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Jumping back onto the giants' shoulders: Why emotional memory should be considered in a network perspective of psychopathology

Inga Marie Freund, Arnoud Arntz, Renée M. Visser [1], Merel Kindt [*,1]

Department of Psychology, University of Amsterdam, Amsterdam, the Netherlands

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ABSTRACT

Clinical psychology finds itself with a paradox: On the one hand, there is abundant empirical evidence showing that aversive experiences increase the risk for psychopathology. In fact, a learning and memory framework forms the foundation of numerous psychological theories and treatments. For example, various CBT approaches aim to target maladaptive emotional memories (e.g., schemas or cognitions) that are deemed to lie at the core of mental health conditions. On the other hand, a new approach – the network theory – is gaining ground, which ignores underlying causes for mental disorders and instead dictates a focus on symptoms and their causal interactions. While radical shifts are sometimes necessary in science, we argue why completely neglecting common causes, such as emotional memory, is not justified. We critically discuss the strengths and limitations of the network approach: While its transdiagnostic nature and recognition of symptom interactions have the potential to invigorate the field, the framework is merely descriptive, its concepts not well defined, and its clinical utility still to be established. To move forward, we propose an incorporation of latent constructs into the network model, starting with clearer definitions and operationalisations of concepts in both network and latent variable models.

1. Introduction

Over the past century, our understanding of psychopathology has vastly expanded, and at times radically changed. Different trends, from psychoanalysis, to behaviourism, biological psychiatry, behavioural neuroscience, and cognitive psychology, have had significant consequences: They set the clinical research agenda, and defined how psychopathology is studied, explained, and treated. Recently, the network approach to psychopathology has been gaining momentum (Robinaugh et al., 2020), which may indicate that we are currently at the brink of an important transition in the way we think about mental disorders. This new trend proposes a radical move away from explaining disorders in terms of latent constructs and instead brings symptoms to the foreground, positing that "symptoms are causally active ingredients of the mental disorders themselves" (Borsboom & Cramer, 2013, p. 96). With this new perspective, mental disorders are conceptualised as dynamic networks of interacting symptoms, *without* an underlying latent entity. Thus, the network approach neglects latent constructs, such as emotional memory.

As clinical scientists and memory researchers, the rejection of common causes – such as emotional memory - surprises us, given their central role in established clinical theories, in which such constructs are assumed to lie at the root of a broad range of mental health conditions (e.g., Beck, 1976; Brewin et al., 2010; Hackmann & Holmes, 2004; Kindt, 2014). Throughout this article, we use emotional memory as an example of a potential common cause to provide substance to our line of arguments. We would like to stress that apart from emotional memory, our considerations may be applicable to other constructs that may play a role in the aetiology and maintenance of mental disorders as well.

Decades of experimental work in humans and non-human animals corroborate the notion of a causal link between adverse experiences and the development of psychological symptoms, and more generally, show that emotional events shape one's beliefs and predictions. For example, childhood adversities, including emotional abuse and emotional neglect, are strongly related to an increased vulnerability for developing psychopathology (C. Clark et al., 2010; Hovens et al., 2010; Nanni et al., 2012; Nelson et al., 2017; Spinhoven et al., 2010). Not only physical or sexual abuse, but other aversive experiences, like being bullied during childhood and adolescence (Copeland et al., 2013; Pirkola et al., 2005; Wolke et al., 2013) or parental divorce, are associated with mental

* Corresponding author. Department of Clinical Psychology, University of Amsterdam, Nieuwe Achtergracht 129B, 1018WS, Amsterdam, the Netherlands.
E-mail address: m.kindt@uva.nl (M. Kindt).
[1] equal contribution.

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health issues (Afifi et al., 2009; Huurre et al., 2006; Marcussen et al., 2021; Paananen et al., 2013; Størksen et al., 2005).

The representation of these adverse experiences is what we refer to as emotional memory: a psychological construct that allegedly captures the link between adverse experiences and later manifestations of various mental disorders. For the sake of the argument and pending a more precise conceptualisation, we use *emotional memory* as an umbrella term for any construct that refers to a mental representation (see Arntz, 2020 for review) of one, or a series of experiences (imagined or real), that carries an affective load, and that shapes the way one perceives and makes predictions about the self, others, and the world. Emotional memory is not restricted to declarative autobiographical memories of past events but can also extend to the imagination of future events (Schacter, 2019), as well as non-declarative memories resulting from implicit learning (e.g., conditioned fear responses). Activation of emotional memories involves action tendencies, such as approach or avoidance behaviour, subjective feelings, physiological reactions, and certain cognitions. Implicit memories can be triggered in absence of awareness and may constitute habits and learned behaviours that can arise without knowing the origin. In short, emotional memory is a broad concept that may play a role in a myriad of disorders.

