# MENTAL DISORDERS AS COMPLEX NETWORKS:
## AN INTRODUCTION AND OVERVIEW OF A NETWORK APPROACH TO PSYCHOPATHOLOGY*

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## Abstract

Mental disorders have traditionally been conceptualized as latent variables, which impact observable symptomatology. Recent alternative approaches, however, view mental disorders as systems of mutually reinforcing symptoms, and utilize network models to analyze the structure of these symptom-symptom interactions. This paper gives an introduction to and overview of the network approach to psychopathology, as it has developed over the past years.

**Key words**: mental disorders, latent variables, network models, psychopathology, major depression, observable symptoms, network perspective

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Anna suffers from chronic pain that affects her sleep. In the mornings she arrives at work feeling tired and during the day her capacity to concentrate decreases. Because of this, she does not perform as well at work as she used to. She makes mistakes, which makes her feel guilty, causing her to sleep even less. The feelings of guilt become increasingly stronger and at some point she feels depressed most of the day. Anna decides to go and see a therapist. Diagnosis: major depression.

Bart just went through a painful break-up. His appetite disappears, which causes him to lose a lot of weight, and his energy to do things decreases. After a while, he has lost interest in almost all activities he used to enjoy and he starts to feel worthless. He sometimes thinks about suicide and does not leave the house anymore, which increases his feelings of worthlessness. A friend insists that he should see a therapist. Diagnosis: major depression.

Anna and Bart both receive the same diagnosis, even though they suffer from a different set of symptoms. Although clinical psychologists and psychiatrists probably are familiar with this phenomenon and encounter situations like this on a regular basis, this great variety in etiology and symptomatology illustrates the heterogeneity of diagnostic categories such as major depression (MD henceforth) across cases. This pattern is characteristic of many mental disorders: the etiology of most psychopathology often shows heterogeneous patterns, as different people can develop the same disorder in different ways. In turn, the same event (e.g., abuse) can have very different consequences for different individuals (Nolen-Hoeksema and Watkins 2011). In the literature of psychiatry and clinical psychology it is therefore quite widely accepted that psychological disorders are multifactorially determined (Kendler et al. 2010).

This heterogeneity in psychopathology is barely reflected in the way psychological disorders are currently regarded: namely as unobservable, unidimensional phenomena that are the underlying cause of observable symptoms. In this paper, we will first elaborate on the current views on psychopathology, we will then discuss why it is insufficient to view disorders as invisible entities. Finally, we will discuss a new way of conceptualizing psychopathology by introducing a network perspective of psychological disorders.

## The status quo: psychological disorders as latent variables

Currently, researchers often investigate psychological disorders by adding up the symptoms of a disorder and using this sum score as a *measurement* of a so-called *latent variable*. A latent variable is a variable that cannot be directly observed (such as MD), but has to be inferred from indicators that can be observed (such as symptoms). By using a latent variable model, it is assumed that the obscure and complex patterns of empirically observable symptoms can be translated into a clear and elegant mathematical structure (see e.g., Krueger 1999). An example of a latent variable is temperature. We cannot observe temperature itself, but we can observe that when the temperature changes, the thermometric fluid expands

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in the thermometer.

To understand where the latent variable model came from, let us go back to the fundamental assumption behind the diagnostic process, the assumption that directs both research as practice in an important way. Anna and Bart both show symptoms that the DSM-5 (American Psychiatric Association 2013) attributes to MD and they can therefore both be diagnosed as depressed. But what does this actually imply?

The idea behind *diagnosis* has roots in medicine: it focuses on the determination of the cause of a set of symptoms. In other words, you want to be able to trace "which disease a person has". The success of modern medicine is largely based on this very specific line of reasoning: a diffuse pattern of symptoms can be traced back to a *common cause*. For instance, imagine a person that suffers from severe headaches, blurred vision, and dizziness. A physician will first look for a single physical cause of these symptoms – for instance a brain tumor. When a cause has been found, the (curative) treatment almost always entails eliminating this common cause (e.g., by cutting out the tumor). The consequence is that the symptoms (that were merely caused by tumor) will disappear (Hyland 2011). The importance of this strategy for modern medicine can hardly be overstated.

Superficially, the diagnostic process based on the DSM-5 appears to do the same: Anna and Bart can both be diagnosed with MD and treated. There is a diffuse pattern of symptoms that appears to be traced back to an underlying disorder and this disorder should therefore be the focus of treatment, either through medication or through psychotherapy. This line of reasoning seems to assume that, in a crucial way, MD is similar to a brain tumor: namely, it acts as a common cause of the symptoms. That is, Anna feels sad *because* she is depressed. The label "MD" then serves as an explanation for the appearance and cohesion of the symptoms (Borsboom and Cramer 2013).

