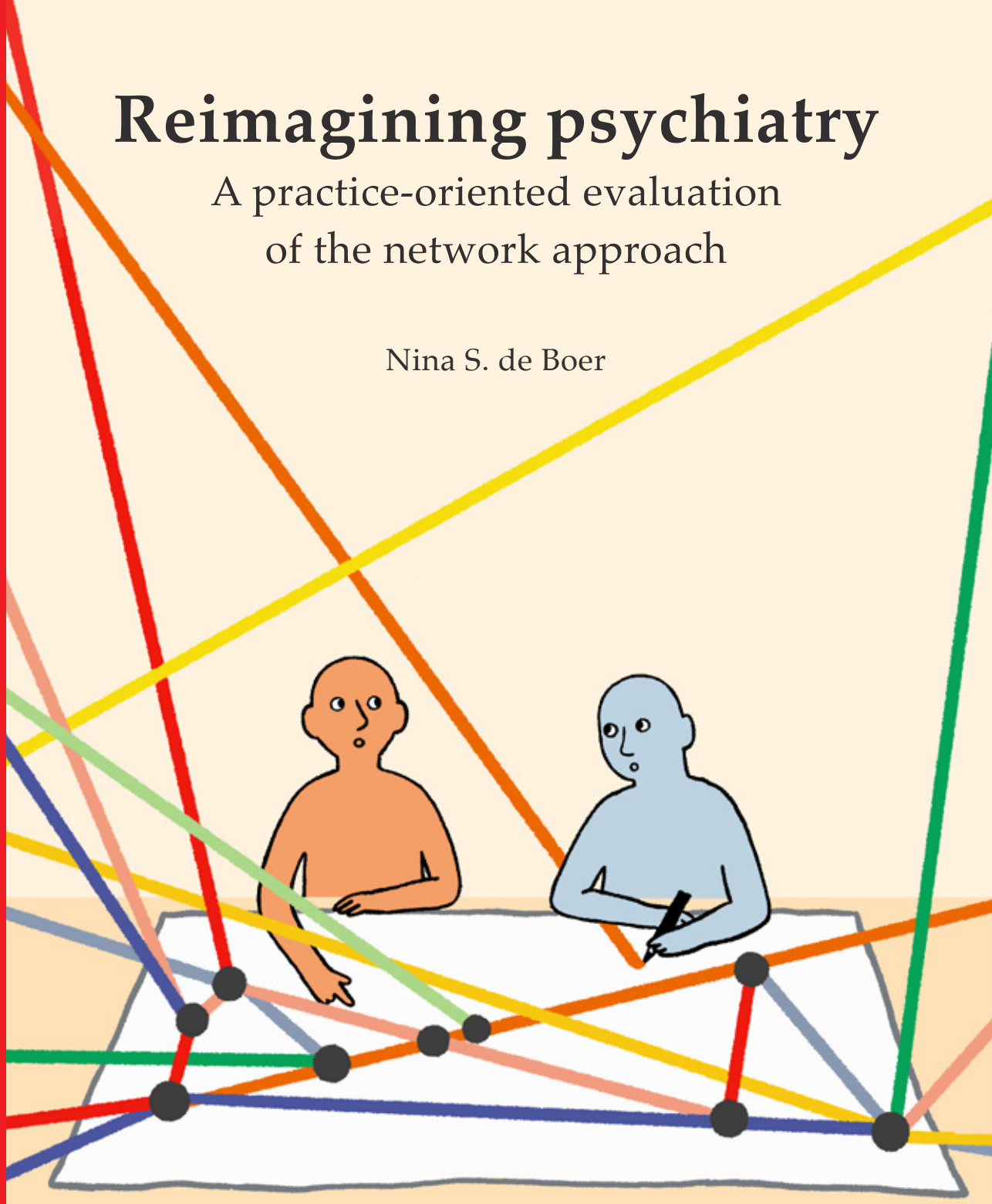
Reimagining psychiatry

A practice-oriented evaluation of the network approach

Nina S. de Boer



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REIMAGINING PSYCHIATRY

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Table of contents

Publications included in this thesis

Chapter 3 is based on the following publication: De Boer, N. S., De Bruin, L. C., Geurts, J. J. G., & Glas, G. (2021). The network theory of psychiatric disorders: A critical assessment of the inclusion of environmental factors. *Frontiers in Psychology*, *12*, 623970. https://doi.org/10.3389/fpsyg.2021.623970

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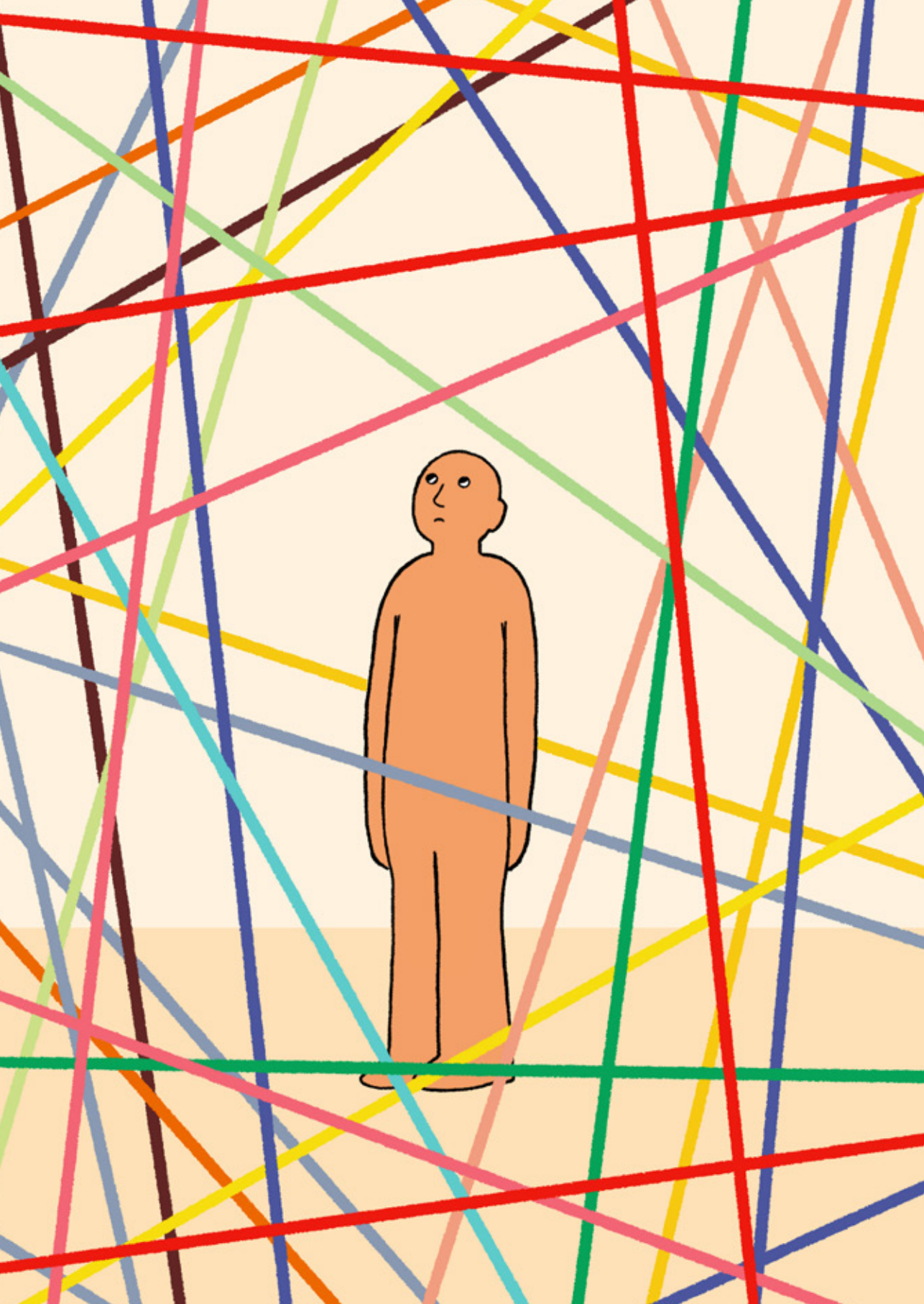
List of figures



Introduction

Chapter 1

Introduction

Abstract

Over the centuries, psychiatry has gone through *theoretical reinventions* (i.e., changes in how we think about the nature and causes of mental disorders) and *empirical reinventions* (i.e., changes in how we study mental disorders). Currently, psychiatry is on the brink of another reinvention: The *biomedical approach* to mental disorders that has been dominant since the 1980s has not led to the epistemic success that it had promised. In response, systemic alternatives to the biomedical approach, including the *network approach*, have emerged. The network approach argues that we should conceptualize mental disorders as (stable sets of) causally interacting symptoms – i.e., *network theory* – and advocates using *network analysis* to study mental disorders. In this thesis, I evaluate the epistemic potential of the network approach from a *practice-oriented* perspective, focusing on how the network approach is used in scientific practice, clinical practice, and daily life to obtain knowledge about possibilities regarding mental suffering. In this chapter, I sketch the context of the network approach and justify my practice-oriented account.

1.1 The intractable and idealized world of mental suffering

In 1998, Gerald N. Grob, historian of medicine and psychiatry, delivered a Presidential Address at the seventy-first annual meeting of the American Association for the History of Medicine. In his address, titled *Psychiatry's Holy Grail: The Search for the Mechanisms of Mental Diseases*, Grob reflects on the history of psychiatry since the early nineteenth century. He starts his analysis with the following metaphor:

> As human beings we generally inhabit two different worlds simultaneously. The first is characterized by contingency, indeterminacy, and an inability to comprehend or control the numerous variables that shape our environment; our judgements, analyses, and actions often represent a pragmatic response to a seemingly intractable and partially incomprehensible universe. The second is an imaginary and idealized world – one characterized by clarity, and where pure and precise knowledge leads to a kind of understanding that enables human beings to cope with or solve perennial problems. (p. 189)

Grob uses this metaphor to highlight an ambiguity that is present in how psychiatry deals with mental suffering.[1] Mental suffering belongs to the *intractable world*: it is "rarely amendable to therapeutic clarification or simple prescriptions" (p. 190). Everyone who has lived through periods of mental suffering or has experienced them up close will recognize this intractability. We have difficulty making sense of our periods of crisis or those of our loved ones. Evnine (1989) argues that mental disorder can be characterized "precisely by the difficulties it presents for understanding" (p. 11). However, how we conceptualize, study, and talk about our mental suffering belongs to the *idealized world*: We abstract away from the lived experience of mental suffering, we generalize and simplify to find patterns, explanations, or entry points for therapy. We want to know what causes us to think, behave, or feel in "mad" ways; why others think, behave, or feel madly. We hope that more knowledge of mental suffering will lead to better treatments or prevention and will make it easier for others to empathize with or relate to us. We hope that scientific and theoretical developments in the idealized world will provide knowledge that will make our lived experience more tractable. When we deal with mental suffering – whether through lived experience, in our clinical practice, or our scientific research – we have to navigate the intractable and the idealized worlds simultaneously.

The landscape of the idealized world of mental suffering has shifted throughout the years. Grob (1998) continues his presidential address by discussing the ways that psychiatry has reshaped its "idealized landscape" over the last centuries. Indeed, our conceptualizations and ways of studying mental disorders have shifted frequently and, in many instances, quite dramatically. Historical analysis shows that every couple of decades, psychiatry *reinvents* itself (Whooley, 2019). Such psychiatric reinventions encompass a variety of developments, two of which I highlight in this thesis. First, *theoretical reinventions*, i.e., changes in how we conceptualize the nature and causes of mental disorders. What causes us to feel depressed, sleep poorly, or lose our pleasure in daily activities? Is this because of our biology, traumatic events in earlier life, existential angst, or relational conflicts? Second, *empirical reinventions*, i.e., changes in how we obtain knowledge about mental disorders and, subsequently, what skills

1. I start with some terminological clarifications. First, I use "psychiatry" to refer to scientific and clinical psychiatric practice while acknowledging that a variety of professions is involved in the alleviation of mental suffering. Second, I use "mental disorder" to refer to the collections of mental problems for which people seek professional help. This does not imply that everyone who has been diagnosed does or should resonate with this term and that there is no harm associated with referring to specific mental differences as disorders (e.g., neurodiverse conditions). I use "disorder" in this thesis to do justice to the severity of the mental suffering that people can experience. When referring to people's (intractable) lived experiences, I use "mental suffering." Third, I use "client" to refer to people who are treated for their mental suffering in clinical practice. When referring to daily life, I use third-person language such as "person who experiences mental suffering."

are required to study these conditions. Should we use clinical neuroimaging to figure out why we feel depressed, sleep poorly, or lose pleasure in daily activities? Should we use psychological research, qualitative interviews, or sociological research? These theoretical and empirical reinventions, these new idealized landscapes, come together in what I refer to as *psychiatric approaches*.

Currently, psychiatry is on the brink of another reinvention because the psychiatric approach that has been dominant since the 1980s, the *biomedical approach*, has not led to the epistemic success it had promised. In response, alternative, systemic approaches to the biomedical approach have come to the fore, including the *network approach*. Proponents of the network approach have set their hopes on this new, idealized landscape, hoping it will provide knowledge where previous approaches were lacking. But how should we evaluate the ability of the network approach to provide knowledge of mental suffering, i.e., its *epistemic potential*? This question is important to address in light of psychiatry's history. Each psychiatric reinvention promised to provide knowledge that would show the causes, constituents, and risk factors of mental disorders, aid clinical practice and help people who suffer mentally make sense of their suffering and be empowered. Grob (1998) argues that with every new psychiatric approach, its proponents insisted that psychiatry "stood on the threshold of fundamental breakthroughs that would revolutionize the ways in which mental disorders were understood and treated" (p. 217). However, thus far, such "fundamental breakthroughs" have not materialized. This finding could make us worry about the epistemic potential of new psychiatric reinventions, such as the network approach. In this thesis, I will present a nuanced and productive way to evaluate the epistemic potential of the network approach, i.e., a *practice-oriented* account. My practice-oriented account focuses on how scientists, clinicians, and people with lived experience use the idealized landscape the network approach sketches to reason about possibilities regarding the intractable world of mental suffering. In doing so, this account avoids unrealistic expectations of the epistemic potential of the network approach without succumbing to unbridled relativism. In this introductory chapter, I sketch the context of the network approach and justify my practice-oriented account.

This chapter will be structured as follows. In **Section 1.2**, I discuss the rise of and disappointment with the biomedical approach, arguing that the biomedical model and biomedical science did not lead to their anticipated epistemic benefits in scientific practice, clinical practice, and daily life. In **Section 1.3**, I discuss earlier systemic alternatives to reductionistic psychiatric approaches, claiming that they did not suffice as complete idealized landscapes because they did not encompass theoretical *and* empirical reinventions. In **Section 1.4**, I claim that the network approach may be

a suitable systemic alternative to the biomedical approach because it encompasses a theoretical reinvention (network theory) and an empirical reinvention (network analysis). In **Section 1.5**, I reflect on the *similarity-based* account, i.e., the intuition that the epistemic potential of the network approach depends primarily on whether this approach "accurately reflects" or "maps onto" the intractable word. I show that recent developments in the network approach reflect this intuition but that evaluating the epistemic potential of this approach based on similarity alone is unfruitful. In **Section 1.6**, I present my alternative, *practice-oriented* account that provides a nuanced and realistic view of the network approach's epistemic potential. In **Section 1.7**, I provide an overview of the practices and chapters I discuss in this thesis.

1.2 The biomedical approach

When Grob (1998) held his presidential address, the biomedical approach was at the height of its popularity. The biomedical approach arose from nosological (i.e., classification-related) and technoscientific developments. In this section, I sketch the context in which the biomedical approach developed and the reinventions it encompassed.[2]

The biomedical approach became dominant with nosological changes in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III, American Psychiatric Association, 1980). The DSM-I and -II were based on psychoanalytic principles: They conceptualized mental disorders as the expression of unconscious, repressed conflicts in our wishes, desires, and beliefs that had their basis in our psychosocial (and psychosexual) development. From the 1960s onward, clinicians and scientists expressed concerns about the reliability and validity of the DSM-I and -II (Beck, 1962; Schwartz & Wiggins, 1986, 1987). Such concerns were aggravated by studies such as the Rosenhan Experiment (1973), in which eight healthy volunteers submitted themselves for evaluation at psychiatric institutions with feigned hallucinations: All were admitted, and the only individuals questioning their mental health status were the patients residing in the institutions. In response, data-oriented psychiatrists gained influence in the task force responsible for designing the DSM-III from 1974 onwards. These psychiatrists were inspired by Kraepelin, a contemporary of Freud who advocated for systematic clinical observations and psychiatric diagnoses and classifications (Lamb, 2014). These data-oriented psychiatrists – or Neo-Kraepelinians

2. For a historical account of the development of the biomedical approach, see Whooley (2019, Chapter 5) and Horwitz (2002, Chapter 3). While not the focus of this thesis, institutional forces have also played an important role in the popularity of the biomedical approach. For instance, the pharmaceutical industry and insurance companies are inextricably linked to diagnostic psychiatry, for they use psychiatric classifications to monitor treatments and provide coverage.

– argued that the DSM could improve its reliability and validity by grounding mental disorder classifications in observable and measurable features (i.e., symptoms) instead of theoretical explanations. To this end, DSM-III introduced *diagnostic psychiatry*. The Neo-Kraepelinians hoped that diagnostic psychiatry would "form the basis for a better understanding of specific etiological mechanisms and greater specificity and efficacy in the treatment of patients suffering from psychiatric illness" (Feighner, 1979, p. 1174). Indeed, diagnostic psychiatry was spurred by technoscientific developments in *biomedical science*. In the 1990s, the *Human Genome Project* was launched, which promised to generate the first DNA sequence of the human genome. Around this time, genetics was "expected to reveal much about the etiology and pathogenesis of mental illness" (Pardes et al., 1989, p. 435). Moreover, developments in neuroimaging – most notably in functional magnetic resonance imaging (fMRI) – promised transparent access into the mechanisms of the brain that would "blow away old barriers to knowledge" (Pardes, 1990, p. 1115). These nosological and technoscientific developments together led to the development of the *biomedical approach*.

The biomedical approach encompasses two types of reinventions. First, the *theoretical reinvention* concerns the shift from a psychoanalytic to a *biomedical model* of mental disorders. The biomedical model assumes that the symptoms of a mental disorder are caused by an underlying (neuro)biological dysfunction. For instance, Insel and Quirion (2005) claim that "[o]ne of the fundamental insights emerging from contemporary neuroscience is that mental illnesses are brain disorders" (p. 2221). To illustrate, let us consider depression. The diagnostic criteria for depression – or major depressive disorder according to the fifth edition of the DSM (DSM-5, American Psychiatric Association, 2013) – can be found in **Table 1**.[3] The biomedical model explains the co-occurrence of depressive symptoms by claiming that they have a shared, underlying (neuro)biological cause (**Figure 1**). An example of the biomedical model is the chemical imbalance theory of depression (Schildkraut, 1965), which states that depression is caused by low levels (or a decreased reuptake) of the neurotransmitter serotonin. Such a lack of serotonin (reuptake) causes one to experience insomnia, weight loss, suicidal ideation, anhedonia, and a depressed mood, for instance. The biomedical model is *reductionist*, for it assumes that all aspects of mental disorders can be reduced to or grounded in their biological origins, and encourages *essentialist* thinking**,** i.e., the belief that our biology (and its dysfunctions)

3. In the DSM-5, depressive disorders encompass a set of conditions characterized by "the presence of sad, empty, or irritable mood, accompanied by somatic and cognitive changes that significantly affect the individual's capacity to function" (American Psychiatric Association, 2013, p. 155). Major depressive disorder is the "classic condition in this group of disorders" (p. 155). In this thesis, I use "major depressive disorder" when explicitly referring to the mental disorder classification; in other instances, I refer to "depression."

determines our identity. Second, the *epistemic reinvention* concerns the shift from a lack of scientific research into mental disorders under the psychoanalytic approach to the adoption of biomedical science as the scientific discipline that would be best suited to study (and discover) the causes of mental disorders. Both aspects of the biomedical approach are exemplified in the following quote by Andreasen (1984): "*The major psychiatric illnesses are diseases* (…) *caused principally by biological factors, and most of these factors reside in the brain*. (…) *As a scientific discipline, psychiatry seeks to identify the biological factors that cause mental illness*" (pp. 29-30, emphasis in original). Or, as Guze (1989) stated in his article *Biological Psychiatry: Is There Any Other Kind?*, "[t]here can be no such thing as a psychiatry which is too biological" (p. 316).

Table 1. A summary of the DSM-5 criteria for major depressive disorder (American Psychiatric Association, 2013)

A. Five (or more) of the following symptoms have been present nearly every day during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.
1. Depressed mood most of the day 2. Diminished interest or pleasure in almost all activities most of the day (i.e., anhedonia) 3. Decrease or increase in appetite, or significant weight loss or weight gain (e.g., more than five percent of body weight in a month) 4. Insomnia or hypersomnia 5. Psychomotor agitation or retardation 6. Fatigue or loss of energy 7. Feelings of worthlessness or excessive or inappropriate guilt 8. Diminished ability to think or concentrate, or indecisiveness 9. Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide
B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The episode is not attributable to a substance's physiological effects or another medical condition.

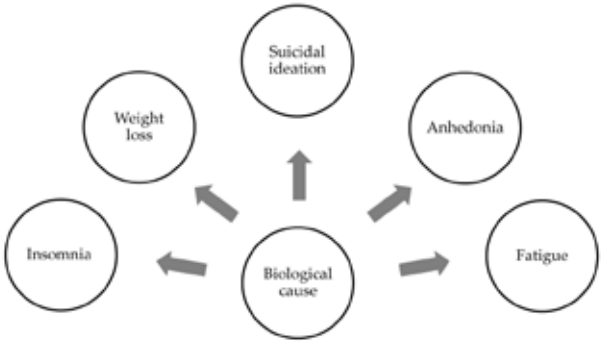


Figure 1. A visual representation of the biomedical model of depression

Proponents of the biomedical approach hoped that the idealized landscape sketched by this approach would provide epistemic benefits in different psychiatry-related practices. First, they hoped it would provide epistemic benefits for *scientific practice*, allowing psychiatric scientists to obtain generalizable scientific knowledge about the causes of mental disorders. Grob (1998) describes this sentiment as follows:

> [M]any contemporary psychiatrists – as well as those associated with genetics and neuroscience – perceive themselves as standing on the threshold of a new era. Laboratory findings will presumably shed light on the physiological and genetic etiological mechanisms that shape normal and abnormal behavior, and will thus set the stage for the development of effective curative interventions. (p. 216)

Second, and related, they hoped the biomedical approach would provide epistemic benefits for *clinical practice*, providing knowledge that clinicians could use to treat or diagnose their clients. Third, they hoped the biomedical approach would provide epistemic benefits in *daily life*, that is, in improving the lives of people who experience mental suffering. Amongst others, they hoped the biomedical approach would positively influence how people with lived experience would see themselves and be perceived. Proponents of the biomedical approach assumed that attributing our mental suffering to a biological cause would remove its moral connotations by giving it a similar status to physical suffering (e.g., Corrigan et al., 2000). Indeed, the American grassroots mental health organization *National Alliance on Mental Illness,* involved in advocacy work, explicitly stated in 2006 that "mental illnesses are biologically based brain disorders. They cannot be overcome through 'will power' and are not related to a person's 'character' or intelligence" (see Deacon & Baird, 2009). So, proponents of the biomedical approach hoped it would positively affect the stories that people with lived experience told about themselves, i.e., their *self-narratives*.

Has the biomedical approach lived up to these epistemic hopes? In his address, Grob (1998) is skeptical of the biomedical approach's ability to provide "*final* answers" about mental disorders (p. 218, emphasis in original). In contrast, around fifteen years after Grob's address, Kapur et al. (2012) presented an optimistic reading of the approach's epistemic successes:

> By several measures [the biomedical approach] has been a tremendous success – thousands of scientific papers and hundreds of books devoted to this subject; legions of dedicated scientists and over 60 dedicated

> professional societies worldwide; and a profound impact on the public's perception of mental disorders. (p. 1174)

Indeed, the biomedical approach has continued to be a dominant psychiatric approach, shaping how we conceptualize mental suffering. Empirical research shows that – at least in the Western world – the biomedical model in general (Schomerus et al., 2012) and of depression specifically (Pescosolido et al., 2010; Pilkington et al., 2013) has become increasingly popular over the past decades. Moreover, the chemical imbalance theory is commonly used to explain depression on popular websites (Demasi & Gøtzsche, 2020). However, despite this popularity, the biomedical approach has not lived up to its anticipated epistemic expectations. First, psychiatric scientists have not been able to identify a distinct biological correlate – let alone cause – for any mental disorder. As noted by Adam (2013), "[d]espite decades of work, the genetic, metabolic, and cellular signatures of almost all mental syndromes remain largely a mystery" (p. 417). Kendler (2005) summarizes it as follows:

> We have hunted for big, simple neuropathological explanations for psychiatric disorders and have not found them. We have hunted for big, simple neurochemical explanations for psychiatric disorders and have not found them. We have hunted for big, simple genetic explanations for psychiatric disorders and have not found them. (pp. 434-435)

Second, the knowledge provided by biomedical science has not straightforwardly led to better interventions or clinical care. Some neuroscientific-research-inspired treatments have been developed, such as deep-brain stimulation (Figee et al., 2022). However, the success rates of interventions across the full range of mental disorders in adults and adolescents are still modest (Holmes et al., 2018; Reynolds et al., 2012), and only about 40% of people with a psychiatric diagnosis achieve sustained recovery (Clark, 2018). Moreover, biomedical science has not led to the development of clinical tests that can be used to diagnose and treat mental disorders (Kapur et al., 2012).[4] Third, the biomedical approach has not provided its intended epistemic benefits in daily life: It has not univocally reduced stigma and empowered those with lived experience. For instance, empirical research on the influence of the biomedical model of depression on people's self-perception (Deacon & Baird, 2009; Kemp et al., 2014; Kvaale et al., 2013; Lebowitz et al., 2013) and self-narration more specifically (Kangas, 2001; Laegsgaard et al., 2010; Lafrance, 2014; Ridge & Ziebland, 2006; Schreiber &

4. At the same time, there have been calls for integrating neuroscience into psychotherapy (Holmes et al., 2014) and initiatives that promote neuroscientific education for mental health care professionals, such as the *National Neuroscience Curriculum Initiative* (https://nncionline.org/).

Hartrick, 2002) shows that it does not only have positive effects on how people relate to their mental suffering. Related, empirical research on public responses to depression (Kvaale et al., 2013; Pescosolido et al., 2010; Schomerus et al., 2012) shows that the biomedical model increases certain aspects of stigmatization. So, the biomedical approach has not lived up to its epistemic hopes in scientific practice, clinical practice, and daily life.

To date, there is increased recognition that the biomedical approach is not psychiatry's Holy Grail. There is growing disillusionment with the biomedical approach, and psychiatry is ready for a new reinvention. Some suggest that psychiatry requires a *nosological reinvention* while remaining within its reductionist, biomedical framework. This option is endorsed, for instance, by the Research Domain Criteria (RDoC) initiative launched by the *National Institute of Mental Health* (Cuthbert, 2014; Insel, 2013; Insel et al., 2010). The assumptions driving RDoC are reflected in the title of one of its position papers: *Brain disorders? Precisely: Precision medicine comes to psychiatry* (Insel & Cuthbert, 2015). RDoC sticks to the biomedical approach's theoretical and empirical framework but proposes a new nosology consisting of transdiagnostic domains, such as negative and positive valence systems and cognitive systems, comprised of specific constructs that can each be studied by different (biological and behavioral) research domains.

However, some critics argue that nosological reinvention alone does not go far enough: Improving psychiatry's epistemic potential in scientific practice, clinical practice, and daily life requires an entirely new psychiatric approach, including a theoretical and empirical reinvention. This sentiment is echoed by Demazeux (2015): "Perhaps the fact that the DSM has stayed unsuccessful thus far is not the result of some methodological shortcoming but instead may depend on the general model the DSM has endorsed from the 1980s on, i.e., the medical model of mental illness" (p. 21). These critics claim that we should zoom out beyond the biomedical context: The idealized world sketched by the biomedical approach is too narrow and should be replaced by a *systemic alternative*. In the next section, I will discuss this option in more detail.

1.3 Systemic alternatives

Recently, clinicians, psychiatric scientists, and people with lived experience have expressed interest in systemic alternatives to the biomedical approach (Fried, 2022; Köhne, 2020). These alternatives share the assumption that focusing on one

cause or domain only does not suffice when making sense of mental disorders; we should consider factors from multiple domains and their interrelations (Kendler, 2008; Nolen-Hoeksema & Watkins, 2011). Systemic alternatives to reductionist psychiatric approaches have been proposed in the past. In this section, I discuss two of these earlier systemic alternatives – the *psychobiology movement* and the *biopsychosocial model* – and what prevents them from being suitable alternatives to the biomedical approach.

The psychobiology movement arose in the United States in the 1890s when developments in neuroanatomy, histological staining, and germ theory renewed scientific interest in mental disorders (Porter, 2002).[5] The movement, led by the psychiatrist Meyer, developed in response to the lack of knowledge about mental disorders the predating asylum period had provided. Instead of the myopic approach that was dominant in the asylum period, the psychobiology movement wanted to reconstruct psychiatry "as a non-reductionistic, humanistic medical science that maintained an appreciation for the kaleidoscopic character of mental illness" (Eyman, 1920, p. 4). The psychobiology movement advocated for a *pluralistic* approach to psychiatric science: Psychiatric scientists should study the biological, psychological, and social facets of mental disorders using different research methods. However, while Meyer (1908) assumed that mental disorders are comprised of social, biological, and psychological facets that cannot be neatly separated, the psychobiology movement was explicitly empiricist and anti-theoretical. That is, the movement was founded on the hope that multisource data and experimentation alone would organize themselves into "facts" about mental disorders. This lack of a theoretical framework led the psychobiology movement to unbridled, unconstrained pluralism that developed into an incoherent research program, contributing to the movement's fall in the 1940s (Whooley, 2019, pp. 85–90). The psychobiology movement shows that empirical reinvention alone does not suffice when wanting to provide a systemic alternative to reductionism.

The biopsychosocial model provides a more developed, systemic alternative to reductionist psychiatric approaches. The biopsychosocial model proposed by Engel (1977, 1979, 1980) developed around the same time as the biomedical approach and can be considered a direct response to the latter. Similar to the psychobiology movement, the biopsychosocial model assumes that making sense of mental disorders requires considering their biological, psychological, and social dimensions. Contrary to the psychobiology movement, it was explicitly founded on theoretical principles, i.e.,

5. Lamb (2014) and Whooley (2019, Chapter 2) provide a detailed historical account of the psychobiology movement.

general systems theory (Von Bertalanffy, 1950, 1969).[6] General systems theory presented a theoretical, transdisciplinary account of *systems*, i.e., "complexes of elements standing in interaction" that produce systemic behavior (Von Bertalanffy, 1969, p. 33). More specifically, general systems theory was concerned with "the formulation and derivation of those principles that are valid for 'systems' in general, whatever the nature of their component elements and the relations or 'forces' between them" (Von Bertalanffy, 1969, p. 37). In line with general systems theory, the biopsychosocial model assumes that mental disorders are "symptom clusters" where "the biochemical defect constitutes but one factor among many," and the "complex interaction" of biopsychosocial factors ultimately culminates into a mental disorder (Von Bertalanffy, 1969, p. 131). Specifically, the biopsychosocial model claims that the biological, psychological, and social domains are "different levels of a 'hierarchically arranged continuum'" (Engel, 1980, p. 536). Each domain is its own subsystem, requiring its own methods, research questions, and explanations, but the domains are combined into one higher-order system; each domain has its boundaries "across which material and information flow" (p. 537) (**Figure 2**). So, the biopsychosocial model encompasses a theoretical reinvention based on the principles of general systems theory and suggests an empirical reinvention based on epistemic pluralism.