In response to the emerging trend of the network approach in clinical science, we critically discuss aspects of the current network perspective that we view as potential shortcomings and question whether latent constructs should be entirely ignored in clinical science of mental disorders. Perhaps we are moving too fast with shifting the clinical research agenda from one extreme to the other. Instead, we explore a middle ground by considering how latent constructs, such as emotional memory, can be conceptualised in network models of psychopathology.

1.1. Box 1: Definitions

Emotional memory: An umbrella term for any construct that refers to a mental representation of one, or a series of experiences (imagined or real), that carries an affective load, and that shapes the way one perceives and makes predictions about the self, others, and the world.

Common cause: A construct that plays a causal (mono- or multicausal) role in the development and maintenance of (multiple) symptoms of psychopathology. We also refer to this as 'latent construct'.

Latent variable: A statistical variable in factor analyses, often used as a label to capture the shared variance of a set of items, e.g., depression or the g factor in intelligence. A latent variable can be a common cause or latent construct, but it does not necessarily have to be.

Disease/medical model: A model of pathology that assumes that there is an underlying cause responsible for the symptoms (this can either be mono- or multicausal).

Network theory of psychopathology: A theory on how symptoms constitute mental disorders (Borsboom, 2008). Symptoms interact with each other, such that activation can spread from symptom to symptom, which can cause the network to shift to a state of full activation. We use the term 'network perspective' as a synonym for the network theory.

Network model: A statistical model that represents items as nodes connected by edges. We also refer to this as the 'network approach'.

Node: In the original network theory of Borsboom (2008) a node is viewed as a symptom, represented by a circle, and may be causally or statistically connected to other nodes by edges.

Edge: A relationship between two nodes, represented by a line or a one or two-sided arrow (if there is a specific directionality of the relationship).

External field: A field outside of the network itself, from which other events or characteristics may influence nodes or edges within the network.

Hysteresis: A network is a bistable system, such that it can shift from one (e.g., a functional) to another (e.g., a dysfunctional) state and vice versa. Hysteresis is when the network remains in the activated state even upon removing the initial stressor that triggered the shift.

2. The network perspective

By claiming limited empirical evidence for common causes, the network theory places symptoms into the spotlight. As such, it provides a new hypothesis for how mental disorders may develop. A symptom, represented as a *node* in the network may be activated by a stressor or factor in the *external field*, see Fig. 1. For example, the loss of a spouse may trigger the onset of symptoms (i.e., nodes) within the network, such as depressed mood. It is important to note that a factor in the external field is outside of the network itself, but not necessarily external to the person. Besides life events, Borsboom (e.g., 2017; Borsboom et al., 2019) refers to processes inside the person as external factors, such as neurobiological imbalances (e.g., inflammation). The nodes in a network are connected by *edges* that can represent causal or statistical relations, through which the activation can spread to neighbouring symptoms, initiating a vicious cycle that can cause the whole network to shift to a state of symptom activation (Borsboom, 2017). If symptoms have a high connectivity, the threshold for activation is lower, activation spreads faster, and the shift from healthy to unhealthy state occurs more abruptly than in a weakly-connected network (Cramer et al., 2016; Nuijten et al., 2016; but see; Bos et al., 2018; Fried et al., 2016). Moreover, if connections are strong, the network may remain in a prolonged state of activation, even when circumstances may have improved, or the original stressor has been eliminated, which is a phenomenon called *hysteresis* (Borsboom, 2017; Cramer et al., 2016; McNally, 2017). In other words, a network that is more densely connected is associated with an inferior prognosis (Lee Pe et al., 2015; Van Borkulo et al., 2015; but see Bos et al., 2018; Fried et al., 2016).

Besides symptom severity and frequency, the network theory and model introduce new dimensions for assessing the severity of a disorder. Aspects such as network structure, type of external stressor(s), connectivity strength and the causal sequence of symptom activation may vary across individuals, thereby offering new ways to capture heterogeneity within one diagnostic category. Incorporating such idiosyncratic components of a mental disorder may pave the way for formalised individualised treatment approaches. Network theory, thus, presents the development of a mental disorder as a complex and dynamic system over time, where every individual may have their own unique system.