## Methodological deadlock

Although it is an elegant and simple idea that a single entity causes a specific set of psychopathological symptoms, one can wonder whether it is necessary and useful to search for such a common cause. One reason to doubt this is for instance that, after a century of scientific research, and contrary to successful identification of medical conditions, there is not a single method to establish the presence of any of the common mental disorders *independent of their symptoms*. As opposed to the tumor (an entity one can observe with a brain scan without knowing that the patient suffers from headaches), mental disorders – ranging from MD to schizophrenia – cannot be observed without appealing to symptomatology. A doctor can coincidentally see on a brain scan that you have a tumor while you yourself did not notice a thing yet. However, a psychologist or a psychiatrist will not discover MD on a brain scan. There is no laboratory test for disorders such as MD, and it does not appear that such a lab test is forthcoming either.

The leading idea behind the practice of adding up symptoms to obtain a single diagnosis is that these latent variables can be measured and quantified in the same way as physical entities such as length and weight (Borsboom 2005). However, this only seems to hold in theory, as few of the characteristic payoffs associated with successful measurement – prediction, explanation, and control – have been successfully realized in psychopathology research. In practice, the results of research based on latent variable models are quite limited. For instance, we still do not know how a disorder such as MD arises, or how we can predict which individuals are particularly vulnerable and which ones are not. These limited results may be due to the implausibility of the assumptions of latent variable models for psychopathology research (Cramer et al. 2010); for instance, the question of whether complex phenomena, such as MD, are even measurable in the first place has been a topic of an ongoing and unresolved discussion (Michell 1999, McGrath 2005). It seems as if we have every reason to doubt the standard model of disorders as common causes yet almost all research is predicated on it. The main reason for this is the simplicity of the current psychometric interpretation of the latent variable model. This model assumes that the (co-)variance in observable pathological symptoms is caused and explained by a latent "invisible" variable. If true, then a simple count of the number of symptoms can be reliably used as an approximation of this latent variable. And that is exactly what almost all (clinical) psychology, whether it is neuroscientific, genetic, or psychological in nature, is based on: the counting of symptoms. Statistically, this translates in models such as factor models or IRT models, which are easy to use and interpret. This simplicity is very appealing, but limited – a methodological deadlock.

## Heterogeneity

The latent variable model also struggles with the empirical reality of heterogeneity in psychopathology. Why is heterogeneity hard, even impossible, to accommodate within a common cause framework?

Because diagnoses are often based on sum-scores – simple counts of how many symptoms a person displays – not everyone with the same diagnosis has the same symptoms. For instance, according to the DSM-5 (American Psychiatric Association 2013) MD is diagnosed using nine symptoms that function as diagnostic criteria: depressed mood, diminished interest or pleasure in (almost) all activities, significant weight gain or loss, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or excessive guilt, diminished ability to think or concentrate, and recurrent thoughts of death or suicide. A person is diagnosed with MD when (s)he shows at least five of these symptoms, and one of these symptoms is depressed mood or lack of interest. This means that there are hundreds of possible combinations of symptoms that all lead to the label "major depression". Theoretically, two people could both be diagnosed with MD even though they have only a single symptom in common. On top of that, many symptoms are also heterogeneous themselves: sleep problems comprise both sleeping too little and sleeping too much, and a change in the activity levels may signal either restlessness or psychomotor retardation.

Furthermore, the way in which separate disorders are distinguished is often disputable: the DSM-5 contains many arbitrary discriminations and empirical analysis has shown that some disorders co-occur so often that it is almost impossible to defend a distinction between such disorders (e.g., Generalized Anxiety Disorder and Major Depression; Cramer et al. 2010). Thus, behind a seemingly simple diagnosis there can be a world of difference.

To date, these debates have remained largely academic, as there was no feasible alternative to the standard psychometric approach in clinical research. However, these days are over now. Novel methodological techniques, originating from studies of complex

networks, have come to offer a viable alternative strategy for pursuing research in psychopathology.

## A new approach: symptom networks

If we take another look at Anna and Bart, despite the large differences in their symptom pattern and development, we see also a common element in that development. That is, the mechanism behind the pattern of symptom development seems to be the same. In both cases, the condition started with a particular symptom (trouble sleeping for Anna and lack of appetite for Bart), which affected another symptom (tiredness for Anna and decreased energy for Bart). That symptom then caused another symptom, and so on, until a spiraling pattern of vicious circles arose.

