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Chapter 1

Network Perspectives

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1.1 Introduction

The application of network thinking to psychometric questions has led to an eruption of network approaches in several subfields of psychology, most notably clinical psychology and psychiatry (Robinaugh et al., 2020) where the idea that mental disorders are composed of interactions between components (symptoms or other problems) in a multifactorial system is plausible (Kendler et al., 2011). In many such cases, the statistical application of network models to empirical data is motivated by theoretical concerns: substantive considerations that render the conceptualization of a construct as a network plausible (e.g., because causal connections between relevant variables stand to reason; Cramer et al., 2010; Dalege et al., 2016; Isvoranu et al., 2016; Lange et al., 2020). This entanglement of statistical modeling and substantive concerns is typical of the network literature, and

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arguably in part responsible for its success: the combination of systems thinking with methodological tools to analyze empirical data are important drives behind the popularity of network approaches. However, the close relation between substance and statistics can also lead to problems, because the distinctions between statistical, conceptual, substantive issues are not always clear. Thus, one may unwittingly mistake conceptual questions for statistical ones, or statistical questions for substantive ones, which may hamper progress. The present chapter aims to make the distinction between substantive network theories and statistical network models explicit, and to discuss ways in which they can be connected.

Consider the network approach to psychopathology. Network theory posits that symptoms of psychopathology are causally related (i.e., activating one symptom increases the likelihood of connected symptoms to arise; Borsboom et al., 2017). However, this is not an assumption that underlies the estimation of the popular pairwise Markov random field (PMRF) as explained in Chapter 6. The assumptions of the estimation procedure are purely statistical and hold only that the joint probability distribution can be described by a set of main effects (node thresholds) and pairwise interactions (edges). Because the network theory and the network model are not equivalent, it is important not to confuse assumptions of one for assumptions of the other. Such confusion arises, for instance, when researchers think that application of the network model requires that the network theory is true. This is incorrect: the PMRF, as network *model* can be successfully estimated in situations where the network *theory* is false; for instance, when all dependencies in the data arise from a latent common cause rather than from causal interactions between components (Fried, 2020; Marsman et al., 2017). In this case, mistaking theoretical assumptions for statistical ones may inadvertently hold back the researcher, who may incorrectly think that it is unjustified to apply a network model in cases where relations between network components are not causal in nature. The converse problem arises when researchers think that the successful application of network models indicates that network theory is true. This is incorrect because the network model only contains statistical relations, and the interpretation of such relations in terms of causality requires a stronger inference than data analysis by itself can provide (i.e., one has to provide causal assumptions as well; Pearl, 2010). Interpretation of network results in terms of network theory may, in such cases, overstep the evidence (Bringmann et al., 2019; Epskamp et al., 2017; Fried, 2020).

For these reasons, it is crucial that the student of network analysis learns to distinguish between different ways in which network thinking can be applied, and to get a clear view of the distinctions between theory and statistical modeling. This chapter aims to clarify these distinctions by discussing three ways in which network thinking may be methodologically useful. First, we discuss network approaches, which simply entails 'viewing' a construct as a network of interacting components as a way of developing one's thinking and creating new ways to understand and investigate the construct. Second, we describe psychometric network models (hereafter, referred to simply as network models): systems of statistical relations between variables defined on empirical data. Third, we describe network theory, where both the components of the system and their connections are substantively interpreted and specified, so that they are able to explain characteristic phenomena involving the construct. We then discuss how these conceptualizations of networks are related, identifying network models and network theory as specializations

under the rubric of a network approach that, ideally, serve to inform one another. Finally, we analyze a number of strategies that have been applied to connect network theory and network models.

1.2 Network approaches

The network approach to a given empirical domain simply entails conceptualizing that domain as a network: that is, as a set of components and the relationships among those components. Central to this approach, which others have referred to as 'systems thinking,' is the notion that network behavior is closely tied to network structure (Meadows, 2008). The network approach often entails conceptualizing a given phenomena of interest as an emergent property, with the components of a network 'working together' to generate emergent phenomena that feature surprising levels of organization (Barabási, 2012; Wright & Meadows, 2012). For example, consider the complex and seemingly highly organized behavior exhibited by a flock of birds. A network approach conceptualizes such behavior as an emergent property arising from the interrelationships among the individual birds that constitute the flock. This same lens can be applied to numerous empirical domains. For example, we can apply it in the domain of psychiatry, considering whether the behavioral characteristic of the depression syndrome (i.e., chronically elevated depression symptoms) might arise from interrelationships among the components of the syndrome (i.e., the symptoms themselves). This process can be facilitated using *analogical abduction* (Haig, 2014): establishing a systematic correspondences between a source domain (e.g., a flock of birds) and a target domain (e.g., a set of depression symptoms) so that one can use explanatory models from the source (e.g., flocking models that explain why flight courses of birds are correlated) to better understand phenomena in the target (e.g., symptom network models that explain why depression symptoms are correlated). Indeed, because similar features are consistently observed across networks taken from a wide range of empirical domains, the network approach provides fertile ground for productive analogical abduction (Barabási, 2012; Scheffer, 2020).