This new conceptualisation also provides a way to portray the unclear borders between single disorders. Instead of explaining the commonly observed overlap in symptoms with respect to a common cause, the network perspective rather refers to the causal relations between symptoms that form bridges across different symptom networks (Borsboom & Cramer, 2013; Cramer et al., 2010). An example of how two symptom networks can be connected is shown in Fig. 1. In essence, such bridging symptoms have a transdiagnostic nature (Borsboom et al., 2011). While the transdiagnostic approach to psychopathology is not necessarily novel (e.g., Harvey et al., 2004), a unique asset of the network approach is that it provides a statistical technique to capture comorbidity.

Applying a network approach to investigate the changes that occur in different individuals over time and across conditions presents opportunities for understanding how disorders evolve and where to intervene. Nonetheless, empirical science is only beginning to explore the clinical utility of the network theory. As a result, it currently remains elusive whether the network perspective has the potential to significantly improve treatment outcomes. What is clear is that instead of targeting underlying causes, such as emotional memory, interventions in line with the network theory would most likely target the symptoms and their causal relations.

While the clinical utility remains uncertain, the unique view of the network perspective may spark new possibilities in the field. There are many aspects of the network approach that we embrace, such as the way it describes relationships between symptoms, the spread of activation, and its transdiagnostic nature. At the same time, we wonder whether the network perspective must be as black-and-white such that these assets

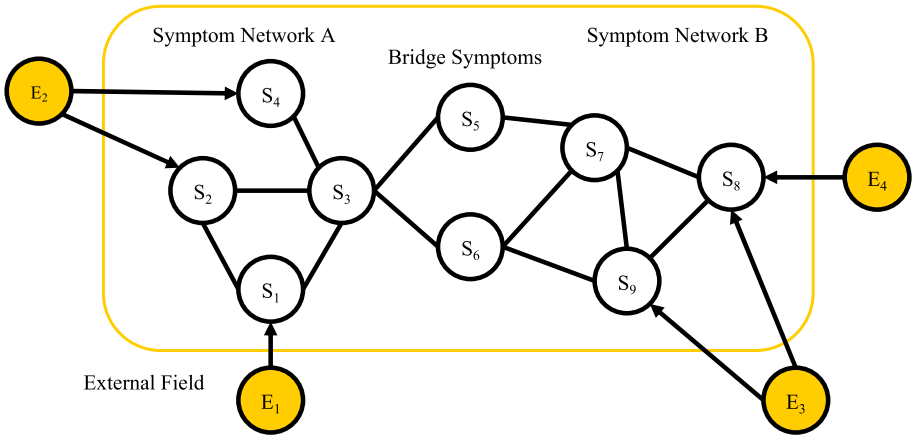

Fig. 1. Connected Symptom Networks According to the Original Network Theory
Note. Illustration adapted from Borsboom et al. (2017). Nodes are activated by factors in the external field, which can either be specific for one symptom (E_1 and E_4) or shared across multiple symptoms (E_2 and E_3). Symptom networks A (S_1–S_4) and B (S_7–S_9) are connected via two bridging symptoms (S_5 and S_6).

can only exist by negating latent constructs in psychopathology.

3. Current shortcomings of the network perspective

With its new take on mental disorders, the network theory establishes a radical shift in how disorders are explained. The current stagnation in treatment advancement and the so-called theory crisis in psychology (Borsboom et al., 2021; Eronen & Bringmann, 2021; Fried, 2020; Oberauer & Lewandowsky, 2019) may indicate that such a radical shift could be beneficial in order to induce new momentum in clinical science. We have previously witnessed radical transitions in the conceptualisation of mental disorders causing significant headway in how we understand and approach psychopathology, as for example the paradigm shift from psychoanalysis to behaviourism. Yet, it was later acknowledged that the behaviouristic conceptualisation of mental disorders was not as clear cut; solely focusing on stimulus and response left a range of phenomena unexplained. It appeared that the conceptualisation of mental disorders was incomplete without the integration of mental representations (Rescorla, 1988). In consequence, concepts such as schemas, introspection, and childhood experiences were rediscovered and re-evaluated (Beck, 1976). This ultimately led to the reconcilement of the cognitive and behavioural theoretical models, responsible for far-reaching developments in the conceptualisation and treatment of disorders that are still used today (e.g., D. M. Clark, 1986; Foa & Kozak, 1986; Rachman, 2015). With respect to the original network theory, we may find ourselves in a similar situation, where latent constructs are, once more, completely ignored. By reviewing potential shortcomings of the network approach, we question whether its radical view of psychopathology is justified.