In both the cases of Anna and of Bart, the development of their problems resembles a chain reaction that at some point starts reinforcing itself (see **figure 1**). Based on this observation, one may suppose that mental disorders *in general* consist of such chain reactions between problems. Because some problems can affect each other more easily (e.g., trouble sleeping  tiredness) than others
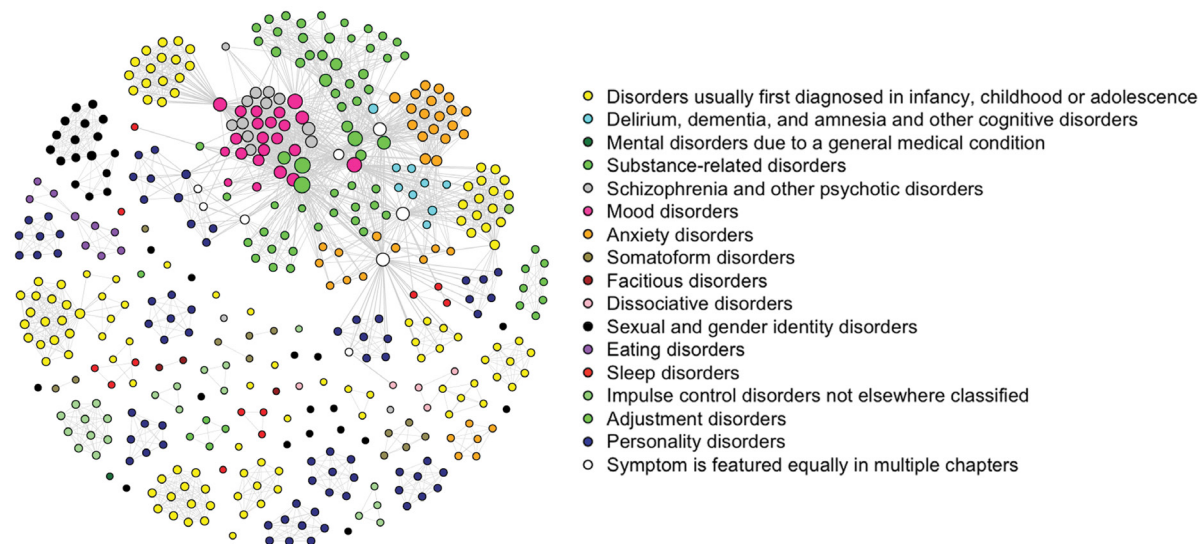
(e.g., trouble sleeping → panic attacks) it is then sensible to speak of an "architecture" of problems.

This architecture forms a *network* of symptoms that stand in certain direct relations toward one another. In our example we focused on symptoms that are currently associated with MD, but other sets of symptoms can be depicted as a network with stronger and weaker relations (e.g., panic disorder and posttraumatic stress disorder; Borsboom 2008, McNally 2014). Some symptoms can be considered closer together in the network and because they have stronger connections, they can therefore affect each other more easily – we often observe these problems together. For example, you could imagine that we often observe guilt and depressed mood together, or hallucinations and delusions, but we do not often observe guilt and hallucinations together. Groups of problems that often occur together are classified in diagnostic systems such as the DSM-5 and are called symptoms of a certain disorder. In our example, guilt and depressed mood would be symptoms of MD, and hallucinations and delusions symptoms of schizophrenia. **Figure 2** shows a representation of the DSM (this figure is based on the fourth edition; see Borsboom et al. 2011) that is designed according to this idea: every symptom

**Figure 1.** *Graphical representation of the causal relations between the symptoms of Anna and Bart*



Anna

Bart
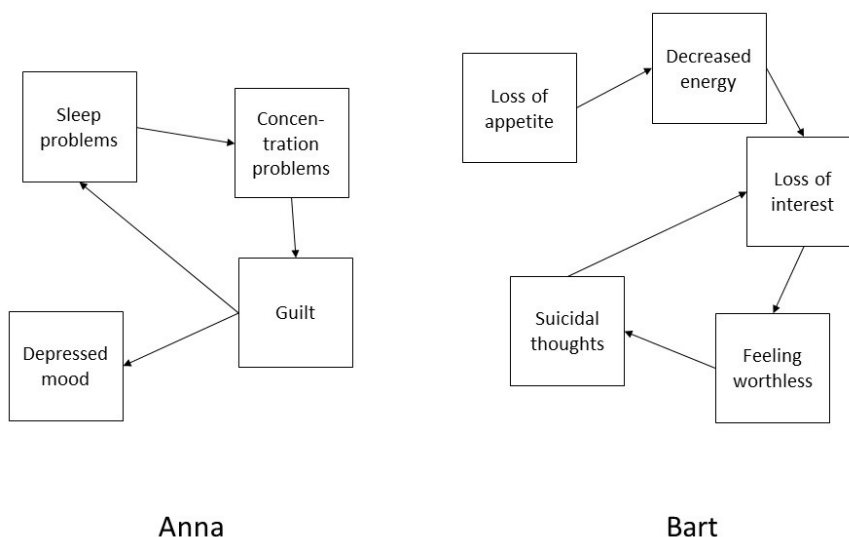
**Figure 2.** *The network structure of the DSM-IV. The figure depicts each individual symptom as a node. Two nodes are connected whenever they feature as diagnostic criteria for the same disorder*