When one is initially considering the viability of network approaches, it is useful to consider the applicability of its central concepts. Can one identify at least some of the important components and links between them? Do these components behave as a coherent whole? Do the links between them offer *prima facie* plausible explanatory resources, in the sense that a network structure would 'make sense' of a given behavior or pattern of findings (e.g., correlations in one's data)? Do we see synchronized behavior that may emerge out of a network structure? Are there signs that the system shows behavior commonly exhibited in complex systems, such as non-linear behavior in which there are sudden changes in the state of the system (Scheffer, 2020)? Systematically investigating such questions can help in assessing whether a network approach is plausible and worth pursuing (Fried & Robinaugh, 2020).

If these or other initial considerations suggest the network approach may be a suitable conceptual framework, the researcher can put on a pair of network glasses and start exploring whether network science may offer further possibilities. When embarking on such a discovery process, one typically does not make a particular choice on exactly

which components are in play or how they work together; rather the researcher chooses to view an empirical domain through the 'lens' of networks. Thus, network approaches usually do not single out precisely which factors act as components in the system at the outset, nor do they typically specify precisely how they interact. Instead, these are thought of as open questions worthy of discussion and research. For example, in the network approach to psychopathology, symptoms enumerated in diagnostic criteria for a given disorder were put forward as possible components of the network structure. However, it has subsequently been argued that this viewpoint may be too narrow and that other components should be included as well (Fried & Cramer, 2017; Jones et al., 2017). Similarly, in the mutualism model of intelligence, interactions between cognitive processes were posited (van der Maas et al., 2006), but the processes themselves were not directly identified. In both these examples, network approaches were initially 'open'; with the components and links between them subject to discussion and research.

As one is considering which components may be present in the network and how they may relate to one another, it is important to recognize that the nature of components and links is not uniform across empirical domains. Indeed, in network science we see a diverse set of network components: organizations, species, actors, neurons, computers, geographical areas, and medical diseases, to name only a few. There is similar diversity in the connections between these components, with links defined by e.g., financial loans (between organizations), competition (between species), co-occurrence (between actors in movies), physical connections (axons connecting neurons), information exchange (between computers), travel (between geographical areas), and comorbidity (between psychopathological conditions or medical diseases). Thus, it is important to realize that network approaches do not require a particular physical structure, but rather require the applicability of a mode of representation, and therefore the question whether a domain 'really is' a network is often moot. The reason that network approaches are so generative and interesting is precisely that they can apply to different systems in different ways.

1.3 Network models

If a network approach to an empirical domain is taken, the researcher will arrive at a (possibly rough) idea of which components would plausibly feature in a network. A natural next step is to assess which of these components are linked. In some cases, one can observe network relations directly (e.g., in traditional social networks, friendship links between individuals are typically treated as 'observed'). In other cases, links between nodes can be assessed on the basis of prior research. For instance, Wittenborn et al. (2016) constructed a network model for depression based on existing knowledge about relations between relevant components. However, in many cases connections are not directly observable and existing research is insufficient to assess whether links are present. In such cases, one can assess how relevant components covary empirically. It is for this purpose that *network models* are especially useful.

Network models are statistical structures that characterize a multivariate probability distribution as a network. In a network model, nodes represent *variables* and links between nodes represent *statistical relations* between these variables. Note that variables

are typically abstract entities, not concrete things, and statistical relations are estimated, not observed. In this sense, network models as used in the approach covered in this book are different from, for instance, social networks in which network nodes represent concrete individuals and relations between individuals are typically observable (or treated as such).

The simplest way of assessing the network structure in a set of variables is to simply calculate correlation coefficients between variables, and interpret these as (weighted) links between nodes (Epskamp et al., 2012). However, a downside of this approach is that, *if* data are indeed generated from a network of pairwise interactions, the correlation matrix will include many spurious links. For instance, if variables A and C are linked through B, but not directly, a correlation between A and C will nevertheless be observed. A solution to this problem is to calculate the correlation between A and C while conditioning on ('controlling for') B. This approach will correctly return the network structure, because the correlation between A and C will vanish (van Borkulo et al., 2014). Generalizing this idea, one can compute the association between any two variables while conditioning on all other variables in the data. This then returns an estimate of the PMRF (see Chapter 6). In such a PMRF, the absence of a link between nodes represents conditional independence between the corresponding variables and the presence of an edge represents conditional dependence. These PMRF models, also known as graphical models in the field of statistics (Cox & Wermuth, 2014), are the type of network model used most often in this book.

