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\title{The effects of mindfulness and fantasizing on rumination and MDD: A network perspective}

\shorttitle{Master Thesis - Kaiser}

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\abstract{}

\keywords{Major Depressive Disorder, rumination, network analysis, experience sampling, mindfulness}

\begin{document}

\maketitle

\section{Introduction}

About one in ten people currently suffer from Major Depressive Disorder \parencites[MDD;][]{kessler2013epidemiology, tolentino2018dsm}, and more than one in five will be affected by this debilitating mood disorder at some stage in their lives \parencite{hasin2018epidemiology}. 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 \parencites[][]{hardeveld2010prevalence, mueller1999recurrence}. These numbers are not surprising to anyone that has engaged with the abundant literature on the topic. MDD has long been a subject of investigation for many scientific fields, and consequently, a wealth of knowledge about its detrimental effects, risk factors, moderators and prevalence has been generated. However, while we have discovered many pieces of the puzzle that is MDD, we have yet to put them together into one coherent picture. That is, we know much \textit{about} depression, but we lack a straightforward answer to the truly fundamental question of what depression really is \parencite{borsboom2013network}.

The lack of a satisfactory answer to this fundamental question is reflected in the diagnosis criteria of the prevailing diagnostic tool, the \textit{Diagnostic and Statistical Manual of Mental Disorders} \parencites[5th ed.; DSM-5;][]{american2013diagnostic}. The process of diagnosing depression is based on a checklist of symptoms as well as a number of additional criteria and exceptions (see Table~\ref{tab:MDD}). There are a number of issues with this approach. First, as \textcite{fried2017moving} point out, DSM-5 diagnoses like MDD are highly heterogeneous. For example, \textcite{fried2015depression} identified 1,030 unique depression symptom profiles in 3,703 depressed individuals. In addition, many mental disorders, as defined by the DSM-5, exhibit high comorbidity rates. For example, close to 75\% of depressed individuals meet the criteria for at least one more mental disorder, most often an anxiety disorder (Kessler et al., 2003). Regarding MDD in particular, the DSM-5 heavily focuses on affective symptoms, neglecting some of the cognitive symptoms that are routinely observed in depressed patients such as poor executive control, attentional biases towards negative information, and impaired working memory. These cognitive deficits are sometimes viewed as mere epiphenomena of the symptoms of low mood. However, a meta analysis by \textcite{rock2014cognitive} shows that cognitive impairments remain detectable even in-between low-mood episodes. In sum, all of this implies that we have yet to find better ways to make sense of depression and how its affective and cognitive puzzle pieces fit together.

%Such a checklist approach is necessarily reliant on subjective judgements, and therefore, prone to errors.

%The persistence of these cognitive deficits indicates that a complete picture of MDD requires an understanding of how the affective and cognitive puzzle pieces fit together.

%generate more doubt about DSM-5? --> Moving Forward: Challenges and Directions for Psychopathological Network Theory and Methodology (p.6)

\begin{table\*}[]

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

\label{tab:MDD}

\begin{tabular}{{l}{l}} \toprule

\textbf{Depressive symptoms} & \textbf{Explanation} \\ \midrule

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 specific plan \\ \midrule

\multicolumn{2}{l}{\textbf{Required criteria}} \\ \midrule

\multicolumn{2}{l}{Symptoms cause significant distress or impairment in social, occupational, or other important areas of functioning.} \\

\multicolumn{2}{l}{Episode not attributable to physiological effects of a substance or another medical condition.} \\

\multicolumn{2}{l}{Episode not better explained by, for example, schizophrenia, delusional disorder, or other psychotic disorders.} \\