Firstly, the theoretical foundation of the network perspective may be a result of two major misconceptions. As previously conveyed, the network theory views mental disorders as "syndromic constellations of symptoms that hang together empirically, often for unknown reasons" (Borsboom, 2017, p. 5), rather than disease entities that exist independently of symptoms. A comparison that is used to illustrate this point is that of cancer versus a psychological disorder: While it is possible to have a brain tumour without enduring any accompanying symptoms, Borsboom and Cramer (2013) reasoned that it is impossible to be diagnosed with major depression in absence of the typical symptoms of depression. Note that this comparison is a bit of a strawman, as not many clinical psychologists would have argued otherwise. Thus, the first misconception that has emerged, is that the prevailing models of psychopathology (at least in psychology), before the network approach came in vogue, would have claimed that the disorder itself refers to one clear-cut underlying cause, see Fig. 2A. Another misconception is that many clinical researchers would have subscribed to the idea of there being a distinct common cause for each diagnostic entity listed in the DSM-5. Instead, the majority of models of psychopathology place multiple causal factors at the root of psychological disorders, without implying a one-to-one mapping of cause and DSM diagnosis, see Fig. 2B for an example with emotional memory. Moreover, most models are inherently transdiagnostic in that they regard common causes as being responsible for symptoms belonging to multiple diagnostic categories. We suggest that a fairer comparison between the common cause model and the network model could be achieved if a *true* possible common cause is applied in the model, such as emotional memory, which may give rise to various symptoms.

Secondly, the network approach seems more descriptive and illustrative rather than mechanistic, as described by Borsboom (2017, p. 11): "Because the network model is not tied to a particular level of explanation (e.g., biological, psychological, or environmental), and does not single out particular mechanisms that generate the network structure, it is perhaps best interpreted as an organizing framework – an explanatory scheme with broad use across sub-domains of psychopathology." This exploratory nature of the network approach offers great flexibility, nonetheless, it also brings about uncertainty, which may hinder theory

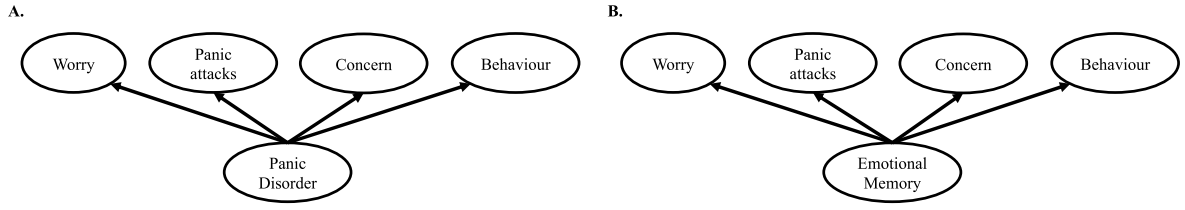

Fig. 2. Simplified Models of Panic Disorder: Latent Variable Model à la Network Literature (A) and Latent Variable Model with Emotional Memory (B)
Note. (A) shows how the network literature illustrates panic disorder using a latent variable model, based on the work of (Borsboom, 2008). (B) illustrates a latent variable model with what we consider a more "just" presentation.

construction. While there has been a rapid surge in the use of network modelling in clinical science, the theoretical gains so far seem limited: Network modelling has been described as reading "like an exercise in model fitting" and falling "short of theory building and testing" (Fried, 2020, p. 271). We would assume that this lack may be due to the vague mechanistic foundation, giving rise to questions such as: What exactly causes the initial activation of a symptom? What causes connectivity strength and central symptoms to vary across individuals? What part of the network should be targeted in treatment? And if there are external factors that activate one or more symptoms, does this not overlap with the idea of an underlying cause? Solely focusing on the causal relations between symptoms, does not seem to provide a full mechanistic explanation of the first spark that causes to ignite a network nor of what leads to these causal relations between symptoms. In that sense, the network theory has been criticised for relying on tautological reasoning: "the causes of the connectivities involved are not specified except by indicating that the symptom interactions are the cause." (Young, 2015, p. 8). Perhaps our view is tainted by the long-standing causal model of experimental psychopathology, however, we propose that developing models that focus on mechanisms underlying networks of symptoms may enhance theoretical development and clinical utility, e.g., by indicating what to target in treatment and how.