Like any statistical model, network psychometric models are generic: they are not about any given substantive phenomena but rather are tools for representing patterns in data. Because these models are generic, they are not about any empirical application in particular. This is obvious from the fact that PMRFs, for instance, have been used in a wide range of research domains, ranging from physics to neuroimaging all the way to clinical psychology. This generality makes network models useful because it allows researchers to construct software that can estimate network models across many different contexts. However, it also means that the interpretation of statistical network models in terms of an empirical domain requires assumptions. For instance, if one wants to interpret the model in terms of causal relations between network components, this requires one to buy into a nontrivial set of assumptions about the data-generating process. One has to be prepared to assume that measurements of the components have sufficient reliability and validity, that causal relations between components are indeed bidirectional and symmetric, and that one has succeeded in capturing the most important components of the network in one's data. Thus, the interpretation of psychometric networks in terms of conditional associations relies only on statistical model adequacy, but the interpretation that such networks represent the targeted attributes and causal relations between them requires additional assumptions of a theoretical nature.

The application of network models shows which variables are so strongly associated that the association cannot be explained away by other variables in the data, which yields an exploratory approach to detecting network structures. Models that operate in this way have become popular in recent years (Robinaugh et al., 2020); here, we highlight examples of how network models can characterize and visualize patterns of dependency in multivariate data, focusing specifically on the network approach to psychopathology.

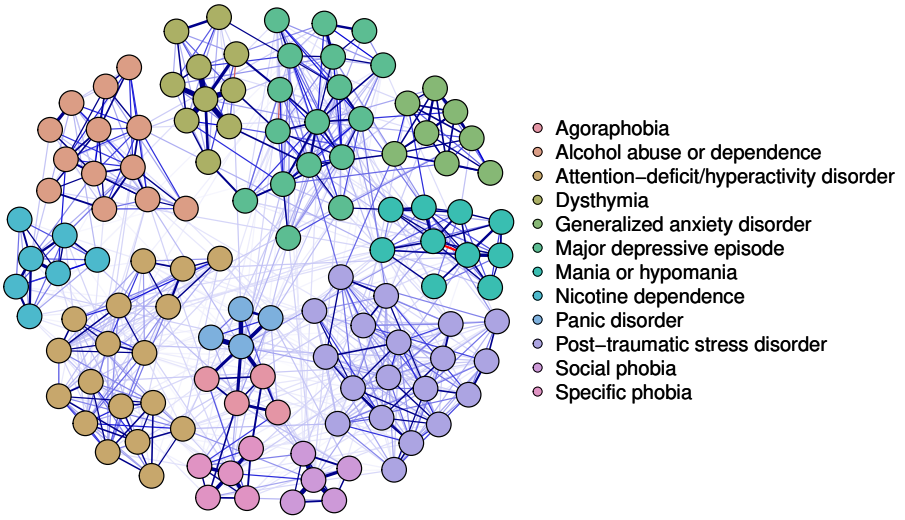

Figure 1.1. DSM-IV symptomatology network structure, adapted from Boschloo et al. (2015).

First, network models have been used to assess the structure of psychopathology networks. An example of this approach is given in Figure 1.1, which displays a network of symptoms defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, as estimated on a large U.S. community sample; Boschloo et al., 2015). As is evident from the figure, the great majority of symptoms feature positive pairwise interactions (i.e., the presence of symptoms increases the probability of connected symptoms, when controlling for the remaining variables). In addition, the figure shows that symptoms of the same disorder cluster more strongly, while these clusters are not neatly separated (i.e., symptoms between different disorders are conditionally associated). This is consistent with (but does not prove) the idea that comorbidity between disorders may arise through causal connections that cross-cut the borders separating different disorders in systems such as the DSM.

Second, network models have been used to study relations between psychopathology symptoms and external factors (Isvoranu et al., 2016). An example concerns the relation between child abuse and psychotic disorders. Figure 1.2 displays the network of psychotic symptoms (e.g., hallucinations, flat affect), general psychopathology symptoms (e.g., depressed mood, anxiety), and childhood trauma (e.g., sexual abuse, emotional neglect). The network shows all types of trauma are linked to positive and negative symptoms of psychosis through general psychopathology, suggesting general psychopathology symptoms as potential mediators between trauma and psychosis. This aligns well with research showing childhood trauma is connected to a wide array of symptoms present across many mental conditions, and is thus not specific to psychotic symptoms. Further, the trauma nodes themselves are highly interconnected, indicating that the effects of trauma to symptoms can also occur via other types of trauma. Overall, network models including external factors can provide information on how such external factors relate to each other, as well as give insight into potential pathways to a disorder state.