\multicolumn{2}{l}{No history of manic or hypomanic episode.} \\

\bottomrule

\end{tabular}

\end{table\*}

\subsection{Rumination}

A concept that may be at the heart of a more comprehensive understanding of the interplay of affective and cognitive factors in depressed individuals is rumination. Rumination is a form of perseverative cognition and is characterized by a repetitive, uncontrolled stream of negatively valenced thoughts and memories that follow a common theme \parencite{lyubomirsky2015thinking}. To clarify what rumination is, it may be helpful to contrast it with a related concept {--} worry. Like rumination, worry is a form of perseverative cognition. And indeed, they are highly correlated \parencites{watkins2004appraisals, watkins2005comparisons}, and share many features. For example, both are repetitive and self-focused \parencite{borkovec2004avoidance}, abstract and overgeneral \parencites{watkins2001rumination, watkins2000decentring}, associated with cognitive inflexibility \parencite{nolen1999thanks}, difficulties in concentration and attention, poor problem solving, and inadequate solution implementation \parencites{lyubomirsky1999ruminators, ward2003can, watkins2002rumination, watkins2005distinct}. And crucially, both are associated with depression and anxiety \parencites{kocovski2005ruminative, muris2005mediating}. Nevertheless, they differ from one another in three key respects. First, whereas rumination focuses on the past and the present, worry is oriented towards the future (temporal orientation). Second, while rumination revolves around themes of loss, meaning, and lack of self-worth, worry is concerned with anticipated threats (topic orientation). Third, the conscious motive behind rumination is to gain insights, while the conscious motive underlying worry is to anticipate and prepare for future threats (conscious motive). Finally, rumination involves viewing events as certain and uncontrollable, whereas worry involves viewing events as uncertain and potentially controllable (degree of certainty) \parencite{nolen2008rethinking}.

Recently, \textcite{watkins2020reflecting} proposed the H-EX-A-GO-N model to explain the onset and perpetuation of pathological 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 \parencite{watkins2014habit}. 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 in executive control (EX) may further facilitate the development of trait 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 \parencites[for example,][]{siegle2007increased,johnstone2007failure}. 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 \parencite{steffens2011change}. This neurobiological chain reaction not only results in episodic negative changes in mood, but also a number of cognitive deficits, among them executive control \textcite{rock2014cognitive}.

In their review, \textcite{watkins2020reflecting} come to the conclusion that the evidence by and large supports a causal contribution from impaired executive control to depressive rumination, especially in the context of stress. \textcite{koster2011understanding} cogently summarize the evidence for and explain the link between trait rumination and poor executive control in their impaired disengagement hypothesis. Executive control may be viewed as consisting of three main components: shifting, updating and monitoring representations in working memory and inhibition of irrelevant information \parencite{miyake2000unity}. As \textcite{koster2011understanding} 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 \parencite{joormann2011sticky}. 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 \parencite[N;][]{gotlib2010cognition} 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 \parencites{whitmer2013attentional, beckwe2016attentional, donaldson2007rumination}. Such biases, in turn, increase the frequency and accessibility of negative thinking, and therefore, prolong rumination \parencite{watkins2020reflecting}. Stated more generally, rumination and depression appear to be linked to a universal bias for negatively-valenced information \parencite{dalgleish1990biases}.

The final mechanism is particularly important to understand why pathological rumination becomes largely 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 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 \parencite[the "how" aspects;][]{watkins2008constructive}. Consequently, pathological rumination fails to alleviate goal discrepancies (GO), leading to longer and more frequent periods of rumination, further promoting habit formation (H).

In combination, the mechanisms identified by the {H-EX-A-GO-N} model (habit formation, executive control, abstract processing style, goal discrepancies, negativity bias) give some indication of the complex interaction of affect and cognition summarized under the term (pathological) rumination. Given this complexity, 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 \parencite{mellings2000cognitive}, PTSD \parencite{mayou2002posttraumatic}, and symptoms of generalized anxiety disorder \parencite{watkins2009depressive}. Furthermore, it has been strongly implicated with substance abuse, alcohol abuse, and eating disorders \parencites{aldao2010emotion, caselli2010rumination}. As a result, \textcite{nolen2011heuristic} have proposed a transdiagnostic model featuring rumination as a main risk factor for a number of disorders (i.e., rumination shows multifinality). The precise psychopathology that ultimately manifests is determined by specific moderators (e.g., environmental context factors or biological characteristics). Instead of "just" improving our understanding 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 comprising 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.

%rumination may be a central guide in our redrawing of the map of mental disorders

%put MINDCOG here? -->

\subsection{MINDCOG}

%https://www.rug.nl/news/2020/06/umcg-start-nieuwe-studie-naar-piekeren-bij-gevoeligheid-voor-depressie

%The research participants will practice both techniques, i.e. mindfulness and positive fantasizing. This will make it possible to investigate which technique works best for whom. This is also why the individual characteristics of the participants will be mapped out. On the basis of this information, the researchers hope to learn which personal characteristics can predict the technique that works best for which patients.