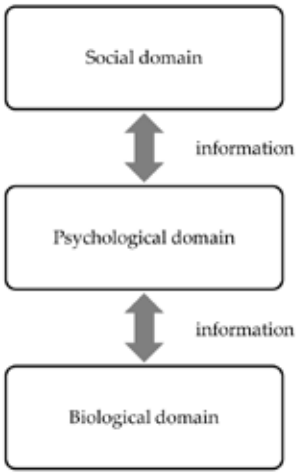


Figure 2. A graphical representation of the biopsychosocial model

However, the biopsychosocial model has been criticized, and many of its criticisms boil down to its lack of clarity and constraints (Benning, 2015; De Haan, 2020; Farre & Rapley, 2017; Ghaemi, 2009; Van Oudenhove & Cuypers, 2014). First, critics argue that

6. It is telling that Engel (1977, 1979, 1980) barely references the psychobiology movement.

the biopsychosocial model is not scientifically testable because it is vaguely defined. For instance, it is unclear how the domains interact: What is meant by "information" that "flows" amongst domains? Second, critics claim that the biopsychosocial model is challenging to implement in clinical practice, for it is unclear how to treat people in a biopsychosocial manner. In a critique that echoes earlier criticisms of psychobiology, Ghaemi (2009) claims that the biopsychosocial model leads to "additive eclecticism" or eclectic pragmatism (p. 4): Clinicians can prioritize whichever domain they want, meaning they can use the biopsychosocial model to enact their own assumptions about the primary causes of mental disorders. Third, critics argue that the biopsychosocial model does not inform scientists on how to identify relevant biopsychosocial data. So, while being more theoretically informed than the psychobiology movement, the biopsychosocial model only provides weak theoretical and empirical constraints for scientific research or clinical practice.

This section demonstrated that we need systemic *approaches* that encompass a theoretical and empirical reinvention to present a suitable epistemic alternative to the biomedical approach. In the next section, I will show that the network approach may suit this task.

1.4 The network approach

In recent years, systemic approaches based on *complexity studies* have been developed. Theoretically, these approaches conceptualize mental disorders as *complex systems*, i.e., systems comprised of factors from different domains that interact non-linearly over different time scales and demonstrate complex properties (Ladyman et al., 2013). Empirically, these approaches promote studying mental disorders using *complexity science*, i.e., research methods that study the properties of complex systems. Examples of complexity-driven systemic approaches are the enactive approach by De Haan (2020) and the complexity theory of psychopathology by Olthof et al. (2023).[7] In this thesis, I focus on a third complexity-driven systemic approach: the *network approach*. The network approach has skyrocketed since its first publications in 2008 (Berta et al., 2022; Robinaugh et al., 2020) and is increasingly taken up in scientific and clinical practice. To illustrate, in 2020, the Dutch government invested nearly 20 million euros in the ten-year research program *New Science of Mental Disorders*, which attempts to improve the treatment of mental disorders based on the network approach (Roefs

7. These alternatives can also accommodate a different critique of the biopsychosocial model, namely that the model provides a too-linear account of how the three domains interact (De Haan, 2020).

et al., 2022). In this section, I discuss the context in which the network approach developed and the theoretical and empirical reinventions it encompasses.

In its most basic form, a network consists of a collection of factors or variables (nodes) and their relations (edges). For instance, railroad systems can be described as networks of stations connected via tracks, social groups as networks of people connected via friendship, and the brain as a network of neurons connected via synapses. The network approach has its foundations in *network science*, a branch of complexity science that studies real-life phenomena as networks. Network science is based on *graph theory*, a mathematical, theoretical discipline founded by Euler (1736) that studies formal representations of networks or *graphs*. Network science had little bearing on empirical data until the 1990s when data availability increased with the rising popularity of the Internet and computational developments in sensors and high-throughput technologies.[8] Technologies that enabled processing large amounts of data allowed network scientists to perform simulation studies to test their theories and to empirically study the network properties of real-world phenomena. Two seminal network science studies from this period stand out. First, Watts and Strogatz (1998) developed a formalization of *small-world networks*, i.e., networks with low average path lengths and high clustering. Second, Barabási and Albert (1999) developed a formalization of *scale-free networks*, i.e., growing networks with new nodes preferentially attaching to existing nodes with many connections. Both studies showed that many real-life phenomena demonstrate small-worldness and scale-freeness.

The technoscientific developments in the late twentieth century not only boosted network science but also spearheaded the intuition that "new ideas, new approaches – really, a new way of *thinking* – were direly needed to help make sense of the highly complex, intricately connected systems that increasingly affect human life and well-being" (Mitchell, 2009, p. 233, emphasis in original). Barabási (2002) referred to this intuition as *network thinking* and claimed that networks "will dominate the new century to a much greater degree than most people are yet ready to acknowledge" (p. 7). Barabási was right: The *Oxford English Dictionary* shows that "network" is one of the thousand most common words in modern written English, similar in frequency to "memory" and "opinion" (Oxford University Press, 2024). Hence, it makes sense that network thinking also entered psychiatry in the early twenty-first century.

8. Two network science developments in the earlier twentieth century stand out. First, Moreno (1934) developed network-based *sociograms* to explore social relations in the classroom. Second, Erdős and Rényi (1959) and Gilbert (1959) developed formalizations of *random networks*, i.e., mathematical models that can generate networks using probabilistic rules. For more information about the history of complexity science, see Ladyman and Wiesner (2020, pp. 11–17) and Mitchell (2009, pp. 295–298).

The network approach was spurred by technoscientific developments and associated developments in our thinking about systems, as well as by a new way of approaching psychiatric nosology. The first article that serves as the basis for network thinking in the psychiatric context is *Psychometric Perspectives on Diagnostic Systems* by Borsboom (2008).[9] This article discusses four ways to conceptualize the relationship between symptoms and mental disorders in the DSM; here, I highlight two. First, the "diagnostic perspective" assumes that a disorder underlies a set of symptoms in line with the biomedical model (**Figure 1**). Second, the "causal systems perspective" assumes that symptoms and their interactions *are* – or constitute – a disorder: Symptoms of specific mental disorders covary because they mutually influence each other. Building on this causal systems perspective, Borsboom and Cramer (2013) argue that one can have an underlying physical dysfunction, such as a tumor, without having symptoms but that this is conceptually impossible for mental disorders. They draw an analogy between scientists looking for the essence of mental disorders and the visit to Cambridge in Ryle's (1949) infamous thought experiment. Just like the visitor to Cambridge makes a category mistake by asking where the University is after receiving a campus tour, we make a category mistake if we ask what the essence of mental disorders is after observing its symptom dynamics. Together with developments in network science, this new perspective on nosology shaped the network approach.

The network approach encompasses two dimensions. First, its *theoretical reinvention* concerns using the *network theory* as an alternative to the biomedical model. In line with the causal systems perspective mentioned earlier, the network theory states that mental disorders are constituted by (stable sets of) causally interacting symptoms (Borsboom, 2017a; Borsboom et al., 2019a). To illustrate, let us go back to depression and its symptoms. Whereas the biomedical model states that all depression symptoms share one underlying (neuro)biological cause, the network theory claims these symptoms cause each other. For instance, experiencing insomnia can cause one to have a depressed mood, which can cause one to eat less and thereby lose weight, which can lead to a lack of energy and, hence, pleasure in daily activities (**Figure 3**). **Table 2** shows the principles of the network theory. Second, its *empirical reinvention* concerns using *network analysis* to study mental disorders.[10] Network analysis comprises statistical techniques that estimate dependencies between variables in a dataset to create *network models* of variables related via edges (Borsboom, Deserno, et al., 2021).[11] The main variables in network analysis studies of mental disorders are

9. Network psychometrics has its roots in earlier work by Molenaar (1987, 2003, 2004).

10. To indicate the popularity of network analysis studies of mental disorders: Only one such study was published in 2010, compared to 127 studies in 2020 (Berta et al., 2022).

11. Unless indicated otherwise, I use "network model" to refer to *data-driven, statistically estimated* network models of *mental disorders*.

psychiatric symptoms. What sets network analysis of mental disorders apart from latent variable analysis is that the former takes each symptom as a relevant score in its own right instead of aggregating their values into sum scores.[12] So, since the network approach encompasses a theoretical *and* empirical reinvention, it could provide an alternative to the biomedical approach.

Table 2. The principles of the network theory of mental disorders, as formulated by Borsboom (2017a, p. 7).

Principle 1. Complexity. Mental disorders are best characterized in terms of the interaction between different components in a psychopathology network.
Principle 2. Symptom-component correspondence. The components in the psychopathology network correspond to the problems that have been codified as symptoms in the past century and appear as such in current diagnostic manuals.
Principle 3. Direct causal connections. The network structure is generated by a pattern of direct causal connections between symptoms.
Principle 4. Mental disorders follow a network structure. The psychopathology network has a non-trivial topology, in which certain symptoms are more strongly connected than others. These symptom groupings give rise to the phenomenological manifestation of mental disorders as groups of symptoms that often arise together
Principle 5. Hysteresis. Mental disorders arise due to the presence of hysteresis in strongly connected symptom networks, which implies that symptoms continue to activate each other, even after the triggering cause of the disorder has disappeared.

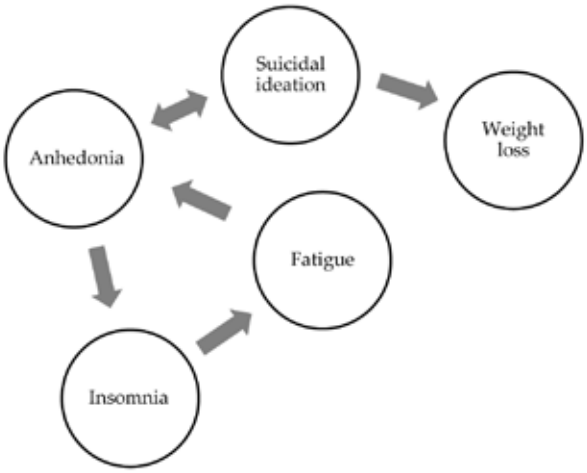


Figure 3. A visual representation of the network theory of depression

12. The network approach does not reject latent variable analysis altogether. For instance, psychometricians have developed statistical methods that combine latent variable- and network analysis (Epskamp, Rhemtulla, et al., 2017). However, focusing on the interactions between variables instead of aggregating them is a defining element of the network approach.

Indeed, proponents of the network approach express the hope that this approach can fill the epistemic lacunae left by the biomedical approach. For instance, Borsboom (2017b) claims that

> It is a fascinating idea that our understanding of mental disorders is not caused by an inadequate understanding of gene-brain-behavior pathways, but by an inadequate conceptualization of what we are doing. A more holistic analysis of symptom networks may offer a road forward in the understanding of mental disorders, where biological reductionism has so far failed. (pp. 92-93)

First, proponents of the network approach assume it has more epistemic potential in scientific practice than the biomedical approach, improving our scientific knowledge of mental disorders. For instance, Borsboom (2022b) expresses the hope that the network approach may "not only realize a new science of mental disorders, but a better one" (p. 5). Second, its proponents assume the network approach may have more epistemic potential in clinical practice, proving knowledge and methods that can improve the treatment of mental disorders, in line with the *New Science of Mental Disorders* project mentioned before (Roefs et al., 2022). Finally, the network approach may have more epistemic potential in daily life, positively affecting how people with lived experience see themselves and are perceived. For instance, Borsboom et al. (2019a) emphasize that the network theory makes specific aspects of mental disorders more understandable, and Meier et al. (2022) suggest that promoting the network theory via psychoeducation could positively affect people's self-understanding. So, the network approach may provide knowledge of mental suffering where the biomedical approach has been lacking.

However, a historical perspective could make us skeptical about the network approach's epistemic potential. Grob (1998) concludes his presidential address by stating that the history of psychiatry presents "a striking example of a cyclical pattern that has alternated between enthusiastic optimism and fatalistic pessimism" (pp. 216-217). In a similar vein, Whooley (2019) argues that every psychiatric reinvention can be analyzed in light of a *hype-disappointment cycle* (**Figure 4**):

> Crises give birth to entirely new paradigms, new identities, and new ways of thinking about mental distress. When the various promises lead to dead ends, a professional crisis erupts. In response, psychiatric reformers shift gears and, fueled by often ostentatious hype, pu rsue another course. As before, when the promised breakthroughs never materialize, malaise sets in.

> Disappointment and nihilism are followed by another crisis and, eventually, another reinvention. The cycle repeats anew every few decades. (pp. 6-7)

So, looking at psychiatry's history provides reason for caution. If we want to examine the epistemic potential of the network approach, we should first focus on an underlying question: *How* should we evaluate its epistemic potential? In the next section, I will present the first possible answer to this question: We can evaluate the epistemic potential of the network approach based on how *similar* the idealized world it sketches is to the intractable world.

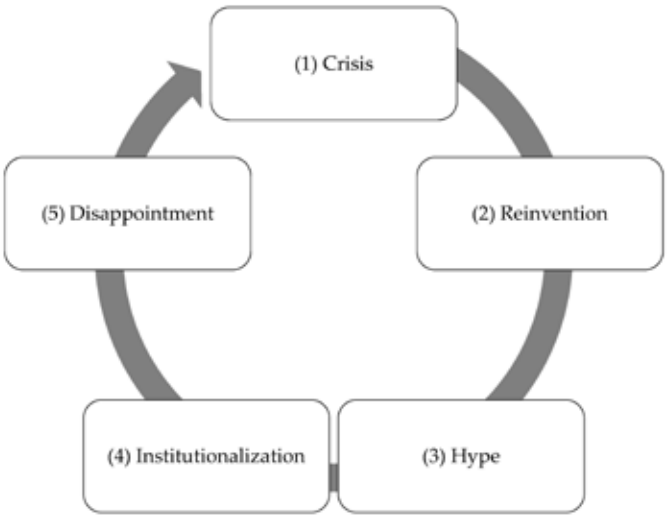


Figure 4. The hype-disappointment cycle, based on Whooley (2021)

1.5 A similarity-based account

A *similarity-based* account assumes that the epistemic potential of the network approach depends primarily on whether this approach "accurately reflects" or "maps onto" mental suffering.[13] In other words, the network approach is epistemically advantageous in scientific practice, clinical practice, and daily life when the network

13. Instead of "similarity," one could also use other terms, such as "correspondence" or "representation." Note that the similarity-based account is not synonymous with a realist interpretation of the network approach. One could argue that scientific representations ultimately say something about reality whilst acknowledging that *how* they represent reality should not be judged based on a similarity criterion. My intuitions align with an instrumentalist interpretation of the network theory and network models, but my claims do not hinge on this intuition. Hence, I will not engage with the realism versus instrumentalism debate in this thesis.

theory and network models accurately reflect or map onto the intractable world of mental suffering. This account is comparable to the *similarity view* of scientific representations that assumes that scientific representations represent their targets by virtue of being similar (Giere, 1988, 2004, 2010; Godfrey-Smith, 2006; Weisberg, 2013) or isomorphic (Van Fraassen, 1980, 2008) to them. The similarity-based account is reflected in claims by proponents of the network approach. For instance, Borsboom (2022a) argues that the network approach has

> mainly contributed to a better understanding of psychological phenomena, in the sense that the conceptualization of psychological constructs as networks has offered a new frame of thinking about them that, in many cases, seems to carve nature at least slightly closer to its joints. (p. 254)

In this section, I show that recent developments in the network approach reflect the similarity-based account but that evaluating the epistemic potential of this approach based on similarity alone is unfruitful.

First, recent developments in the network approach attempt to better reflect the *multifactorial* nature of mental suffering. The building blocks of the network approach are symptoms and their causal relations, but Borsboom and Cramer (2013) already acknowledged that "the reality of psychopathology involves a Russian doll of networks nested within networks in several layers of complexity" (p. 104). In recent years, the network approach has attempted to do more justice to the multifactoriality of mental suffering. Recent formulations of the network theory have explicated the relationship between non-symptom factors and the symptom network (Borsboom, 2017a; Borsboom et al., 2019a). Moreover, psychometricians, i.e., scientists concerned with quantitative measurement practices in psychology (*What Is Psychometrics?*, n.d.), explore how network analysis can be *extended* beyond symptom networks. Borsboom and Cramer (2013) already argued "that the simultaneous analysis of social, symptom, and physiological networks is one of the main research challenges for the near future" (p. 104). Around ten years later, Borsboom (2022a) claims that psychometricians should "start engaging with the tsunami of data that is approaching us," which "will require new extensions [of] the network paradigm" (p. 260). One possible extension that has received interest is the use of *multilayer network analysis* to integrate network psychometrics with other types of network science, such as network neuroscience (Blanken et al., 2021; Borsboom, 2022a, 2022b; Kästner, 2022; Riese & Wichers, 2021; Roefs et al., 2022). These developments reflect the assumption that it is epistemically advantageous to do justice to the multifactorial nature of mental disorders, to make the idealized world more similar to the intractable – multifactorial, interdependent – world.

Second, recent developments in the network approach attempt to do justice to the *idiosyncratic* nature of mental suffering. Wearables such as smartphones and smartwatches allow scientists to collect personalized data over multiple time points via *experience sampling methods* (ESM). Psychometricians can use ESM data to estimate *personalized network models* that could be used in mental health care (David et al., 2018; Epskamp, Van Borkulo et al., 2018; Frumkin et al., 2021; Roefs et al., 2022; Rubel et al., 2018; Von Klipstein et al., 2020). This is in line with the recent interest in introducing data science in clinical practice (Russ et al., 2018; Rutledge et al., 2019; Torous & Baker, 2016). Specifically, Glas (2019) suggests that "personalized network analysis comes closest to the ideal of a truly person-centered approach to psychopathology" (p. 182) and promises "a blended form of science and clinical practice: science *within* practice" (p. 182, emphasis in original). Again, this development reflects the assumption that it is epistemically advantageous to do justice to the idiosyncrasies of mental suffering, to make the idealized world more similar to the intractable – personal and idiosyncratic – world.

At first glance, the idea that the epistemic potential of the network approach primarily depends on its similarity to the intractable world is intuitive. However, there are also relevant counterarguments. First, it is unclear what kind of similarity the network approach should demonstrate to the intractable world. Everything is similar to everything else in some abstract shape or form; what type of similarity is *relevant* in this context? For instance, should a network model be similar to our observable lived experience of mental suffering or the potential non-observable factors *underlying* such suffering? Second, even if the idealized world becomes more similar to the intractable world, the two will not meet: The network approach will always be an idealization of mental suffering. Indeed, Borsboom (2022a) argues that "[w]hile networks probably do a better job of representing the complexity of psychological constructs than traditional psychometric approaches, in many applications they are still likely to be gross oversimplifications" (p. 257). Third, the similarity-based account does not do justice to the fact that we idealize *because* of the intractability of mental suffering. We simplify the intractable world precisely because it is difficult – if not impossible – to make sense of it without doing so. In other words, the idealized world provides scientists, clinicians, and people with lived experience *epistemic access* to the intractable world of mental suffering. This implies that the epistemic potential of the idealized world can also be reduced if we focus too much on its need for similarity.

So, a similarity-based account may not be a fruitful way to evaluate the network approach's epistemic potential. In the next section, I will present my *practice-oriented* account as a more realistic and productive alternative.

1.6 A practice-oriented account

Instead of focusing primarily on whether the network approach "accurately reflects" mental suffering, I present an alternative, *practice-oriented* account that assumes the network approach's epistemic potential depends on whether and how people can *use* it to *reason about possibilities* regarding mental suffering in their specific practices.[14] In this section, I sketch the philosophical background of this account and describe it in more detail.

My practice-oriented account is inspired by positions in the philosophy of science that claim that science's epistemic value is use- and context-dependent. Van Fraassen (1980) acknowledged the pragmatic dimension of science, describing pragmatic factors as those that are "brought to the situation by the scientist from his own social, personal, and cultural situation" (pp. 87–88). Van Fraassen (1980) argued that these pragmatic factors play a role in theory choice but that this pragmatic dimension of scientific inquiry is not part of the epistemic dimension of science, i.e., "cannot rationally guide our epistemic attitudes and decisions" (p. 87). Thus, he acknowledged that a theory may have particular *pragmatic virtues*, such as mathematical elegance and simplicity, that do not contribute to its epistemic potential. Contrary to this claim, more recent pragmatist philosophers of science have argued that science's pragmatic and epistemic dimensions are intertwined. As stated by De Regt and Dieks (2005), "the generation of scientific knowledge of empirical reality is inherently bound up with pragmatic skills, evaluations, and decisions" (p. 142). This intertwining of science's epistemic and pragmatic dimensions is especially visible in scientific modeling, where the distinction between obtaining knowledge via models and using models is not straightforward (e.g., Morgan & Morrison, 1999). In my thesis, I will follow this line of analysis: We cannot separate the pragmatic and epistemic virtues of scientific models and theories – they are always intimately related. This also implies that the epistemic potential of scientific representations cannot be established in a context-independent fashion. Longino (1990, 2002) argues that communities play an important role in science and that different scientific communities may have different values that guide their epistemic inquiry. De Regt and Dieks (2005) suggest that scientific communities shape what skills scientists acquire and to which theoretical virtues they are attuned. Thus, the epistemic value of scientific theories and models is context- and use-dependent.

14. Similarity-based views of scientific representations can also do justice to context-dependent, pragmatic considerations. For instance, Giere's (2010) agent-based view is committed to representation *as* similarity whilst claiming that scientists (1) intend, (2) to use a model, (3) to represent a part of the world (4) for a specific purpose (p. 274). My position on the epistemic value of context-dependency and pragmatic considerations is arguably more radical.

Moreover, my practice-oriented account is inspired by accounts in the philosophy of modeling that emphasize the pragmatics of scientific modeling. In response to the similarity view of scientific representations, Suárez (2004, 2009, 2015a, 2015b) developed an *inferentialist view* according to which models represent their targets by allowing "competent and informed agents to draw specific inferences about" them (2004, p. 773). The *artifactual view* is an alternative to the inferentialist view that focuses on how scientists use and interact with scientific models (Currie, 2017; Knuuttila, 2009, 2011, 2021a, 2021b; Knuuttila & Merz, 2009).[15] The artifactual view states that scientific models are like *tools*, i.e., intentionally produced artifacts constructed using specific representational means for specific epistemic purposes. The artifactual view does not deny that *what* models represent is important: Scientific models are constructed based on existing theoretical and empirical knowledge and, therefore, bear on empirical reality (Knuuttila, 2021b). However, the artifactual view claims that the epistemic value of scientific models lies in scientists' ability to use and interact with them to obtain knowledge about phenomena; scientific representations are scaffolds or "external tools for thinking" (Knuuttila, 2011, p. 263). The epistemic value of a scientific model depends on its idealizations and abstractions, context of use, and material features. Moreover, the artifactual view is modest about the knowledge that scientific models can provide. In line with *modal* views of scientific models (e.g., Massimi, 2018b, 2019, 2022; Verreault-Julien, 2019), the artifactual view claims that scientific models help scientists reason about *possibilities* regarding the phenomena they study instead of showing what is actually the case (Knuuttila, 2021b, 2021a).[16]

Bringing these two lines of work together, I formulate my practice-oriented account as follows: The epistemic potential of the network approach depends on the ability of scientists, clinicians, or people with lived experience to use the network theory and network models to reason about possibilities regarding mental suffering. The specific *practice* in which the network approach is used determines this ability

15. The artifactual view focuses on scientific "models" specifically. However, as I will demonstrate in this thesis, its principles can also be applied to theories (or theoretical models) such as the network theory.

16. Note that a modal view of scientific models does not necessarily exclude a similarity-based view.

and the possibilities that are relevant to consider.[17] My practice-oriented account conceptualizes the idealized world not as a poor reflection of our mental suffering but as a *tool* that can help its users explore what may be possible in the intractable world. So, it focuses on the epistemic potential of the network approach, *given* their place in the idealized world. This does not deny that the network approach's epistemic potential depends, in part, on *what* it represents. However, my practice-oriented account emphasizes that this epistemic potential also depends on *how* the network approach represents, *who* uses it, and for what *goals*.

The idea that the network approach should be judged from a pragmatic angle is echoed in the psychometrics literature. For instance, Borsboom (2022a) argues the following when discussing the rise of different network analysis techniques:

> I would be hesitant to buy into a general dogmatic position here, and would favor the idea that the choice of model should be dependent on the goals of the researcher and the substantive context. (…) However, it will likely be unclear to many researchers exactly how goals and context should be coordinated with choice of analysis. There is, in my view, therefore a need for papers that assist researchers in making this choice. (p. 256)

My thesis can be read as an attempt to aid in these decisions within and outside the scientific context. My practice-oriented account provides a nuanced and productive conceptualization of the network approach's epistemic potential that may, amongst others, present a way out of the hype-disappointment cycle. Moreover, it can highlight practice-dependent criteria to assess the (relative) epistemic potential of the network approach and its recent developments that the similarity-based account overlooks. To this end, my thesis addresses the following question:

> *What is the epistemic potential of the network approach to mental disorders in scientific practice, clinical practice, and daily life when evaluated from the perspective of a practice-oriented account?*

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17. My practice-oriented account is compatible with specific interpretations of *perspectivism*, a philosophical position emphasizing the importance of perspective-dependent factors in scientific theorizing and inquiry. Perspectivism acknowledges that we cannot study the world independently of our perspective and that multiple perspectives can characterize each system (Wimsatt, 2007). Perspectivism presupposes that our scientific theories and models serve specific goals of interest. Each has a limited range, so the ones scientists use should depend on their specific research questions and goals. Hence, perspectivism allows for – and even promotes – epistemic pluralism, i.e., using diverse scientific methods to study complex phenomena.

In the upcoming section, I will discuss these specific practices in more detail and provide an overview of the chapters in this thesis.[18]

1.7 Overview of the practices and chapters

Throughout the chapters of this thesis, I will explore the epistemic potential of the network approach in three different, albeit overlapping, practices: scientific practice, clinical practice, and daily life. Here, I use the term *practice* in a broad fashion, referring to collections of people using comparable (or compatible) skills, methods, background assumptions, activities, and goals to obtain knowledge about a topic of interest (in this case, mental suffering).[19] In this section, I describe each practice and how it will be discussed in the chapters of my thesis.

In **Part I** of this thesis, I will focus on the epistemic potential of the network approach in *scientific practice*. In this context, psychometricians use the network theory and network models to obtain scientific, generalizable knowledge about psychiatric phenomena. I use "network psychometricians" to refer to the community of scientists who quantitatively study psychological phenomena – including psychiatric phenomena – using similar theoretical principles (including the network theory) and statistical methods (network analysis and adjacent methods) and have received similar scientific training (e.g., statistics, formal modeling, and theoretical psychology).[20] In this thesis, I will discuss three epistemic functions that the network theory or network models may fulfill within network psychometrics: *explaining*,

18. While my thesis focuses on the network approach, this should not be taken to imply that I am committed to a monist view of psychiatric approaches. First, monism is not a realistic standpoint for psychiatric approaches: Even when specific approaches – such as the biomedical approach – are dominant, many different approaches are available at a given time. Second, acknowledging that every psychiatric approach idealizes and abstracts mental suffering differently implies that different approaches can make different aspects of mental suffering salient. Hence, my intuitions align with *pluralism*, i.e., the commitment that "[a] more complete representation of some phenomena requires multiple accounts, which cannot be integrated with one another without loss of content" (Kellert, 2006, p. xiv).

19. This description is akin to "epistemic culture" (Knorr Cetina, 1999), although the latter term focuses on the scientific context.

20. This does not imply that every network psychometrician has the same skillset or that network psychometricians cannot resort to other theoretical principles or methods; this description applies to the scientific *community*. Moreover, this description does not imply that network psychometrics can only be used to study psychological phenomena: Psychometrics – and network psychometrics specifically – is also used in educational and social sciences (*What Is Psychometrics?*, n.d.).

understanding, and *exploring* psychiatric phenomena.[21] In **Chapter 2**, I will analyze the network theory's explanatory claims, focusing on the roles of symptoms and environmental factors and the explanatory potential of a "multilayer network theory." This chapter is not yet practice-oriented – it focuses on the knowledge the network theory provides independently of *how* it is used – but will inform my practice-oriented analyses in the following chapters. In **Chapter 3**, I will characterize in practice-oriented terms how network psychometricians use the network theory and network models to obtain *scientific understanding* of mental disorders and what determines the understanding-providing potential of these network representations. In **Chapter 4**, I will use a similar practice-oriented perspective to characterize how network models can help psychometricians *explore* mental disorders and to evaluate the relative exploratory potential of multilayer brain-symptom network models. Specifically, I will show how psychometricians' ability to understand or explore mental disorders using the network approach depends on an interplay between practice-independent and practice-dependent features.

In **Part II**, I will address the epistemic potential of the network approach in *clinical practice*, focusing on a specific application of the network approach: *personalized network models*. In this context, clinicians – who have received psychometric training in constructing and interpreting personalized network models – and clients use personalized network models to obtain knowledge about clients' mental problems. In these chapters, I will show how an interplay of practice-independent and practice-dependent factors determines the *content* and *interpretation* of personalized network models. Specifically, I will discuss how the construction and interpretation of personalized network models intimately relate to clients' goals, values, and personal stories. In **Chapter 5**, I will discuss the epistemic potential of personalized network models in person-centered care from a practice-oriented perspective, focusing on how practice-independent and practice-dependent factors help demarcate these models. In **Chapter 6**, I will examine the relationship between personalized network models and self-narratives by addressing their structural and functional similarities and epistemic complementarity.