- ○ Disorders usually first diagnosed in infancy, childhood or adolescence
- ○ Delirium, dementia, and amnesia and other cognitive disorders
- ● Mental disorders due to a general medical condition
- ● Substance-related disorders
- ○ Schizophrenia and other psychotic disorders
- ● Mood disorders
- ● Anxiety disorders
- ● Somatoform disorders
- ● Facitious disorders
- ○ Dissociative disorders
- ● Sexual and gender identity disorders
- ● Eating disorders
- ● Sleep disorders
- ● Impulse control disorders not elsewhere classified
- ● Adjustment disorders
- ● Personality disorders
- ○ Symptom is featured equally in multiple chapters

is depicted as a node (a circle in the figure) and two symptoms are connected with a line if they belong to the same diagnostic category in the DSM. For a similar network structure, based on empirical symptom patterns rather than the DSM itself, see Boschloo et al. (2015).

A network such as the one in **figure 2** can be analyzed with similar techniques that are in use for the analysis of complex networks such as the Internet. For instance, one can explore the network architecture by investigating which symptom has the most connections (insomnia), which symptoms most often lie on the path between two random other symptoms (depressed mood), and whether the "distance" between disorders in the figure predicts how often they appear together in a client. The latter appears to be true: Borsboom et al. (2011) report a correlation of -.7 between the distance between disorders in the network, and their empirical comorbidity rates. Thus, the closer the disorders are, the more often they appear together. If we assume such a (causal) network of symptoms we could question the necessity of pointing out an underlying common cause for any set of symptoms. The set of symptoms that is currently associated with a disorder such as MD might be analyzed and explained in more detail if we consider it as a complex system. A network of symptoms "synchronizes" because of their underlying causal connections: if sleep problems increase, the connected symptom tiredness will increase as well, which then causes an increase in concentration loss, but if tiredness then decreases again, the connected symptoms will probably decrease as well. We see this type of systems every day, for instance in the synchronized behavior of a school of fish or a flock of birds: if one bird moves to a certain direction, the birds next to it will probably also move to that same direction. To explain that behavior we do not need a "latent bird" that directs the observable birds in a certain formation, their movements are simply determined by their neighbors (see also Cramer et al. 2012).

The network approach of disorders is based on the very same idea: in network models psychological disorders are not considered latent constructs that cause symptoms but they are considered networks of symptoms that are connected by causal relations. This novel perspective provides us with a promising new psychometric toolbox to study the complex dynamics of psychopathology.

## A new approach to old problems

The network approach offers a plausible explanation for the fact that psychopathology research has had virtually no success in identifying disorders with common causes of symptoms: for most disorders, these common causes simply do not exist. Thus, rather than being reflections of "brain diseases", which researchers still hope to identify once we have sufficient observational power (Insel and Cuthbert 2015), correlations between symptoms arise from their direct influences on each other.

The network approach is in line with research that shows that clinical psychologists intuitively interpret relations between symptoms as causal networks already. In 2002, Kim and Ahn showed that clinicians spontaneously create causal relations between symptoms when asked how these symptoms relate to each other. In addition, the way diagnostic systems such as the DSM-5 are set up matches better with this perspective than the medical model. In fact, the DSM-5 is a diagnostic system brimming with all kinds of causal relations between symptoms. Take the example of panic disorder. The criteria in the DSM-5 for this disorder entail: (1) returning panic attacks, (2a) persisting concern that there will be another attack, (2b) worries about the possible implications of another attack, and (3) a significant change in behavior related to the attacks – for instance the avoidance of public spaces (Borsboom 2008).

If we interpret such symptoms according to the standard model, in which they are the consequence of an "underlying disorder" then our conceptualization corresponds to the representation left in **figure 3**: all symptoms can be traced back to a common latent cause. An alternative representation according to the network approach is depicted in the right panel of **figure 2**: (1) a person gets a panic attack in a public space, which causes him/her to (2b) worry about the possible implications of this and (2a) to worry that it might happen again with the result that (3) (s)he will not go outside anymore. Here, it is apparent how each symptom is caused by the previous symptom in a causal system.

This, of course, provides no answer to the question *why* someone has panic attacks or *how exactly* a panic attack causes worrying (there are also people that have