The current study is part of an ongoing project (MINDCOG) at the University Medical Center Groningen (UMCG) and the University of Groningen (RUG) investigates rumination, and repetitive negative thinking more generally, and how it can be influenced positively in individuals at risk for depressive relapse. In particular, it investigates the effects of mindfulness and fantasizing, two interventions that have proven successful at alleviating depressive symptoms. However, they might exert their effects on MDD-related symptoms through different mechanisms. Mindfulness focuses on accepting thoughts and learning to let go of them. 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). Fantasizing, by contrast, focuses more on stimulating positive thoughts. It is a main constituent of Preventive Cognitive Therapy – a therapeutic approach that has been shown to successfully prevent the recurrence of depressive episodes and mitigate depressive symptoms (Bockting et al., 2009).

For each cohort of participants, the experiment lasts at least eight weeks. Throughout the experiment cognitive, behavioral, and (neuro-)physiological measures are gathered from remitted MDD patients and healthy controls. The experiment is split into five periods: two pre-intervention periods, two peri-intervention periods, and one wash-out period. The experiment starts with a pre-intervention period of one week. Thereafter, in the first peri-intervention period, participants receive one of the two interventions at random. This period also lasts one week, with participants receiving a two-hour-long professional training pertaining to their respective interventions on the first day of the period. For the remainder of the period, participants are asked to perform a 10-minute home exercise of the same intervention daily. This is followed by a wash-out period of at least one month. Subsequently, participants go through another round of pre- and peri-intervention periods, this time receiving the intervention they had not yet received.

Throughout the entire duration of the experiment a range of data is collected to guarantee a comprehensive picture. Experience sampling method (ESM), a sustained attention to response task (SART; Robertson et al., 1997), and actigraphy data are recorded daily. A 24-hour ECG and EEG measures are taken on the last day of each period (excluding the wash-out period). In addition, participants are asked to fill in several questionnaires regarding rumination, depression, and mindfulness among other things.

The primary objectives of MINDCOG are (1) to test whether psychological and psychophysiological indices of rumination are differentially affected by fantasizing vs. mindfulness in remitted MDD patients (at risk for depressive relapse) and healthy controls, and (2) to identify individual characteristics that may predict the effectiveness of the interventions in reducing rumination.

The current study combines the ESM and the SART data. ESM refers to the collection of self-reports regarding a participant’s ongoing experience \parencite{kahneman2004survey}. 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 \parencite{smallwood2006restless}. While ESM is used to collect self-reports, i.e., subjective measures, SART is method for obtaining objective cognitive data. Developed by \textcite{robertson1997oops}, 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 have been shown to be predicted by questionnaires pertaining to mind wandering \parencite{smallwood2006encoding}.

The SART lends itself well to mind-wandering due to low attentional demands and a highly repetitive response. Errors on No-Go trials of the SART (i.e., responding to the target when one should withhold the response) are typically preceded by relatively fast Go trials, suggesting that the participant is simply responding automatically/rhythmically to the trial stimulus. SART errors have been shown to predict and be predicted by questionnaires related to absentmindedness and mind-wandering (Manly et al., 1999; Robertson et al., 1997; Smallwood, Davies et al., 2004; Smallwood & Schooler, 2006). Further, response latencies are associated with errors in SART, and the probability of errors occurring is related to blocks of trials in which mind-wandering is reported, but not by blocks of trials in which subjects report being on task (Smallwood et al., 2004). In addition to pre-error speeding, there is also post-error slowing which may reflect several task-related cognitive actions, such as reengagement of the task after the mind has wandered (Cheyne et al., 2009; Rabbit, 1966), a return of attention to the external environment (Smallwood et al., 2006), or even negative mood (Smallwood et al., 2009 ). In keeping with the spirit of MINDCOG, the current study combines the subjective and objective measures obtained via ESM and SART and analyses them holistically.

\subsection{Network Analysis}

%use Borsboom et al., 2021 as structure guide for this section!!!