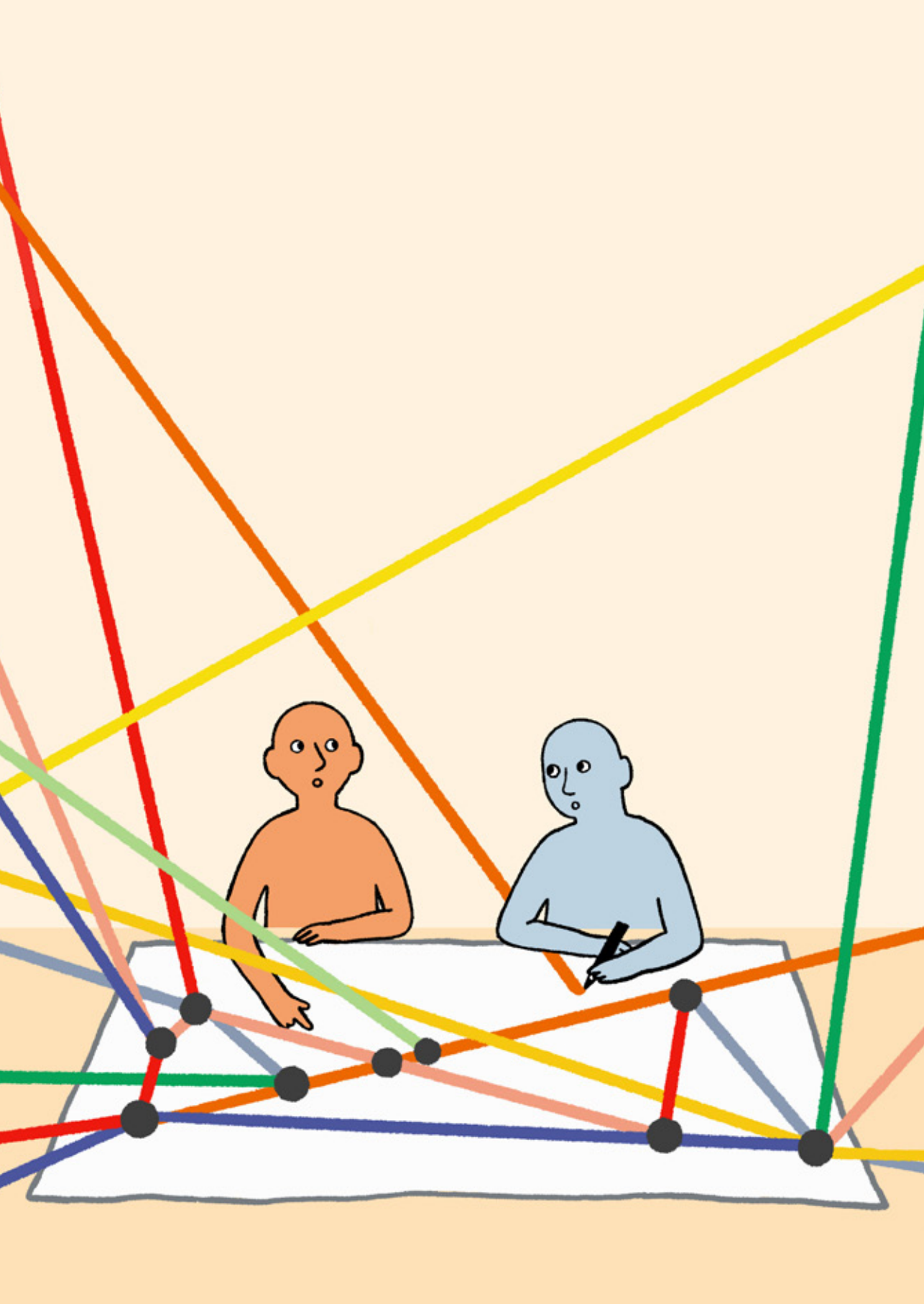
In **Part III**, I will examine the epistemic potential of psychiatric theories in *daily life*, exploring how people with lived experience could use such theories to make sense of their mental problems. In line with my practice-oriented account, I focus on the *function* that psychiatric theories may play in the *self-narratives* of people who experience mental problems and the role of the *sociocultural context* herein. In

21. Note that this list is not exhaustive. For instance, the network theory and network models may also have *predictive* functions.

Chapter 7, I will use the biomedical model of depression as a case study to examine how psychiatric theories could influence our self-narration. In **Chapter 8**, I will apply these insights to the network theory, focusing on how its influence on our self-narration depends on the strategies it promotes for "embracing the complexity" of our mental problems.

I finish this introduction with two additional considerations. First, I will use depression as the primary case study throughout this thesis. Most network analysis studies focus on depression (Robinaugh et al., 2020), and depression is used as an example in seminal papers on the network theory (Borsboom, 2008, 2017a; Borsboom et al., 2019a; Borsboom & Cramer, 2013). Moreover, my personal experiences with depression make this a case study I feel compelled to and comfortable addressing. Whether the epistemic potential of the network approach differs when applied to other mood disorders, compulsivity-related conditions, psychosis, neurodiverse conditions, or dimensional approaches to mental disorders is beyond the scope of this thesis.

Second, the network approach and its applications have developed and expanded tremendously since I started working on this thesis in 2020. These are exciting times for the network approach and for writing a philosophical thesis on this subject. However, this does imply that my thesis cannot cover all recent developments in the field. Amongst others, I will not discuss new ways to study the effects of interventions in network models via intervention modeling or means to incorporate different data types into network analysis other than multilayer network analysis (Borsboom, 2022a). Moreover, I will not discuss how – or whether – the network theory could be improved via formal modeling (Borsboom, Van der Maas, et al., 2021; Oude Maatman, 2024; Robinaugh et al., 2024). Such developments expand and fine-tune the network approach but do not undermine its general principles. Hence, my practice-oriented account can extend beyond the case studies and applications that I describe in this thesis to shed light on these and future developments.



Part I
Scientific practice

Chapter 2

The explanatory potential of the network theory

Abstract

The network theory attempts to explain psychiatric phenomena by conceptualizing mental disorders as (relatively stable) networks of causally interacting symptoms. Moreover, it emphasizes the explanatory role of non-symptom factors, such as *environmental factors*, in mental disorders. In this chapter, I address the explanatory potential of the (extended) network theory in more detail by examining what explanatory claims related to symptoms and non-symptom factors it makes and whether these claims can be justified. First, the claim that symptoms and environmental factors provide *causal* explanations cannot be corroborated using network analysis alone, and justifying causal claims using Woodward's interventionist theory requires meeting criteria for suitable interventions, which may not be met for mental disorders. Additional assumptions should also be met when claiming that the symptom network and environmental factors provide *mechanistic* explanations. The network theory could provide *topological* explanations, but this also poses some issues. Finally, conceptualizing mental disorders as *multilayer networks* could improve the explanatory breadth of the network theory, for it may accommodate mechanistic/causal and topological explanations.[22]

2.1 Introduction

Different psychiatric theories provide different explanations of why and how symptoms of mental disorders arise. The biomedical model explains psychiatric phenomena by conceptualizing symptoms as the effects of a (neuro)biological common cause. However, this explanatory model has been scrutinized on epistemic grounds, as shown in **Chapter 1**. As an alternative, the network theory conceptualizes mental disorders as (relatively stable) networks of interacting symptoms (Borsboom, 2017a; Borsboom et al., 2019a). By reconceptualizing the nature of mental disorders, the network theory attempts to provide alternative *explanations* for psychiatric phenomena.[23] The main premises of the network theory are discussed in **Table 2**.

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22. This chapter is based on the co-authored article by De Boer et al. (2021). As first author, I was responsible for the original article's conceptualization, drafts, review and editing, and final structure. I contributed to the content of all sections. Amongst others, changes to this article include using the first person to unite the style of the thesis, streamlining the terminology with the other chapters, changes to the structure, and removing claims discussed in more detail in later chapters (e.g., about network analysis). I would like to refer the reader to the original article for an exact overview of how the article and chapter compare.

23. I will discuss "psychiatric phenomena" in more detail in **Chapter 3**.

However, what explanations the network theory provides and how its explanatory claims are justified requires a more detailed discussion.

While the network theory places its main explanatory burden on symptoms and their relations, it highlights that non-symptom factors are relevant to developing, maintaining, and treating mental disorders. Indeed, Borsboom (2017a) elaborates on the second principle of the network theory – *symptom-component correspondence* – by stating that non-symptom factors play a role in the "psychopathology network" if they constitute symptoms, constitute a symptom-symptom connection, or act as variables in the "external field" that affects the symptom network. One relevant set of non-symptom factors is *environmental factors*, including adverse life events, social relations, as well as external objects such as gambling machines in gambling addiction (Borsboom et al., 2019a). The motivation for incorporating environmental factors into the network theory is that mental disorders are *multifactorial*; we can only fully understand and *explain* these disorders if we take different types of factors and their relation to each other into account (Kendler, 2008; Nolen-Hoeksema & Watkins, 2011). In this chapter, I examine the explanatory potential of the network theory in more detail by addressing 1) what explanatory claims related to symptoms and non-symptom factors the network theory makes and 2) whether these claims can be justified. Addressing these questions is important because proponents of the network theory want to provide a theory of what mental disorders *are* (Borsboom et al., 2019b). In later chapters, I will reflect on the status of psychiatric explanations (**Chapter 3**) and how the explanations the network theory provides influence scientists' ability to understand (**Chapter 3**) and explore (**Chapter 4**) psychiatric phenomena. Here, however, I examine the explanatory claims of the network theory in a practice-independent matter. I focus on the accounts of Borsboom (2017a) and Borsboom et al. (2019a) because these seminal articles on the network theory make various claims about the causal and constitutive role of symptoms and environmental factors in explaining (phenomena related to) mental disorders.

The chapter is structured as follows. In **Section 2.2**, I explore whether the claim that symptoms and environmental factors provide *causal* explanations of psychiatric phenomena can be corroborated using network analysis and Woodward's interventionist criteria. In **Section 2.3**, I focus on the constitutive claims of the network theory, exploring whether the symptom network and environmental factors can provide *mechanistic* explanations of psychiatric phenomena. They can only do so when additional assumptions are met. In **Section 2.4**, I address whether the network theory can provide *topological* explanations of the dynamics of psychiatric phenomena. In **Section 2.5**, I claim that conceptualizing mental disorders as *multilayer networks* could

improve the explanatory breadth of the network theory, for it could accommodate causal/mechanistic and topological explanations.[24]

2.2 Causal explanation

The network theory provides two types of causal explanations. First, the network theory states that *psychiatric symptoms* causally interact. Specifically, the third principle of the network theory – *direct causal connections* – emphasizes that symptoms directly cause each other. Network theory proponents justify this claim by referring to *folk psychology*: It *makes sense* for specific symptoms, but not others, to be causally related (Borsboom et al., 2019a). For instance, it makes sense that rumination causes insomnia, insomnia causes tiredness, and tiredness causes a lack of interest in activities we previously enjoyed. Borsboom et al. (2019a) justify this causal claim using an *interpretivist* interpretation of folk psychology: We attribute beliefs, emotions, and desires with specific content to ourselves and others to explain and predict behavior (Dennett, 1987). We can understand why symptoms cause each other by referring to their *intentional content* – i.e., what they are about – and people's *basic rationality*. For example, if we believe we may be spreading germs, it makes sense that we wash our hands excessively since hand washing is a reasonable strategy to prevent the spreading of germs.[25] However, those critical of the network theory could argue that intuition and sense-making are not necessarily reliable criteria for determining causal relationships. For instance, there are instances in which folk psychological reasoning alone cannot establish the directionality of a causal relationship: Did anhedonia cause rumination, or vice versa? So, the network theory's causal claims could benefit from additional justification.

Second, the network theory states that environmental factors cause symptoms, but this causal relation differs from symptom-symptom causation in two ways. The

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24. Colombo and Heinz (2019) also present an integrative theoretical framework for mental disorders. Specifically, they present an explanatory account of alcohol use disorder that integrates computational phenotypes and phenomenological information. Colombo and Heinz (2019) suggest that a dimensional framework is the best option for integration because it can accommodate multiple layers. They also discuss the network theory as an option but argue that it cannot include multiple layers. While a dimensional framework may be of interest when integrating symptom and environmental factors, the network theory (and Borsboom and Cramer (2013) they reference) does not reject a multilayer interpretation. This will be discussed in more detail in **Section 2.5**.

25. This example will also be discussed in **Chapter 8**. Note that issues have been raised with this interpretation. For instance, it requires a broad and pluralistic conception of folk psychology, with sense-making strategies expanding beyond standard interpretations of rationality (Slors et al., 2019). However, discussing this in detail goes beyond the scope of this thesis.

first difference is that causal effects of environmental factors on symptoms are considered *unidirectional*, whereas symptom-symptom causation may demonstrate feedback loops. Indeed, environmental factors are typically presented as catalysts or "background elements" of the symptom network: Symptoms can be "activated by factors external to the person" (Borsboom et al., 2019a, p. 4), such as losing one's partner. However, this symptom-symptom activation can spread, making the symptom network self-sustaining; this implies that removing the catalyst will not cause the symptom network to return to its earlier state (i.e., *hysteresis*) (Borsboom, 2017a). The second difference is that network theory proponents claim that environmental factors can also directly influence and determine the strength of symptom-symptom relations (Borsboom et al., 2019a). However, it is unclear on what grounds such causal effects of environmental factors on symptoms are justified, especially because environmental factors are also presented as *constitutive* of symptoms (or their relations), as I will discuss in **Section 2.3**.

In this section, I present two possible ways to corroborate and justify the claims that symptoms cause each other and that environmental factors cause symptoms network analysis and Woodward's (2003) interventionist theory of causation.

Network analysis

Using *network analysis* to corroborate the network theory's causal claims seems like a logical starting point since network theory originates in network psychometrics (Borsboom, 2008). I will discuss network analysis in more detail in **Chapters 3** and **4**, but in short, network analysis refers to statistical methods that estimate (corrected and robust) *conditional associations* between variables in a dataset, i.e., their statistical association when controlling for the other variables in the dataset. For example, Beard et al. (2016) demonstrate a statistically significant conditional association between "depressed mood" and "diminished interest" in a symptom network of people with a depression diagnosis. This conditional association can be interpreted as mood changes in depression predicting changes in interest and vice versa. Similarly, researchers have found conditional associations between environmental factors, such as living in an urban environment (Isvoranu et al., 2016) and spousal loss (Fried et al., 2015), and psychiatric symptoms. For instance, Fried et al. (2015) found a strong conditional association between "spousal loss" and "feeling lonely."

However, network analysis should not be conflated with the network theory, as Fried (2020) and Robinaugh et al. (2020) highlight. First, the network theory does not inform all decisions required for estimating network models. For instance, the network theory alone does not inform psychometricians what the threshold should be for statistical

significance or what regularization techniques to use to correct for false positives (Epskamp, Kruis, et al., 2017). Second, most network analysis studies – including the abovementioned studies – use cross-sectional, between-subject data, whereas the network theory refers to within-person phenomena. Identifying a relation in a between-subject design does not necessarily provide information on whether this relation is present *within* a person (Fisher et al., 2018). Although within-subject network analysis studies are being conducted (Bringmann et al., 2013), they still constitute the minority of the network analysis studies available.[26] So, network analysis and network theory are not necessarily *coordinated*.[27] Third, network models may be *statistically equivalent* to latent variable models, i.e., fit the same dataset equally well (Bringmann & Eronen, 2018). In that case, network models alone do not provide evidence for the network theory over theories that assume that symptoms have a common cause. Finally, we cannot assume that conditional associations imply causality: Covariance does not necessarily imply that one of the variables *influences* the other. As the classic example of the barometer and the storm goes, one can predict a storm using a barometer, but changing the pressure readings will not prevent the storm from happening. Thus, network analysis alone cannot corroborate the (symptom- or environmental factor-based) causal claims of the network theory: Network models generate findings that need to be explained but do not provide such explanations on their own.[28]

A possible solution is using other network analysis techniques that demonstrate causal rather than correlational associations between variables. For instance, causal inference methods estimate *directed acyclic graphs* (DAGs) based on correlational data. DAGs are causal networks without bidirectional effects or feedback loops (Pearl, 2000). DAGs have been used to demonstrate specific causal relations between psychiatric symptoms (e.g., Borsboom & Cramer, 2013) and between environmental factors and symptoms (e.g., Moffa et al., 2017). However, causal inference methods require certain assumptions to be satisfied: The variables in the DAG should include all relevant causal factors, there should be no unobserved confounding, and there should be no causal feedback loops.[29] In the next section, I show the difficulties in meeting these assumptions when applied to mental disorders.

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26. I will discuss within-subject network analysis using time-series data, or personalized network analysis, in **Chapter 5**.
27. I will discuss "coordination" in more detail in **Chapter 3**.
28. Network analysis also cannot provide evidence for the claim that environmental factors can influence the relationship between two symptoms, given that network analysis cannot model this.
29. Statistical methods that estimate *directed cyclic graphs* can account for feedback loops in causal graphs (Richardson, 1996; Spirtes, 1995). Recently, Park et al. (2024) explored the applicability of such methods in psychological research. However, since the assumptions of directed cyclic graphs are stricter than DAG assumptions, it is unlikely that these will be met when studying mental disorders.

Interventionism

A second option for justifying the network theory's causal claims is to refer to (hypothetical) interventions. Indeed, Borsboom (2017a) argues that "such causal interaction between symptoms can be interpreted using interventionist theories of causation" (p. 6). Woodward's (2003) interventionist theory of causation has become one of the most influential approaches to causation in the past decades. Interventionism presents a counterfactual approach to causation that conceptualizes causal relations in terms of changes resulting from possible (i.e., hypothetical) interventions. If there is a possible intervention on *X* that leads to a change in *Y* while holding fixed all other variables that could change *Y*, then *X* causes *Y*. A good intervention meets the following criteria:

1) It causes *X*;
2) It acts as a switch for other variables that cause *X*;
3) It does not cause *Y* via any other path than via *X* and
4) It is independent of any variable *Z* which causes *Y* and is on a directed path that does not go through *X* (Woodward, 2003, p. 98).

Interventionism allows us to make claims on the relations between variables that go beyond correlation. It may not always be empirically possible to construe interventions on symptoms or environmental factors, but this is not necessarily problematic: Interventionism requires hypothetical interventions that meet the abovementioned conditions (Woodward, 2008a). So, if hypothetical interventions on symptoms or environmental factors that adhere to Woodward's criteria can be construed, we can justify the network theory's causal claims. In this section, I show the difficulties in coming up with such interventions based on the characteristics of mental disorders.

If we focus on justifying symptom-symptom causation based on interventionist criteria, we see that this may not be as easy as posed.[30] First, it is uncertain whether we can truly eliminate the possibility of a common cause in symptom networks, for this requires us to know (and include) all factors causally related to a mental disorder. If not, the causal relationship may be ultimately due to confounding. However, given the intractability of mental suffering, it is unlikely that we can know all the causal determinants of specific mental disorders. The network theory acknowledges this: The first principle of the network theory – *complexity* – highlights that mental disorders are "multifactorial in constitution, etiology, and causal background" (Borsboom, 2017a, p. 7). If we do not know these different factors, the causal relation between symptoms may ultimately be due to confounding.

30. Eronen (2020) and Eronen and Bringmann (2021) provide a general account of the difficulties in corroborating causal relations between psychological variables using interventionist criteria.

Even if we knew all relevant causal variables, we would still be left with a second problem: It is uncertain whether we can come up with *surgical* hypothetical interventions on psychiatric symptoms, i.e., interventions that do not influence other variables in the symptom network. Can we intervene on a symptom while keeping other variables in the network stable? Many symptom interventions likely have effects on *Y* that do not go through *X* (violation of the third criterion) or influence a variable *Z*, which causes *Y* and is not on a directed path through *X* (violation of the fourth criterion; Romero, 2015). For example, a peer support group may not be a good surgical intervention to assess whether using medication causes a stable mood because the peer support group may enhance one's motivation to use medication but also facilitate participation in meaningful activities and interaction with helpful group members, which could influence one's mood (cf. De Bruin, 2020). We could solve this problem by allowing for *fat-handed* rather than surgical interventions, i.e., interventions that not only affect *X* and other variables on the route from *X* to *Y* but also affect variables affecting *Y* which are not on this route (Eberhardt, 2014; Romero, 2015; Woodward, 2008b, p. 209).

However, even if we allow fat-handed interventions, a third problem arises: We cannot take for granted that psychiatric symptoms are distinct and non-overlapping entities. The second principle of the network theory – *symptom-component correspondence* – states that symptoms are defined at the right level of detail and specificity and "successfully identify the important components in the psychopathology network" (Borsboom, 2017a, p. 7).[31] However, it is difficult to pinpoint individual mental states – including psychiatric symptoms – as suitable targets for intervention (Woodward, 2008a). For example, there may be a conceptual overlap between the symptom "depressed mood" and the symptom "diminished pleasure." This is problematic for applying interventionism to symptoms: If we cannot clearly differentiate between two symptoms, we cannot come up with an intervention that does not directly affect both.[32] Thus, the nature of mental disorders and psychiatric symptoms, more specifically, makes it difficult to corroborate symptom-symptom causality using interventionist criteria.

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31. Borsboom (2017a) uses "granularity" rather than "detail and specificity," but I assume this was implied.

32. Interestingly, Woodward (2008a) argues that the multiple realizability of psychiatric symptoms (i.e., the assumption that multiple different physical/neural states could realize psychiatric symptoms) could be problematic for applying interventionism to mental disorders, whereas Borsboom et al. (2019a) refer to interventionism and use multiple realizability as an argument against the biomedical model (for multiple realizability would hamper the possibility of reducing symptoms to brain states).

Finally, interventionism can account for *acyclic* networks but not for *feedback loops*. In real life, symptoms likely influence each other via feedback loops; the network theory acknowledges this with its fifth principle, *hysteresis*. For example, a feedback loop may be present between insomnia, fatigue, concentration problems, and stress (insomnia causes fatigue, which causes concentration problems, which causes stress, which causes insomnia, et cetera). If this is the case, an intervention on insomnia that affects fatigue does not act as a switch for concentration problems and stress, thereby violating Woodward's (2003) second criterion for good interventions. It may be possible to circumvent this problem by considering the temporal relations between symptoms (cf. Dijkstra & de Bruin, 2016), but these relations may not always be easy to discern. Relatedly, if symptoms are too dependent on each other to discern their individual contributions, our ability to make claims about their causal relationships is hampered. So, the interventionist criteria that should be satisfied to call a relationship between symptoms causal cannot always be met, given the nature of mental disorders.

The causal effects of environmental factors on symptoms could be more easily corroborated using interventionist criteria. Some problems that apply to symptom-symptom causation apply here, too. For instance, we cannot ascertain that hypothetically intervening on an environmental factor only affects one symptom (e.g., people from a stressful home environment may affect both their mood and agitation), and we cannot ascertain that all relevant causal factors are considered. However, the proposed relationship between environmental factors and symptoms is unidirectional rather than cyclical, with environmental factors serving as catalysts or background elements of the symptom network. Relatedly, it may be possible to establish the temporal order of events for some environmental factors. For example, certain adverse life events may have happened before the present-day symptoms arose. So, for environmental factors that are temporally distinguishable from the onset of symptoms and under some weak interpretations of interventionism (e.g., allowing for fathandedness), we could establish a causal relation between environmental factors and symptoms using interventionist criteria. However, this does not apply to the causal effect of environmental factors on the relationship between symptoms. Since intervening in a symptom-symptom relationship would likely lead to changes in both symptoms, coming up with hypothetical interventions in this context that meet interventionist criteria seems highly improbable.

In response, proponents of the network theory could still justify their claim that symptoms cause each other by referring to folk psychological principles. However, this section shows that network analysis alone cannot corroborate the network theory's causal claims and that justifying these causal claims using interventionist

criteria requires specific assumptions to be met. These assumptions may be met for some effects of environmental factors on symptoms but may be more difficult to meet for symptom-symptom relations and the relationship between environmental factors and symptom-symptom relations. In the next section, I will examine the network theory's mechanistic explanatory potential.

2.3 Mechanistic explanation

The network theory suggests two types of constitutive relations. First, it states that *symptoms and their causal interactions* constitute mental disorders.[33] However, like the causal relations the network theory poses, this constitutive claim is not straightforward. For instance, there is considerable variation in the type of symptom combinations one can have to receive a psychiatric diagnosis. Given this observation, how can we claim that all these diverse combinations constitute the same disorder?[34] So, the network theory may benefit from justification criteria for these constitutive claims. Second, proponents of the network theory claim that *environmental factors* are constitutively related to (aspects of) mental disorders. Borsboom et al. (2019a) claim that environmental factors can be part of the mechanisms that constitute mental disorders: Network structures "rest on or invoke mechanisms in the environment" (p. 8). Moreover, Borsboom et al. (2019a) state that "we should expect to find interactions between symptoms to be grounded in an even more complex set of biological, social, and cultural factors involved in psychopathology" (p. 10). To illustrate this, they examine the role of a Roulette table in gambling addiction. They state that the relationship between excessive gambling and debt – both symptoms of gambling addiction – is realized by the gambling setups that require a monetary investment, such as, Roulette tables. If we imagine a world without Roulette tables or with Roulette tables that are operationalized differently, there would not be a link between excessive gambling and debt. Hence, they claim that environmental factors – such as Roulette tables – are an integral part of the relationship between these symptoms. Finally, Borsboom et al. (2019a) claim that environmental factors can co-constitute a mental disorder:

> [T]he environment itself may become part of the network structure, and hence part of the disorder. More or less by definition, this means that (…)

33. This claim is not made explicitly by Borsboom (2017a) or Borsboom et al. (2019a) but has been endorsed and explained by Borsboom (2008), Fried and Cramer (2017) and Oude Maatman (2020).

34. Disorder heterogeneity also poses a problem for the biomedical model: How can we justify referring to a common cause when there is substantial heterogeneity in the way mental disorders are manifested?

> cultural and historical factors as well as external mechanisms, to some extent, shape mental disorders (p. 8).

So, network theory proponents claim that environmental factors can constitute symptoms, symptom-symptom relations, and the disorder itself. Again, these claims could benefit from additional justification. Interestingly, the constitutive role of environmental factors is explicitly discussed in terms of *mechanisms*. In this section, I explore whether the network theory's constitutive claims can be justified using the criteria for mechanistic explanations.

Mechanistic explanations are concerned with the representation of mechanisms underlying a phenomenon or system, i.e., a phenomenon's components, the components' operations, and their causal organization (Craver & Kaplan, 2020). A mechanistic explanation of chemical neurotransmission, for example, appeals to entities (or components such as ions, neurotransmitters, vesicles, and membranes) and their operations (or activities such as depolarizing, diffusing, priming, docking, and fusing) organized together so that they do something – in this case, reliably preserve a signal across the space between cells (Piccinini & Craver, 2011). Providing mechanistic explanations is the primary explanatory strategy in the life sciences, but this does not imply that it necessarily goes hand in hand with the biomedical model. Although mechanistic explanations are reductionist insofar as they appeal to entities and operations at a lower level of organization, they do not advocate a sole focus on (neuro)biology. Indeed, mechanistic explanation typically involves multiple levels of organization and does not privilege the lowest level. This means that the network theory could theoretically provide mechanistic explanations, even without including (neuro)biological information.[35] However, assumptions should be met before we can claim that the (extended) network theory has mechanistic explanatory potential.

First, claiming that symptoms *and* environmental factors are part of the mechanisms underlying mental disorders assumes that these mechanisms can be *extended*. However, this assumption is not uncontroversial. This is reflected in discussions on the possible extension of cognitive mechanisms. Some philosophers have argued that cognitive mechanisms are situated in and dependent on the environment but that we should not consider environmental factors part of the mechanism that explains cognitive phenomena. For example, Bechtel (2009) states that

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35. Some may argue that the network theory cannot provide mechanistic explanations because of its "flatness": Mechanistic explanations require the presence of multiple layers. I address this claim in **Section 2.5**.

> for mental phenomena it is appropriate to treat the mind/ brain as the locus of the responsible mechanism and to emphasize the boundary between the mind/brain and the rest of the body and between the cognitive agent and its environment. (p. 156)

However, Craver (2007) suggests that

> many cognitive mechanisms draw upon resources outside of the brain and outside of the body to such an extent that it is not fruitful to see the skin, or surface of the central nervous system (CNS), as a useful boundary. (p. 141)

If we extrapolate Craver's (2007) claim to mental disorders, we may assume that their mechanisms can be extended if we define mental disorders in an extended sense to include the brain, body, and environment – as the network theory does. However, doing so introduces a related problem: Where should we draw the boundary of the disorder and the mechanism we want to describe? Recall the example by Borsboom et al. (2019a), where gambling machines are part of the mechanism that explains gambling disorder. Why should the mechanism not also include other external entities or events, such as gambling legislation, entry tickets, or socio-cultural norms regarding gambling? To use another example, having an opioid use disorder depends heavily on the availability of opioids. Does this mean that the person who provides these drugs should be considered part of the disorder mechanism? These examples show that claiming that environmental factors are part of a mental disorder's mechanism raises questions on the *boundaries* of mental disorders: Where to draw the line between 1) factors that are explicitly part of the mechanism and thus constitutive of the phenomenon that we want to explain, and 2) other external factors that simply causally influence the mechanism or are preconditions for the mechanism's emergence?

As a possible solution, we could resort to Craver's (2007) *mutual manipulability* criterion to decide whether a part or its activity is constitutively relevant to a phenomenon. According to this criterion, the behavior of a spatiotemporal part *X* of a system *S* is constitutively relevant to *S*'s behavior if, and only if, the behaviors of *X* and *S* can be mutually manipulated. Craver (2007) defines manipulability as a change in behavior brought about by an intervention à la Woodward (2003). This demarcation criterion is attractive because it could transform the philosophical debate about whether environmental factors cause or constitute symptoms into a tractable, empirical debate (Kaplan, 2012). However, Craver's mutual manipulability criterion has been criticized on conceptual grounds: It undermines the fundamental

distinction between causation and constitution, for constitution is typically treated as a *non-causal* dependency relation between lower-level parts and higher-level mechanisms. Some suggest using the fat-handed intervention criterion to define constitutive relationships in interventionist terms (Baumgartner & Casini, 2017; Baumgartner & Gebharter, 2016; Romero, 2015). However, as shown in **Section 2.2**, coming up with fat-handed interventions for mental disorders already faces challenges. Hence, it is uncertain whether adding this demarcation criterion would help to decide on the issue when it concerns mental disorders.

Moreover, providing mechanistic explanations requires that a phenomenon is *decomposable* in terms of components (*structural decomposition*) and operations (*functional decomposition*). Recall the example of chemical neurotransmission: This phenomenon is mechanistically explainable because it is structurally decomposable in terms of ions, neurotransmitters, vesicles, and membranes and functionally decomposable in terms of depolarization, diffusion, priming, docking, and fusion. However, it is unclear whether we can assume that mental disorders are decomposable. We can distinguish between two types of systems with different levels of decomposability. In a *nearly decomposable system*, the behavior of the system's components is integrated, but the components can still be understood and studied independently. Bechtel (2009) argues that cognitive systems are nearly decomposable and can thus be explained mechanistically. In a *non-decomposable system*, the (short-term) behavior of the system's components highly depends on the behavior of other individual components. Since no subsystems of components are (nearly) independent, the system cannot be explained mechanistically (Rathkopf, 2018). It is unclear whether mental disorders can be best described as nearly- or non-decomposable systems; this is an open-ended question.[36] If we assume that mental disorders are nearly decomposable systems, then the network theory's description of mental disorders in terms of symptoms and environmental factors provides a mechanism sketch that can be filled in with more (structural) details as more research becomes available (Piccinini & Craver, 2011). However, we can also assume that mental disorders are non-decomposable systems, especially if we focus on the presence of causal feedback loops. If mental disorders are characterized by circular causality – i.e., a given component of the system is both continuously affecting and simultaneously being affected by activity in another component – it is difficult to identify the contribution of the component in question in terms of the underlying structural entities (Lamb & Chemero, 2014).[37] Even if this were possible,

36. As I will discuss in **Chapter 8**, the network theory is compatible with both interpretations of decomposability.

37. Note that the concept of "circular causality" has been criticized (Bakker, 2005).

we still face the problem discussed previously: Individual symptoms may not be easily differentiated on conceptual grounds, limiting the decomposability of mental disorders. If we assume, based on these considerations, that mental disorders are non-decomposable systems, we cannot explain them mechanistically nor substantiate the network theory's constitutive claims using mechanistic explanatory criteria.[38]

So, the (extended) network theory's constitutive claims can only be interpreted mechanistically if we assume that the network theory's mechanisms can be extended, that causal and constitutive relations can be differentiated and that mental disorders are nearly decomposable. In the next section, I will build upon this latter criterion to address whether the network theory provides *topological explanations*.