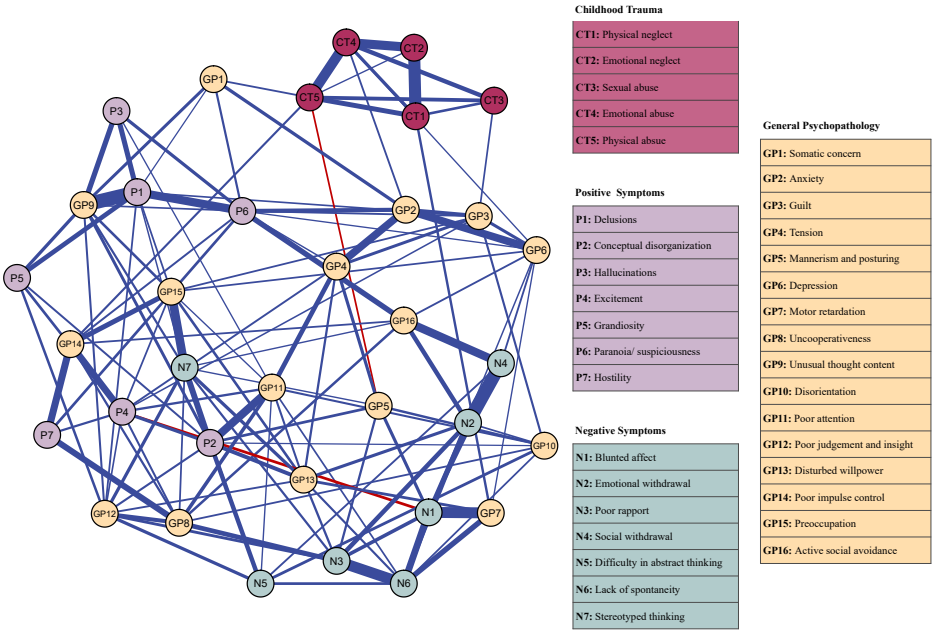

Figure 1.2. Network structure of positive and negative psychotic symptoms, general psychopathology symptoms, and factors involving childhood trauma, adapted from Isvoranu et al. (2016).

Third, network models have been used to examine relations between external shocks and psychopathology. For example, Figure 1.3 shows the relation between the loss of a spouse and a number of symptoms of depression (Fried et al., 2015). The analysis is suggestive of an indirect effect of losing one's spouse on depressive symptoms through feelings of loneliness. Thus, this type of application can lead to hypotheses on the way external shocks may impact the network, which can in turn inform theory formation and further empirical research.

In each of these examples, the application of network models sheds light on the structure of psychopathology networks and the possible ways in which they are influenced by external factors. By constructing such representations, researchers can inform the network approach taken through empirical data analysis. Network models are useful because they can function to inform the researcher with respect to the generic features of the network structure (e.g., is the network dense or sparse?), the position of particular nodes in that structure (e.g., central versus peripheral nodes), and the places in the network that are likely to be perturbed by particular external events (e.g., which variables in the network connect to external shocks?).

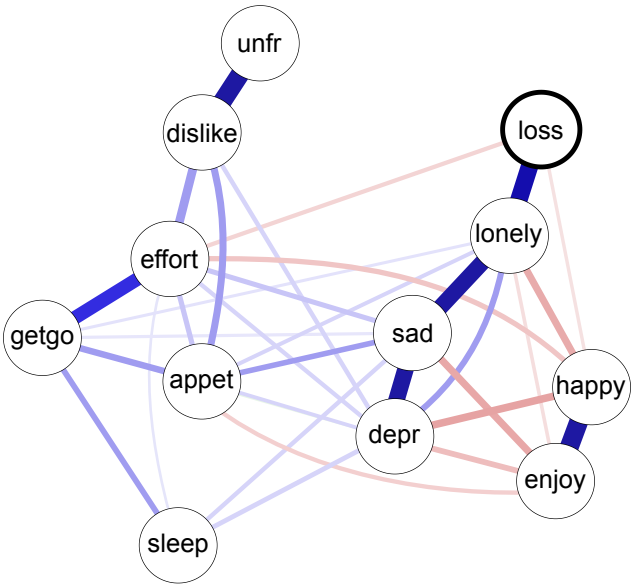

Figure 1.3. Network structure of depression symptoms, feelings of loneliness, and spousal loss, adapted from Fried et al. (2015).

It is important to note that statistical models have limitations. First, it is notoriously hard to establish causal relations from statistical models, especially in quasi-experimental and correlational settings (see also Chapter 12). Thus, although PMRFs may be suggestive of causal structure, they do not establish causal relations directly. Second, although in some circumstances network models *can* correctly estimate networks *if* the data are generated by a particular network model, this does not mean that the application of a network model *will* correctly estimate a generating network in general. For example, it is possible that the links between childhood trauma and psychotic symptoms in Figure 1.2 reflect the operation of an unmodeled common cause (e.g., a genetic disposition that increases both the likelihood of trauma and the likelihood of psychotic symptoms). Third, network models are often explorative statistical tools that try to optimize a search through a very large model space. In general, this means that modeling results should be viewed as hypothesis generating, so that independent research is generally needed to test the hypotheses suggested by the network model. Thus, exploratory uses of network models generate hypotheses, which open up a line of research, rather than firm conclusions that confirm a theory.

In sum, network models are explorative tools that can serve to chart the rough outlines of a network based on empirical data. This renders them highly useful for informing network approaches empirically. By conjoining such empirical results with theoretical considerations, network approaches can gradually become better informed and as such can form the groundwork for building network theories.