**Figure 3.** *Panic disorder as a latent variable that influences a set of observable symptoms (left panel) and as a causal network, in which symptoms influence each other (right panel)*
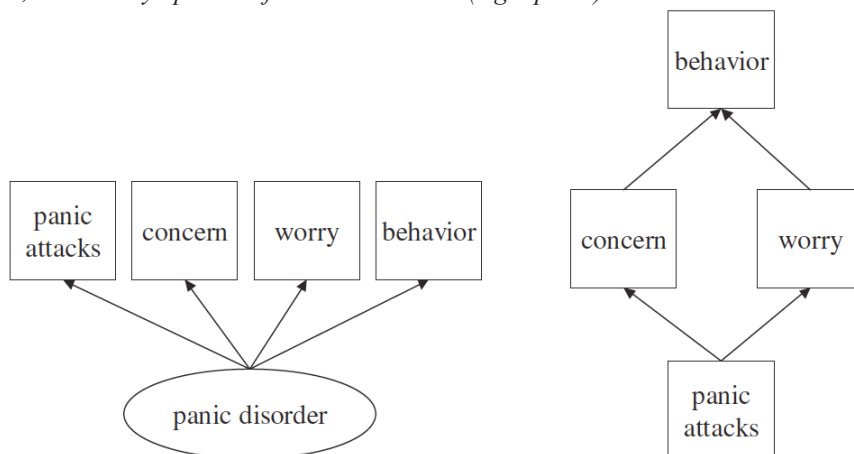
panic attacks but not a panic disorder: they may not have the connection between panic attacks and feelings of concern and worry). However, it does lead to a completely different approach to the question what kind of entity a disorder is and how we should investigate it (Kendler et al. 2010): in a network model the symptoms *themselves* are causing differences and changes in the way someone feels, thinks and acts; instead of a mysterious underlying disorder.

Based on this idea it is also relatively simple to create a simulation model that implements a disorder such as MD as a network of reinforcing symptoms. Van Borkulo, Borsboom, Nivard, and Cramer (2011) built such a system in the programming environment NetLOGO (Wilensky 1999). A simulation model such as the one in **figure 4** offers a scientific starting point for the studying of depression that did not exist before. That is, for the first time in history researchers can work with a dynamic formal model in which the etiology of MD can be modeled. To give one example: in this novel system one can investigate how the transition between a depressed and a normal state arises when the model is put under stress. Stress is modeled as *external* symptom activation. For instance, Anna does not sleep (symptom of MD) because she experiences pain (external factor).

The transition from normal to depressed in these simulations turns out to be dependent on the strength with which symptoms affect each other: the connectivity of the network. For weakly connected networks (in which the occurrence of one symptom does not have many consequences) the transition is continuous and linear: the more stress the worse the MD. However, for strongly connected networks (in which the occurrence of one symptom easily activates a new symptom) this turns out not to be the case. In these networks a very small increase in stress at a certain point can result in a complete depression. This conceptualization offers a new answer to the very old question whether MD is the end of a continuum or qualitatively differs from the normal state (Waller and Meehl 1998): this will differ between individuals, depending on the connectivity of the system of symptoms (Cramer et al. *submitted*, Borsboom et al. 2016).

Furthermore, it is interesting that under certain circumstances an episode of MD can arise through an increase of stress but does not disappear when the stress is decreased to its original level. This phenomenon, which occurs in strongly connected networks, is called *hysteresis*. In daily life we encounter this for instance in the phase transition in water, which freezes at 0 degrees Celsius but melts at +4 degrees. Possibly this hysteresis effect can explain why disorders such as MD are so hard to treat: some people get depressed very easily but getting out of the MD seem more difficult for them. On the other hand: if you wait long enough many simulated networks show spontaneous recovery. This too is a well-established phenomenon in clinical practice: many depressions just pass.

## Bad experiences and depression networks

So what can cause the dominos of a disorder to fall; what triggers the symptom network? In fact, this can be anything. Often there is a certain external event that triggers the development of problems. MD, for instance, is often preceded by an unpleasant experience (an *adverse life event*) such as chronic stress or the end of a relationship.

As the nature of those experiences differs an episode of MD can evolve in different ways just like Anna and Bart. Most people will intuitively sense this: the