2.4 Topological explanation

The network theory not only refers to individual relations between factors but also to the characteristics of symptom networks *as a whole*. For instance, the fourth principle of the network theory – *mental disorders follow a network structure* – claims that the psychopathology network, an inter-diagnostic network including all possible psychiatric symptoms, "has a non-trivial topology, in which certain symptoms are more tightly connected than others. These symptom groupings give rise to the phenomenological manifestation of mental disorders as groups of symptoms that often arise together" (Borsboom, 2017a, p. 7). So, the psychopathology network features *clustering*, i.e., groups of strongly related nodes (Borsboom et al., 2011). However, the network theory also implies that the characteristics of symptom networks as a whole can *explain* how mental disorders develop and maintain. Indeed, building on the fifth principle – *hysteresis* –, Borsboom (2017a) argues that high symptom network connectivity can explain the dynamics of mental disorders (i.e., symptoms continuing to activate each other after the initial activation of one symptom). In this section, I explore whether these claims are compatible with a topological explanatory strategy.

Topological explanations explain the dynamics of complex systems using *topological properties*, i.e., systemic properties that are mathematically quantified using graph theory (Kostić, 2019a). **Table 3** provides a (non-exhaustive) overview of topological

38. One could argue that (structural) decomposition is not essential for mechanistic explanations (Zednik, 2014) and that it is more important that mechanistic explanations demonstrate how phenomena are "situated in the causal structure of the world" (Craver, 2013, p. 134). However, as argued previously, demonstrating causal relationships for mental disorders also poses issues.

properties. To illustrate these properties in more detail, I build upon the case study of Watts and Strogatz (1998) introduced in **Chapter 1**. Watts and Strogatz (1998) used network science to study two topological properties: *characteristic path length* (i.e., the average shortest path length between all pairs of nodes in the network) and *clustering coefficient* (i.e., the number of pairs of neighbors of a node that are directly connected, divided by the number of potential pairs of nodes in that neighborhood) (**Table 3**). They created formal network models with short characteristic path lengths and high clustering coefficients, i.e., *small-world networks*. First, they discovered empirically that many real-world phenomena demonstrate a small-world property when represented as networks. Second, they used the small-world property to study, amongst others, how infectious diseases spread. They construed a simulation network model where nodes refer to individuals in the population and disease spreads along the edges. This simulation model showed that infectious diseases spread easily and quickly when populations demonstrate the small-world property. This example illustrates that topological properties can explain the dynamics of a system constituted by interacting parts. However, what exactly is meant by *explaining* in this context?[39]

39. Some philosophers have questioned the explanatory potential of topological properties. For example, Craver (2016) argues that topological explanations are *exploratory* because they cannot distinguish good from bad explanations. Moreover, one could argue that topological explanations do not provide information on *why* specific topological properties, and not a relevant contrast class, yield these network dynamics. In **Chapter 5**, I will discuss topological explanations in more detail and touch upon some of these critiques.

Table 3. A non-exhaustive overview of topological properties of networks

Local topological properties	
Path length	The number of edges required to get from one node to another.
Node degree	The sum of edges maintained by a single node.
Node strength	The sum of the weights of edges maintained by a single node (Barrat et al., 2004).
Edge betweenness	The number of the shortest paths between pairs of nodes that go through that specific edge (Girvan & Newman, 2002).
Betweenness centrality	The relative number of shortest paths between any pair of nodes passing through a node (Freeman, 1977). This measure has been taken as an indication of the role of a node in information flow or communication in a network.
Closeness centrality	The average shortest distance from a node to all other nodes in a network (Borgatti, 2005).
Eigenvector centrality	The extent to which a node is connected to central nodes. It is proportional to the sum of the degrees of a node's neighbors (Bonacich, 1972, 1987).
Cluster topological properties	
Clustering coefficient (or local clustering)	The number of pairs of neighbors of a node that are directly connected, divided by the number of potential pairs of nodes in that neighborhood (Watts & Strogatz, 1998). A measure of the cliquishness of a "neighborhood" in a network.
Community structure	The presence of groups of nodes with strong internal and weak external connections. Community detection refers to means of detecting whether a network is subdivided into separate (non-overlapping, interconnected) modules (Newman, 2006).
Global topological properties	
Characteristic path length	The average shortest path length between all pairs of nodes in a network.
Global degree	The average sum of edges maintained by nodes in a network (Barrat et al., 2004).
Global topological properties	
Network connectivity (or global strength)	The weighted absolute sum of all edges maintained by nodes in a network (Opsahl et al., 2010).
Network density	The edges present in a network, relative to the number of potential edges (Bullmore & Sporns, 2009).
Average clustering coefficient	The mean of clustering coefficients (Watts & Strogatz, 1998), indicates a network's robustness.
Small-worldness	The ratio of clustering coefficient to path length (Watts & Strogatz, 1998). Networks that demonstrate small-worldness are more efficient than randomly connected networks.

According to Kostić (2020), a topological explanation supports a counterfactual dependency between a topological property and a phenomenon related to a system's dynamics: The phenomenon would have been different if the topological property had not been there. Kostić distinguishes between two types of topological explanations: a *vertical* topological explanation, in which a global topological property (characteristic of the network as a whole) determines a phenomenon related to a system as a whole, and a *horizontal* topological explanation, in which a local topological property (characteristic of a part of the network) determines a phenomenon related to a system's local dynamical properties. Kostić (2020) illustrates the difference between these two modes of explanation by focusing on *cognitive control*, i.e., the ability of the brain – as a dynamical system – to transition between internal states efficiently. Suppose we want to answer the question, "Why can the brain achieve cognitive control?". In that case, we can provide the following relevant vertical counterfactual: "If the brain had not been a small-world network, it would not have been able to achieve cognitive control." Suppose we want to answer the question, "How and why can the brain efficiently transition between states?". In that case, we can provide the following relevant horizontal counterfactual: "Had the brain's local topological properties not determined the energy requirements for those transitions, these energy requirements would have been different." Kostić (2020) proposes three ways such counterfactual dependencies can account for *explanatory asymmetry*, i.e., the topological property explaining the phenomenon and not vice versa. First, *property asymmetry*: The explanandum (e.g., cognitive control) is not mathematically quantified, whereas the topological property (e.g., small-worldness) is. Second, *counterfactual asymmetry*: The explanandum depends on the topological property, but the topological property does not depend on the explanandum. Third, *perspectival asymmetry*: Reversing the direction of the explanation makes the claim non-explanatory. Suppose we want to answer the question, "Why does a system demonstrate a certain topological property?" If so, referring to the phenomenon is not a scientifically relevant answer. So, a good case can be made for the explanatory asymmetry of topological explanations.

The network theory could provide topological explanations. For instance, its fifth principle – *hysteresis* – can be interpreted as a vertical topological explanation: A global, mathematically quantifiable network property (i.e., high network connectivity, or a high weighted absolute sum of all the network's edges, **Table 3**) explains the phenomenon that some people are more vulnerable than others to developing a mental disorder and relapsing. Suppose we want to answer the question, "Why are some people vulnerable to relapsing into their depression?". In that case, the fifth principle provides the following relevant vertical counterfactual: "If their

symptoms had not been strongly connected, their likelihood of relapsing would have been smaller." This counterfactual dependency is supported by empirical evidence. For instance, Borsboom (2017a) refers to the within-subject simulation study by Cramer et al. (2016) that demonstrates that in depression, altering a parameter that determines symptom network connectivity changes the network's vulnerability. When the nodes are highly connected, this increases the likelihood that activation of one symptom leads to activation of other symptoms, making it less likely for these symptoms to disappear. Other empirical studies have also suggested that increased network connectivity may predict the transition from a healthy to a disordered state (Van de Leemput et al., 2014; Wichers et al., 2011). Relatedly, Van Borkulo et al. (2015) demonstrate that high symptom network connectivity in depression is associated with having a persistent diagnosis after two years. So, the network theory can resort to topological explanations that counterfactually explain phenomena related to the dynamics of mental disorders.

An appealing feature of topological explanations is that they can – and should – be used to explain phenomena related to non-decomposable systems (Rathkopf, 2018). To illustrate this, Rathkopf (2018) uses the topological property *edge betweenness*, i.e., the number of the shortest paths between pairs of nodes that go through that specific edge (Girvan & Newman, 2002) (**Table 3**). Betweenness is a measure of the centrality of an edge in the network. To compute edge betweenness, the shortest path length between all pairs of nodes in the network is determined, after which the proportion of those paths incorporating that edge is calculated. So, the edge betweenness value indirectly refers to the rest of the network. Rathkopf (2018) claims that this shows how topological properties combine complex patterns of interaction into one meaningful variable with explanatory power, thereby making phenomena related to non-decomposable systems "epistemically accessible" (Rathkopf, 2018, p. 72). So, topological explanations help explain psychiatric phenomena even if we assume that mental disorders are non-decomposable.

However, the topological explanatory strategy poses some difficulties when applied to mental disorders. First, providing the right topological explanation requires that the topological property and the psychiatric phenomenon it aims to explain are "empirically adequate," i.e., conform to the observable world or empirical evidence (cf. Kostić, 2020).[40] As I mentioned in **Section 2.2** and will discuss in more detail in

40. Throughout this thesis, I use "empirically adequate" rather than "approximately true" (which is the terminology used by Kostić (2020) in this specific context) to refer to the assumption that theories, models, and the aspects of the world they study should conform to the observable world (De Regt, 2017; Douglas, 2009). I do so because "empirically adequate" carries fewer metaphysical associations.

Chapter 3, network analysis studies can only provide evidence for topological counterfactual dependencies if they are adequately coordinated with the network theory. Hence, not every network analysis study can substantiate (topological) explanatory claims. Second, what constitutes a *relevant* counterfactual for a topological explanation of a psychiatric phenomenon is not always clear. Take the previous example: "If their symptoms had not been strongly connected, their likelihood of relapsing would have been *smaller*." Is the relevant counterfactual a scenario in which people do not relapse at all, relapse less frequently, or develop less severe symptoms in case they do relapse? Third, interpreting what psychiatric phenomena are explained by global and local topological properties is not straightforward. For example, Borsboom (2017a) suggests that *centrality* measures could signify "the functional role and importance of specific symptoms in maintaining disorders" (p. 10). However, Bringmann et al. (2019) suggest that certain centrality measures (e.g., betweenness and closeness centrality) may not have meaningful interpretations when applied to mental disorders because they depend on assumptions that symptom networks may not meet. So, the network theory could provide topological explanations, although this does require additional considerations.

A final issue is that, thus far, I have only focused on how topological properties of the *symptom network* may explain psychiatric phenomena. Can non-symptom factors – such as environmental factors – also provide or contribute to topological explanations of psychiatric phenomena? Network analysis studies have shown that symptom network dynamics may change depending on the presence of specific environmental factors (e.g., Hasmi et al., 2018). However, multiple environmental factors and their dynamics may also influence symptom network dynamics. In the next section, I will explore how the network theory could accommodate non-symptom factors in a way that could improve its causal/mechanistic and topological explanatory potential, i.e., by conceptualizing mental disorders as *multilayer networks*.

2.5 Mental disorders as multilayer networks

A multilayer network can be defined as a network of networks, i.e., a network comprised of multiple layers with connections between and within the layers. In recent years, statistical techniques have been developed to estimate such networks (Bianconi, 2018; Boccaletti et al., 2014; Kivelä et al., 2014). Multilayer network analysis has been used to study, amongst others, social, biological, and transport systems (Boccaletti et al., 2014; De Domenico et al., 2014, 2016; Mucha et al., 2010). It is also increasingly used in network neuroscience to integrate different neuroimaging

modalities (e.g., comparing brain regions' structural and functional connectivity) or to study brain networks over different time points (De Domenico, 2017; Vaiana & Muldoon, 2018). An advantage of multilayer network analysis over "standard" network analysis is that the latter often requires data to be aggregated, for instance via averaging, or ignored. Multilayer network analysis can retain this information by including it in different layers, making it better suited to deal with multidimensional data and allowing for analyses that could not be performed when focusing on one layer of analysis only. So, multilayer network analysis may offer epistemic benefits that standard network analysis does not.

Braun et al. (2018) suggest that multilayer network analysis can also be beneficial for studying mental disorders. For instance, multilayer network analysis could be used to study psychopathology networks over different time points. However, I want to focus on a second function: using multilayer network analysis to integrate information from different dimensions (e.g., symptoms, neurobiological factors, and environmental factors) as layers in the multilayer network structure. Multilayer network analysis typically requires nodes to be replicated over the different layers, but scientists are developing multilayer network analysis methods that do not require node replication (e.g., Brooks et al., 2020). Such methods could enable scientists to statistically estimate the dynamics amongst and between different domains relevant to the development of mental disorders, e.g., by construing multilayer networks with a symptom, environmental, and neurobiological layer.[41] In **Chapter 4**, I will assess the *exploratory* potential of multilayer network analysis of mental disorders in more detail. In this section, however, I focus on the *explanatory* potential of conceptualizing mental disorders as multilayer networks. Indeed, Braun et al. (2018) state that statistical innovations in multilayer network analysis techniques should be paired with *theoretical* innovations, i.e., our psychiatric theories should do justice to the dimensional and multilayered nature of mental disorders. Although network theory proponents do not explicitly conceptualize mental disorders as multilayer networks, their claims are compatible with this view. First, conceptualizing mental disorders as multilayer networks is compatible with the claim that "basically every element of the system is dependent on a heterogeneous set of biological and external

41. This type of multilayer network analysis cannot be used to study the association between non-symptom factors, such as environmental factors, and symptom-symptom relationships.

factors" (Borsboom et al., 2019a, p. 9).[42] Second, proponents of the network theory claim that environmental factors could be part of the *mechanism* that *constitutes* symptoms or symptom-symptom relations, as discussed in **Section 2.3**. A multilayer network interpretation of mental disorders could account for this claim. Moreover, conceptualizing mental disorders as multilayer networks may also improve the network theory's *explanatory breadth*: It could allow the network theory to provide (extended) causal/mechanistic and topological explanations. I will discuss this statement in more detail.

A multilayer interpretation of the network theory could enhance the theory's mechanistic explanatory potential by incorporating different domains that may be part of the mechanisms underlying mental disorders. This interpretation of the network theory is compatible with the claim that mental disorders are *mechanistic property clusters* (MPCs): clusters of properties that span multiple layers and are maintained by interacting, dysfunctional, and self-sustaining mechanisms (Kendler et al., 2011). The MPC account assumes that complex and multi-layer causal mechanisms – including genetic, cellular, neural, psychological, environmental, and sociocultural factors – produce, underlie, and sustain mental disorders (Kendler, 2008). Various proponents of the network theory claim that it is compatible with the MPC account (Borsboom & Cramer, 2013; Robinaugh et al., 2020).[43] A multilayer interpretation of the network theory would further highlight these similarities. However, as claimed in **Section 2.3**, providing causal/mechanistic explanations using the network theory requires decomposability. A multilayer interpretation of the network theory could accommodate layers with a high degree of decomposability, such as a structural neurobiology layer consisting of brain regions and their anatomical connections (based on diffusion-weighted magnetic resonance imaging, for instance). Such layers may allow for structural decomposition – as the mechanistic explanatory strategy requires – and may meet more criteria for good interventions than purely functional layers; hence, their inclusion could provide local causal explanations of specific psychiatric phenomena. So, a multilayer interpretation of the network theory may enhance its causal/mechanistic explanatory potential, but this depends on the decomposability of the layers that such a theoretical framework would incorporate.

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42. As mentioned in **Chapter 1**, network theory proponents seem sympathetic to the idea that different domains that play a role in mental disorders may represent different network structures. Borsboom et al. (2019b) argue that symptom networks may relate to biological networks in a part-whole relationship or with biological networks being nested in a symptom network. They acknowledge that it is unclear how nested networks could be modeled statistically. The fact that multilayer networks can be statistically modeled is an additional advantage.

43. The compatibility between the network theory and MPCs will also be discussed in **Chapter 8**.

A multilayer interpretation of the network theory could enhance the theory's topological explanatory potential by accommodating topological explanations beyond symptom networks. Such an interpretation can do justice to the idea that interactions between non-symptom factors are relevant for explaining psychiatric phenomena.[44] Specifically, a multilayer interpretation of the network theory may allow for topological explanations that span multiple layers. Scientists have extended traditional topological property measures to multilayer networks and developed methods to quantify topological properties specific to multilayer structures (see Vaiana & Muldoon, 2018 for an overview). Multilayer network analysis studies show that multilayer topological properties provide information that could not have been provided when focusing on one network layer only. For example, De Domenico et al. (2015) demonstrate that hubs in multilayer neural networks differ dramatically from hubs in separate network layers, and Battiston et al. (2014) show that two layers in a multilayer network can exhibit different network properties but share specific hubs and motifs (i.e., characteristic recurrent connection patterns). This suggests that multilayer topological explanations may explain different psychiatric phenomena than standard topological explanations. What could a multilayer topological explanation look like in the context of mental disorders? A topological property that could be exploited is *community structure*, i.e., the presence of groups of nodes with strong internal and weak external connections (**Table 3**). A multilayer interpretation of the network theory could refer to multilayer clustering as a possible explanation of the heterogeneity within mental disorder classifications by identifying subtypes of people with different multilayer topologies (e.g., including different symptoms and neurobiological factors, similar to a suggestion in the context of personality science, Brooks et al., 2020).[45]

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44. Relatedly, the topological properties of non-symptom layers may provide information about the topological properties of symptom networks and vice versa. As discussed in **Section 2.4**, the fifth principle of the network theory – *hysteresis* – refers to high symptom connectivity to explain the vulnerability to developing mental disorders. Mental disorder-related changes in connectivity patterns have also been demonstrated in neural networks at multiple layers of brain organization (Van den Heuvel et al., 2019; Van den Heuvel & Sporns, 2019). So, the dynamics of non-symptom layers of the multilayer network structure may provide information about the dynamics of the symptom network.

45. If a multilayer interpretation of the network theory focuses on different dimensions *and* time points, i.e., mental disorders are (stable sets of) causally interacting symptoms that develop over time (Kästner, 2022), we can also use (changes in) community structure over time as an entry point for multilayer topological explanations. This aligns with Braun et al. (2018), who suggest that multilayer community structures in brain networks of people with a psychiatric diagnosis could be associated with critical time points in their clinical development.

So, a multilayer interpretation of the network theory could accommodate multilayered causal/mechanistic and topological explanations of mental disorders; it could address various explanation-seeking questions. This implies that conceptualizing mental disorders as multilayer networks could improve the *exploratory breadth* of the network theory. More theoretical and empirical (statistical) research is needed to explore the epistemic potential of a multilayer interpretation of the network theory; I will touch upon this in **Chapter 5**. Theoretically, it is unclear how the different layers should be defined, how the nature of the different relations should be conceptualized, and to what extent the multilayer network framework is compatible with other theoretical frameworks in psychiatry. For instance, a multilayer network framework may also be applied to the RDoC initiative mentioned in **Chapter 1**: Different research domains may be represented as different layers of a multilayer network. Empirically, further research should explore what methods are best suited to statistically estimate multilayer networks of mental disorders.[46]

2.6 Conclusion

In this chapter, I critically examined the explanatory potential of the (extended) network theory. Proponents of the network theory claim that causally interacting symptoms constitute mental disorders and that environmental factors causally and mechanistically influence symptoms and mental disorders in general. Hence, they suggest that the (extended) network theory could provide causal and mechanistic explanations of mental disorders. In this chapter, I showed that to justify these claims, various assumptions should be satisfied. We cannot make causal claims based on network analysis alone, and justifying causal claims using Woodward's interventionist theory requires meeting criteria for suitable interventions, which may not always be met for mental disorders. Moreover, the (extended) network theory can provide mechanistic explanations if we assume that the mechanisms underlying psychiatric phenomena can be extended, that causal and constitutive relations can be differentiated and that mental disorders are nearly decomposable. The network theory could provide topological explanations of psychiatric phenomena, but this also poses some issues. Finally, conceptualizing mental disorders as multilayer networks could improve the explanatory breadth of the network theory, for it may accommodate causal/mechanistic and topological explanations.

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46. Another open-ended question is how a multilayer network interpretation of the network theory can be applied beyond scientific practice, for instance, in clinical practice. In the clinical context, consultation with clinicians and experts by experience could help examine which layers and relations are relevant to include.

In this chapter, I highlighted the difficulties in making explanatory claims about psychiatric phenomena in general and within the network theory more specifically. Amongst others, I showed that ensuring the empirical adequacy of the network theory's explanatory claims is not straightforward. Moreover, I suggested that the network theory's epistemic potential could be heightened by improving its explanatory breadth. However, the network theory's empirical adequacy and explanatory breadth alone do not drive its epistemic potential. In line with my practice-oriented account sketched in **Chapter 1**, we should focus on how scientists *use* the explanations provided by the network theory to *reason* about possibilities for psychiatric phenomena. I will do so in the upcoming chapters, addressing the network theory *and* network models.

Chapter 3

The network approach and scientific understanding

Abstract

In this chapter, I characterize in practice-oriented terms how network psychometricians use the network approach to obtain *scientific understanding* of mental disorders. The network theory provides how-possibly explanations of psychiatric phenomena, and network models highlight empirical patterns that provide evidence for the possible existence of psychiatric phenomena. Psychometricians can use the network approach to understand psychiatric phenomena scientifically if they can reason about possible ways to reduce the mental suffering associated with a phenomenon by physically or mentally manipulating the dependency relationships demonstrated by the network theory or network models. The understanding-providing potential of the network approach depends on the empirical adequacy and quantity of the dependency relationships that the network theory and network models demonstrate, their coordination, psychometricians' skills, and the pragmatic virtues of network representations.

3.1 Introduction

Scientists study mental disorders to obtain knowledge about these conditions. However, psychiatric scientists do not merely want to have information about what makes some more vulnerable to developing a mental disorder than others, how we can prevent mental disorders from arising, or why specific treatments do (not) work. Scientists study mental disorders to *understand* them. However, it is rarely specified how (improvements in) scientific understanding of mental disorders should be characterized.

The role of understanding in scientific practice has only relatively recently been acknowledged by philosophers of science.[47] Traditionally, understanding was considered irrelevant to scientific practice. For instance, Hempel (1965) argued that "expressions as 'realm of understanding' and 'comprehensible' do not belong to the vocabulary of logic, for they refer to the psychological and pragmatic aspects of explanation" (p. 413). However, this sentiment changed when science's pragmatic and value-laden nature was increasingly emphasized and embraced in the second half of the twentieth century, as mentioned in **Chapter 1**. Theories of scientific understanding were developed in the late twentieth and early twenty-first century (De Regt & Dieks, 2005; Friedman, 1974; Schurz & Lambert, 1994), and to date, most philosophers agree that "[u]nderstanding is an inextricable element of the aims of science" (De Regt & Dieks, 2005, p. 142). However, what scientific understanding entails and how it is

47. For a historical overview of the value of understanding in the philosophy of science and epistemology, see Baumberger et al. (2017) and Grimm (2021).

achieved is still debated. While making general claims about scientific understanding is possible, De Regt and Dieks (2005) recognize that philosophically studying scientific understanding should be practice-specific (p. 138). Philosophers have explored the specifics of understanding in different human sciences that share characteristics with psychiatric science, including biology (Leonelli, 2009), psychology (Eigner, 2009), and medicine (Varga, 2023). Moreover, Kästner (2022) argues that "[f]or a model to be successfully employed in diagnosis, treatment, and prevention of mental illnesses, it should ideally also be interpretable, viz. elicit understanding in clinicians and researchers" (p. 6). However, the criteria for *psychiatric scientific understanding* have not yet been explicated, let alone how different psychiatric approaches contribute to such understanding. The network approach is frequently characterized as a psychiatric approach that can improve scientists' understanding of mental disorders, as I alluded to in **Chapter 1**. For instance, Guloksuz et al. (2016) state that "[t]he conceptualization of psychopathology as a dynamic network of symptoms may provide a particularly useful tool to understand pathways to mental illness" (p. 1). However, *how* the network approach provides psychiatric scientific understanding is not discussed. In this chapter, I characterize in practice-oriented terms how network psychometricians use the network approach to obtain *scientific understanding* of mental disorders.

The chapter is structured as follows. In **Section 3.2**, I describe the object of psychiatric scientific understanding, i.e., psychiatric phenomena, in more detail. In **Section 3.3**, I examine what type of information about psychiatric phenomena the network theory and network models can provide. The network theory can provide how-possibly explanations of psychiatric phenomena, whereas network models can highlight empirical patterns that provide evidence for the possible existence of psychiatric phenomena. In **Section 3.4**, I present an initial characterization of the understanding-providing (noetic) potential of the network approach based on the observation that the network theory *and* network models are said to have noetic potential. The network theory and network models provide objectual understanding of psychiatric phenomena via the dependency relationships they demonstrate; these relationships should be empirically adequate. In **Section 3.5**, I discuss which interpretation of *grasping* aligns with the noetic potential of the network approach. I advocate for a pragmatic, modal interpretation of grasping, i.e., the ability to reason about possible ways to reduce the mental suffering associated with a phenomenon by physically or mentally manipulating the dependency relationships demonstrated by the network theory or network models. In **Section 3.6**, I argue that the noetic potential of the network approach depends on the empirical adequacy and quantity of the dependency relationships that the network theory and network models demonstrate, their coordination, psychometricians' skills, and the pragmatic virtues of network representations.

3.2 Psychiatric phenomena

The object of "scientific understanding" can be characterized in different ways (Baumberger et al., 2017). In this chapter, I focus on scientists' ability to understand *psychiatric phenomena*. A phenomenon is a robust and observable feature of the world, i.e., a feature that occurs over a wide variety of situations or contexts (Bogen & Woodward, 1988). Psychiatric phenomena, in turn, are robust, observable features of mental suffering. The most straightforward psychiatric phenomena are *psychiatric symptoms*, such as "anhedonia" or "insomnia" (Fellowes, 2021). However, they can also encompass robust, observable features related to the development or dynamics of mental suffering, such as "feeling stuck in a negative state" or "the co-occurrence of anhedonia and depressed mood." In this section, I highlight two characteristics of psychiatric phenomena.

First, "psychiatric phenomena" are not synonymous with "mental disorder classifications." Mental disorder classifications describe *collections* of psychiatric phenomena. For instance, the classification "major depressive disorder" describes a set of co-occurring, depression-related phenomena such as depressed mood, anhedonia, insomnia, eating disturbances, suicidal ideation, et cetera (**Table 1**). How psychiatric phenomena are grouped in a classification can change over time. Instead of being *natural kinds*, i.e., groups of robust phenomena with relatively fixed boundaries, there is increasing consensus that mental disorder classifications are *practical kinds*, i.e., groups of robust phenomena whose grouping is grounded in practical considerations such as clinical, scientific, or administrative ones (Zachar, 2000, 2002, 2014, 2015).[48] In other words,

> The concept of natural kind orients us to regularities in psychiatric phenomena that exist irrespective of our wishes or preferences; they are the result of causal processes that scientists seek to discover. The concept of practical kind orients us to the variety of the decisions we make in order to classify an indeterminate world. (Zachar, 2015, p. 289, emphasis in original)

Second, psychiatric phenomena do not exist "out there" in the world; they are human-dependent and less stable than natural phenomena such as gravity. The first reason is that psychiatric phenomena are *thick phenomena*, i.e., they encompass both evaluative and descriptive elements (Alexandrova, 2018). Their "thickness" is visible on the level of mental disorder classifications. The DSM-5 defines a mental disorder

48. For a discussion on the status of "symptoms" in the network approach, see Wilshire et al. (2021) and Borsboom (2022b).

as "a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (American Psychiatric Association, 2013, p. 20). This definition highlights that "mental disorder" inevitably invokes value-laden notions such as disability, harm, and suffering (Fulford, 2001; Fulford et al., 2005; Sadler, 2002). Given that our ideas about what levels of disability, harm, and suffering are considered "clinically significant disturbances" depend on social and practical considerations, what we consider mental disorders may shift over time. A similar claim about "thickness" can be made for psychiatric phenomena: Whether they reflect a "clinically significant disturbance" will depend on social and practical considerations (Fellowes, 2021; Wilshire et al., 2021). The second reason why psychiatric phenomena are relatively unstable is that they are *reactive*. Hacking (1996, 2007) argues that mental disorder classifications are *moving targets*: Individuals respond to their mental disorder classification, which in turn influences disorder categories (i.e., looping effects). Such looping also applies to psychiatric phenomena: Individuals may respond to their behavior being classified as instances of a symptom, e.g., being someone with insomnia. So, the behavior of individuals classified and what we consider "psychiatric phenomena" may change over time. Thus, psychiatric phenomena are, to some extent, "constructed" (Fellowes, 2021) and are less stable than other natural phenomena.[49] In the next section, I will describe what information about psychiatric phenomena the network theory and network models can provide.

3.3 Network theory and network models

Scientists study psychiatric phenomena by building and using *psychiatric theories* and *models*. Psychiatric theories and models represent *target systems*, i.e., features of the world that scientists consider relevant to the psychiatric phenomenon they study (Elliott-Graves, 2020). For instance, a theory of insomnia will represent those features of the world that scientists reasonably believe contribute to insomnia, such as caffeine intake, exercise levels, biological features related to the circadian rhythm, stress levels, et cetera; it will not include bed sheet colors. How scientists demarcate their target system will depend on their metaphysical (Hochstein, 2019) or ontological commitments (Oude Maatman, 2024), i.e., their (often implicit) ideas about the nature of these phenomena. These commitments are part of scientists' *background*

49. This view of psychiatric phenomena is compatible with Massimi's (2011) "Kantian stance" on phenomena as being "conceptually determined appearances," where "[p]henomena are not ready-made in nature, instead we have somehow to *make* them" (p. 109, emphasis in original).

theories, i.e., their (often implicit) intuitions about what the world is like that are not directly tested or evaluated but nonetheless interact with and drive scientific inquiry (Oude Maatman, 2024). Psychiatric theories and models represent their target systems in a simplified manner via *idealizations* (i.e., incorporating unrealistic features that do not align with the target system) and *abstractions* (i.e., omitting features of the target system). For instance, a theory of insomnia may group together different types of exercise (while higher-intensity exercise may have a different effect on sleep than lower-intensity exercise) or leave out information on biological contributors to insomnia. The kinds of idealizations or abstractions that scientists deem permissible will depend on their background theories (which features are *most relevant* to include), epistemic considerations (e.g., the scientific questions they wish to answer), or practical considerations (e.g., simply lacking certain data). So, psychiatric theories and models provide information about psychiatric phenomena by representing their target systems in an idealized and abstract manner. In this section, I expand on this taxonomy to discuss what information about psychiatric phenomena the network theory and network models can provide.[50]

Network theory

A theory is a collection of statements that address *why* a phenomenon – or a collection of phenomena – occurs. In other words, a theory represents an explanandum (i.e., the phenomenon to be explained), an explanans (i.e., what does the explaining), and the relationship between the two.[51] The *network theory* consists of five theoretical principles outlined in **Table 2**. Together, these principles provide explanations for different psychiatric phenomena, including "the co-occurrence of specific symptoms," "the comorbidity of mental disorders," and "the difficulties people have in recovering from their condition." In **Chapter 2**, I addressed the *types* of explanations provided by the network theory; here, I focus on their *status*.