%Rumination is a core mechanism of MDD and can explain the development and maintenance of negative affective states. However, rumination is not bound by our definitions of mental disorders. Therefore, to better understand rumination, and by extension depression, we must look at the bigger picture, beyond the margins of MDD as defined by the DSM-5. Instead, we need to investigate it in a more holistic manner, taking into account not only affective symptoms (of MDD) but also cognitive ones.

%As we shall see in section (\nameref{section:ruminationNetwork}), substantial evidence supports rumination's link to many undesirable outcomes. However, so far this body of evidence largely relies on studies looking at individual relationships or small sets of relationships.

Network analysis is an analytical framework that allows us to study the complex, interdependent interactions of multiple symptoms at the same time. It has entered the field of psychopathology because of the aforementioned shortcomings of the prevalent diagnostic approach \parencite{borsboom2013network}. 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 boundaries between them might need to be redrawn. In fact, the exorbitant rates of comorbidities in psychopathology \parencites[e.g.,][]{andrews2002deconstructing, kessler2005prevalence} are likely explained in the first place by unfortunate features of the prevalent diagnostic criteria \parencite{maj2005psychiatric}. Network analysis might lead to greater precision in the delineation of mental disorders and illuminate the pathways connecting them. Rumination may turn out to be a central feature of such a network structure.

More generally, network analysis is concerned with the measurement, description, and visualization of relational structures \parencite{furht2010handbook}. The end product of such an analysis is typically a graph consisting of both nodes and the 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.

%Add adapted (simplified) verison of borsboom (2021) figure 2

Following \textcite{borsboom2021network}, we can regard network analysis as consisting of three main phases: network structure estimation, network description, and network stability analysis. 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, a number of statistical methods can be employed. Model selection methods based on fit indices, such as regularized estimation procedures \parencite{epskamp2018tutorial}, 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 \parencite{robinaugh2020network}. 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-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 a number of ways, and consequently, there are many measures for this property \parencite{das2018study}. The most basic of these is referred to as degree, which measures how many connections a node possesses. In psychological research, strength is in high demand for specifying a node’s centrality as it is generally more stable than other common metrics \parencite{bringmann2019centrality}. In a graph, centrality is often depicted by varying the size and/or the color of a node. 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 comparisons of these networks is another step in the network description phase.

%Frequently, network analysis also aims at extracting sub-communities – i. e., sets of highly connected nodes – from a network \parencite{blondel2018fast}. A range of algorithms can be employed towards that goal (e.g., the spinglass algorithm).

The purpose of the final phase, network stability analysis, is to ensure reproducibility of the estimated networks. 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 case-dropping bootstrap, in which fewer and fewer cases are sampled from the original data set to obtain subsamples, may be used. Various approaches have been devised to assess such robustness measures, including approaches based on bootstrapping \parencite{epskamp2018estimating} and Bayesian statistics \parencite{williams2020bayesian}. 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 \parencite{borsboom2021network}.

%Given that such data involves taking measurements only at one point in time, it is generally speaking easier to collect.

Naturally, the interpretation of a network is dependant on the data used to inform its structure. Generally speaking, we can either use cross-sectional or time series data. In network studies within the fields of psychology and psychopathology, cross-sectional data is most commonly used \parencite{borsboom2021network} {--} resulting in \textit{contemporaneous networks}. Estimating contemporaneous networks is simpler than estimating networks from time series data because for cross-sectional data independence of measurements can typically be assumed. Because 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 \textit{temporal networks}. First, data collected on two (or more) subsequent measurement points may be correlated (if somebody is happy right now, they have a high chance of still being happy in two hours). Second, responses from one person may correlate with one another more strongly in general (as 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 researcher have voiced concerns regarding the replicability of contemporaneous networks \parencite[for example,][]{fried2017moving}, and have argued that they cannot necessarily be assumed to generalize to the level of the individual \parencite[for example,][]{bringmann2018don}.

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 it. Vector autoregressive models \parencite[see for example,][]{haslbeck2015mgm} have been devised for the purpose of dealing 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 \parencite{borsboom2021network}. Therefore, when using time series data, one receives two network structures, one depicting lagged associations {--} or temporal effects {--} and a sort of contemporaneous network that depicts the associations unaccounted 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 causal interpretation. However, such interpretations from temporal networks are not straightforward \parencite{fried2017moving}. The contemporaneous network will capture effects that occur at timescales different to those defined by the spacing between repeated measurements. Clearly, time series data contains more information about the associations of variables than cross-sectional data.