**Figure 4.** *Screenshot of the NetLOGO depression simulation model. The model allows one to vary the parameters of a depression network and to put it under various levels of stress*
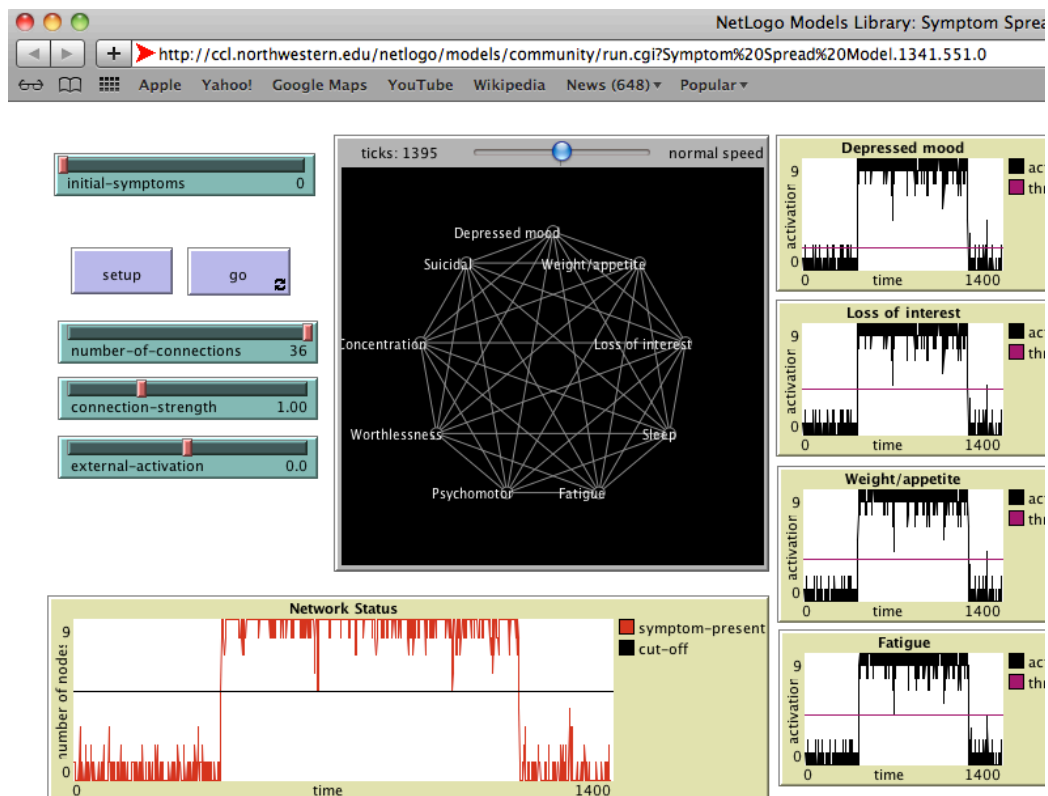
consequences of having trouble sleeping will differ from those of an ended relationship and the consequences of chronic stress will differ from those of pain. This can therefore lead to different network structures and different "routes" through the symptom network.

Keller et al. (2007) already showed that these differences can be detected in symptomatology patterns. For instance, MD preceded by chronic stress often includes tiredness whereas MD preceded by a break-up is more often characterized by sadness. These structural differences are hard to explain along the lines of a latent variable model in which the complaints are only indicators of an underlying disorder. Furthermore, Cramer et al. (2012) also showed that the mutual connections between symptoms differ as a function of the nature of the cause of the MD.

With a so-called measurement invariance analysis it is possible to establish whether these differences in symptom prevalence and correlational structure can be explained with a model in which MD is the common cause of the symptoms (i.e., the latent variable). This appears not to be the case: in research in which the effect of life events is modeled as mediated by a latent variable, the fit of such a model to the data is such that the model should be rejected (Cramer et al. 2012, Fried et al. 2015). In other words, both the prevalence of symptoms and their connections differ depending on the events that precede a MD and this difference cannot be explained by differences in a possible latent variable. This suggests that the network approach as compared to the latent variable approach does a better job in explaining the differences in symptom patterns, although more research is needed before we have a clear picture of this. This research should mainly focus on following the dynamics of symptoms over time.

## Comorbidity

Psychopathological research is largely organized along the DSM categories. Some studies are focused on MD, others on fear, and again others on schizophrenia. In this design psychological disorders are presented as independent categories. A big theoretical and practical problem for this approach is that certain symptoms of two supposedly distinct disorders appear together that often that research about these disorders can hardly be conducted independently. This co-occurrence of disorders is called comorbidity. Comorbidity is highly prevalent: the *National Comorbidity Survey*, a large representative study on mental disorders in the USA, showed for example that three quarters of the respondents who ever had a MD also met the criteria for at least one other disorder (Kessler et al. 2007). This