To explain a psychiatric phenomenon, a psychiatric theory has to formulate *difference-makers*. That is, "[a]n explanation ought to be such that it enables us to see what sort of difference it would have made for the explanandum if the factors cited in the explanans had been different in various possible ways" (Woodward, 2003, p. 11).[52] However, coming up with empirically adequate difference-makers for psychiatric phenomena is not straightforward. This is partly due to the relative

50. Borsboom, Van der Maas, et al. (2021) and Haslbeck et al. (2022) provide comparable, albeit slightly different taxonomies.

51. In this thesis, I focus on the verbal representation of the network theory; formal representations, such as the network theory of panic disorder (Robinaugh et al., 2024), are beyond the scope of this thesis.

52. Reutlinger (2016) presents a unifying counterfactual account of causal and non-causal explanations.

instability of these phenomena, as highlighted in **Section 3.2**, and reflected in the challenges of meeting requirements for hypothetical Woodwardian interventions discussed in **Chapter 2**. In short, we cannot fully eliminate confounding variables; hypothetical interventions will always be fat-handed, and there may be conceptual overlap between symptoms (cf. Eronen, 2020; Eronen & Bringmann, 2021). So, there are principled reasons that make it difficult for psychiatric theories to formulate empirically adequate difference-makers.

I argue that this inability to identify clear explanantia has implications for the explanatory potential of psychiatric theories: They provide *psychiatric how-possibly explanations*. Verreault-Julien (2019) states that *how-actually* explanations provide knowledge of what is actually the case (*X* is the case because of explanation *Y*), whereas *how-possibly* explanations provide knowledge of what may be the case (it is possible that *X* is the case because of explanation *Y*). So, the difference between actuality and possibility is one of *modality*. My definition of psychiatric how-possibly explanations is broader. Psychiatric theories provide how-possibly explanations because they provide information on how psychiatric phenomena may arise, *and* the explanations themselves are less empirically adequate than those for other natural phenomena. Translating this to the network theory, I argue that it provides how-possibly explanations of psychiatric phenomena.

Network models

Network models are estimated using *network analysis*. I will provide a case study of network analysis in **Chapter 4**, but, in short, it consists of the following steps (**Figure 5**). First, psychometricians collect a dataset with quantitative data about psychiatric phenomena, such as self-report questionnaires about depressive symptoms. Second, psychometricians use network analysis methods that estimate the *conditional associations* between these variables; these associations are corrected for false positives and checked for stability. Third, these conditional associations are summarized in two equivalent types of network models. The *adjacency matrix* is a data structure that includes these conditional associations between the variables and allows scientists to calculate the network's *topological properties*, i.e., mathematically quantifiable connectivity patterns (see **Chapter 2** and **Table 3** specifically). The *visual network model*, which represents the data as a collection of nodes corresponding to the variables and edges corresponding to their conditional associations, allows scientists to visually inspect these conditional associations.

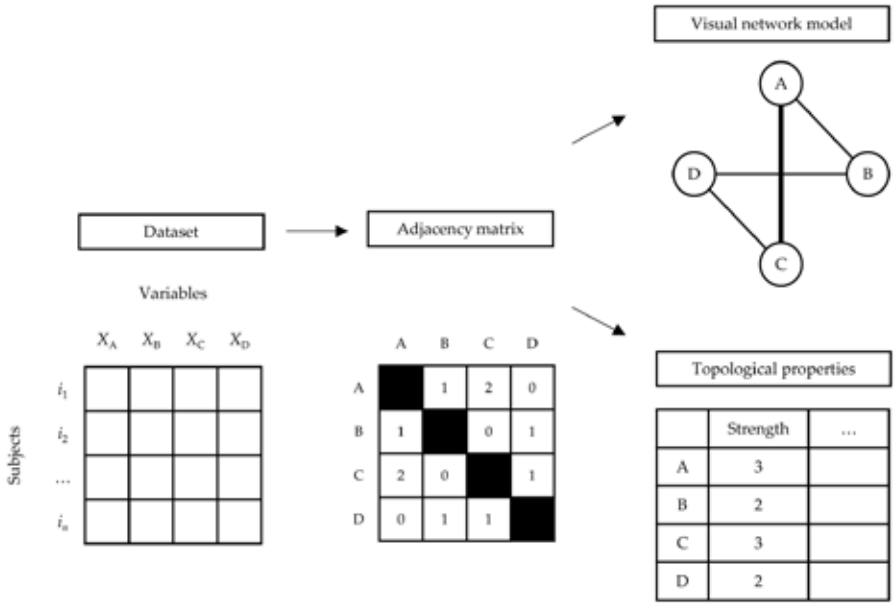

Figure 5. A simplified representation of network analysis

Network models are *data models*, i.e., representations that summarize and describe (corrected) data about phenomena gathered via measurements or observations (Suppes, 1962). Data are records produced by measurements that represent phenomena (Bogen & Woodward, 1988). Data models, in turn, are summaries of that data that highlight *empirical patterns*, i.e., robust patterns in a dataset that serve as *evidence* for the possible existence of psychiatric phenomena. For instance, if a visual network model estimated based on the data of a group of individuals with a depression diagnosis shows a strong edge between "experiencing worthlessness" and "sleeping poorly," this provides evidence for the possible existence of the phenomenon "people with a depression diagnosis experience both worthlessness and sleeping poorly." If this connection does not exist, it provides evidence that this phenomenon may not exist.[53]

So, the network theory provides how-possibly explanations of psychiatric phenomena, while network models provide empirical patterns that serve as evidence for the possible existence of these phenomena. In the next section, I will present my initial characterization of the noetic potential of the network approach by highlighting what the network theory and network models have in common.

53. In **Chapter 4**, I will go beyond this initial characterization to show how network models – when seen in their broader (theoretical) context of use – serve as a starting point for exploratory reasoning about psychiatric phenomena.

3.4 Understanding dependency relationships

Although the network theory and network models provide different types of information about psychiatric phenomena, psychometricians claim that both representations can aid scientists in understanding these phenomena. First, they discuss the noetic potential of the *network theory*. Borsboom and Cramer (2013) argue that all psychiatric theories, including the network theory, "share a deep desire to understand the inner workings of mental disorders" (p. 117). As shown in **Chapter 1**, Borsboom (2017a) claims that "our current lack of understanding of mental disorders may not have resulted from limited observational capacities, noisy measurement instruments, or inadequate data" but that "we may have simply lacked a theoretical framework [i.e., the network theory] to organize the available empirical facts" (p. 11). Finally, Borsboom (2017b) states that "[i]t is a fascinating idea that our understanding of mental disorders is not caused by an inadequate understanding of gene–brain–behavior pathways, but by an inadequate conceptualization of what we are doing" (pp. 92-93). Second, psychometricians claim that *network models* have noetic potential. Borsboom, Deserno, et al. (2021) state that understanding systemic phenomena, such as mental disorders, requires us to "study the organization of the system's components, which can be represented in a network" (p. 1). Guloksuz et al. (2017) state that "network methods have been utilized to understand the interplay between symptoms of mental disorders" (p. 1). Even those critical of network analysis claim that "[t]he goal of psychometric network analysis is to understand the relationships among measured psychological variables" (Neal et al., 2022, p. 1).[54] Let us take these claims seriously and assume that both the network theory and network models can provide psychometricians with scientific understanding of psychiatric phenomena. If this is the case, it has three implications for characterizing the noetic potential of the network approach.

First, we can characterize the *type* of scientific understanding that the network approach provides as *objectual understanding*. Objectual understanding can be defined as understanding a phenomenon, subject matter, or object (Kvanvig, 2003, 2009). This type of understanding is an alternative to *explanatory understanding*, i.e., understanding why a phenomenon is the case based on an explanation. The network approach provides objectual understanding because it encompasses exploratory *and* non-exploratory ways to understand phenomena (cf. Elgin, 1996, 2017a, 2017b). In other words, framing the noetic potential of the network approach in terms

54. The noetic potential of network models is also implied by philosophers reflecting on the network approach. Van Loo and Romeijn (2019) argue that symptom networks should be understood "as tools to improve our understanding of different aspects of these disorders" (p. 42).

of objectual understanding suggests that network models can provide scientific understanding of psychiatric phenomena irrespective of their non-explanatory nature, and that the network theory can offer scientific understanding irrespective of whether how-possibly explanations are considered "genuine explanations" (Verreault-Julien, 2019).

Second, both the network theory and network models provide scientific understanding based on the *dependency relationships* they demonstrate.[55] Dependency relationships are robust relations between features *A* and *B*, where *A* changes when *B* changes, and vice versa. The network theory and network models demonstrate comparable dependency relationships. First, they demonstrate *nodal* dependency relationships between individual nodes. For instance, a network model can demonstrate a dependency relationship (i.e., conditional association) between an insomnia- and an anhedonia-related questionnaire item. Second, they demonstrate *topological* dependency relationships between topological properties and psychiatric phenomena (or empirical patterns). For instance, the network theory demonstrates a dependency relationship between relapse vulnerability and network connectivity. There are important differences between the dependency relations that the network theory and network models can offer. The network theory demonstrates "explanatory" dependency relationships that are generalizable, have a specified nature (e.g., causal or constitutive), and are evidentially robust. Network models demonstrate "statistical" dependency relationships that are more specific, whose nature is not specified, and are less evidentially robust. Given these differences, we might argue that the scientific understanding provided by network models is "weaker" than that offered by the network theory. However, both the network theory and network models show how psychiatric phenomena could possibly be situated within a broader network of dependency relations. So, the network theory and network models both afford scientific understanding of psychiatric phenomena via the dependency relationships they demonstrate.

However, this does not imply that every dependency relationship goes. The dependency relationships that the network theory and network models demonstrate should be *empirically adequate*, i.e., conform to the observable world (cf. De Regt, 2017). This is in line with Borsboom (2022a) who argues, as highlighted in **Chapter 1**, that

55. This position differs from Dellsén (2020), who argues that having objectual understanding of a phenomenon depends on having a *mental model* of its (non-)explanatory dependence relations. My account does not concern mental models but focuses on the dependency relations afforded by *scientific* representations.

> [N]etwork approaches have mainly contributed to a better understanding of psychological phenomena, in the sense that the conceptualization of psychological constructs as networks has offered a new frame of thinking about them that, in many cases, *seems to carve nature at least slightly closer to its joints* [emphasis added]. (p. 254)

Here, it is important to note that, contrary to what this quote implies, I do not assume that these dependency relationships must be *true, factive* representations of what the world actually is like. The *factivity criterion* refers to the assumption that scientific representations should be truth-conducive descriptions of their target systems to provide understanding of a phenomenon; understanding requires meeting a truth condition (Kvanvig, 2003; Pritchard, 2010). However, various philosophers have criticized the factivity criterion for scientific understanding. Amongst others, it cannot account for the noetic potential of idealizations (Elgin, 1996, 2007, 2017b), outdated scientific theories (De Regt, 2015), and non-propositional representations, such as diagrams (Riggs, 2003). These concerns also apply to the network theory and network models, as both rely on diagrammatic representations and idealizations. Moreover, both representations convey information about *possibilities* regarding *relatively unstable* phenomena. Given these considerations, truth-conduciveness is too strict a criterion to assess the noetic potential of the network approach. Instead, the dependency relationships demonstrated by the network theory and network models should be *weakly factive*.[56] So, we can start with the following definition:

> Psychometricians understand a psychiatric phenomenon scientifically based on the dependency relationships demonstrated by the network theory or network models; these dependency relationships should be empirically adequate.[57]

However, this definition does not cover how psychometricians *use* the network theory and network models to understand psychiatric phenomena scientifically. In the next section, I will introduce a pragmatic, modal criterion in my definition of psychiatric scientific understanding by focusing on *grasping*.

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56. Alternatively, one could argue that the noetic potential of the network theory and network models depends on whether *the explanatory or predictively essential elements they represent* are *true* (Dellsén, 2016; Mizrahi, 2012; Strevens, 2013). However, this alternative may also be difficult to uphold in psychiatric science, given the difficulty in establishing difference-makers for mental disorders.

57. Note that empirical adequacy is not an all-or-nothing criterion, i.e., dependency relations can be more or less empirically adequate.

3.5 A pragmatic, modal interpretation of grasping

The *grasping criterion* refers to the assumption that (scientifically) understanding phenomena requires an additional cognitive achievement: *seeing* how isolated pieces of information hang together. In other words, understanding depends on "seeing the way things fit together" (Riggs, 2003, p. 218) and the "grasping of explanatory and other coherence-making relationships in a large and comprehensive body of information" (Kvanvig, 2003, p. 192). However, the literature on (scientific) understanding does not agree on what such grasping entails. The first option is to characterize grasping as a *feeling* of understanding. However, most philosophers argue that the phenomenology of understanding, the "Aha!" or "Eureka!" feeling, is strongly influenced by cognitive biases and, therefore, neither necessary nor sufficient for (scientific) understanding (De Regt, 2004, 2009b, 2017; Grimm, 2010; Trout, 2002, 2005; Ylikoski, 2009; but Lipton, 2009). The second option is to characterize grasping as a skill or ability. For instance, the contextual theory of scientific understanding (*contextual theory*, in short, De Regt, 2009a, 2009b, 2017; De Regt & Dieks, 2005) claims that an explanatory scientific understanding of phenomena is achieved via intelligible theories, defining intelligibility as "the value that scientists attribute to the cluster of qualities that *facilitate the use* [emphasis added] of the theory" (De Regt, 2017, p. 12). In this section, I discuss which interpretation of grasping aligns with the network approach, defending a pragmatic, modal interpretation.

It makes sense to conceptualize grasping pragmatically if we take psychiatric science as a starting point. As alluded to in **Chapter 1**, psychiatric science aims to understand mental disorders with the hope that this will inform *interventions to reduce mental suffering*.[58] Indeed, research is *psychiatric*, not psychological or medical, if it – at least distally – aims to reduce mental suffering. If we zoom in on the network approach, we see a similar association between (scientific) understanding and intervening. For instance, after stating that psychiatric theories aim at understanding, Borsboom and Cramer (2013) suggest that the network theory may help answer the most pressing *practical* questions in psychiatry, i.e., "finding out why some people are more vulnerable to developing mental disorders than others, how we can protect vulnerable people from harm, and how we can effectively treat people who have already fallen into the abyss of mental dysfunction" (p. 117). Moreover, the Dutch research program *New Science of Mental Disorders* mentioned in **Chapter 1** uses the network approach to "develop a novel approach to understand, diagnose and treat mental disorders"

58. I use "reducing mental suffering" instead of "reducing psychiatric phenomena" because the latter is not the only way to reduce mental suffering (e.g., mental suffering can be reduced by developing the ability to cope with one's symptoms).

(Roefs et al., 2022, p. 2). Building on these statements, I argue that scientifically understanding psychiatric phenomena *requires* interventions. This claim can be interpreted in two ways. First, understanding psychiatric phenomena scientifically requires their direct manipulation. However, this criterion is too strong: It suggests that scientists can only understand psychiatric phenomena if their research aims to directly change them and that research that has not led to psychiatric interventions has not contributed to scientific understanding. Instead, I opt for a second interpretation: Understanding psychiatric phenomena scientifically requires the ability to reason about *possible* ways to reduce the mental suffering associated with a phenomenon by *manipulating psychiatric theories and models*. I will elaborate on this interpretation below.[59]

The idea that scientific understanding requires manipulating scientific representations is not new (cf. Morrison & Morgan, 1999). For instance, Knuuttila and Merz (2009) claim that "[o]ne devises the model in such a way that one can learn and gain understanding from using or 'manipulating' it" (p. 154). Similarly, Leonelli (2009) argues that having scientific understanding of phenomena depends on scientists' ability to manipulate models: Scientific models are tools for acting and thinking, and understanding and manipulation are "two sides of the same coin" (p. 193). Leonelli (2009) conceptualizes such model manipulation in two ways. First, it can involve *physical manipulation*, such as physically removing, adding, or changing elements of a model (for instance, when constructing a scale model). Second, it can involve *mental manipulation*, "as when mentally shifting the terms of an equation" without actually rewriting it (Leonelli, 2009, p. 192). Indeed, by creating, manipulating, adapting, and evaluating models, scientists can make inferences about phenomena, i.e., engage in *model-based reasoning* (Nersessian, 1999, 2008).[60] Conceptualizing reasoning as model manipulation echoes Ryle (1949), who argued that the distinction between knowing-that and knowing-how is an "intellectualist legend" (p. 18). So, we can conceptualize the grasping involved in *psychiatric* scientific understanding as the ability to reason about *psychiatric* phenomena by physically or mentally manipulating *psychiatric* models or theories.

The type of reasoning involved in psychiatric scientific understanding is reasoning about *possibilities*. As I described in **Section 3.3**, the network theory and network models

59. This position differs from Wilkenfeld (2013), who claims that having objectual understanding involves possessing a *mental representation* of the phenomenon that we can manipulate in useful ways. My account focuses instead on the understanding provided by manipulating *scientific* representations.

60. This is comparable to the statement that scientific representations allow scientists to engage in *surrogative reasoning*, i.e., to draw inferences about a target system based on a scientific representation (cf. Haslbeck et al., 2022).

provide information about possibilities. So, manipulating these representations provides *modal* information about how reductions in the mental suffering associated with psychiatric phenomena *could be possible*. The idea that understanding involves exploring or formulating (non-actualized) possibilities builds upon earlier work in the philosophy of (scientific) understanding (Haugeland, 2017; Humphreys, 2006; Le Bihan, 2017; Lipton, 2009; Nozick, 1981; Reutlinger et al., 2018). For instance, Lipton (2009) argues that how-possibly explanations can provide actual understanding, even outside of "possible worlds where they are true" because they provide knowledge of possibility rather than necessity (p. 50). Le Bihan (2017) formulates a modal account of understanding, where "[o]ne gains modal understanding of some phenomena when one has some idea of how possibly the phenomena *might* arise" (p. 112, emphasis in original). Specifically, Le Bihan (2017) characterizes modal understanding as an ability, i.e., "[o]ne has some *modal understanding of some phenomena* if and only if one *knows how to navigate some of the possibility space* associated with the phenomena" (p. 112, emphasis in original). This ability is characterized as scientists' ability to know how 1) a particular dependency structure gives rise to a phenomenon, 2) dependency structures are related to one another, or 3) some constraints apply to the whole possibility space (p. 117).[61] In fact, Knuuttila (2021a) argues that Le Bihan's (2017) modal understanding account aligns with the artifactual view of scientific modeling, which inspires my practice-oriented account (see **Chapter 1**). This leads to the following characterization of the noetic potential of the network approach:

> Psychometricians understand a psychiatric phenomenon scientifically if they can reason about possible ways to reduce the mental suffering associated with the phenomenon by physically or mentally manipulating the dependency relationships demonstrated by the network theory or network models. These dependency relationships should be empirically adequate.

Let us suppose that a psychometrician is interested in how specific symptoms co-occur in people with a depression diagnosis. They ask people with a depression diagnosis to fill out a self-report questionnaire to obtain data about depression symptoms, perform network analysis on this dataset, and construe a network model. In the (visual) network model, the psychometrician observes a robust dependency relationship between the items representing anhedonia and insomnia. The psychometrician scientifically *understands* the phenomenon that this dependency relationship provides evidence for – "anhedonia and insomnia frequently co-occur"

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61. Le Bihan's (2017) interpretation of "dependency structure" is similar to my interpretation of "dependency relationship": Both encompass two factors and their relation and claim that these elements should stand in an adequacy-relation to the possible world.

– if they can use the network model to reason about possible ways to reduce the suffering associated with this phenomenon; they do so by physically or mentally manipulating this dependency relationship. For instance, they can imagine a scenario in which the value of the anhedonia node is increased and reason about what this may imply for the insomnia node. Alternatively, they can imagine a scenario in which the relationship between the nodes is severed and reason about what this implies for other symptoms in the network. So, the ability to reason about possibilities to reduce the suffering associated with this phenomenon is intimately related to acts of mental manipulation. Other examples can also be considered. The network theory shows a dependency relationship between "network connectivity" and "relapse vulnerability," as stated in **Chapter 2**. Psychometricians can use this dependency relationship to *understand* relapse vulnerability scientifically if they can reason about possibilities to reduce this vulnerability by manipulating the network theory.

Note that my conceptualization of the noetic potential of the network approach shows similarities to the criteria for scientific understanding as proposed by the contextual theory; here, I use its latest formulation as a reference (De Regt, 2017). First, the contextual theory connects scientific understanding to the ability to *use* scientific representations. As mentioned earlier, De Regt (2017) argues that understanding phenomena scientifically requires *intelligible* scientific representations, defining intelligibility in pragmatic terms. Second, the contextual theory conceptualizes intelligibility as the ability to *reason with* scientific representations. For instance, De Regt (2017) formulates his *Criterion for the Intelligibility of Theories* as follows: "A scientific theory *T* (in one or more of its representations) is intelligible for scientists (in context *C*) if they can recognize qualitatively characteristic consequences of *T* without performing exact calculations" (p. 102). Moreover, De Regt (2017) acknowledges that this intelligibility criterion may vary depending on the discipline to which it is applied (pp. 261-262). Given these considerations, my characterization of the noetic potential of the network approach may differ from the contextual theory only in emphasis. However, the contextual theory focuses on *explanatory understanding*, i.e., understanding *why* a phenomenon is the case based on a scientific explanation (see **Section 3.4**). Specifically, De Regt (2017) states that exploratory research "does not aim at understanding-why but merely at understanding-that or understanding-how" (p. 96). Hence, it remains unclear whether the contextual theory would ascribe noetic potential to the information provided by the network theory and network models. Therefore, not automatically subsuming my account under the contextual theory is justified. In the next section, I will explore what practice-independent and practice-dependent features determine the noetic potential of the network approach.

3.6 Determinants of scientific understanding

A commonly accepted feature of understanding is that it is a matter of degree: We can have more or less, or better or worse, understanding of something. Similarly, noetic accounts of scientific progress characterize progress as improving our scientific understanding of phenomena (e.g., Dellsén, 2016, 2018, 2021; McCoy, 2022). However, making claims about *improving* scientific understanding requires clarifying what *determines* scientific understanding. In line with my practice-oriented account, I claim that the noetic potential of the network approach depends both on the dependency relationships demonstrated by the network theory and network models (i.e., practice-independent features) as well as their context of use (i.e., practice-dependent features). This echoes Leonelli (2009), who argues that the ideal type of scientific understanding of a phenomenon is *integrated understanding*, i.e., the ability of a scientist to understand a phenomenon via the coordination of their theoretical and embodied knowledge and skills. Similarly, Robinaugh et al. (2020) argue that "[t]he central task for the next decade of the network approach is to build on [its] theoretical, methodological, and empirical foundations (…) and advance our understanding of how specific mental disorders operate as causal systems" (p. 361). In this section, I present four determinants of the network approach's noetic potential.

First, the noetic potential of the network approach depends on the *dependency relationships* that the network theory and network models demonstrate. Psychometricians can enhance this potential by having these representations present a greater number (*quantity*) of dependency relationships, encompassing a broader range of phenomena. In **Chapter 2**, I proposed a way to improve the explanatory breadth of the network theory; this could also enhance its noetic potential, provided practice-dependent considerations are in place. Moreover, psychometricians can improve their scientific understanding of psychiatric phenomena if these dependency relationships have more *empirical adequacy* or evidential support. The network theory's empirical adequacy can be improved through additional studies to test its premises. The empirical adequacy of network models can be enhanced by improving their robustness, for example, by increasing sample sizes or by performing robustness and stability analyses (Bringmann et al., 2022).

Second, the noetic potential of the network approach depends on the *coordination* of the network theory and network models. In **Chapter 2**, I highlighted that these representations should not be conflated. However, the network theory and network models can, in principle, represent similar dependency relationships, with the network theory providing information on their nature and network models on their statistical

association. First, we can assume that the network theory and network models represent similar target systems, as both are part of the network approach described in **Chapter 1**, which characterizes mental disorders as systemic, multifactorial, and complex. Second, both represent their target system using similar network-based *idealizations* and *abstractions*. For instance, both idealize their target system by assuming we can neatly differentiate symptoms into nodes. Third, psychometricians will use comparable *background theories* when construing and interpreting these representations. So, the network theory and network models can, in principle, be coordinated (Bradburn et al., 2017; Eronen & Romeijn, 2020). This coordination could be enhanced by using network models that align with the explanatory claims made by the network theory (e.g., within-subject network analysis) or by improving the *explanatory precision* of the network theory to ensure that its claims do not lead to multiple interpretations. I will elaborate on this in **Chapter 4**.

Third, the noetic potential of the network approach depends on *psychometricians' skills* in engaging with the dependency relationships demonstrated by the network theory and network models. For instance, through scientific training, network psychometricians can enhance their programming and statistical skills or become skilled in formal modeling and theory-building. By refining their ability to skillfully interact with the network theory and network models, psychometricians can improve their ability to physically or mentally manipulate the dependency relationships these representations demonstrate, thereby reasoning about (im)possibilities for reducing mental suffering.

Fourth, the noetic potential of the network approach depends on the *pragmatic virtues* of network representations. This echoes discussions of intelligibility by De Regt (2017) and the emphasis of Knuuttila and Merz (2009) on the relationship between understanding phenomena and the material embodiment of models. Here, I define pragmatic virtues as those features of the network theory and network models that facilitate scientists' ability to *use* them.[62] Pragmatic virtues are context-specific, i.e., depend on scientists' skills, background knowledge, and assumptions. For instance, some scientific communities prefer mathematical simplicity – i.e., simple equations – while others prefer logical simplicity, i.e., theories with few independent postulates (De Regt, 2017). Alternatively, scientists in scientific community *A* may

62. There is an elaborate debate within the philosophy of science on the role of virtues in scientific practice. These discussions often concern *theoretical virtues*, i.e., the virtues of a good scientific theory (e.g., Kuhn, 1977; Longino, 1990). As highlighted in **Chapter 1**, Van Fraassen (1980) posed a distinction between theoretical virtues that are epistemic (i.e., the virtues of a scientific theory that make it truth-conductive) and pragmatic (i.e., the virtues of a scientific theory that concern its use). Instead, following De Regt (2017), I argue that the virtues that improve scientists' ability to use a theory or model also improve scientists' knowledge.

be trained in specific skills that allow them to reason more easily with abstract formulas than with visualizable models, whereas this may be reversed for scientists in community *B* (for a historical case study in physics, see De Regt, 2017, Chapter 6). The psychometrics literature highlights various pragmatic virtues valued in the psychometrics community. For instance, Borsboom, van der Maas, et al. (2021) argue that the "explanatory goodness" of a scientific psychological theory depends not only on its explanatory breadth but also on its simplicity and the presence of analogies. They suggest that the latter virtue can help psychometricians in reasoning with psychological theories (p. 762). The network approach frequently resorts to analogies between mental disorders and the climate (Borsboom, 2022b) or ecosystems (Borsboom, 2017b; Lunansky et al., 2021), amongst others. Indeed, mentally manipulating, interpreting, and reasoning with dependency relationships becomes easier when they align with our background knowledge of "analogous phenomena." Moreover, network models are praised for their *visualizability* and *transparency* (Borsboom, 2022a). As I will illustrate in **Chapter 4**, both pragmatic virtues improve psychometricians' ability to interact with and "see" dependency relationships.

So, the noetic potential of the network approach depends on the quantity and empirical adequacy of the dependency relationships that the network theory and network models demonstrate, their coordination, psychometricians' skills, and the pragmatic virtues of network representations. Note that these determinants may not always be complementary and could trade off against one another.

3.7 Conclusion

One epistemic aim of the network approach is to improve scientists' understanding of mental disorders. In this chapter, I characterized the scientific understanding that network psychometricians can obtain through the network approach and what determines this understanding. The network theory and network models provide dependency relationships that offer information about possibilities regarding psychiatric phenomena. In line with my practice-oriented account, I claimed that psychometricians understand a psychiatric phenomenon scientifically if they can reason about possible ways to reduce the mental suffering associated with the phenomenon by physically or mentally manipulating the dependency relationships demonstrated by the network theory or network models. Moreover, I presented practice-independent and practice-dependent criteria that can determine the noetic potential of the network approach, offering suggestions for its improvement.

Let us return to the two-worlds metaphor by Grob (1998) introduced in **Chapter 1**. Here, Grob argues that in the intractable world of mental suffering, "our judgements, analyses, and actions often represent a pragmatic response to a seemingly intractable and partially incomprehensible universe" (p. 189). In contrast, the idealized world of mental disorders contains "pure and precise knowledge leads to a kind of understanding that enables human beings to cope with or solve perennial problems" (p. 189). My account of the noetic potential of the network approach is a mix of both perspectives. The idealized world that the network approach sketches will not help psychometricians comprehend the intractable world of mental suffering directly. However, the network approach provides information that can help psychometricians identify possible entry points for responding pragmatically to the intractable world. This modest interpretation recognizes both the limitations of making sense of the intractable world of mental suffering via psychiatric science and the importance and value of the idealized information that psychiatric science provides.

This chapter provided an initial exploration of the noetic potential of the network approach and opened up avenues for further research. For instance, the extent to which different determinants of the noetic potential of the network approach may trade off against each other should be explored in more detail. Another question that follows from my analysis is what *constraints* should be in place when scientists reason about possibilities for reducing the mental suffering associated with psychiatric phenomena. This chapter showed that psychometricians can use the dependency relations that the network theory or network models demonstrate to *imagine* possible scenarios in which mental suffering is reduced. However, what guides and constrains the possibilities that scientists can imagine and are willing to take seriously should be explored in more detail. In the next chapter, I will explore the role of *bounded imagination* in the exploratory potential of network models; this analysis may also shed light on the type of imagination required for scientific understanding.

Chapter 4

The exploratory potential of network models

Abstract

Network models are generally presented as exploratory models, but what gives these models their exploratory potential is rarely explicated. In this chapter, I examine the exploratory function of network models in greater detail based on my practice-oriented account. Network models can serve as a starting point for reasoning about possibilities for psychiatric phenomena. I use Massimi's (2018b, 2019, 2022) work on perspectival modeling to claim that network models help psychometricians reason about *epistemically relevant* possibilities for psychiatric phenomena if these possibilities are *physically conceivable*, i.e., consistent with available empirical and theoretical background knowledge. Such background knowledge can only guide and constrain psychometricians' exploratory reasoning with network models if it is adequately *coordinated* with the dependency relationships that a network model demonstrates and is *empirically adequate*. Moreover, the exploratory function of network models depends on their pragmatic virtues, including their visualizability and (functional) transparency. Finally, I use these criteria to argue that multilayer brain-symptom network models provide more dependency relationships related to psychiatric phenomena than standard network models, but are not necessarily more exploratorily advantageous.