%In general, we can distinguish networks based on the type of data they can represent. Contemporaneous networks utilize cross-sectional data, i.e., measurements from multiple participants at a single point in time.

%write about contemporaneous networks, their limitations --> temporal network's adavantages --> ESM data usage will come up naturally --> fairly brief ESM info --> then interventions? Or better to go into MINDCOG and deal with interventions in that context

%say something about inference / network interpretations (again see Borsboom, 2021), including limitations?

%Write about limitations here or only later on?

%Exploratory nature + issues of replicability / reproducibility --> important to have and state hypotheses about network!!!

\subsection{Theoretical rumination network}

\label{section:ruminationNetwork}

The associated high degree of certainty indicates that rumination does not lead to attempts to change the circumstances one’s thoughts are circling around. To the contrary, rumination is inherently focused on perceived problems and the negative feelings associated with them, thereby leading to a host of undesirable consequences. Most importantly, rumination exacerbates and prolongs negative affective states such as sadness, anger, and depressed mood \parencites{lavender2004rumination, lyubomirsky1998effects, lyubomirsky1995effects, lyubomirsky1999ruminators, rimes2005effects}. Similarly, it has been shown that both increased negative affect and decreased positive affect predict subsequent rumination \parencites{hjartarson2021daily, moberly2008ruminative}. These bidirectional effects can lead to a vicious cycle between affective and cognitive symptoms, with each amplifying the other \parencite{ciesla2007rumination}. 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.

%Rumination focuses attention on the discrepancy between a desired state and the actual situation, magnifying the discrepancy.

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 interpersonal characteristics, such as dependency \parencite{spasojevic2001rumination}, neediness \parencite{spasojevic2001rumination}, and sociotropy \parencites{gorski2002sociotropy, nolen2001mediators}. By eroding social support, rumination increases an individual's risk for slipping into depression. Additionally, rumination interferes with sleep, both lowering sleep quality and increasing sleep latency {--} the time it takes somebody to fall asleep \parencite{thomsen2003rumination}.

Although the hallmark symptom of generalized anxiety disorder (GAD) is worry, rumination has also been linked to increased anxiety \parencites{kirkegaard2006association, nolen2011heuristic}. Even though this relationship may be indirect, with rumination affecting levels of anxiety only via its effects on other negative affective symptoms, such as anger and sadness \parencite{thomsen2003rumination},

\subsection{Interventions}

\subsubsection{Mindfulness}

\subsubsection{Positive Fantasizing}

%Not symptomwise effects but global

%In addition, current treatments for MDD have at best demonstrated a small positive effect on cognitive deficits \parencites{rock2014cognitive, bernhardt2019longitudinal}, highlighting the need for supplementary intervention.

%This neurobiological chain reaction not only results in episodic negative changes in mood, but also in a number of cognitive deficits in executive control, memory and attention .

% maybe write about how rumination relates to other psychopathologies. Then you can talk about how rumination is so central to many pathologies and yet finds no mention in the DSM-5 --> this is just one point that has led many to question the assumptions underlying the DSM-5 diagnosis criteria. (A Heuristic for Developing Transdiagnostic Models of Psychopathology : Explaining Multifinality and Divergent Trajectories, Nolen-Hoeksema)

% ######################################################################

\section{Methods}

\printbibliography

\newpage

\appendix

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%% This work is "maintained" (as per LPPL maintenance status) by

%% Daniel A. Weiss.

%%

%% This work consists of the file apa7.dtx

%% and the derived files apa7.ins,

%% apa7.cls,

%% apa7.pdf,

%% README,

%% APA7american.txt,

%% APA7british.txt,

%% APA7dutch.txt,

%% APA7english.txt,

%% APA7german.txt,

%% APA7ngerman.txt,

%% APA7greek.txt,

%% APA7czech.txt,

%% APA7turkish.txt,

%% APA7endfloat.cfg,

%% Figure1.pdf,

%% shortsample.tex,

%% longsample.tex, and

%% bibliography.bib.

%%

%%

%% End of file `./samples/longsample.tex'.