**Figure 5.** *Estimated network structure for generalized anxiety and major depression, as estimated on NCS-R data (Kessler et al. 2007). Figure reproduced from Borsboom and Cramer (2013), with permission*
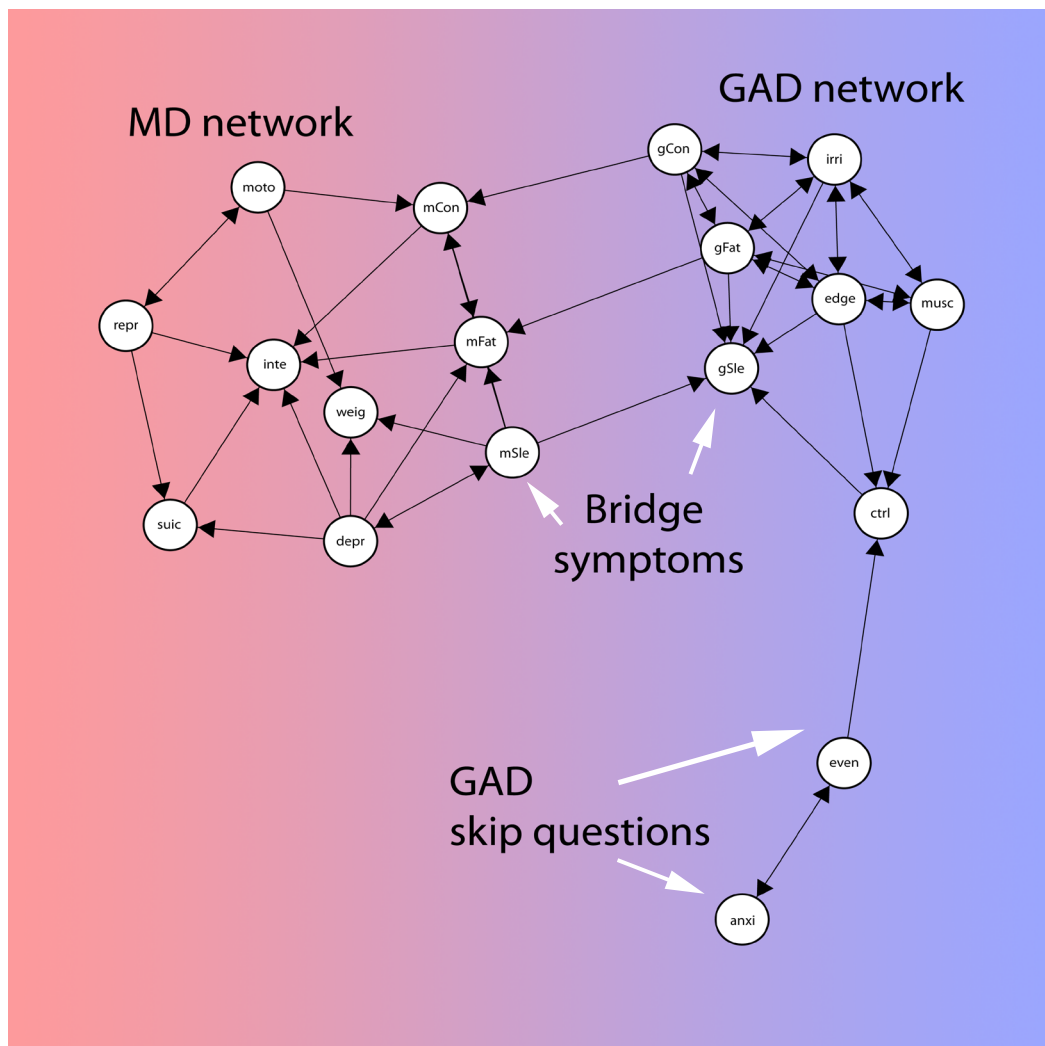
observation holds for most other disorders as well.

From the network perspective comorbidity can clearly be explained with so-called bridge symptoms. Bridge symptoms are symptoms that occur in two or more disorders (Cramer et al. 2010). In the DSM-5 such symptoms are often criteria for multiple diagnoses. Take for instance the prevalent comorbidity between MD and generalized anxiety disorder. These disorders have four important symptoms in common: sleeping problems, tiredness, restlessness, and concentration problems. These symptoms play a role in two networks at the same time and can therefore cause the effects of these problems to "spill over" to the other side of the network.

This is consistent with empirical data. For example, **figure 5** shows an empirically established network in which symptoms are depicted as connected when they have a significant partial correlation (the correlation between two symptoms when we control for the influence of all other symptoms); this can be interpreted as a clue for the existence of a direct relation. The figure shows that the MD and anxiety networks are indeed connected by their common symptoms. From this perspective, it is no wonder that the comorbidity between anxiety and MD is excessively high.

## Networks and research

When problems spread through a network structure there is no "underlying disorder". Instead, symptoms synchronize much like a flock of birds or a set of coupled ecosystems. This idea offers possibilities for the way we investigate psychological symptoms.

At the moment a lot of clinical research is focused on finding a genetic or neurobiological cause for psychological symptoms, for instance with brain scans or genetic research. This type of research costs a lot of money and has not led to any clear results to date. This lack of substantial results can be explained with the network theory: there *is* no physical central cause other than the physical realization of the symptoms and their connections.

Furthermore, we are used to measure the severity of for instance MD through questionnaires such as the *Beck Depression Inventory* (BDI, Beck et al. 1961). This type of questionnaire is meant to measure a latent trait as reliably as possible. To this end the questions are meticulously formulated (often in multiple ways, for instance both indicative and contra-indicative) and all kinds of statistical tests are performed to establish the internal consistency of the test.