4.1 Introduction

There is increased recognition that scientific models can have an *exploratory* function and that exploration serves an essential epistemic purpose in scientific practice (Gelfert, 2016, 2018; Massimi, 2018b, 2019, 2022; Shech & Gelfert, 2019). One set of models whose exploratory function is frequently acknowledged by its users is *network models* (Borsboom, 2022a; Borsboom, Deserno, et al., 2021; Epskamp & Fried, 2018; Epskamp & Isvoranu, 2022). Network psychometricians have reflected on what makes network models of psychological phenomena (Borsboom, Deserno, et al., 2021) and mental disorders specifically exploratorily advantageous (Borsboom, 2022a). However, it is unclear from these accounts *how* network models can help psychometricians explore mental disorders. This echoes Gelfert (2016), who observes that "scientists are ready to acknowledge the exploratory role of modeling, yet this acknowledgment is not usually followed up by a more detailed analysis of exploratory strategies and their specific functions" (pp. 80-81). Similarly, various authors have applied insights from the philosophy of modeling to network models (De Boer et al., 2022; Eronen & Bringmann, 2019; Van Loo & Romeijn, 2019; Ward & Fischer, 2019) but have not explicitly focused on their exploratory function. In this chapter,

I examine the exploratory function of network models in greater detail based on my practice-oriented account. I use insights from Massimi's (2018b, 2019, 2022) work on perspectival modeling, a type of exploratory modeling that focuses on modeling *possibilities*.[63] Massimi (2022) applies perspectival modeling to the human sciences when discussing scientific models of language development and dyslexia (p. 126-140). However, this account has not yet been applied to psychiatric science, let alone to the network approach. Clarifying the exploratory potential of network models can help scientists assess the relative exploratory potential of recent developments in network psychometrics, such as multilayer brain-symptom network models.

The chapter is structured as follows. In **Section 4.2**, I describe how network models are estimated and constructed using a case study. In **Section 4.3**, I describe the different functions of exploratory models and highlight one specific exploratory function of network models: They provide a starting point for reasoning about possibilities for psychiatric phenomena. In **Section 4.4,** I use Massimi's (2018b, 2019, 2022) work on perspectival modeling to formulate criteria for the exploratory potential of network models: Network models help psychometricians reason about *epistemically relevant* possibilities for psychiatric phenomena if these possibilities are *physically conceivable*, i.e., consistent with psychometricians' available empirical and theoretical background knowledge. In **Section 4.5**, I address what criteria such background knowledge should meet to aid the exploratory function of network models, claiming that it should be adequately *coordinated* with a network model and *empirically adequate*. These criteria are not always met for the types of background knowledge that play a role in psychometricians' reasoning with network models, i.e., the network theory, empirical studies, and background theories. In **Section 4.6**, I address the exploratory function of network models in light of their pragmatic virtues, i.e., their visualizability and (functional) transparency. In **Section 4.7**, I use my analysis to assess the *relative* exploratory potential of network models by comparing them to multilayer brain-symptom network models. Multilayer brain-symptom networks provide more dependency relationships than "standard" network models but are not necessarily exploratorily advantageous because they have less constraining background knowledge and more functional opacity.

63. Massimi (2022) distinguishes two types of perspectivity: perspectival$_1$ and perspectival$_2$. Perspectival$_1$ emphasizes that models are situated, i.e., drawn *from* a specific vantage point. Perspectival$_2$ emphasizes that models also aim *towards* a vantage point. My analysis focuses on perspectival$_1$.

4.2 Network models: A case study

In **Chapter 3**, I outlined how network models are constructed and estimated. In short, network models are the output of *network analysis*, i.e., statistical methods that estimate conditional associations between variables in a dataset and subsequently (1) correct these associations to limit false positives and (2) assess their robustness and stability (Borsboom, Deserno, et al., 2021). Network analysis provides two types of network models: a data structure summarizing these conditional associations, i.e., an *adjacency matrix*, which is subsequently used to create a *visual network model* (**Figure 5**). In this section, I illustrate these different steps of network analysis using a case study.

Mullarkey et al. (2019) use network analysis to examine the relationships between depression symptoms in adolescents.[64] The aim of their study was to evaluate what (associations between) depression symptoms are most central to adolescent depression. Mullarkey et al. (2019) asked their study participants – a community sample of adolescents between 13 and 19 years old – to fill out the Children's Depression Inventory (CDI, Kovacs, 1978), a 27-item self-report questionnaire that investigates depressive symptoms on a 3-point Likert scale (e.g., "I don't feel alone" = 0, "I often feel alone" = 1, "I always feel alone" = 2). The scientists collected CDI responses from 1409 adolescents. To estimate the adolescent depression network model, the scientists converted the CDI items in their dataset to binary values (symptom absent = 0, symptom present = 1). Based on this converted dataset, they estimated the conditional dependencies between these variables using an Ising model (Van Borkulo et al., 2014). To limit false positive associations, Mullarkey et al. (2019) regularized these conditional associations in their adjacency matrix by fitting them with an extension of the least absolute shrinkage and selection operator (eLasso).

Mullarkey et al. (2019) used their regularized adjacency matrix to provide various network descriptions. They created a visual network model using the R package *qgraph* (Epskamp et al., 2012), with nodes corresponding to CDI items and edges to their regularized conditional associations. Moreover, they calculated various *topological properties* of their adolescent depression network model.[65] First, they calculated each CDI item's *node strength*, *closeness centrality*, and *betweenness centrality* (**Table 3**). They found that four items had significantly higher node strength than other nodes in the network: self-hatred, loneliness, sadness, and pessimism. Second,

64. This study is illustrative for network psychometrics, given that it is the most cited empirical study that has performed network analysis to study depression since 2018, according to the keywords "network analysis depression" in *Web of Science* (233 citations in the *Web of Science Core Collection* between March 2018 and November 2024).

65. Stability and accuracy tests were performed to evaluate the robustness of their findings.

Mullarkey et al. (2019) identified the strongest, i.e., most strongly connected, edges in the network model. These included the edges between sadness and crying, between anhedonia and disliking school, and between sadness and loneliness. Third, they explored gender differences in network connectivity by creating a male-only and female-only dataset, estimating network models for both, and comparing their *global strength* (Van Borkulo et al., 2023) – they found no significant differences. In the next section, I will build on this case study to characterize the exploratory function of network models in more detail.

4.3 The functions of exploratory models

The term "exploratory model" is frequently used in the empirical literature, but more often than not, the exploratory *function* of such models is not explicated. Sometimes, scientists say a model is "exploratory" because they do not want to fully commit to its results or underlying assumptions. In those instances, "exploratory" is used as a placeholder for "poor epistemic value" or "non-explanatory." Similarly, the epistemic value of exploratory models has received comparatively little interest in the philosophy of science literature. Traditionally, exploratory models are considered part of the arbitrary *context of discovery*, the generation of new ideas, instead of the *context of justification*, the defense of these ideas. Various authors have criticized the arbitrariness of the context of discovery (Arabatzis, 2006) and the sharp distinction between these two contexts (Gelfert, 2018). In line with these authors, I argue that exploratory models have important epistemic functions in scientific practice and psychometrics specifically.

The term "exploratory model" was first introduced in the philosophy of modeling literature by Gelfert (2016). Gelfert (2016, Chapter 4) highlights that exploratory models can have four related and non-exhaustive functions. First, exploratory models can serve as a *starting point for future inquiry*, where the future models that build upon them are increasingly sophisticated (cf. Wimsatt, 2007, p. 104). Second, exploratory models can *provide proof-of-principle demonstrations*: They can (a) show that it is possible to represent a phenomenon's target system using a particular method or (b) propose a specific mechanism or process that turns out to be associated with the phenomenon in question (such as the Lotka-Volterra model's finding that the size of predator-pray populations can oscillate without external forces). Third, exploratory models can *generate potential explanations* of a phenomenon, "for example by envisaging scenarios that, if true, would give rise to the kinds of phenomena that

constitute the explanandum" (p. 87).[66] Fourth, exploratory models can help scientists determine the *suitability of the target system* by identifying the best way to delineate it.

If we examine the psychometrics literature, we see that the epistemic function ascribed to network models fits the description of exploratory models.[67] For instance, Borsboom, Deserno, et al. (2021) argue that network psychometrics can help scientists *explore* patterns of conditional associations in a dataset when a strong theory of how variables are related is absent. The exploratory functions of network models also come to the fore in Mullarkey et al. (2019). For instance, they present their findings as a starting point for future inquiry: Based on the centrality of self-hatred, loneliness, sadness, and pessimism, they suggest that future studies should "examine the temporal dynamics of activating and deactivating" these central symptoms and their associations (p. 665). However, this case study also highlights an exploratory function of network models that is adjacent to but not wholly similar to the functions discussed by Gelfert (2016): Network models can be used as *a starting point for reasoning about possibilities for psychiatric phenomena*.

To illustrate, Mullarkey et al. (2019) use the strong edge between loneliness and sadness as a starting point to explore why or how this edge may be present, hypothesizing that the symptoms may mutually reinforce each other. So, they use the network model, which provides evidence for the possibility of the phenomenon "loneliness and sadness frequently co-occur in adolescents with depressive tendencies," to subsequently explore possible explanations for this dependency relationship. The network model shapes the specific possibilities for psychiatric phenomena that researchers can "see," constraining, fine-tuning, or expanding them. This aligns with Borsboom, Deserno, et al. (2021), who argue that network models "function as a gateway that allows the researcher to assess the plausibility of different generating models that may produce the relevant conditional associations" (p. 15).[68]

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66. Gelfert's (2016) use of "potential explanations" is not synonymous with my interpretation of "how-possibly explanations" as presented in **Chapter 3**. Gelfert (2016) implies that potential explanations are how-actually explanations that have not yet been empirically confirmed. How-possibly explanations, on the other hand, also have a different internal structure than how-actually explanations, explaining what is possible rather than what is actual.

67. Serban (2020) addresses the exploratory function of network models in a different context: molecular and developmental biology.

68. Other network psychometric methods could have different exploratory functions. For instance, the simulation study by Cramer et al. (2016) provides a proof-of-principle demonstration of hysteresis in the development of depression. Borsboom, Deserno, et al. (2021) also suggest that network representations "may be of interest in [their] own right" (p. 15). While exploration for the mere sake of examining the data structure may be of interest during the early stages of the research process, studies with network models as their primary output use them for a more substantial epistemic purpose.

This exploratory function is not synonymous with using network models as starting points for future inquiry, for it does not directly inspire building new models. It is also not synonymous with generating potential explanations: Network models do not provide an *explanans* but provide evidence for an *explanandum*, e.g., the possibility of the phenomena that loneliness and sadness co-occur. Psychometricians subsequently use this explanandum as the *object* of how-possibly explanations. Hence, this exploratory function is not entirely reducible to the aforementioned functions. In the next section, I will characterize this specific exploratory function in more detail using Massimi's (2018b, 2019, 2022) notion of *physical conceivability*.

4.4 Exploration and physical conceivability

The idea that network models can be used as a starting point for reasoning about possibilities for psychiatric phenomena shows similarities with Massimi's (2018b, 2019, 2022) account of the exploratory function of *perspectival models*. Massimi states that perspectival models are a specific set of exploratory models that provide modal knowledge of what might be possible about their target system. Specifically, perspectival models are situated, exploratory models that support relevant and appropriate modal inferences about phenomena, i.e., they allow researchers to reason about indicative conditionals ("if *X* is the case") with suppositional antecedents ("then *Y* may be the case").[69] Here, *X* refers to a scenario that scientists can *physically conceive* based on the model, and *Y* refers to possibilities for a phenomenon. Massimi (2022) uses the term *physical conceivability* to describe the specific type of bounded imagination that perspectival models afford, defining it as follows:

> *p* is physically conceivable for an epistemic subject *S* (or an epistemic community *C*) if *S*'s (or *C*'s) imagining that *p* not only complies with the state of knowledge and conceptual resources of *S* (or *C*) but is also consistent with the laws of nature known by *S* (or *C*). (pp. 150-151)

So, a scenario that scientists can imagine based on a model is physically conceivable when imagining that scenario (1) complies with their knowledge and conceptual resources and (2) is consistent with known laws of nature. In this context, laws of nature refer to *lawlike dependencies* that do not necessarily have to be deductive-

69. Note that these statements are not the same as counterfactual statements, i.e., subjunctive conditionals with an antecedent that is assumed or known not to obtain (Godfrey-Smith, 2020). Massimi's (2022) modal inferences invite scientists to reason about what may be the case instead of how things could have been differently.

nomological. For instance, when discussing a theoretical model of dyslexia in light of perspectival modeling, Massimi (2022) argues that "[t]here are lawlike dependencies (probabilistic ones) between specific cognitive deficits and performance of associated behavioural tasks. Psychologists routinely use these to make diagnoses (…) and could not do so without them" (p. 169). Moreover, Massimi (2022) highlights that different types of physically conceived scenarios can help scientists in reasoning modally about different types of possibilities for their phenomenon of interest. For instance, they can imagine scenarios that help them reason about the phenomenon's causal possibilities (i.e., what its possible causes might be), non-causal explanatory possibilities (i.e., what its possible non-causal explanations might be), or objective possibilities (i.e., what may actually be the case about the phenomenon).

To illustrate, Massimi (2022) discusses climate scientists who use climate models to study global warming (see pp. 110-125 for various examples of perspectival modeling related to climate modeling). Let us consider that climate scientists create a climate model that represents different greenhouse gas concentrations for 2100. The scientists can physically conceive of scenarios based on the climate model, their knowledge about greenhouse gas concentrations, and their knowledge of lawlike dependencies between greenhouse gases and global warming (*X*). For instance, a scenario where the greenhouse gas concentration keeps increasing until 2100 and steadily drops afterward is not consistent with their available theoretical knowledge on greenhouse gases and, hence, not physically conceivable. The scientists use these scenarios to explore different causal possibilities for global warming (*Y*), i.e., how changing concentrations of greenhouse gases may causally affect global warming.

If we translate this example to network models, differences emerge. Most importantly, climate models are simulation models, whereas network models are data models. Indeed, Massimi (2018b) contrasts perspectival models with phenomenological models, where the latter "are designed to model observed occurrences rather than possibilities, as is the case with perspectival models" (p. 339). This claim about phenomenological models can be applied to data models. Indeed, I am hesitant to claim that network models *are* perspectival models in Massimi's interpretation of the term. However, we can use elements of Massimi's analysis to formulate criteria for assessing the exploratory potential of network models. Specifically, network models help psychometricians reason about possibilities for psychiatric phenomena that are *epistemically relevant* if these possibilities are *physically conceivable*, i.e., consistent with psychometricians' available empirical and theoretical background knowledge.

To illustrate this, let us go back to the case study by Mullarkey et al. (2019). They use their finding that the variable "self-hatred" has high node strength to make a possible explanatory claim about a psychiatric phenomenon, i.e., self-hatred may play an important causal role in adolescent depression. They do so based on empirical evidence that negative self-referent thinking is a risk factor for depression (Connolly et al., 2016) and the theoretical assumption that symptoms with high node centrality play an important role in depression (alluded to by, e.g., Borsboom, 2017a). So, what Mullarkey et al. (2019) consider epistemically relevant how-possibly explanations of their network model findings is guided and constrained by their available theoretical and empirical background knowledge of adolescent depression.

This description shows similarities to the proposed role of network models in *hypothesis generation*. Indeed, various psychometricians have alluded to the hypothesis-generating function of network models (e.g., Borsboom & Cramer, 2013; Epskamp, van Borkulo, et al., 2018; Epskamp, Waldorp, et al., 2018; Fried & Cramer, 2017). Borsboom and Cramer (2013) argue that "[b]etween-subjects psychopathology networks are useful in, for instance, investigating the general structure of psychiatric disorders as they can generate testable hypotheses about trajectories toward developing a psychiatric disorder that are shared by individuals" (p. 108). Moreover, Fried and Cramer (2017) argue that "[c]ross-sectional network models are capable (…) of generating hypotheses at the group level: for example, the potential hypothesis that women—as a group—have a more strongly connected depression network than men—as a group" (p. 12). However, psychometricians could formulate many different hypotheses based on network models; what makes a hypothesis afforded by a network model "good" or "epistemically relevant"? My account implies that network models provide epistemically relevant hypotheses about possibilities for psychiatric phenomena if these hypotheses are consistent with psychometricians' available theoretical and empirical background knowledge. In the next section, I will elaborate on the criteria for such background knowledge.

4.5 Exploration and background knowledge

In the previous section, I argued that the possibilities for psychiatric phenomena that psychometricians can envision based on network models – or the hypotheses they can formulate – should be guided and constrained by their theoretical and empirical background knowledge to ensure their epistemic relevance. However, more remains to be said about such background knowledge. What criteria should it meet to aid the exploratory function of network models, i.e., enhance the epistemic relevance

of the hypotheses that network models afford? As shown in **Section 4.4**, Massimi (2022) claims that model-based scenarios are physically conceivable when they comply with scientists' knowledge and conceptual resources and are consistent with known lawlike dependencies. This description presents the first criterion, already mentioned in **Chapter 3**, for psychometricians' background knowledge: *empirical adequacy*. We can also formulate a second criterion for the background knowledge of psychometricians based on **Chapter 3**: It should be adequately *coordinated* with the dependency relationships demonstrated by network models, i.e., represent similar dependency relationships. Phrased differently, psychometricians' background knowledge can only guide and constrain their exploratory reasoning with a network model if this knowledge can *speak to* the dependency relationships the network model affords. In this section, I describe three types of background knowledge that guide and constrain the exploratory potential of network models and examine whether these criteria are met. Again, I use Mullarkey et al. (2019) as a case study.

First, the exploratory potential of network models is guided and constrained by the *network theory*. Network theoretical principles (implicitly) inform how network models are construed and estimated. Indeed, we see the network theory's principles reflected in Mullarkey et al.'s (2019) network model estimation. The second principle – *symptom-component correspondence* – is reflected in their use of depression symptoms (as measured with CDI) as nodes. The first principle – *complexity* – and fourth principle – *mental disorders follow a network structure* – are reflected in the use of network analysis rather than, for instance, latent variable analysis. Moreover, network theoretical principles inform what dependency relationships the researchers consider relevant to investigate. Mullarkey et al. (2019) refer to the theoretical work of Borsboom (2017a) to justify their decision to explore node centrality, and their exploration of the strength of individual edges reflects the third principle, i.e., *direct causal connections*. Finally, network theoretical principles shape how psychometricians translate network analysis findings into possibility claims about psychiatric phenomena, as discussed in **Section 4.4**. Indeed, the explanations provided by the network theory can shape the explanatory possibilities for psychiatric phenomena that network models afford. How well can these explanations do so?

I stated in **Chapter 2** that the network theory explains within-person phenomena, whereas cross-sectional studies demonstrate group-averaged dependency relationships. This implies that the network theory and cross-sectional network models are not adequately coordinated. Indeed, for various dependency relationships that network models demonstrate, network theoretical principles only weakly guide or constrain the possibilities for psychiatric phenomena that psychometricians can

imagine. The third principle – *direct causal connections* – suggests that the dependency relationships between nodes are causal, but this principle does not provide a specific directionality for these relationships. For instance, the edge between the nodes "loneliness" and "sadness" in Mullarkey et al.'s (2019) network model could reflect the dependency that "feeling lonely causes sadness" or "sadness causes feelings of loneliness." Given the difficulties in justifying the network theory's causal claims based on the nature of mental disorders, as discussed in **Chapter 2**, these dependency relationships could also be interpreted as "feeling lonely and sadness mutually cause each other" or "feeling lonely and sadness overlap conceptually." Moreover, network theoretical principles do not fully guide psychometricians' reasoning about centrality measures**.** Centrality measures are hypothesized to refer to the relative importance of specific nodes in the development and maintenance of mental disorders (Cramer et al., 2010), but what such "importance" entails is unclear (Bringmann et al., 2019). Relatedly, there is a weak correlation between the causal influence of a node and its centrality (Dablander & Hinne, 2019). So, the network theory alone may only weakly guide and constrain the possibilities that psychometricians can envision based on their network models.[70]

Second, the exploratory potential of network models is guided and constrained by *empirical studies* that focus on specific nodes, edges, or topological properties**.** As mentioned before, Mullarkey et al. (2019) claim that self-hatred may play an important role in adolescent depression based on the finding that negative self-referent thinking is a risk factor for depression (Connolly et al., 2016). However, to what extent can such empirical evidence help psychometricians reason about *epistemically relevant* possibilities based on their network models? First, we can question whether such scientific evidence is adequately coordinated with the network model. Connolly et al. (2016) demonstrated that in a community sample of adolescents, negative information processing biases on a self-referent encoding task were associated with concurrent depressive symptoms. Moreover, they found that these negative information processing biases predicted increases in depressive symptoms after a follow-up period. Can these findings be used in conjunction with the high centrality of self-hatred in the network model to support the claim that self-hatred may play an important causal role in adolescent depression? This depends, amongst others, on whether the variables "self-hatred" and "negative information processing biases in a self-referent encoding task" relate to the same phenomenon. Moreover, given

70. Mullarkey et al. (2019) also interpret their findings in light of Beck's (1967) cognitive model of depression, where negative thoughts about the self, the world, and the future, along with negative mood, mutually influence each other to maintain depressive symptoms. I will highlight the similarities between the network theory and the cognitive-behavioral theory in **Chapter 8**.

the replication crisis in psychology, assessing the empirical adequacy or *robustness* of such findings requires further confirmation.

Third, the exploratory potential of network models is guided and constrained by *background theories*, i.e., psychometricians' (often implicit) intuitions about what the world is like that are not directly tested or evaluated but nonetheless interact with and drive scientific inquiry (Oude Maatman, 2024) (see **Chapter 3**). The constraining function of background theories echoes Phillips and Kratzer (2024), who argue that the possibilities we consider in modal reasoning should conform to *normality*, i.e., our knowledge of natural laws (or lawlike dependencies), statistically likely events, and actions we deem morally good, rational, and in line with conventional norms. For instance, we are more likely to interpret the strong edge between sleep disturbance and fatigue that Mullarkey et al. (2019) found as indicating the possibility that "sleep disturbances may cause fatigue," rather than vice versa. Related, folk psychological considerations can help determine what possibilities are (not) epistemically relevant. However, as suggested in **Chapter 2**, folk psychology alone will not always help determine the directionality of these relations.

In conclusion, the network theory, empirical studies, and background theories can guide and constrain the exploratory potential of network models by informing what possibilities for psychiatric phenomena are *epistemically relevant* to imagine. However, they can only provide such constraints when they are empirically adequate and adequately coordinated with the dependency relationships that network models demonstrate; such criteria are not always met. Thus far, I have focused on how the *information* that network models provide and their background knowledge drive the exploratory function of network models. In the next section, I will focus on network models' *pragmatic virtues*.

4.6 Exploration and pragmatic virtues

Scientific exploration is a practice-dependent endeavor. Indeed, Massimi (2022) highlights the context-dependency of perspectival modeling by claiming that the physical conceivability of model-based scenarios depends on the knowledge and resources *available to a specific scientist or scientific community*. However, the practice-dependency of exploration also requires us to focus on how scientists *use* scientific models in their epistemic endeavors. In line with **Chapter 3**, I argue that reasoning with models about (physically conceivable) possibilities for psychiatric phenomena is an *ability*. This implies that the exploratory – or hypothesis-generating – potential of

network models depends, in part, on how well psychometricians can *use* these models to reason; that is, on network models' *pragmatic virtues*. This pragmatic dimension of exploration has been acknowledged in the literature on exploratory modeling. It builds upon the idea that perspectival representations are pragmatic (Massimi, 2022, Chapter 2.5) and Gelfert's (2016) claim that exploration is an activity involving "subjective" elements (e.g., getting a feel for a model or target system), skills, and the ability to manipulate. However, to my knowledge, the role of specific pragmatic virtues has thus far not been explicitly addressed in the literature on exploratory modeling or hypothesis generation. As stated in **Chapter 3**, what features of scientific representations are considered pragmatic virtues is practice-specific. In this section, I focus on two pragmatic virtues of network models put to the fore by network psychometricians.

Visualizability

The first pragmatic virtue of network models is their *visualizability*. The epistemic value of visualizable scientific models has been acknowledged previously (Griesemer, 2004; Nagel, 1961), and the visualizability of network models is often considered the main reason for their appeal (Borsboom, 2022a; Borsboom, Deserno, et al., 2021; Luke & Stamatakis, 2012). Specifically, Borsboom (2022a) argues that visualization is an "essential part of the network psychometric paradigm" (p. 255) and an important feature of the exploratory function of network models, for the original aim of network representations in psychology was to aid in visually generating hypotheses (e.g., Cramer et al., 2010; Epskamp et al., 2012).

The visualizability of network models supports their exploratory function because it allows psychometricians to directly *see* the dependency relationships that serve as the starting point for imagining possibilities for psychiatric phenomena. Visual network models show directly how symptoms are related, how strong these relations are, and what symptoms may be strongly or weakly connected. To illustrate, let us look at the hypothetical network model presented in **Figure 6**. Just by glancing at this model, we can already *see* that anhedonia is a central node, the dependency relationship between insomnia and fatigue is strong, and weight loss does not demonstrate relevant connections to the other nodes. In other words, visualizations highlight the model's epistemically relevant features.

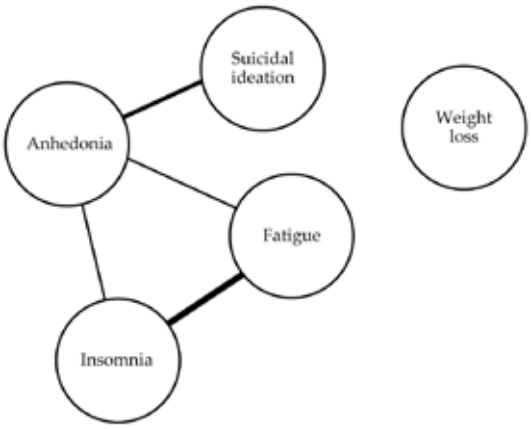


Figure 6. A hypothetical depression network model

Indeed, as argued by Leonelli (2009) in the context of biological databases, images help scientists to "better assess the significance of those data and descriptions" (p. 198) and "offer the possibility to match the researcher's sensory experience of a phenomenon (and of the system in which the phenomenon manifests itself) with his or her hypotheses and background knowledge" (pp. 198-199). Similarly, in their work on mechanistic explanations, Bechtel and Abrahamsen (2005) argue that diagrammatic representations of complex mechanisms are more tractable than linguistic (or, in this instance, numerical) ones.[71]

As shown in **Chapter 3**, grasping is defined as *seeing* how isolated pieces of information hang together. Indeed, like grasping, the epistemic value of network model visualizations is closely tied to model manipulation. This again echoes Leonelli (2009), who argues that images in biological databases "constitute models with which biologists can work" (p. 198). The relationship between network model visualizations and manipulation is illustrated by *qgraph*, an *R* package developed for network analysis and visualization in psychometrics (Epskamp et al., 2012). The package "accommodates capacities for spotting patterns by visualizing data" as networks by allowing psychometricians to play around with the visualizations (p. 1). They can change the settings to group nodes according to a pre-fixed criterion or based on their correlation patterns (i.e., shorter edges for stronger weights), make stronger edges thicker, and use different colors for different nodes, amongst others. Such tweaking with the network visualizations provides a "powerful exploratory tool" (p. 6) that enables psychometricians "to approach their data from a new perspective"

71. For a discussion on the role of visualizations in theory intelligibility in physics, see De Regt (2017, Chapter 7).

(p. 14). We can put this more strongly: Actively engaging with network visualizations allows psychometricians to *see* what dependency relationships are most salient. As a related example, Hayles (1991) describes an interaction between a researcher and their computer simulation of a dynamical nonlinear system as follows:

> With her own responses in a feedback loop with the computer, she develops an intuitive feeling for how the display and parameters interact. She notices that small changes in initial values can lead to large changes in the display. She also sees that, although the displays are complex, there are underlying symmetries that impart a pleasing, sometimes a startlingly beautiful quality to them. (p. 6)

So, the visualizability of network models allows psychometricians to directly *see* (and engage with) the epistemically salient dependency relationships that serve as the starting point for reasoning about possibilities for psychiatric phenomena.

Transparency

The second pragmatic virtue of network models is their *transparency*. The epistemic value of transparent scientific models – and the risks of epistemic opacity – has been discussed previously in the philosophy of science (e.g., Humphreys, 2004, 2009). Similarly, in the network psychometrics context, Borsboom (2022a) argues that "[t]he value of transparency in the explorative research process should not be underestimated" (p. 255). Various forms of transparency can be distinguished in scientific modeling (Creel, 2020). Here, I focus on *functional transparency*, defined by Chirimuuta (2021) as scientists' ability to know "the mathematical functions mapping inputs to outputs" (p. 775). Indeed, network psychometricians imply *functional* transparency when discussing the importance of transparency in their modeling practice. In network model estimation, functional opaqueness could arise when transforming the dataset into the adjacency matrix or the adjacency matrix into topological properties. However, Borsboom (2022a) argues that one of the exploratory benefits of network analysis is that the transformation from dataset into adjacency matrix is "tractable." That is, the regularization methods that are applied to the data – such as lasso – operate "by following a *tractable* [emphasis added] search path through the model space on the basis of neighborhood selection" which makes it "clear what mode of exploration is followed" (p. 255). This contrasts with other psychometric modeling practices, such as structural equation modeling, "where researchers sometimes follow an *opaque* [emphasis added] search path through the model space" (p. 255).

The functional transparency of network models supports their exploratory function because it can help psychometricians become acquainted with and assess the model's relevant epistemic features. Like visualization, functional transparency can improve psychometricians' engagement with network models. For instance, if they know how the dependency relationships in a network model came about, it is easier for them to spot possible mistakes in the modeling process. This helps them assess what dependency findings are actually possible evidence for psychiatric phenomena and which findings are less robust. Moreover, being able to trace the modeling steps from the initial formulation of questionnaire items and data collection to the output of network models could make it more salient for psychometricians with what background knowledge the network model should comply in formulating possibilities for psychiatric phenomena. Psychometricians can formulate (epistemically relevant) possibilities based on network models if they do not know how their dependency relations came about. However, a functionally transparent model will give psychometricians more opportunity to engage with its epistemically salient dependency relationships.

So, the exploratory function of network models is facilitated by their pragmatic virtues, specifically their visualizability and functional transparency, because this highlights what network model features are most epistemically salient. In the next section, I will address the *relative* exploratory potential of network models.

4.7 Comparing exploratory models: Multilayer networks

Based on my analysis thus far, I can identify two features of the modeling context that determine the exploratory potential of network models: (1) the coordination of network models with available, empirically adequate background knowledge and (2) the pragmatic virtues of network models. In this section, I use these features as a starting point to reflect on the relative exploratory potential of developments in network psychometrics. As a case study, I focus on the integration of network psychometrics and network neuroscience via *multilayer brain-symptom network models* (Blanken et al., 2021; Brooks et al., 2020; De Boer et al., 2021).

As mentioned in **Chapter 2**, multilayer networks are networks comprised of multiple layers of nodes connected via edges, with connections between and within the layers. Multilayer network analysis is increasingly used in *network neuroscience* to study the interactions between different neuroimaging modalities (Vaiana & Muldoon, 2018). For instance, multilayer network analysis allows neuroscientists to explore

the relationship between structural and functional connectivity brain networks or between the functional connectivity networks of different frequency bands. However, researchers are also investigating the possibility of integrating brain network models with network models of cognitive factors (Simpson-Kent et al., 2020), personality factors (Brooks et al., 2020), social factors (Bassett & Sporns, 2017), and *psychiatric symptoms* (Blanken et al., 2021). Such multilayer brain-symptom network models would include (1) symptom nodes and their conditional associations, (2) brain region nodes and their structural or functional conditional associations, and (3) *inter-layer associations* between symptom and brain region nodes. To date, no studies have estimated such multilayer brain-symptom network models based on empirical data, but Blanken et al. (2021) have suggested how such analyses could take place. First, scientists estimate the symptom- and brain network models separately based on psychometric and neuroimaging data.[72] Afterward, they estimate the inter-layer edges between the brain regions and symptoms, representing correlations between symptom and brain region values. Next, the adjacency matrices of the symptom network model, brain network model, and inter-layer correlations are combined into a multilayer network data model or *supra-adjacency matrix*. This supra-adjacency matrix is subsequently used to estimate a visual multilayer network model and to quantify multilayer network properties.

