for specifying a node's centrality as it is generally more stable than other common metrics (Bringmann et al., 2019). Other local features of interest include clustering of nodes into subcommunities, providing insight into potentially unobserved causes and the dimensionality of the system, and shortest paths between nodes, possibly yielding insights into the most predictive pathways within the network. Frequently, network analysis involves the estimation of multiple networks, using multiple statistical methods with different advantages, or comparing groups. Hence, the comparison of different networks is another step in the network description phase.

The purpose of the final phase, network stability analysis, is to ensure reproducibility (obtaining the same conclusions from the same data) and increasing replicability (obtaining the same conclusions from new data) of the estimated networks – topics that have received ample attention in the psychometric network literature recently (see for example, Bringmann et al., 2022). The main targets of statistical tools for robustness analyses in networks are individual edge weight estimates, differences between edges in a network, and topological metrics, such as node centrality. In determining the robustness of edge weight estimates, measures of sensitivity to sampling errors, such as confidence intervals, credibility intervals, and bootstrapped intervals are indicated. The degree to which such intervals overlap for the relevant coefficients gives insights into the robustness of differences between edge weights. To investigate the robustness of other network properties, such as node centrality, various approaches have been devised, including approaches based on bootstrapping (Epskamp et al., 2018) and Bayesian statistics (Williams & Mulder, 2020). The final product generated with these steps is then interpreted. To make substantive inferences, however, it is necessary to combine the output with general methodological considerations and domain-specific knowledge (Borsboom et al., 2021).
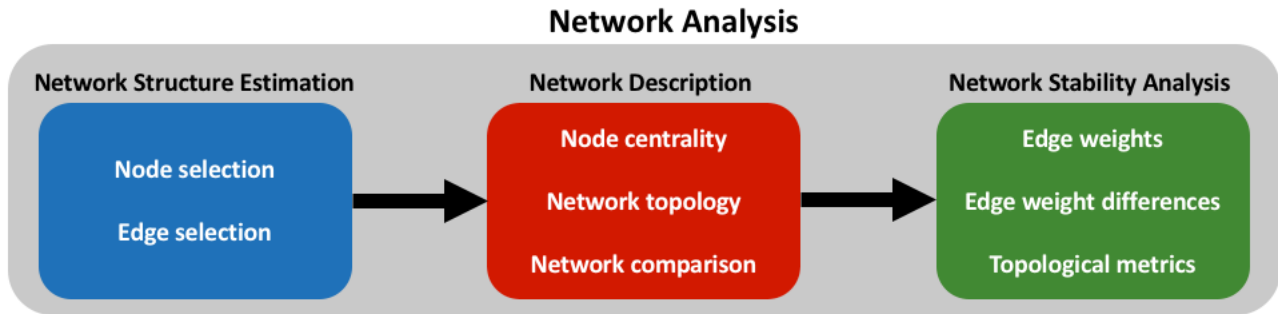
Network Analysis

Figure 2

Schematic representation of the workflow used in network analysis. Adapted from Borsboom et al. (2021).

Naturally, the interpretation of a network is dependent on the data used to inform its structure. Generally speaking, we can either use cross-sectional or longitudinal data. In network studies within the fields of psychology and psychopathology, cross-sectional data is most commonly used (Borsboom et al., 2021) – resulting in *contemporaneous networks*. Estimating contemporaneous networks is simpler than estimating networks from longitudinal data because for cross-sectional data independence of measurements can typically be assumed. Since independence is given, the application of population-sample logic is warranted, lowering the requirements for an estimation model. In time-series data, two kinds of interdependence are introduced that complicate the estimation of *temporal networks*. First, data collected on two (or more) subsequent measurement points may be correlated (if somebody is happy right now, there is a high chance they are still happy in two hours). Second, responses from one person may correlate with one another more strongly in general (somebody of a more happy nature will on average report more happiness). The obvious downside of contemporaneous networks, however, is that they contain no information about the direction of effects between variables. Additionally, some researchers have voiced concerns regarding the replicability of contemporaneous networks (for example, Fried & Cramer, 2017), and have argued that they cannot necessarily be assumed to generalize to the level of the individual (for example, Bringmann & Eronen, 2018). Overall, temporal networks may be more advantageous than contemporaneous networks. Although the violation of independence assumptions places greater demands on models, the additional insights that can be extracted from it may well be worth the effort. Vector autoregressive models (see for example, Haslbeck & Waldorp, 2015) have been devised to deal with temporal data. When using such models to estimate a temporal network, one ends up with a structure of associations that remain after taking the temporal effects into account (Borsboom et al., 2021). Therefore, when using time series data, one receives two network structures, one depicting lagged associations – or temporal effects – and a kind of contemporaneous network that depicts the associations unac-counted for by the temporal effects. The temporal network, generally, can be interpreted in terms of carry-over effects at the timescale defined by the time between repeated measurements. The temporal ordering may allow for a causal interpretation. It is important to note, however, that causal interpretations are not straightforward (Fried & Cramer, 2017). The contemporaneous network will capture effects that occur at timescales different from those defined by the spacing between repeated measurements.

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