1.4 Network theories

Network theories specify a set of components and interactions among components and posit that the resultant network can explain a phenomenon of interest. Network theories are distinguished from simply adopting a network approach by the specification of components and inter-component relationships in the network and by the presence of falsifiable conditions. In other words, whereas the network approach is a lens through which to view a construct of interest, a network theory goes further: it posits what the relevant entities in the network are, it posits how they interact, and, in doing so, it makes predictions that can be corroborated or falsified.

For example, a network approach to depression would entail looking at depression as a system of components and considering whether those components may work together to produce depression akin to the way in which individual birds interact to produce flocking behavior. Such a network approach may offer a tentative set of components and a rough idea of the relations between them, but these are typically sketchy and open-ended. For this reason, network approaches have no clear falsification conditions; they represent a theoretical perspective, rather than a theory. A network theory goes further: it identifies what the relevant entities in the system are and how they interact. These specifications are made by identifying the component set (i.e., deciding which components to include in the network), the type of interaction between components (i.e., specifying what the links in the network represent), and the phenomena to be explained by the theory. The greater the specificity with which the components and inter-component relationships are expressed, the better equipped theorists will be to determine precisely what behavior is predicted by the theory and, thus, the better equipped they will be to determine whether the theory can indeed account for the phenomena of interest. For example, in the context of depression, a very simple kind of network theory could state that depression arises from instantaneous pairwise causal interactions among DSM-5 symptoms. This theory generates an *interpreted* network structure: rather than being an abstract conceptual framework or a statistical model for a data set, the network structure now aims to represent particular entities (DSM-5 symptoms) and their interactions (i.e., causal ones). To strengthen this theory, one can go further, identifying the functional form of those interactions (e.g., linear or nonlinear) and time scale on which they take place (e.g., minutes, days, years). By doing so, theorists can evaluate whether the network theory can indeed account for the core phenomena of interest in depression research: the tendency for depression symptoms to cohere and persist as a syndrome in some individuals.

Naturally, while this step of model interpretation is an important phase towards the construction of a theory, it does not magically render an *adequate* theory. For instance, as previously noted, DSM-5 symptoms may not be optimal components (Fried, 2020; Jones et al., 2017) and several of the observed correlations between them may not be causal but reflect, for instance, semantic overlap or the influence of unmodeled common causes (Fried & Cramer, 2017). Here again, it is important to note that many different choices are possible in the identification of component sets and interactions. For example, the mutualism theory in intelligence research is also a network theory, but it has an entirely

different set of components (cognitive processes and abilities) as well as a different type of interactions (mutualistically coupled growth rather than direct causal interaction).

Network theories are often used to explain particular types of empirical phenomena, such as correlation structures, bimodality, and hysteresis. We will examine some of these explanations in detail. First, a network theory of general intelligence has been proposed in van der Maas et al. (2006). The main phenomenon in the study of individual differences in intelligence is the so-called positive manifold, which is the well replicated fact that scores on sub-tests of intelligence tests tend to correlate positively. This phenomenon is traditionally explained by a general factor, which suffices from a statistical point of view in describing the data but lacks a generally accepted substantial interpretation. In the mutualism model, the positive manifold is explained by positive reciprocal interactions between many different cognitive functions during cognitive development. There is ample evidence for such interactions, of which only a few weak ones are needed to lead to the positive manifold data found in intelligence research (van der Maas et al., 2019). The mathematical model is simple. Cognitive functions grow logistically until some limited capacity. The speed of growth and the capacity are strengthened by mutual interactions. van der Maas et al. (2006) also proposed to add a simple linear model for the genetic and environmental effect on the limited capacities, such that the heritability of intelligence could be modeled. Interestingly, this addition to the model naturally leads to an increase of heritability with age, an important finding in genetic studies of intelligence (Haworth et al., 2010).

Second, the network theory of psychopathology (Borsboom et al., 2017) identifies the nodes in the network as clinical symptoms as encoded in diagnostic manuals like the DSM-5. The links between nodes are interpreted as (possibly, but not necessarily) reciprocal causal relations, such that connected symptoms may activate each other. For some disorders, such as panic disorder, such relations have been explicated in some detail (Robinaugh et al., 2019), while for others they remain sketchy. Similar to the mutualism case, this theoretical interpretation naturally explains the fact that psychopathology symptoms form a positive manifold (Caspi et al., 2014; van Bork et al., 2017). In addition, the theory is consistent with the presence of extensive comorbidity between disorders, as symptoms that belong to multiple disorders may act as bridge symptoms that transfer activation from one problem area to another (Cramer et al., 2010). Finally, the theory offers a potential explanation of the onset and maintenance of mental disorders. For instance, Cramer et al. (2016) found that strongly connected networks, in which symptoms easily activate each other, potentially generate sudden transitions into a disorder state, in which they can get stuck due to a hysteresis effect. This naturally leads to the conjecture that mental disorders *are* stable states of symptom activation in strongly connected symptom networks (Borsboom et al., 2017).