At first glance, we may assume that integrating neuroscientific data into symptom network models enhances the latter's exploratory potential. That is, it adds new information that could open up new routes for exploring brain-behavior relationships (Blanken et al., 2021; Hilland et al., 2020). Indeed, multilayer brain-symptom network models can provide two types of information about psychiatric phenomena that "standard" network models cannot. First, multilayer brain-symptom network models can provide information about the presence, strength, and direction of the association between brain regions and symptoms via inter-layer edges. Second, these models can provide information on multilayer topological properties, such as multilayer clusters that include both brain- and symptom nodes (De Domenico, Lancichinetti, et al., 2015). Following my earlier terminology, we could argue that multilayer brain-symptom network models have more *exploratory breadth* than standard network models. However, considering the criteria for the exploratory potential of network models outlined at the start of this section, we see that multilayer network models do not necessarily enhance scientists' ability to reason about possibilities concerning psychiatric phenomena.

72. For more information on (estimating) brain network models, see Bullmore & Sporns (2009).

First, multilayer brain-symptom network models are not (yet) accompanied by a theory that is adequately coordinated with these models. Specifically, there is insufficient theoretical knowledge about the dependencies between brain regions and symptoms, as well as about multilayer brain-symptom network dynamics. As a result, the hypotheses that scientists can currently formulate about why these dependency relationships may hold may lack empirical relevance. Although the network theory is compatible with a multilayer conceptualization of mental disorders, as argued in **Chapter 2**, its principles do not *speak to* these multilayer dependency relationships. Similarly, the theoretical principles guiding network neuroscience focus on network topology, i.e., brain connectivity patterns, rather than individual edges (Van den Heuvel et al., 2019; Van den Heuvel & Sporns, 2019). So, neither psychometric- nor neuroscientific network theory provides explanations for the dependency relationships *between* these two domains. Let us suppose that scientists find a strong positive association between anhedonia and the medial prefrontal cortex in their multilayer brain-symptom network model. If so, no adequately coordinated theoretical background knowledge is available that can guide or constrain their reasoning on why this may be the case. Borsboom (2022a) claims that psychometricians should develop theoretical models that can integrate different data sources. In line with this call, I argue that the lack of adequately coordinated theoretical background knowledge hampers the exploratory potential of multilayer brain-symptom network models.

Second, multilayer brain-symptom network models will not necessarily have more *pragmatic virtues* than standard network models. In **Section 4.6**, I stated that visualizability and transparency are pragmatic virtues in network psychometrics. Are these pragmatic virtues also relevant for multilayer brain-symptom network models? Interpreting and estimating multilayer brain-symptom network models involves two distinct but related scientific communities: network psychometrics and network neuroscience. These scientific communities differ because their scientists have different background knowledge – psychological science versus neuroscience – and are trained in different skills. However, both use similar statistical methods, i.e., network analysis. Moreover, scientists who work in (network) neuroimaging also highlight the importance of visualization (Albensi et al., 2004) and transparency (Badrulhisham et al., 2024) in their scientific practices. So, focusing on these two pragmatic virtues is justified for multilayer brain-symptom network models. Multilayer brain-symptom networks are visualizable in principle, and Vaiana and Muldoon (2018) specifically argue that an advantage of multilayer network models is that they retain the visualizability of standard network models. So, I will focus on the relative *functional opacity* of multilayer brain-symptom network models.

Functional opacity may arise at two steps in the multilayer network analysis pipeline. First, functional opacity could be introduced when transforming supra-adjacency adjacency matrices into multilayer topological properties, specifically when estimating multilayer clustering. Blanken et al. (2021) argue that one of the benefits of multilayer brain-symptom network models is their ability to demonstrate cross-layer communities using multilayer clustering algorithms (De Domenico, Lancichinetti, et al., 2015) (see **Chapter 2**). However, clustering algorithms are driven by machine learning functions that are not entirely tractable. Second, even earlier in the multilayer network estimation process, functional opacity may arise when estimating brain region–symptom associations. Indeed, estimating these associations requires a high degree of *kludging*. A kludge is a software fix that is made locally to make a model work (Clark, 1987; Wimsatt, 2007), creating a "piece of program or machinery which works up to a point but is very complex, unprincipled in its design, ill-understood, hard to prove complete or sound and therefore [has] unknown limitations, and hard to maintain or extend" (Clark 1987, p. 278). When a scientific model includes too many unprincipled modeling decisions and future alternations of the model build on those pre-existing kludges, it becomes increasingly difficult to explore how model input and -output are related, introducing functional opacity (Creel 2020). Integrating brain- and symptom network models into one model will introduce unprincipled decisions, exacerbated by the separate modeling histories of network neuroscience and network psychometrics. For instance, to estimate associations between symptoms and brain regions, brain regions should have a group-level mean value; however, due to how brain network models are estimated, brain regions do not automatically have this value. This means that scientists should decide in an unprincipled manner how to calculate this value, for instance, using the node's mean strength (Brooks et al., 2020). Also, inter-layer edges should be controlled for false positives. However, it is unclear what method to use: Symptom network models use regularization, but brain network models use thresholding (i.e., omitting edges that do not meet a predetermined statistical significance criterion). Relatedly, there is no principled way to control for the fact that intra-layer variables will correlate more strongly than inter-layer variables. All these statistical hurdles require scientists to make decisions that are, to some extent, unprincipled. Hence, multilayer brain-symptom network models will be less functionally transparent than standard network models, hampering scientists' ability to see what features of multilayer network models are epistemically salient.[73]

73. For a comparable account of the role of (entrenched) kludges in climate models, see Lenhard and Winsberg (2010).

In conclusion, multilayer brain-symptom network models may have more *exploratory breadth* than standard network models. However, this will not directly translate to improvements in their exploratory function unless they are accompanied by theory-building and efforts to improve their functional transparency.

4.8 Conclusion

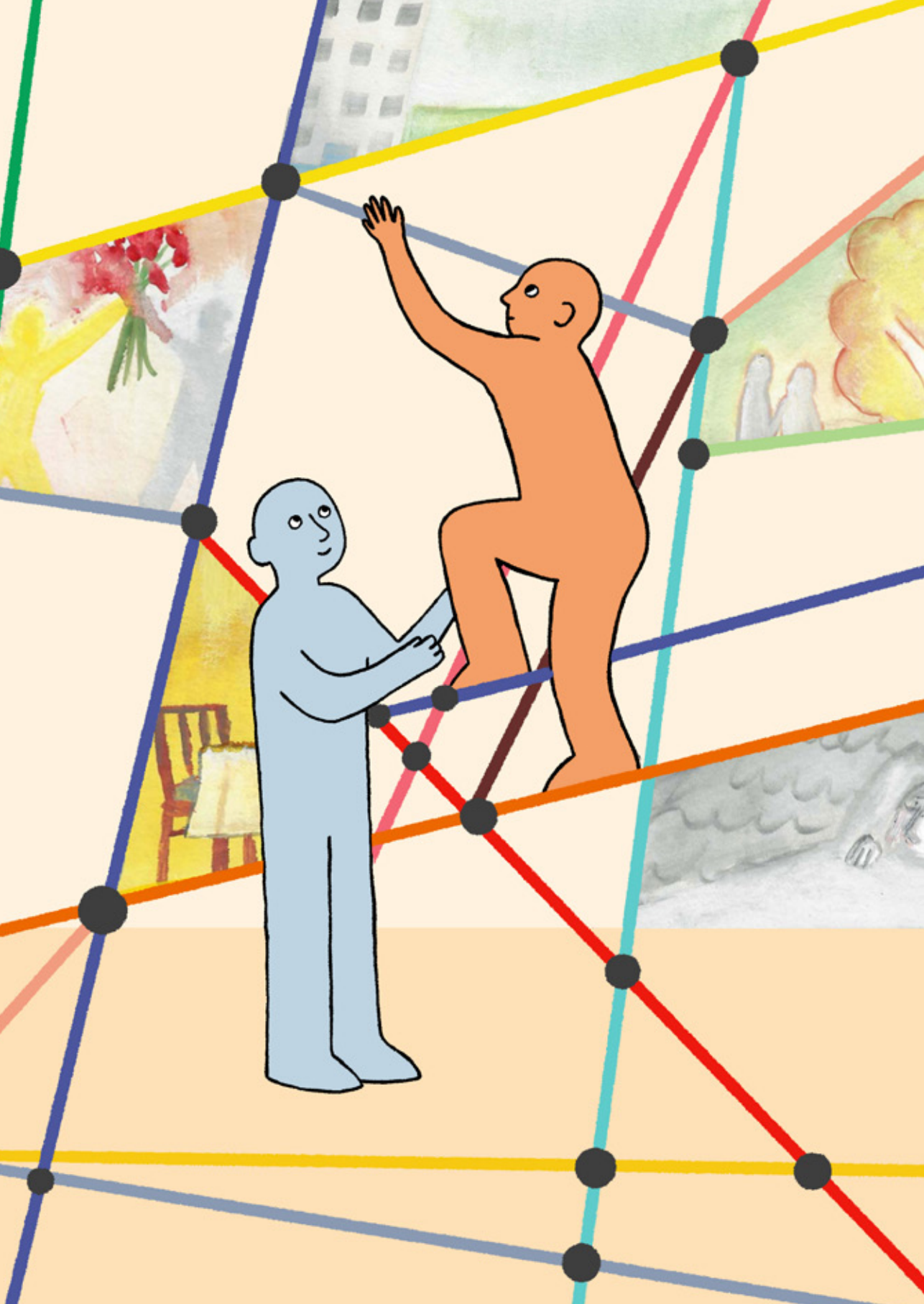
In this chapter, I characterized the exploratory function of network models based on my practice-oriented account. Network models fulfill their exploratory function by allowing psychometricians to reason about possibilities for psychiatric phenomena based on the dependency relationships these models demonstrate. However, for these possibilities to be epistemically relevant, they should be physically conceivable, i.e., comply with psychometricians' available background knowledge. To guide and constrain what possibilities we can imagine based on dependency relationships in network models, such background knowledge should be adequately coordinated with these dependency relationships and empirically adequate. Based on these criteria, I showed that the network theory, empirical studies, and background theories do not always sufficiently guide and constrain psychometricians' ability to reason about possibilities for psychiatric phenomena. Moreover, the exploratory function of network models depends on their visualizability and functional transparency: Both features allow scientists to engage with these models and thereby highlight salient dependency relationships. Given these considerations, I concluded that integrated multilayer brain-symptom network models may have more exploratory breadth than standard network models but will not necessarily have more exploratory potential.

This chapter showed that exploration is not a poor derivative of other epistemic functions of network models but rather a relevant epistemic endeavor that can be assessed and improved. Psychometricians should ensure that their network models are coordinated with empirically adequate theories and empirical studies and that network models' pragmatic virtues are considered when pursuing developments in network analysis. Doing so will help ensure that psychometricians' practices align with their exploratory goals. Indeed, this chapter should not be read as a critique of (recent developments in) network psychometrics but rather as an explication of fruitful avenues for future research: It shows scientists what areas they should invest in to make network models more exploratorily advantageous. For instance, the need for theory-building alongside statistical developments in psychometrics is increasingly emphasized (Borsboom, Van der Maas, et al., 2021; Eronen & Bringmann, 2021; Eronen & Romeijn, 2020; Fried, 2020; Oude Maatman, 2024). Such

accounts emphasize the importance of theory-building for the network approach's explanatory potential; my analysis suggests that theory-building could also improve the exploratory potential of network models.

Moreover, my analysis raises new questions about the relationship between the different epistemic goals of the network approach. The claims in this chapter showed similarities to my characterization of scientific understanding presented in **Chapter 3**: Both understanding and exploration are concerned with psychometricians' ability to use network models to reason about possibilities for psychiatric phenomena. Indeed, one could argue that psychometricians' ability to *explore* psychiatric phenomena is a prerequisite for *understanding* them. Alternatively, one could argue that both epistemic represent different phases in the research process. Another interpretation could be that the exploratory reasoning that network models afford provides a different form of scientific understanding than the type I have advocated in **Chapter 3**, one that is not directly concerned with possibilities for reducing our mental suffering. How the noetic and exploratory potential of network models precisely relate is an open question. Also, this chapter focused on the exploratory potential of network *models*, but one could argue that the network *theory* also has an exploratory function. Indeed, **Chapter 3** shows that in the psychiatric context, explaining and exploring are not as opposed as is often suggested. Since the network theory is not presented in the psychometrics literature as a means of exploration, I did not discuss this interpretation here. However, identifying the exploratory differences between the network theory and network models would be an interesting endeavor for future research.

In these past chapters, I have focused on the epistemic potential of the network approach in scientific practice. However, as I already highlighted in **Chapter 1**, this is only one practice in which the network approach is used. In the following chapters, I will build on this analysis to explore the epistemic potential of the network approach in other practices, starting with *clinical practice*.



Part II
Clinical practice

Chapter 5

Personalized network models in person-centered care

Abstract

In this chapter, I address the epistemic potential of personalized network models in *person-centered care* (PCC) from a practice-oriented perspective. Personalized network models can be helpful tools for PCC because they are *personal* and *personalized*. Clinical ESM data used to estimate personalized network models can overcome some standard critiques of quantification in the social domain, i.e., generalization and non-contextuality. However, taking the personalization of personalized network models seriously could raise *boundary problems*, i.e., problems in demarcating what should and should not be included in the model. Personalized network models are constrained by their representational, explanatory, and contextual boundaries. Erotetic reasoning principles can clarify what relevant explanation-seeking questions personalized network models can address.[74]

5.1 Introduction

In the second part of my thesis, I discuss the epistemic potential of the network approach in *clinical practice*, where clinicians and clients use the approach to obtain knowledge about clients' mental problems.[75] Specifically, I focus on the use of *personalized network models* in clinical practice. Focusing on these models is of interest given recent shifts in how the role of scientific evidence in psychiatric clinical care is conceptualized.

The biomedical approach coincided with the birth of *evidence-based medicine* (EBM) as a paradigm for psychiatric clinical care. EBM states that medical practitioners

74. This chapter is based on the co-authored articles by De Boer et al. (2022) and De Boer and Runhardt (2024). As first author, I was responsible for the original articles' conceptualizations, drafts, reviews, editing, and final structure; I contributed to the content of all sections. For De Boer and Runhardt (2024), I specifically contributed the case study (i.e., clinical ESM data). Some general changes to both articles include using the first person to unite the style of the thesis and streamlining the terminology with the other chapters. The main changes to De Boer et al. (2022) involve changes to the structure, removing the term "perspectivism" to avoid confusion with "perspectival modeling" mentioned in **Chapter 4**, and changes to how the explanatory potential of personalized network models is framed. The main changes to De Boer and Runhardt (2024) involve focusing on the case study, for which I was solely responsible. I would like to refer the reader to the original articles for an exact overview of how the articles and chapter compare.

75. In my discussion of scientific practice, I refered to "psychiatric phenomena" as the epistemic object of the network approach, but in clinical practice and daily life, I will refer to "mental problems." In these latter practices, the network approach is used to obtain knowledge about an individual's mental suffering rather than about robust, observable, and generalizable features of mental suffering that occur over a wide variety of contexts.

– including psychiatrists – should conscientiously, explicitly, and judiciously use the current best scientific evidence in making decisions for clinical care (Sackett et al., 1996). EBM creates a hierarchy of evidence, with meta-analyses of randomized clinical trials at the top of the hierarchy and clinical intuition and personal experience at the bottom. Indeed, a similar separation between and hierarchy of "objective" and "subjective" aspects of mental disorders is visible in certain interpretations of the biomedical model. The biomedical model is often interpreted in a *neuroessentialist* fashion, assuming that the brain is the essence of a person and synonymous with concepts like the "self" (O'Connor et al., 2012).[76] This neuroessentialist interpretation obscures the role of personal, contextual factors in mental suffering and ignores the web of relationships between an individual and their context that co-determines their identity.[77] Relatedly, the neuroessentialist interpretation of the biomedical model implies that neuroscientific data alone provides exhaustive insight into psychopathology's "objective" core; personal experience is merely its subjective reflection. In other words, the biomedical approach has facilitated the idea that scientific evidence about disease pathogenesis of mental disorders belongs to the "objective" core, and the values and interests of the person in treatment and clinical intuitions belong to the soft margins.

In recent years, there has been a reappreciation of the idiosyncratic and contextual nature of mental suffering in psychiatric clinical care. This is reflected in the rise of *person-centered care* (PCC) as a guiding vision for diagnosis and treatment in clinical practice.[78] PCC has its origins in nursing and geriatrics (Morgan & Yoder, 2012) and aims to respectfully care for an individual by considering their preferences, needs, and values and ensuring that these aspects guide all clinical decisions (Håkansson Eklund et al., 2019; Morgan & Yoder, 2012). So, PCC emphasizes the alliance between a clinician and a client. Mezzich (2011) gives the following description of person-centered medicine, which applies well to PCC:

> [A] medicine *of* the person (of the totality of the person's health, including its ill and positive aspects), *for* the person (promoting the fulfillment of the person's life project), *by* the person (with clinicians extending themselves as full human beings, well-grounded on science and with high ethical aspirations) and *with* the person (working respectfully, in collaboration

76. Such neuroessentialism is also visible outside the psychiatric context. It is not uncommon to hear phrases such as "You are your brain," or to encounter headlines like "the [adjective] brain," where the brackets are filled in with categories like "female/male," "teenage," "addicted," "hyperactive," et cetera.

77. I will discuss this observation in more detail in **Chapter 7** when I describe how the biomedical model influences the psychiatric threads of our self-narration.

78. In the remainder of this thesis, I will use "PCC" to refer to PCC *in psychiatric clinical practice*.

> and in an empowering manner through a partnership of patient, family, and clinicians). The person here is conceptualized in a fully contextualized manner. (p. 335)

As this quote illustrates, PCC does not reject using scientific evidence in clinical practice. Instead, it wants to place scientific evidence in a clinical framework sensitive to the experience, context, and personal values of the person in treatment (Glas, 2019). However, bringing scientific evidence in "epistemic parity" with personal and contextual factors is no easy task. How can scientific evidence capture PCC's main tenets *and* provide the client and clinician with relevant knowledge? As I alluded to in **Chapter 1**, psychiatric scientists and clinicians have expressed the hope that introducing *data science* into clinical practice can help this endeavor (Russ et al., 2018; Rutledge et al., 2019; Torous & Baker, 2016). Specifically, psychiatric science is finding new ways of bringing quantitative data and personal experience into "epistemic parity" with recent developments in *personalized network analysis*. Indeed, Glas (2019) argues that using personalized network models may be "a promising way to connect scientific evidence, contexts, and person-centered care" (p. 184). In this chapter, I evaluate the epistemic potential of personalized network models in PCC from a practice-oriented perspective. In other words, I examine what may drive or hamper using these models to reason about possibilities regarding a client's mental problems.

The chapter is structured as follows. In **Section 5.2**, I explore why personalized network models could be valuable epistemic tools for PCC. I describe the main differences between cross-sectional and personalized network models and argue that the latter could be helpful for PCC because they are personal and personalized. In **Section 5.3**, I show how the personal nature of the data used for personalized network models – clinical ESM – avoids traditional criticisms of quantification in the social domain by avoiding generalization and being context-dependent. In **Section 5.4**, I describe an epistemic limitation of the personalized nature of personalized network models, i.e., the introduction of *boundary problems*. In **Section 5.5**, I assess how personalized network models' representational, explanatory, and contextual boundaries can help guide and constrain node selection. In **Section 5.6**, I address how erotetic reasoning can illuminate the relevant explanation-seeking questions that personalized network models could answer in PCC.[79]

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79. Note that this chapter will discuss the inclusion of contextual factors in personalized network models but will not address broader sociocultural influences that shape clinical practice.

5.2 Personalized network models

Pilot studies suggest that personalized network models can benefit clinical practice (e.g., Bak et al., 2016; Frumkin et al., 2021; Kroeze et al., 2017; von Klipstein et al., 2023). For instance, clinicians and psychometricians argue that personalized network models can generate questions that serve as a starting point for clinical dialogue (Epskamp, Van Borkulo, et al., 2018; Von Klipstein et al., 2020), improve case conceptualization (Von Klipstein et al., 2020) or suggest treatment targets (David et al., 2018; Rubel et al., 2018). In this section, I explore how personalized network models are estimated and what features of these models could make them valuable epistemic tools for PCC.

Personalized network models are (visualizable) data models that are the output of network analysis. However, their estimation and interpretation differ from the cross-sectional network models discussed in **Chapter 4** (Borsboom, Deserno, et al., 2021). First, personalized network models have different data input than cross-sectional network models. Cross-sectional network models are estimated based on data from a group of individuals (N = large) collected at a single time point (T = 1). In contrast, personalized network models are estimated based on *personal time-series data*, i.e., data from one individual (N = 1) collected over multiple time points (T = large). Such data is provided by ESM, a structured self-report technique that assesses an individual's momentary states, such as their thoughts, feelings, activities, and company at the time of reporting. The principles of ESM are not new (Larson & Csikszentmihalyi, 1983). However, recent developments allow individuals to monitor their experiences several times a day, usually for multiple days or weeks, generally on their smartphones (Shiffman et al., 2008) or other electronic devices (e.g., Timmons et al., 2017).[80] ESM is used in psychological science to test hypotheses that cross-sectional methods cannot address (Myin-Germeys et al., 2009, 2018). In the clinical context, *clinical ESM data* is used to investigate how factors that influence a client's mental well-being develop and co-occur over time.

Second, the conditional associations in personalized network models are interpreted differently from those of cross-sectional network models. Indeed, whereas cross-sectional network models are fitted with Ising, Gaussian, or mixed graphical models, personalized network models are fitted with *graphical vector autoregression* (VAR)

80. ESM can be combined with other measurement tools, such as wearables that generate personal biological data.

models.[81] An edge in a cross-sectional network model represents the (regularized) conditional association between two nodes in a group of individuals, driven by individual differences. For instance, a cross-sectional network model edge represents how, on average, the variables "anhedonia" and "insomnia" are conditionally dependent within a group of individuals with a depression diagnosis. VAR models can be used to estimate two types of network models. In a *temporal network model*, an edge represents a (regularized) correlation association between two nodes over time within an individual. For instance, a temporal network model edge represents how, within an individual, anhedonia is (predictively) related to insomnia at a later measurement window. In a *contemporaneous network*, an edge represents a (regularized) conditional association between variables in the same measurement window (after controlling for the other variables in the same measurement window and all variables at the previous measurement window). For instance, a contemporaneous network edge represents how, within an individual, having anhedonia is predictively related to insomnia at the same time point.[82] So, the dependency relationships that personalized network models demonstrate provide evidence for the possible existence of specific patterns in a client's mental problems.

There are two reasons why personalized network models could serve as valuable epistemic tools in PCC. The first reason is that personalized network models are constructed based on *personal*, clinical ESM data rather than group averages. This aligns with PCC's principles, especially its focus on clients' specific problems and idiosyncrasies. Second, personalized network models can be constructed based on *personalized* rather than pre-determined datasets. Clinical ESM is not bound to a pre-set list of variables but can include different variables considered important for a client's mental health. This also implies that personal network models can include variables *beyond* symptoms, such as contextual and health-promoting factors (Kroeze et al., 2017; Lutz et al., 2018). For instance, the personalized clinical ESM questionnaire developed by Von Klipstein et al. (2023) includes variables ranging from "rumination" to "physical discomfort," "having company," and "gaming."[83] This broad set of variables that personalized network models could accommodate aligns with PCC's *systemic* assumption that clinical care should not only focus on symptom reduction but reflect "the totality of the person's health, including its ill and positive aspects," with the person being "conceptualized in a fully contextualized manner"

81. VAR models should not be confused with dynamical system models, which are based on sets of differential equations and may provide directed (causal) relations between variables (e.g., causal loop diagrams).

82. Epskamp, van Borkulo, et al. (2018) and Epskamp, Waldorp, et al. (2018) provide more information on estimating and interpreting VAR models.

83. This case study will be discussed in more detail in **Chapter 6**.

(Mezzich, 2011, p. 335). Indeed, "salutogenesis," or the study of the origins of health (Antonovsky, 1979), is one of the principles of PCC.[84] With this, PCC aligns with the World Health Organization, which declared almost fifty years ago that health is not merely the absence of disease or infirmity (Callahan, 1973). It also aligns with empirical research indicating that reducing negative mental states does not necessarily lead to an increase in positive mental states (Bradburn, 1969; Keyes et al., 2002). So, personalized network models could be valuable tools for PCC because their variables, and hence the dependency relations they demonstrate, are personally relevant to the client and could include contextual and salutogenic factors.[85]

Hence, personalized network models could be valuable tools for PCC because they are personal and can be personalized. In the next section, I will zoom in on the epistemic potential of their personal dimension, focusing on clinical ESM data specifically.

5.3 Clinical experience sampling methods

Clinicians and psychometricians claim that collecting personal, clinical ESM data could benefit clinical practice. For instance, they suggest that co-constructing ESM items could strengthen the collaboration between the client and clinician (Von Klipstein et al., 2020), and filling out ESM questionnaires may stimulate the client's awareness, reflection, and insight (Bos et al., 2019; Kramer et al., 2014). In this section, I address the epistemic potential of clinical ESM data from a philosophy of measurement perspective, showing how clinical ESM data can overcome some standard limitations of quantification in the social domain, which includes clinical practice.

From the 1990s onward, philosophers of measurement, historians of science, and philosophers of public administration have developed what is now called the "Original Critique" of quantification in the social domain (e.g., Desrosières, 1998; Espeland & Sauder, 2016; Espeland & Stevens, 1998; Hacking, 1990, 1995; Nirenberg & Nirenberg,

84. "Salutogenesis" is a combination of the Latin term for "health" (*salus*) and the Greek term for "origin" (*genesis*).

85. The idea that psychometricians can study well-being and salutogenic factors using network analysis is not new. First, network analysis has been applied to the study of well-being (e.g., Deserno et al., 2017; Giuntoli & Vidotto, 2020). Second, studies have estimated the conditional associations between symptoms, contextual influences, and health-promoting factors in cross-sectional network models (e.g., Deserno et al., 2017). There are different ways to incorporate these different domains into network models: They can be incorporated in "standard" network models (e.g., Deserno et al., 2017), or scientists can use more advanced network analysis methods, such as multilayer network analysis (Bianconi, 2018), to do justice to the differences between these psychometric items.

2021; Poovey, 1998; Porter, 1992, 1994, 1995; Power, 1997; Shore & Wright, 2015). The Original Critique challenges the objectivity and value-free nature of quantification in the social domain, arguing that quantification presupposes categories that encode specific (dominant) worldviews, histories, and goals. More specifically, the Original Critique claims that the infiltration of numbers and quantitative data in the public domain coincides with the erasure of contextual, informal, and qualitative knowledge. Based on this position, we could argue that quantifying mental suffering is epistemically and morally disadvantageous.

However, recent debates in the philosophy of measurement that build on the Original Critique provide a more nuanced perspective on quantification in the social domain (e.g., Alexandrova, 2017; Alexandrova & Fabian, 2022). Such recent views are illustrated in the contributions of *Limits of the Numerical: The Use and Abuse of Quantification* (Newfield et al., 2022b). The contributors to this edited volume argue that values shape how and why we quantify social phenomena but that this does not negate the valuable work that quantification can do in the social world. For instance, quantitative data can mobilize people and expose injustices, i.e., *statactivism*. Instead of highlighting the adverse effects of quantification in the social domain, they call for two related developments in our thinking about such quantification. First, we should acknowledge that the value of quantification in the social domain is goal- and context-dependent: Such quantification presents epistemic and moral/practical risks and benefits that may trade off. For instance, Alexandrova and Singh (2022) argue that philosophers of science should consider both the validity of quantitative measurements of well-being and "the rhetorical and pragmatic role [these numbers] plays in politics, governance, and public debate" (p. 198). Second, we should focus on *(re)contextualizing* quantitative data, i.e., bringing it in epistemic parity with non-numerical, qualitative information. Indeed, a general criticism of quantification in the social domain that builds upon the Original Critique is that relying on quantitative data alone causes us to lose sight of individual nuances and personal context. To illustrate, Newfield et al. (2022a) state that quantification in the social domain "[s]implifies, accompanied by loss of interpretive complexity, local context, and qualitative experience" (p. 13) and "[s]ilences and causes loss of political agency when numbers are used to bypass vernacular, standpoint, or subaltern knowledges" (p. 13). So, quantification in the social domain is not inherently epistemically or morally disadvantageous. We should evaluate the relative epistemic (and moral) potential of such quantification on a case-by-case and context-specific basis, and call for the contextualization of such data. I use this position as a starting point to examine clinical ESM data in more detail.

Zooming in on how clinical ESM data is obtained shows that this process is neither straightforward nor value-free, especially in a PCC context that emphasizes collaboration between client and clinician (cf. von Klipstein et al., 2020). Each measurement step necessary for obtaining clinical ESM data is driven by decisions influenced by epistemic and practical/moral considerations.[86] The first step in clinical ESM measurement is *variable selection*, i.e., deciding what features of the target system we want to measure and eventually represent in our personalized network models. Since clinical ESM is not bound to a pre-set list of variables, epistemic and practical/moral considerations will influence variable selection; this will be discussed in more detail in **Section 5.4**. The second step in clinical ESM measurement is *item selection*, i.e., transforming the selected variables into statements or questions the client should answer (*items*). Again, this transformation is not straightforward. An item's content (i.e., its conceptualization, cf. Cartwright & Runhardt, 2014) can differ depending on whether the client and clinician are interested in that client's current experiences (e.g., "I have a sense of belonging") or their recent experiences (e.g., "Since the last beep, I have felt a sense of belonging"). Moreover, after making this decision, multiple ways exist to transform variables into items. For instance, "feeling alone" can be conceptualized as "I feel like an outsider" or "I would prefer to have company." The final conceptualization that the clinician and client will decide upon will be influenced by epistemic and practical considerations. The third step in clinical ESM measurement is *response selection*, i.e., deciding what type of response the item requires, comparable to the *representation stage* of measurement in Cartwright and Runhardt (2014) and Bradburn et al. (2017). Most commonly, clients respond to an item on a numerical scale. Indeed, most items in the *ESM Item Repository* – an open-access repository of existing ESM items (Kirtley et al., 2023) – use an ordinal Likert scale (e.g., "I feel anxious," 1 = not at all, 7 = very much). The repository also includes items that require nominal responses (e.g., "Are you alone?" yes/no) or open-ended answers that are not on a scale at all (e.g., "Think about the most positive event of today. What was it?"). Again, an item can require different responses: Response selection that meshes with an item is rarely straightforward and will be driven by more than epistemic considerations. So, clinical ESM data inherently depends on variable, item, and response selection decisions, which are influenced by epistemic and practical considerations.