Thirdly, the limited theoretical foundation may also account for the frequent confusion between the network *theory* and the network *model*. Terms like "network approach", "network framework", and "network perspective" are often used ambiguously without explicitly stating whether referring to the theory or statistics. As such, network theory and statistics seem to sometimes be conflated, although each represents fundamentally different aspects. As a result, statistical data models are interpreted as theoretical models. This conflation presents an important issue in terms of inferences and theory building, of which the network literature is well aware (Fried, 2020). This is especially problematic since cross-sectional data, which is the most common type of data collected, can be used to model both a network as well as a factor model (usually applied for common cause models), with high goodness of fit for both (Bringmann & Eronen, 2018; Epskamp et al., 2016; Fried, 2020; Kruis & Maris, 2016; van Bork et al., 2019; Van Der Maas et al., 2006). Thus, although one's data may seem to fit a network model, the data may just as well be explained by a factor model. This statistical equivalence posits that a model with excellent fit is not sufficient evidence for drawing decisive conclusions. The unclarity of what model should be used to explain the data and to ultimately conceptualise mental disorders, stresses why it is crucial to advance the network theory to make more justifiable decisions about whether a network model is indeed a better fit.

A fourth limitation is the selection of variables included as nodes in a network. A major part of the literature depicts symptoms according to the DSM diagnoses as nodes, although the theory itself does not limit its use to a specific type of variable. Instead, "at its core, a network is simply a set of elements (nodes) that are connected through a set of relations (edges). Elements as well as relations between elements can be virtually anything" (Borsboom & Cramer, 2013, p. 98). By focusing on the DSM criteria, most of the network literature ignores variables that may also be important in the development and maintenance of psychopathology. Limiting ourselves to the DSM criteria restricts the ability to attain a complete picture of the aetiology of mental disorders. Similar arguments have been made by some of the prominent scientists in the network field, such as Fried and Cramer (2017), Jones et al. (2017), arguing for other elements like self-efficacy and cognitive control to be included in psychopathological networks. In line with our plea, Bringmann et al. (2022) claim that mental representations play a causal role in the development and maintenance of mental disorders, and should be included in networks of psychopathology. Interestingly, if such variables (e.g., self-efficacy, cognitive control, or mental representations) are included at a similar level as nodes or edges are we not confusing symptoms with underlying psychological constructs?

Related to the fourth limitation, another reason for including other variables besides symptoms, are spurious correlations. When variables that may be causally linked to other nodes in a network structure are missed, there is an increased risk of spurious correlations inflating the symptom network. An edge between two variables in a network could reflect a causal relationship, however, such inference may embody erroneous conclusions, as a third missing variable (i.e., a common factor) could be the cause of the relationship. Thus, including other variables as nodes, besides DSM-5 symptoms, may be useful in terms of creating a representative network and reducing spurious correlations. Nonetheless, if nodes can virtually be any variable, what criteria do we abide by for selecting what variables to include as nodes and to prevent overloaded networks with numerous intercorrelations and too little power? We would argue that theory-based guidelines are needed to select relevant variables as nodes in a network, which also distinguish them from factors in the external field.

Lastly, a limitation that warrants further discussion is that clinical targets according to the network theory remain unclear. With symptoms constituting a disorder, treatment would presumably solely focus on symptoms instead of on the alleged underlying causes. However, in the perspective of the medical common cause model, targeting a symptom, such as taking paracetamol to subdue the pain caused from appendicitis, will not cure the source of the pain. With respect to the network theory, where symptom interactions are the source of the disorder, the therapeutic target remains undefined: Should treatment be targeting edges, the node that has the highest centrality measures (node strength, closeness, and betweenness), or rather the node at the beginning of the causal chain? Research on the clinical utility of the network approach is still in its infancy and such questions are starting to be explored now by a large research consortium in the Netherlands (Roefs et al., 2022; https://www.nsmd.eu/). In a recent article, Henry et al. (2022) explored whether targeting a single symptom in a network is necessarily best suited for treatment. Their simulations show that single target interventions are effective in reducing the targeted symptom but had no meaningful effects on the non-targeted symptoms. A solution could lie in the centrality hypothesis, which may suggest that highly central nodes (i.e., connected to many other nodes) should be targeted in treatment (Robinaugh et al., 2016). It appears quite straightforward that targeting a central node would be a way to restrict the spread and maintenance of activation. Initial results of interventions targeting central nodes seemed promising (Elliott et al., 2020; Robinaugh et al., 2016; Rodebaugh et al., 2018). Nonetheless, the literature is moving away from this idea, as centrality estimates do not appear as very stable nor accurate parameters (Forbes et al., 2017; Fried et al., 2018; Rodebaugh et al., 2018; Terluin et al., 2016). Network connectivity has also been considered as a potential tool for prognosis or determining severity (Cramer et al., 2016). While some evidence suggests that a more densely connected network is indeed associated with an inferior prognosis (Lee Pe et al., 2015; Van Borkulo et al., 2015), findings are inconsistent, as other cross-sectional and longitudinal network studies have found the opposite: Network connectivity increases with symptom reduction (Bos et al., 2018; Fried et al., 2016). We are aware that the network approach is undergoing advancements as we write this, but the current inconsistencies in findings reflect the novelty and uncertain validity of the network theory thus far.