Third, a network theory of attitude dynamics has been proposed in Dalege et al. (2016, 2018). This network theory of attitudes relies on the central principle that pairwise interactions between affective, cognitive, and behavioral attitude elements increase when individuals pay direct attention to the attitude object and when the attitude is important to the individual. As is discussed in detail in Chapter 13, this principle explains several well-established findings in the attitude literature. For example, heightened interactions

between attitude elements lead to a more extreme attitude, because all attitude elements are pressured to assume the same state. This process provides a mechanistic explanation for the finding that attitudes become more extreme when individuals think about their attitudes (Tesser, 1978).

As these examples illustrate, network theories are explanatory structures aimed at improving our understanding of empirical domains. If successful, this increased understanding may contribute to improved prediction and control. For example, in the case of psychopathology, this would imply the development of better treatments; in the case of attitudes, better instruments of persuasion. Therefore, the construction of network theories is an activity with a deeply practical goal; namely, that of increasing both our understanding of complex multivariate processes and our ability to execute controlled interventions on these processes.

1.5 Relations between network approaches, models, and theories

The fact that network approaches, network models, and network theories are rarely separated neatly has the potential to generate confusion, because it is not always clear how they relate to each other. This problem has been exacerbated by the fact that the term 'network model' has been used to describe all three domains we list here: (a) a broad conceptual network model, (b) a psychometric network model, and (c) a theoretical network model. This section outlines how we can think about the relations among these three domains.

The relation between network approaches on the one hand, and network theories and network models on the other, is relatively straightforward: network approaches define an overarching category, and then further *specialize* into network theories and into network models. A very large set of phenomena can be viewed through the lens of networks, systems science, or complexity science. Nested within these approaches are numerous theories, and the methodological network toolbox in turn comprises many statistical models that lend themselves well to investigate phenomena as networks.

On its surface, the relation between network models and network theories is similarly straightforward. Network models are representations of one's data. In contrast, network theories are representations of the real-world components that one believes give rise to a phenomenon of interest. However, when we interrogate this relationship further and consider how network models can best inform, or even serve as the basis for network theories, things become less clear. There are two broad viewpoints we can consider on this matter.

The first viewpoint posits that one can view the relation between models and theories as direct. Here the transition from network model to network theory is simply an act of interpretation, interpreting the network model as a representation of the real-world system of interest. For example, if we fit a PMRF to the data of the DSM-5 depression symptoms assessed once in a sample of depressed patients, we will obtain statistical

relations among these symptoms. We may find, for instance, that 'loss of energy' shows the strongest relations to all other symptoms (or, in network science terminology, has the highest centrality). If we assume the statistical and theoretical models coincide, we can interpret the statistical model *as* a theoretical model, treating the structure of the model as the causal structure of our real-world network of interest. This interpretation, in turn, implies that loss of energy is the most obvious treatment target: the symptom has the largest number of connections; connections are causal relations, according to network theory; hence a successful intervention on loss of energy (i.e., one that persists in deactivating the symptom) should not only alleviate this problem, but also improve connected symptoms.

The direct interpretation of statistical models as theoretical models can be useful in situations where one wants to study the properties that a model could have but has no idea on how to parameterize it. For instance, Borsboom et al. (2011) filled in network relations gleaned from DSM-IV symptom overlap with parameters taken from statistical regression modeling to investigate whether such a model could explain comorbidity patterns (it could). Similarly, Cramer et al. (2016) used an Ising model (see Chapter 6 and Chapter 13) to investigate whether such a model could exhibit hysteresis effects (it did). As a proof-of-principle, they are highly useful because they show that certain phenomena are within the reach of the explanatory model and specify targets for further investigation.

However, a significant number of obstacles must be overcome to justify accepting such a model as a valid description of reality, rather than as a proof-of-principle, which we refer to as an inference gap between statistical and the theoretical domains (Fried, 2020). Broadly speaking, inferences follow from assumptions, and the assumptions for the above type of model are both numerous and implausible. We list but a few here. First, one must assume that a network process generated the data in the first place, yet there are numerous alternative causal mechanisms that can give rise to data that, if analyzed with network models, produce a network—a problem known as statistical equivalence (Fried, 2020; van Bork et al., 2021). More fundamentally, just because one can fit a regression line through the joint distribution of two variables does not mean the resulting coefficient represents a meaningful causal effect, whether in network models or any other statistical model. Second, one must assume that the undirected edges of the PMRFs indicate bidirectional and symmetric causal relationships. However, an undirected edge in a PMRF could arise from a variety of sources. For example, loss of energy could be the causal endpoint of all other symptoms it is connected to, rather than a causal origin. If this were the case, interventions on such a causal endpoint should not be expected to propagate through the system at all. Third, one must assume that the network model estimated from data of multiple people measured once coincides with the within-person structure of causal relationships among depression symptoms. However, this assumption holds under circumstances unlikely to be observed in psychological research (Molenaar, 2004; further discussed in Chapter 9). Further, there is reason to believe that network processes differ (at least to some degree) across people with depression, which would mean that the estimated network structure at the between-subjects level may not be informative about interventions at the individual level (Fisher et al., 2018; Fisher et al., 2017; Fried & Robinaugh, 2020; Henry et al., 2021). Fourth, the PMRF used in the above example estimates linear, pairwise relations. If more complicated processes