If we retain the network model this practice could be changed and improved substantially. For instance, we no longer have to spend time and money on constructing questionnaires as internally consistent as possible; after all, we do not have to measure a latent trait. In the network model we are primarily interested in the relation between symptoms. Accordingly, it is much more important to measure *those* reliably. This shifts the focus from the individual symptoms to the connections between them. Frewen et al. (2011) developed a methodology by which the effects of symptoms on each other can be assessed through self-report. In addition, Bringmann et al. (2013, 2015) developed ways to assess patterns of symptom-symptom influence through time series analysis of experience sampling data. Wigman et al. (2015) showed that these patterns differ meaningfully among patients diagnosed with different disorders.

## Fighting symptoms?

The network model also offers new implications for the treatment of psychological symptoms: successfully targeting symptoms that are influential in the network should in itself lead to improvement of the status of the entire network. One could also focus on 'weakening' crucial connections that cause symptoms to occur repeatedly. Before we can start such a "symptom target" treatment we need to be able to draw everyone's personal network of symptoms.

A promising way to do this is through a digital monitoring system. In such a system the development of separate symptoms is monitored over time. To do this, a practitioner creates a patient-specific questionnaire including statements concerning the symptoms the patient is suffering from, such as "I am tired" or "My concentration is low". The patient then indicates at regular time points to what extent the statement reflects his or her feelings at that moment. This individual approach could elucidate how often specific symptoms occur and whether they co-occur regularly with other symptoms. Based on this information, the practitioner could estimate a personal network.

In such a network we can see which connections are particularly strong and which symptoms play a central role in the network. If we can repress such symptoms with specific treatment interventions, we may stop the spread of influence through the network structure. In the best case scenario, this would switch off the activation across the entire network as a result. Take for instance the case of Anna again. Her depressed feelings began with a chronic pain that caused sleeping problems. If one were able to successfully take away the pain, her sleeping problems may diminish, and her concentration might improve as a result; perhaps, she would do better at work again, and her feelings of guilt would disappear.

This approach could offer many advantages. Practitioners are no longer bound to heterogeneous diagnostic categories but can look at the development of a symptom pattern in detail. Of course it has always been possible to tailor treatments to an individual, but there was no solid methodology for that. We can now start developing such a methodology (see also Wichers 2013).

More fundamentally, we have reached a point in which we do not need to reduce complex systems such as psychological disorders to a single concept. We can approach disorders in their full complexity: psychological disorders *are* sets of symptoms and their interrelations. We may no longer need to establish diagnoses that may fit the group data but do not fit anyone in particular, we may not need to make assumptions about invisible entities hidden in the mind, and we may not need to search for a single underlying cause of disorders and their comorbidities. Instead, we may create monitoring systems with which we can establish personal networks for the individual to describe and model their specific problems and the way they interact. The most important symptoms of the network may be tackled directly, hoping that countering them will have positive effects across the network structure. Above all, we do not need underlying latent disorders to explain psychological symptoms: the disorder is nothing more – but also nothing less – than a causal network of observable symptoms.

Recent empirical evidence suggests that the network perspective provides an adequate and promising approach to study several psychological disorders (Wichers 2013) such as MD. First, intra-individual analyses of multivariate time series data have

demonstrated direct relations between variables that are related to psychopathology (Bringmann et al. 2013). Second, symptoms of MD display distinct responses as a consequence of major life events (such as the loss of a loved one; Cramer et al. 2012) and show different relations to other external variables and to other (distinct) disorders (Lux and Kendler 2010). Another study showed that the direct effects of a specific adverse life event on particular symptoms are inconsistent with the predictions of latent variable models, but seem intuitively likely from a network perspective (Fried et al. 2015). Finally, using recently developed self-report methods, individuals with elevated symptom levels typically report causal interactions between their symptoms of anxiety, posttraumatic stress and MD (Frewen et al. 2012).

After the theoretical consolidation of the network paradigm, the inevitable next step lies in the investigation of the clinical utility. Can we, for example, predict critical transitions from being healthy to being depressed? Research on dynamical systems has shown, for example, that positive feedback loops among causal relations can cause a system to have alternative stable states separated by a so-called tipping point (Scheffer et al. 2009). Earlier research in ecosystems has shown that a large perturbation of the system (i.e., a treatment intervention in our case) might have more impact if timed very near a tipping point instead of further away from that point. Based on this finding, Van de Leemput et al. (2014) have shown that mood system also displays signals of critical slowing down, a phenomenon that emerges when a dynamic systems approaches a tipping point. Clinical relevance of the network approach has also been suggested by results showing that mental state network connectivity could serve as an indicator for individual vulnerability to psychopathology. Wigman et al. (2015) have shown how momentary assessment techniques can be used to expose transdiagnostic processes. Examining an individual's network structure could lead to more individualized treatment strategies sensitive to individual differences in symptom patterns and vulnerabilities.

In conclusion, conceptualizing psychopathology as causal systems provides a promising complement to traditional models and diagnostic classification systems. The network paradigm, combined with momentary assessment technology, offers unparalleled opportunities to expose the underlying structure of psychopathology and develop more personalized treatment schedules.

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