The possible shortcomings of the network perspective, ranging from being built up on two misconceptions that could be perceived as strawmen, not providing sufficient mechanistic insight, frequent confusions between the theory and its statistics, an exclusive focus on symptoms, spurious correlations, and unclear clinical utility, may hinder its application to models of psychopathology. Ultimately, as the network perspective of psychopathology commenced in response to the claimed stagnation in clinical psychology and poor treatment outcomes, the litmus test for the network theory is its clinical utility: whether it will indeed lead to better treatment outcomes and lower relapse rates.

4. Conceptualising common causes in a network model

We propose that integrating common causes within the network approach to psychopathology may help address some of the limitations discussed above, especially the lack of a mechanistic foundation and unclear clinical utility. Rather than ignoring decades of research, we believe that standing on the shoulders of giants is central to making progress in clinical science. In the spirit of epistemic iteration, "a process in which successive stages of knowledge, each building on the preceding one, are created in order to enhance the achievement of certain epistemic goals" (Chang, 2004, p. 224), we will now discuss whether a latent construct, such as emotional memory, can be accommodated in a network model and how.

Incorporating causal factors into models of psychopathology may be facilitated by differentiating between distal and proximal causes (Harvey et al., 2004; Roiser, 2015). Proximal causes are closely related to mechanisms underlying symptom onset and are usually targeted for treatment. For example, a brain tumour is proximally caused by the uncontrolled cell division and is treated by surgically removing or killing the rapidly dividing cells. Distal causes, on the other hand, are indirectly linked to the mechanisms, such as genes or prenatal toxins, and can be addressed for prevention (Roiser, 2015). We hypothesise emotional memory to be both a proximal and distal cause of a spectrum of mental disorders. For example, emotional memory may have a similar working mechanism as observed in the onset of multiple sclerosis, a biological disorder explained by the medical model. In multiple sclerosis, it is the accumulation of multiple micro-inflammations in the brain over time, which cause symptoms, such as the deterioration of cognitive and motor functioning (Musella et al., 2018). We know that emotional memories are collected throughout a lifetime and can remain dormant until activation. Perhaps the accumulation of emotional memories increases the vulnerability to psychological instability, reducing the threshold for activation in a network, such that a small external trigger can cause symptom activation, shifting the system to a dysfunctional state. If emotional memory plays such a role, there are two critical questions that need to be answered: 1) How can emotional memory be measured independently from symptoms? And 2) how would emotional memory be depicted in a network model?

The network architecture consists of nodes and edges and may include factors that influence the network from an external field. As nodes can "be virtually anything" (Borsboom & Cramer, 2013, p. 98), could emotional memory simply be represented as a node, causally connected to specific elements within a network, as seen in Fig. 3A? Upon its activation, emotional memory may lead to the onset of other symptoms. However, as a node, emotional memory would not necessarily influence the activation threshold of the network as a whole. To account for that, emotional memory could possibly be represented as an edge (Fig. 3B), such that highly severe or dysfunctional emotional memories may be associated with the spread of activation between symptoms. Yet, the depiction of emotional memory as edges would indicate that emotional memory is what connects the different nodes, for example depressed mood with suicidality. If emotional memory is the factor that holds the dysfunctional network together, is this any different to a common cause?

Another aspect that should be considered when incorporating emotional memory into a network model is its dynamic nature. Emotional memory may be a variable that may either change slower or faster compared to other symptoms in a network. Fried and Cramer (2017, p. 1005) have suggested that "variables that change much slower over time … to be part of the external field". Albeit unclear what is considered "slower", this could suggest that emotional memory could be represented in the external field (Fig. 3C), where upon activation of the memory, it may trigger the onset of a single or a cluster of symptoms in the network. Nonetheless, similar to the representation as a node, this would not allow emotional memory to influence the vulnerability of the network as a whole. Moreover, in contrast to factors that are commonly presented in the external field, such as specific life events like the loss of a job or a partner, emotional memory may not be a single adverse event with a clearly defined ending.