are part of the data generating structure, such as non-linear dynamics or higher-order interactions, the estimated structure will be biased or incorrect (Haslbeck et al., 2021). Many further assumptions, are described in detail elsewhere (Fried, 2020; Haslbeck et al., 2021; Robinaugh et al., 2020). Drawing inferences from network models thus requires spelling out the auxiliary assumptions a researcher brings to the table that are necessary to make these inferences work. Doing so transparently enables readers to vet whether the assumptions are sufficiently plausible to warrant the conclusions.

The second viewpoint on the network model-network theory relationship posits that the relationship is indirect. From this perspective, the network model is seen as an 'intermediary' between the theory and the empirical data: a data model that organizes multivariate dependencies in an optimal way for network theories to latch onto. Network models provide rich information that serves to inform and constrain the development of network theories, but the theories have a structure that is independent to that of the network model and may include features (e.g., asymmetric and non-linear relationships) not present in the network model. If the theory is able to explain the observed network model derived from empirical data, our confidence in the theory is strengthened. If it fails, we can reason about the best explanation for this explanatory failure, and use this abductive inference to guide improvements to the theory that bring it in line with the network model and other findings from empirical research.

The advantage of the second viewpoint is that it does not require adherence to assumptions that may be implausible in a given domain, such as those reviewed above. The challenge in this perspective is that the systems that give rise to psychometric constructs like intelligence, personality traits, or mental disorders are almost certainly highly complex and heterogeneous biopsychosocial systems whose structure is extremely resistant to discovery. Indeed, a precise and detailed explication of these systems may not be feasible, at least in coming years. This, in our view, does not diminish the role that theories must play in our efforts to unravel the complex systems that give rise to psychopathology. To the contrary, we believe this difficulty elevates the importance of theories. Efforts to simply uncover the structure of such highly complex systems from data models alone will be especially (indeed, perhaps prohibitively) difficult. The key, in our view, is in leveraging the advantages of theories and data models that, by themselves are limited, but can serve to inform and advance one another over time. To illustrate, consider the role of smoking and lung cancer. The statistical relation between smoking and lung cancer, first observed in cross-sectional data and estimated via simple statistical models such as linear regression, did not provide insights into mechanisms, and did not perfectly capture individual relations between the constructs. It failed to take complex interactions with many moderating factors into account. Nonetheless, it was crucial for our research into the causal mechanisms that govern the relationship and the development of theories that could explain this association. Newton's theory of universal gravitation is another example. Newton's theory describes and predicts the motion of the planets in our solar system well, but has important explanatory failures in special cases, e.g., when strong gravitational fields and small distances come together. Because these explanatory shortcomings were clear, a path for improvement was clear and Newton's theory was eventually superseded by Einstein's theory of general relativity, which more accurately explains planetary motion in these special cases. In this way, Newton's theory was not the final word, but a

critical stepping stone toward a theory that could provide a more comprehensive account of the known phenomena. Analogously, initial network theories developed to account for robustly observed network models and other empirical phenomena are all but guaranteed to have important explanatory shortcomings, but can hopefully lay the groundwork for better empirical research and stronger network theories that address these shortcomings. For example, an initial network theory of panic attacks introduced in Robinaugh et al. (2019) will not be the final theory of this phenomenon, but provides a starting point to evaluate what the theory can explain and what it cannot, thereby enabling further theory development and identifying empirical research that must be conducted if we are to further inform the theory.

The direct and indirect paths to connect network theories to network models have somewhat distinct implications for how to proceed in applying the network approach to a given empirical domain. If one is sufficiently confident that one has access to the important nodes in the system, and prepared to assume that the relations between them indeed reflect symmetric causal interactions, then the direct interpretation of the statistical model can be feasible and may be preferable. In such cases, a well-developed theory requires only a well-executed empirical research effort in which a set of components are rigorously measured and the structure of relationships among them is robustly estimated. Even in cases where the assumptions required for direct inference are violated, however, it may be that these methods provide a sufficiently faithful representation of the real-world network that it can be useful for advancing our understanding of that network and informing our ability to predict its behavior and intervene upon it; for instance, by providing 'toy models' that can reveal interesting possibilities for further modeling and research. This direct route then emphasizes the importance of rigorous data collection, precise estimation of network models, and research evaluating how well the inferences made from these models can give us purchase in understanding, predicting, and controlling the phenomena that arise from that network. With improvements in network data collection procedures and in the network model methodological toolbox, this direct route may become increasingly plausible.