Perhaps emotional memory could be better depicted as a latent construct within a network, either influencing several nodes (Fig. 3D) or edges (Fig. 3E). A recent attempt at this has shown promising advancements, integrating risk factors into network models that can either influence the activation threshold of a node (Fig. 3D) or the edge strength (see Fig. 3E; Lunansky et al., 2021). While factors that affect nodes are *causal main effects*, factors that influence the edges are presented as *causal moderators*. In essence, such conceptualisations are hybrid models, accommodating both network and common cause models (Fried & Cramer, 2017; Young, 2015). For example, a causal main effect in a network would entail a common cause to activate one or multiple symptoms, upon which activation precipitates and culminates in a self-sustaining network due to the causal interactions between symptoms.

While the network model has commonly been contrasted with the factor model, the distinction may in fact not be that clear-cut. Latent variable and network models cannot be distinguished so explicitly as "there is no clear boundary where network models end, and latent variable (or common cause) models begin" (Bringmann & Eronen, 2018, p. 606). The work of Cramer et al. (2012) shows that a latent variable model can similarly include direct links between symptoms. However, the more direct the connections are, the less explanatory power of the covariance between symptoms is attributed to the common cause. Likewise, if a node in a network model directly causes two or more nodes, this could be considered a common cause, for example, as seen in the work of McNally et al. (2017), where sadness is a direct cause of guilt and suicidal ideation, plans, and attempts. Epskamp et al. (2017) have

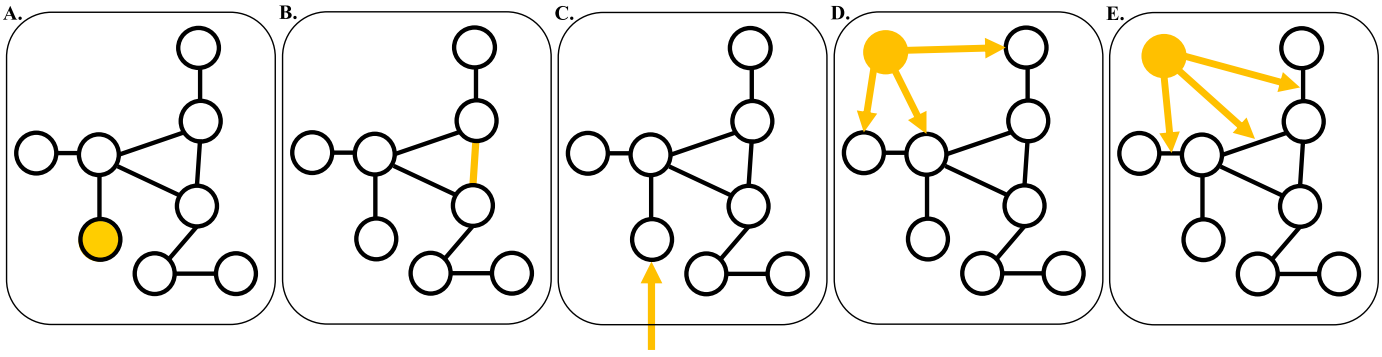

Fig. 3. How could Emotional Memory be Represented in a Network Model?
Note. Emotional memory could be represented as (A) a node; B. an edge; (C) a factor that activates a symptom in the network from the external field; (D) a causal main effect within the network triggering the onset of several elements; or as (E) as causal moderator within the network influencing edge strength.

acknowledged that "network modelling and variable modelling can complement – rather than exclude – one another" (p. 905) and have created generalisations of the network model, introducing latent and residual network models. This overlap in models does not justify the aforementioned unclarity with respect to whether causal or factor models should be applied to explain one's data, but further highlights the need for theory-based guidelines.

It seems that clinical psychology is moving to an understanding that pure models (latent variable vs network models) do not seem fit for explaining the development and maintenance of mental disorders. However, the field lacks the tools and insight for reconciling both the network and latent variable model. With the aim of advancing the field, more specific criteria are needed to determine how and what constructs are best represented in what model and at what level. The network model prides itself in being an exploratory approach, at the same time this flexibility can stand in the way of robust and replicable theoretical output. If the application of the network model would be more precise with respect to what types of variables are best represented with what specific architectural component, it would probably be easier to integrate existing theories and assess its clinical utility. Considering epistemic iteration, we believe that more guidelines at the model level as well as at the construct level are key for moving forward.

5. Conclusion

In this paper we present emotional memory as a promising candidate for causing a range of transdiagnostic symptoms, and therefore, deserving a place high on the clinical research agenda. Meanwhile, the arguments made throughout this article may apply to other putative common causes in psychopathology as well. In contrast to the claims made in the network literature and concerning the so-called theory crisis in psychology, clinical science *does* already have several strong theories that provide substantial evidence in support of a latent cause of mental disorders (e.g., Beck, 1976; Brewin et al., 2010; Hackmann & Holmes, 2004; Kindt, 2014).

The network theory of psychopathology posits otherwise: It rejects all latent constructs in the aetiology of mental disorders. Meanwhile, this new perspective on psychopathology offers a plethora of convincing benefits for studying mental disorders, such as incorporating the relationships between symptoms, the spread of activation, hysteresis, and clarifying comorbidities. While these are all positive aspects, the mechanistic and theoretical foundations of the network theory remain somewhat vague. With this we mean, for example, uncertainties regarding the onset of symptom activation, what can be represented as a node, and the lack of operational tools for clinical practice. We are aware that the exploratory nature of the network model is an asset to this approach, but too many degrees of freedom stand in the way of theory construction, epistemic iteration as well as the integration of network models with existing theories.

Fortunately, advancements are being undertaken to further improve network modelling and therefore its theory. The more contemporary ideas acknowledge that the network theory may not be as black-and-white as presented by Borsboom (2008) and that adding causal structures into a network model may be beneficial. Although it remains unclear how common causes can best be represented in a network model, we suggest that emotional memory, for example, may be best integrated as a factor within the network that influences clusters of nodes or edges (see Fig. 3D and E). We propose that accommodating emotional memory, as latent construct, into the network approach will invigorate the field by offering mechanistic insight for treatment tailoring along with evidence-based treatment methods (e.g., Imagery Rescripting targeting the dysfunctional meaning of emotional memory), and more tangible assessment tools to quantify treatment effects in a transdiagnostic and idiosyncratic manner.

To understand how exactly emotional memory can be depicted in a network, we do not only need more specific criteria in terms of the network architecture, but we also need to clarify the construct at hand and its role in mental disorders. A critical question to be answered is how emotional memory can be measured independently from symptoms. In clinical science, it is often the case that interventions are developed first, and only afterwards do we try to explain their underlying mechanisms of action. This 'methodological iteration' of advancing treatments based on experience with prior interventions, may be seen as a weakness in clinical science, working in reverse, irrespective of theories or constructs. Ultimately, this way of working may obstruct epistemic iteration; rather than building up on prior knowledge, new concepts are developed in absence of a theoretical or explanatory foundation. Furthermore, all interventions have a general effect on an individual, rather than solely influencing one single variable. This inability to finely pick apart what specific psychological variables are manipulated stands in the way of identifying underlying causes (Eronen & Bringmann, 2021). Even though various theoretical and therapeutic frameworks seem to follow from conceptualisations where emotional memory plays a critical role, it is not easy to decipher whether the treatment effects are the result of changing the emotional memory itself. Without an independent test of emotional memory, it is difficult to demonstrate that the treatment effect is due to a change in emotional memory. In the example of PTSD, a disorder that is said to be developed by dysfunctional processing of trauma, the recommended treatments are trauma-focussed approaches, such as EMDR, Imaginal Exposure, and Imagery Rescripting, that focus on processing the trauma. The only way of assessing the quality of the treatment is symptom mitigation. Thus, without an independent measure of the underlying construct, we cannot prove whether it truly is the hypothesised mechanism that accounts for the treatment effect.

To move forward from here, we argue that the research priority should be to expand the conceptualisation and operationalisation of constructs that are viewed as driving factors in psychopathology. With this, we do not solely refer to emotional memory, but rather all constructs that may play a role in the aetiology and maintenance of mental disorders. Only by understanding the concepts in question can we provide a foundation for applying models and advancing theory. Additionally, thoroughly defined and operationalised variables will pave the way for developing and fine-tuning treatments as well as deciphering their mechanistic effects (Eronen & Bringmann, 2021). Therefore, we call for clinical science to invest into strengthening construct validity. For us, the next step is a systematic exploration of the construct of emotional memory to arrive at a definition and operationalisation that allows a potential integration with e.g., the network theory, and more generally, invites the development of better theoretical models.

CRediT authorship contribution statement

Inga Marie Freund: Conceptualization, Investigation, Visualization, Writing – Original Draft, Writing – Review & Editing. **Arnoud Arntz:** Conceptualization, Supervision, Writing – Review & Editing. **Renée M. Visser:** Conceptualization, Writing – Original Draft, Writing – Review & Editing, Supervision. **Merel Kindt:** Conceptualization, Writing – Original draft, Writing – Review & Editing, Supervision.

Declaration of competing interest

All authors declare that they have no conflicts of interest.

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