The indirect path to connect network theories to network models calls for a somewhat different approach, arguing that practical, ethical, and technical limitations may render the prospect of data and models that can directly estimate the causal system unrealistic. In this case, the route forward may not lie primarily in data and model improvements, but in establishing phenomena that are sufficiently robust against model misspecifications and data problems that they can serve as explanatory targets with which to inform and validate theories. For example, we may never gather data good enough to distinguish between different functional forms for the relation between, say, insomnia and depressed mood (e.g., linear, logistic, power functions, etc.) but the fact remains that, whatever form we choose, they remain positively associated. Thus, at a higher level of abstraction, robust phenomena can be identified even if their precise details remain obscure. These phenomena can be represented in many different data models, including networks (Haslbeck et al., 2021) and the challenge of moving from network models to network theory lies in the construction of plausible theoretical models that are able to account for these (Borsboom et al., 2021). Thus, this route towards improvement relies on robustness analyses and theoretical progress. The challenge for this indirect path will be to determine

whether these more precise efforts at theory specification can improve upon the insights we can gain from the rough but more accessible direct path of treating network models themselves as theories. In other words, for both the indirect and direct paths, the ultimate arbiter of success will be whether these theories are able to support the pragmatic aims of explaining, predicting, and controlling the phenomena of interest. Because it is likely to be a longer and more resource intensive process, it will be incumbent on those taking this indirect path to demonstrate the added value it provides in moving toward these practical aims.

Naturally, the two routes sketched here are not mutually exclusive, and progress is most likely to ensue by pursuing them in parallel. Ideally, this also leads to productive interaction between theoretical and empirical advances, where, in a true mutualistic fashion, each can inform and stimulate the other's growth (Chang, 2005).

1.6 Conclusion

In this chapter, we have presented and analyzed the different ways in which networks can be used in scientific research. First, networks can offer a novel perspective on empirical domains, just because they generate a different way of looking at the domain. Especially in areas where the classic method of isolating parts of the system for empirical study are hard, because everything seems to be connected to everything else, adoption of a network approach can be fruitful. Second, one can further inform such network approaches through the application of network models, which are statistical techniques to assess conditional dependence relations between putative components of the network. Such techniques are useful because they can broadly identify connectivity patterns from empirical data, and thus can inform network approaches to a higher level of empirical adequacy. Third, through a suitable combination of theoretical principles and empirical information, network approaches can be further specified into network theories: systems in which both the components of the system as well as their interrelations are specified to a level of detail that allows one to explain empirical phenomena. Ideally, the development of such theories will increase our understanding of the system studied, inform our empirical efforts to investigate it further, and strengthen our ability to plan controlled interventions on the system.

1.7 Exercises

Conceptual

1.1. Consider the following statements:

a "Suppose that Alice displays two depression symptoms—depressed mood and loss of interest—while Bob displays two other depression symptoms—psychomotor retardation and weight problems. On an intuitive level, it is plausible that Alice's symptoms are more likely than Bob's to eventually result in a full-fledged depression." (Cramer et al., 2010)

b "Most links we identified in our data are links between the polygenic risk score and positive psychotic symptoms, especially symptoms related to notions of conspiracy and paranoia." (Isvoranu et al., 2020)

c "We found that particular symptoms such as loneliness, sadness, and loss of appetite were especially elevated in the context of bereavement, and that the effects from loss on these symptoms were not conveyed via a latent variable, but through a network. Loneliness played a key role: bereavement mainly affected loneliness, which in turn activated other depressive symptoms." (Fried et al., 2015)

Discuss for each of these cases whether they are best interpreted in terms of a network model or in terms of a network theory.

1.2. Choose a construct that you think may be plausibly represented as a system. Suggest a putative set of system components and relations between them, and evaluate whether a network approach would be feasible. Next, describe how one could gather data to estimate a network model empirically. Do you think the estimated model would be directly interpretable as a network theory? Why (not)?

1.3. Search the literature for a paper that applies network approaches to an empirical topic. Identify the main conclusions of the paper. Are these conclusions best interpreted as being about network theories or about network models? Why?

1.4. Network theories and network models do not have a one-to-one relationship. One can for instance test implications of network theories without using network models at all. Can you think of examples?

1.5. Network models can be used in cases where network theories are known to be false. For instance, suppose one hypothesizes that a set of variables is determined by a latent variable (a common cause) and there are no direct relations at all between the variables. From this hypothesis, it is possible to deduce the implied network model for the observed variables, which can be used to test the latent variable hypothesis. What is your intuition about the network model that would be implied in this case? Would it be expected to contain many edges, a few, or none?

True or false

1.6. Network models can be thought of as tools for representing patterns in data.

1.7. To apply network models, one needs to assume that a network theory is true.

1.8. The difference between a network approach and a network theory is that a network theory aims to specify precisely which components feature as nodes in the network and what the links between them mean.

1.9. In network models, a link between two nodes represents a causal interaction.

1.10. If one interprets an estimated network as giving a truthful picture of how variables influence each other, one follows a *direct* interpretation of the relation between network models and network theory.

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