However, this observation alone does not show where the epistemic potential of clinical ESM data lies. I argue that clinical ESM data is epistemically advantageous

86. These measurement steps are inspired by Cartwright and Runhardt (2014) and Bradburn et al. (2017). These authors argue that measurement requires *characterization* (i.e., defining the concept or quantity and its features), *representation* (i.e., defining a scale or table of indicators to represent the concept or quantity), and *procedures* (i.e., describing what should be done to carry the measurement out successfully).

because it overcomes criticisms of quantification in the social domain found in both the Original Critique and *Limits of the Numerical*, i.e., the idea that quantitative data causes us to lose sight of individual nuances and personal context. First, clinical ESM data can nuance claims about the *generalization* that comes with quantification. The "depersonalization" that can accompany quantitative data is not necessarily the result of the data's *quantitative* nature; rather, it arises from the *group averaging* that generally accompanies quantitative data. Glossing over or bypassing individual differences within a study population is a broader criticism that many philosophers of science (cf. Steel, 2008) and psychometricians (e.g., Molenaar & Campbell, 2009) have previously raised. Clinical ESM data alleviates this worry by providing personal quantitative data based on self-reports that are not averaged across different individuals. Second, clinical ESM data can nuance claims about the *non-contextuality* of quantitative data, demonstrating that quantitative data *can* take local context into account. Clinical ESM data is collected in an ecologically valid manner, i.e., the data is collected in the "real world' rather than in a laboratory setting. Moreover, clinical ESM items can include questions about the individual's context. Moreover, clinical ESM pilot studies show that quantitative data *can* be embedded directly in a qualitative setting: ESM-based data models, such as personalized network models, are discussed and questioned in the clinical context (e.g., Frumkin et al., 2021; von Klipstein et al., 2023). So, clinical ESM data can overcome traditional criticisms of quantification in the social domain.

Clinical ESM data could still pose epistemic and moral limitations or risks. For instance, there is a risk that clinical ESM data – and its data models – will be excessively weighted by either the client or clinician in the clinical encounter, for "[t]he collection of 'objective' data (…) create[s] an appearance of objectivity" (Von Klipstein et al., 2020, p. 6). However, this analysis shows that clinical ESM data could

be used in epistemic parity with qualitative information in clinical practice.[87] In the next section, I will discuss the epistemic difficulties that may arise if we focus on the *personalized* dimension of personalized network models.

5.4 Boundary problems

Clinicians and psychometricians suggest that collecting *personalized* clinical ESM data *beyond symptoms* could benefit clinical practice. In other words, the fact that personalized network models can include factors of specific interest to the client, including specific symptoms, health-promoting factors, and contextual factors, is presented as an epistemic benefit. However, if we allow network models to be personalized to this degree, does this not amount to drawing their boundaries too broad? Attempts to move beyond symptoms inevitably raise questions about what factors (not) to include. Hence, the personalized and systematic nature of personalized network models may also introduce epistemic difficulties, specifically *boundary problems*.[88]

In its most basic definition, a boundary is present when an entity is somehow demarcated from something else (Varzi, 2013). However, deciding how to demarcate an

87. Clinical ESM data also introduces new epistemic and moral/practical questions about quantification in the social domain. First, the intensity and frequency of ESM assessment may increase the chance of *reactivity*, i.e., an individual changing their attitudes and behavior in response to being measured (Eisele et al., 2023). Psychological measurement could invoke different types of reactivity (Marchionni et al., 2024). Reactivity in ESM assessments could hamper the ecological validity of clinical ESM data, but this is not necessarily the case (Runhardt, 2021). For a more detailed discussion, see **Chapter 6**. Second, clinical ESM frequently resorts to *Ballung concepts* in its questionnaire items, i.e., items whose conceptualization can be interpreted in multiple ways (cf. Cartwright & Runhardt, 2014). For instance, Von Klipstein et al. (2023) includes the following item in their personalized clinical ESM questionnaire: "Since the previous beep, I avoided something." One may say "yes" to this question if one has avoided a confrontation, but also if one avoided working on a cognitively challenging task at work. Using Ballung concepts in clinical ESM places the burden of conceptualization and, to some extent, representation on the client and could thereby address the criticism that individual narratives and values are often ignored in measurement (Newfield et al., 2022a, p. 13). However, it may also introduce epistemic issues. For instance, without further analysis, we cannot be sure that a numerical value means the same thing at different times. Other risks of quantification in the social domain, which may or may not pertain to clinical ESM data, are its ability to 1) undermine the autonomy of and trust in professionals, 2) create a measurement burden and increase inequalities when measurement production is expensive, or 3) create perverse incentives to value numerical outcomes for their own sake (Newfield et al., 2022a, p. 13).

88. **Chapter 2** already touched upon boundary problems while discussing the network theory and *extended* mechanisms.

entity from its surroundings is not always straightforward. In the modeling context, boundary problems can arise when there is no consensus or principled rationale for *target system demarcation*, i.e., deciding what elements are part of or external to the target system. Uncertainties in target system demarcation directly translate to uncertainties in demarcating scientific models: What variables should they include? Target system demarcation is rarely straightforward: Even physical target systems rarely have clearly defined boundaries (Meadows, 2008). Indeed, boundary problems related to target system and model demarcation are not exclusive to personalized network models; they may be widespread in modeling practices. However, when using personalized network models in PCC, boundary problems may be especially salient.

First, PCC takes a systemic approach to mental disorders as its starting point. This systemic intuition that mental disorders are multifactorial and multidimensional can be conceptualized as one of PCC's *background theories* (see **Chapter 3**). However, this background theory does not strongly constrain target system demarcation for personalized network models. If we take the systemic approach seriously, the target system can encompass a person's mental health as influenced by their mental states, activities, environmental factors, and social factors. Thus, the systemic intuition that drives the personalization of personalized network models can actually hinder target system demarcation.

Second, boundary problems have specific epistemic consequences for network models, given that the topological dependency relationships these models demonstrate strongly depend on what variables are included in the model. In other words, node selection can strongly influence the topological properties of network models (Forbes et al., 2017; Hallquist et al., 2021; Neal & Neal, 2021). For instance, the *betweenness centrality* of a node is calculated by measuring the relative number of shortest paths passing through a specific node (Freeman, 1977). Removing or including one node in a network model can strongly impact the betweenness centrality values of individual nodes (Bringmann et al., 2019), as illustrated in **Figure 7**. So, the variables included in personalized network models strongly influence the dependency relationships that these models demonstrate.

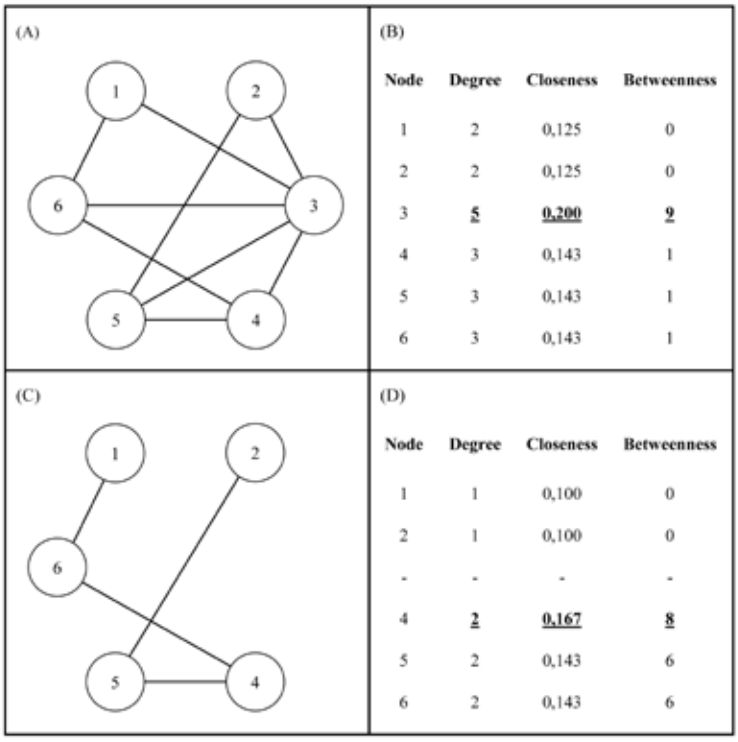

Figure 7. A hypothetical illustration of the effects of node selection on local topological properties in a network model. Panel (**A**) demonstrates a hypothetical network model comprised of six nodes. Panel (**B**) demonstrates the centrality measures of this network model, showing that node 3 has the highest node degree, closeness centrality, and betweenness centrality. Panel (**C**) demonstrates the same hypothetical network model but with node 3 removed. Panel (**D**) shows the influence of removing node 3 on the network model's centrality measures: Nodes 4, 5, and 6 have the highest node degree; node 4 has the highest closeness and betweenness centrality, and the betweenness centrality of nodes 5 and 6 has strongly increased.

Third and related, boundary problems have specific consequences for the context in which personalized network models are used, i.e., clinical practice. A clinician and client can use the dependency relationships that a personalized model demonstrates to reason about (possibilities regarding) the client's mental problems. If model demarcation strongly influences the dependency relationships that personalized network models demonstrate, node selection will influence the content of their therapeutic conversations. Relatedly, deciding *not* to include variables in personalized network models could also indirectly affect the clinical conversation: Excluding a variable could imply that it may enter into the background in these discussions. So, where clinicians and clients draw the boundary of personalized network models has important epistemic and, therefore, clinical consequences.

How, then, should they decide where to draw the boundary of personalized network models of mental disorders and justify their decision? In the next section, I will discuss three features of personalized network models and their context of use that can help demarcate their boundaries.

5.5 Three types of boundaries

Personalized network models have representational, explanatory, and contextual boundaries that guide and constrain the knowledge about mental problems they can provide. In this section, I will discuss these three types of boundaries and whether and how they could constrain node selection.

Representational boundaries

Personalized network models have *representational boundaries,* i.e., constraints provided by the models' representation and construction. ESM data can be analyzed using different types of statistical models that may summarize the data in different ways and, depending on the choice of model, reveal different empirical patterns (Piccirillo et al., 2019; Piccirillo & Rodebaugh, 2019).[89] The statistical techniques used to estimate (and visualize) personalized network models influence how the network model is represented and what kind of inferences about mental problems the model affords. Network representations themselves do not provide many constraints on what can be represented. Network models typically capture global and abstract features of a target system, whereas, for instance, mechanistic models capture more fine-grained and local features (Darrason, 2018; Kostić, 2018b, 2018a, 2019a, 2019b, 2020; Kostić & Khalifa, 2021, 2023; Rathkopf, 2018). However, nodes and edges can, in principle, represent anything. Nonetheless, this does not imply that personalized network models are representationally *boundless*, i.e., that they provide no inherent constraints on what nodes can be included and can be extended indefinitely in size or scale. Instead, network models in general, and personalized network models in particular, provide some, albeit limited, representational constraints. First, network models can represent only those variables that are, in principle, quantifiable and distinguishable from others. Second, VAR models cannot represent how structural relations between variables change over time (Molenaar, 2004). Third, VAR models cannot represent how variables in the network may be related to each other on different timescales. These representational boundaries provide some, albeit limited,

89. Another way to summarize clinical ESM data, besides using personalized network models, is by analyzing it for complexity markers (Olthof, Hasselman, Strunk, et al., 2020).

constraints on node selection: Nodes should be quantitatively measurable factors that are non-stationary and, in principle, distinguishable from other nodes.

Explanatory boundaries

Personalized network models have *explanatory boundaries*, i.e., constraints provided by the types of explanations they can support. I argued in **Chapter 4** that network models have an exploratory function, i.e., psychometricians use them to reason about (explanatory) possibilities for psychiatric phenomena. The theoretical explanations that are part of psychometricians' background knowledge can guide and constrain this ability. We can translate this observation to clinical practice: The theoretical background knowledge of clinicians and clients can inform their reasoning with personalized network models about possibilities for mental problems. However, this requires the dependency relationships in personalized network models to be adequately *coordinated* with relevant theoretical principles. What types of theoretical explanations could coordinate with personalized network models?

The first option is that personalized network models provide evidence for *possible causal explanations* of psychiatric phenomena. Some question whether personalized network models can provide evidence for causal claims (Olthof, Hasselman, & Lichtwarck-Aschoff, 2020). The edges in personalized temporal network models provide evidence for temporal predictions or indications of Granger causality (Granger, 1969), which can be considered an approximation or potential indication of causal relationships. However, it is unlikely that personalized network models, including the temporal ones, will satisfy interventionist criteria for causality (De Boer et al., 2021; Kostić & Khalifa, 2023; Woodward, 2003). So, whether personalized network models provide evidence for possible causal explanations of psychiatric phenomena depends on which criteria for causality we require.

The second option is that personalized network models provide evidence for *possible mechanistic explanations* of psychiatric phenomena. As argued in **Chapter 2**, mechanistic explanations show how the working parts of a phenomenon are organized into a mechanism that either causes the phenomenon or constitutes a phenomenon at a higher level (for instance, how micro-physical atomic structures constitute the macro-physical property of hardness) (Bechtel, 2008; Craver, 2007). The ability of network representations to *provide* mechanistic explanations has been questioned. Craver (2016) argues that if network representations provide any explanation at all, it is a mechanistic one. However, Kostić and Khalifa (2022) claim that network representations do not provide mechanistic explanations if any of the following conditions are violated:

(1) The nodes and edges in a network model should denote the working parts of a mechanism,
(2) The explanandum should be at a higher level than the explanans, and,
(3) Topological properties should be causally responsible for the explanandum.

If we follow this reasoning, personalized network models will not provide evidence for possible mechanistic explanations of mental problems. Personalized network models will likely violate condition (1) because the variables and conditional dependencies that VAR models represent do not reflect the spatiotemporal working parts of a mechanism underlying mental disorders – their organization is merely conventional. Personalized network models will likely violate condition (3) because causation requires that causes precede their effects, and topological properties in personalized network models do not precede the phenomena they want to explain – they are simultaneous.

The third option is that personalized network models provide evidence for *possible topological explanations* of psychiatric phenomena. Network representations are particularly suited to provide (evidence for) topological explanations (Darrason, 2018; Huneman, 2018; Jones, 2014; Khalifa et al., 2022; Kostić, 2019a, 2022; Kostić & Khalifa, 2021, 2023; Rathkopf, 2018). Following Kostić (2020), network models can provide evidence for topological explanations when the topological properties of the network model and the phenomenon (or empirical pattern) it addresses are empirically adequate and have an appropriate counterfactual dependence relationship. Whether this is the case for personalized network models will be discussed in more detail in **Section 5.6**.

So, personalized network models provide explanatory constraints that influence what explanatory hypotheses about mental phenomena these models can(not) support. Explanatory boundaries can inform node selection, as the types of explanations that network models can provide evidence for depend on what the nodes and edges represent (Craver, 2016). For instance, if we argue that the ability of personalized network models to provide evidence for explanations depends on their empirical adequacy and the adequacy of the empirical patterns they address (cf. Kostić, 2022), then nodes should meet this criterion. However, representational and explanatory boundaries do not constrain the *types of variables* personalized network models can include. Here, *contextual boundaries* can play an important role.

Contextual boundaries

Personalized network models have contextual boundaries, i.e., constraints provided by the context in which a model is used. I argued in **Section 5.4** that the systemic background theory driving PCC does not adequately constrain target system

demarcation. In line with my practice-oriented account, I argue that the goals and questions driving clinical practice *can* provide such nodal constraints.

From a PCC perspective, it makes sense that practice drives node selection; ultimately, we want personalized network models to be *useful* for clinical practice. Clinical practice is inherently context-dependent and value-laden, and PCC brings these features to the fore. PCC highlights that a person can enter clinical practice with different goals: feeling better, functioning better, improving their agency, and finding the right balance between dependence and independence (of help). Moreover, PCC emphasizes that the specific goals that clinical practice should focus on may vary depending on the client, their mental problems, and context. So, to be useful for clinical practice, personalized network models should contain nodes that help clinicians and their clients address their specific clinical goals.

The PCC context could guide and constrain node selection in various ways. First, practical considerations can constrain the number of variables measured in ESM questionnaires. For instance, the clinician and client could limit the number of variables to make ESM questionnaires less burdensome (Myin-Germeys et al., 2018; Von Klipstein et al., 2020). Second, clinical goals can guide and constrain node selection. Clinicians and psychometricians recommend that node selection should depend on the specific problems and questions the client and their clinician are interested in (Bos et al., 2019; Von Klipstein et al., 2020). This means that the specific problem a client wants to address – as decided in collaboration with their therapist – or the symptoms they consider most burdensome will determine the variable selection (Bringmann et al., 2022). Various clinical considerations could be weighted in node selection. The first consideration is that the nodes should be relevant to the client and their situational context. If it is hypothesized that someone's depressive symptoms are aggravated by their stressful job, this factor should be included in the model. The second consideration is that nodes should be able to guide treatment. This consideration may limit the variables to those that could be intervened upon, for instance (Frumkin et al., 2021). The third consideration is that nodes should be able to monitor clinical development. This implies that the nodes most relevant for assessing the effectiveness of treatments (Helmich et al., 2021) or predicting the risk of relapse (Smit et al., 2019) should be included. So, contextual boundaries can guide and constrain node selection.

However, the PCC context also does not impose definitive constraints on node selection. My analysis could imply that any variable can, in principle, be included in personalized network models as long as it is *relevant* to the client and clinician. This

concern echoes a general worry that people may have about my practice-oriented account: It invokes relativism by making modeling too dependent on contingent factors such as the inquirer's background knowledge, preferences, or contingent facts about personal circumstances.[90] If we take this worry seriously, contextual constraints may limit personalized network models' ability to provide knowledge about clients' mental problems. One way to address this worry is to focus on how the PCC context can do justice to personalized network models' representational and explanatory boundaries. A clinician and client want to address various *questions* in their clinical encounter. For instance, a person may enter treatment with (one of) the following questions in mind: "How can I feel better?", "How can I function better (in different domains of functioning)?", "What can I do myself to improve my condition?" and "What kind of help do I need?". Personalized network models can help support some, but not all, clinical questions. So, one way to ensure that personalized network models provide *epistemically relevant* information in clinical practice is to be *explicit* about the clinical questions they could address. This echoes the claim that constructing (personalized) network models should be informed by clearly defined research questions and hypotheses that are of personal and clinical relevance (Bastiaansen et al., 2020; Borsboom, Deserno, et al., 2021; Bringmann et al., 2022). The clinical questions we want personalized network models to address should – at least – do justice to their representational and explanatory boundaries. In the next section, I will suggest that principles of *erotetic reasoning* could help with this endeavor.

5.6 Erotetic reasoning

Insights from *erotetic* (or perspectival) reasoning can give us more insight into the clinical questions that personalized network models could, in principle, help clinicians and clients answer. Erotetic reasoning is based on the principle that questions can be conclusions in arguments. More specifically, erotetic reasoning demonstrates how we can logically derive questions from sets of propositions – which may include hypotheses – about a model and empirical observations (Hintikka, 1981; Wiśniewski, 1996). So, we can start from a set of propositions and derive relevant questions based on the syntax (structure) and semantics (meaning) of those statements. To illustrate this, we can use a toy example inspired by Wiśniewski (1996):

90. My emphasis on the need for empirical adequacy and the fact that skills and pragmatic virtues will, by and large, be shared within a specific practice alleviates this relativist concern (cf. De Regt, 2017). This relativist worry has also been voiced for other philosophical positions that do justice to the context-dependency of modeling, such as perspectivism (Giere, 2006; Massimi, 2018a; Massimi & McCoy, 2020; Mitchell & Dietrich, 2006). Discussing perspectivism's counterarguments against these relativist allegations is beyond the scope of this thesis.

(1) If a person writes three books in one year, they are either a paid writer, exceptionally gifted, or live a monastic life.
(2) Mary writes three books in one year.
(3) Is Mary a paid writer, exceptionally gifted, or does she live a monastic life?

This example demonstrates that we can derive a relevant question – and space of possible answers – based on an observation and possible explanations for this observation. In this example, we observe that Mary writes three books in one year and come up with the question, "Is Mary a paid writer, exceptionally gifted, or does she live a monastic life?" based on the possible explanations we have of this observation (i.e., people who write three books in one year are either a paid writer, exceptionally gifted, or a live a monastic life). We do not know which of these answers holds, but we do know what question is sensible to ask given our available background knowledge.[91] Similarly, erotetic reasoning may help explicate what explanation-seeking questions personalized network models could provide evidence for. I focus on topological explanations to illustrate this claim.

What criteria should be met before personalized network models can provide evidence for (how-possibly) topological explanations of mental problems? I discussed this in **Chapter 2** and briefly in **Section 5.5**. Here, I build on these discussions using the account of topological explanations by Kostić (2020) and Kostić and Khalifa (2021, 2022). This account outlines the necessary and sufficient conditions under which a network representation provides a genuine topological explanation and does so by explicitly incorporating perspectival criteria. Kostić formulates his account as follows:

a's being *F* topologically explains why a is *G* if and only if:

(T1) *a* is *F* (where *F* is a topological property);
(T2) *a* is *G* (where *G* is an empirical property);
(T3) Had *a* been *F'* (rather than *F*), then *a* would have been *G'* (rather than *G*);
(T4) *a* is *F* is an answer to the question "Why is *a*, *G*?"

91. This example differs from more familiar examples of deductive arguments in two ways. First, whereas traditional deductive arguments derive a conclusion, which is also a proposition, this argument derives a question. Second, erotetic reasoning requires a disjunction of hypothetical propositions in the first premise, where any disjuncts could be true. The second premise specifies more closely what is the case. Based on both premises, we can derive a relevant question, which also implies a space of possible answers. For the technical details of the logic of this type of argument, see Groenendijk and Stokhof (1994), Millson (2019, 2020), and Wiśniewski (1995, 2013).

Criterion T1 states that a network representation of a system should demonstrate a specific network connectivity pattern expressed as a topological property. Criterion T2 states that the system should demonstrate an empirical property (or phenomenon), e.g., display specific behavior. Criterion T3 describes a counterfactual dependence between the topological property and the phenomenon: The phenomenon should depend on the presence of the topological property. However, if we combine these three criteria, there is still something missing: We do not yet know, based on these criteria, whether the topological property is an answer to the relevant explanation-seeking question. That is why Kostić's account provides the perspectival criterion T4: For a topological property to explain a phenomenon, the topological property should answer the relevant explanation-seeking question. This shows how asking the relevant questions makes it intelligible why some empirical property or phenomenon *G* counterfactually depends on a network connectivity pattern, which is expressed as its topological property *F*.

Let us apply these considerations to an example relevant to using personalized network models in PCC. Here, I build on the example put to the fore in **Chapter 2** regarding hysteresis and network connectivity. Various studies have examined the global topological property network connectivity (or global strength) in personalized network models to predict whether someone is vulnerable to developing or relapsing into a mental disorder. In line with the idea that mental disorders behave like complex dynamic systems (Cramer et al., 2016; Olthof, Hasselman, & Lichtwarck-Aschoff, 2020; Wichers, 2014), it is supposed that our mental health may shift from a healthy to a disordered state following perturbations. Perturbations to the healthy state may not have any effects until a tipping point is reached and the system (abruptly) shifts to a disordered state. Psychometricians have suggested that increased network connectivity may predict this transition from a healthy to a disordered state (Van de Leemput et al., 2014; Wichers et al., 2011). This hypothesis has been examined in simulation studies (Cramer et al., 2016) and in small samples of time-series data of individuals with a depression diagnosis (Wichers et al., 2011, 2020). Hence, this translates to the following how-possibly explanation: If someone has a personalized symptom network model that is more strongly connected, they are possibly more vulnerable to developing a mental disorder or relapse.

We can use Kostić's scheme to formulate what criteria should be met before we can claim that a strongly connected personalized network model provides possible explanatory evidence for a client's vulnerability to developing depression:

> The observation that person *X* has a personalized symptom network model with high network connectivity provides possible explanatory evidence for their vulnerability to developing depression if and only if:
>
> (T1) *X* has high symptom network connectivity (topological property *F* in the schema above);
> (T2) *X* is vulnerable (or has demonstrated vulnerability) to developing depression (empirical property *G* in the schema above);
> (T3) *X* would not have demonstrated such vulnerability if their symptom network connectivity had been lower;
> (T4) Symptom network connectivity is the relevant answer to the question, "Why is *X* vulnerable to developing depression?"

Can the principles of erotetic reasoning help us determine whether (T4) could be a relevant answer to this explanation-seeking question? We can assess whether the counterfactual relationship (T3) holds and combine this with the empirical observation (T2). However, combining the counterfactual relationship (T3) with the empirical observation (T2) also allows us to come up with a relevant explanation-seeking question and space of possible answers (T4). That is, it shows why it is relevant to appeal to the counterfactual relationship (T3) as a (how-possibly) explanation of the empirical observation (T2) and why appealing to other (non-)topological properties is not explanatory, i.e., why they are not included in the space of possible answers (Lange, 2018). So, erotetic reasoning can help us determine what explanation-seeking clinical questions personalized network models can address. This also suggests that personalized network models should include (types of) nodes about which clinicians and clients have specific (explanatory) hypotheses.

5.7 Conclusion

In this chapter, I examined the epistemic potential and limitations of using personalized network models in PCC from a practice-oriented perspective. Personalized network models could be tools for PCC because they are personal and personalized. The personal nature of clinical ESM data can be epistemically advantageous because it avoids standard critiques of using numerical data in the social domain. However, personalized network models' personalized and systemic nature can introduce boundary problems. I showed that the type of knowledge that personalized network models can provide – and node selection – can be constrained by their representational, explanatory, and contextual boundaries. So, these

constraints can help address the boundary problems that could arise when using personalized network models. Finally, these constraints can be used to explore what clinical questions these models are (not) able to address.

This chapter showed that considering the content of personalized network models and their context of use could help clinicians and their clients in justifying their decisions on what variables (not) to include in these models. However, even when these considerations are taken into account, construing and using personalized network models in PCC is not straightforward. For instance, one critical issue is determining the clinical and personal relevance of the dependency relationships that personalized network models demonstrate. The relevance a clinician assigns to a pattern may differ from the relevance the client assigns to it, as both may attribute different values to these findings. Indeed, this chapter has assumed that the goals of clinicians and their clients align, which is not necessarily the case. Clinical practice is messy, and while this chapter may serve as an example of how network demarcation could work in practice, its claims cannot be directly translated into clinical guidelines. At the least, this chapter emphasized the importance of making the client's context and goals explicit when using data models in clinical practice.

This chapter has not touched on one aspect of clinical practice that is crucial for how personalized network models are constructed and interpreted: clients' stories about their mental problems. In the next chapter, I will focus on this relationship between personalized network models and clients' self-narratives.

Chapter 6

Personalized network models and self-narratives

Abstract

In this chapter, I explicate the relationship between personalized network models and self-narratives in clinical practice. When we develop mental problems, we may formulate a *psychiatric narrative thread* as part of our self-narrative to explain and understand our mental problems and to help others understand them. Case studies in clinical research show that clients' self-narratives play an important role in the construction of personalized network models. Personalized network models and self-narratives demonstrate structural and functional similarities: Both can be characterized as idealized, abstract, and reflexive representations of our lived experiences that help us reason about our mental problems. However, personalized network models and psychiatric narrative threads are not epistemically equivalent. Instead, they are epistemically complementary: Psychiatric narrative threads are part of the background knowledge that guides and constrains how clients and clinicians reason with personalized network models.

6.1 Introduction

When we enter clinical practice, we may do so with specific goals in mind. We may want to feel less anxious, find ways to deal with our suicidal thoughts or learn how to cope with our debilitating insomnia. However, more primarily, we enter clinical practice as individuals with a personal story about our mental suffering, its development, and its causes. These stories can serve as the basis of the therapeutic encounter and may change throughout the clinical process: They are an important element of the epistemic context in which the clinical encounter takes place. Hence, if we want to explore the epistemic potential of personalized network models in clinical practice in practice-oriented terms, we should also focus on how these models shape – and are shaped by – clients' self-narration.[92]

The relationship between narratives and scientific models is a topic that has traditionally received relatively little interest in the philosophy of science (but see Hartmann, 1999; Morgan, 2001; Wise, 2011). Indeed, Morgan and Wise (2017) argue in the editorial of their special issue on narrative science that "philosophers of science have not given narrative much credence as having any ontological or epistemological functions and, if anything, have been deeply suspicious that it could have any

92. In this chapter, I do not want to make the normative claim that everyone who experiences mental suffering *should* engage in such self-narration. Relatedly, I acknowledge that people may differ in their self-narrating abilities.

such functions" (p. 1). Even though the epistemic value of narratives in science is increasingly recognized – as is reflected by the articles in this special issue –, the relationship between scientific models and *self-narratives* has not yet been explicated. Moreover, this relationship has not been addressed in the philosophy of personal identity literature. Finally, self-narratives are not explicitly mentioned in discussions on integrating personalized network models in clinical practice. In this chapter, I attempt to fill this gap in the literature.

This chapter will be structured as follows. In **Section 6.2**, I describe the role of self-narration in mental suffering. Developing mental problems calls for the formulation of a "psychiatric narrative thread" as part of our self-narrative; we use this narrative thread to understand our mental problems and make them understandable to others. In **Section 6.3**, I use a case study to show that clients' self-narratives play a role in constructing their personalized network models. In **Section 6.4**, I provide a practice-oriented interpretation of self-narratives, arguing that – like personalized network models – they can be characterized as idealized, abstract, and reflexive representations of our lived experiences that help us reason about mental problems. In **Section 6.5**, I characterize the relationship between self-narratives and personalized network models. Personalized network models are not epistemically equivalent to psychiatric narrative threads because they have different target system demarcations and representational boundaries. Instead, they are epistemically complementary: Clients' psychiatric narrative threads are part of the background knowledge that guides and constrains how clients and clinicians reason with personalized network models.

6.2 Psychiatric narrative threads

A narrative can be defined as an organizing structure that explains actions and events by integrating them into a meaningful and coherent pattern or sequence (Mackenzie, 2014). Indeed, a narrative is not just a list of events but "a representation of those events which is shaped, organized, and coloured, presenting those events, and the people involved in them, from a certain perspective or perspectives, and thereby giving narrative structure (…) to what is related" (Goldie, 2012, p. 2). For instance, a biography about a famous actor may include details about his upbringing, his first performance in a local theater, the time he got discovered by an agent, and the rumors in which he was subsequentially involved. This biography will not merely list these events but connect them, show how earlier experiences led to later ones, and help explain why the actor made certain life choices. The biography helps the

readers get a sense of the most important features of the actor's life and connects these features in a way that helps readers describe how the actor's life developed, explain his decisions, and understand his thoughts, feelings, and actions. Moreover, the biography influences how the readers will interpret the actor's future actions.

Narratives also play an important role in how we relate to our own lives. *Self-narratives* are self-referential narratives that explain and integrate autobiographical events and experiences – such as our thoughts, feelings, actions, and context – into a meaningful and coherent pattern or sequence (Fabry, 2023; Smith & Watson, 2010).[93] Self-narratives include the texts we write about ourselves, for instance, in our diaries or memoirs, and how we spontaneously describe our lives in conversations with others (Fabry, 2023). The content of our self-narratives may differ depending on the format and context. For instance, a written self-narrative is likely to be more thorough and reflective than a spontaneously generated verbal self-narrative, and the self-narratives we share with our colleagues will differ from those we share with our therapist. In practice, a self-narrative refers to a collection of interweaving narrative threads. We use these threads to "interpret and make sense of our past histories, project ourselves into the future via plans and intentions and make sense of our actions, emotions, desires, beliefs, character traits and relations to others" (Mackenzie, 2014, p. 158). Our self-narratives can help us explain and understand ourselves, prescribe how we see ourselves, and help others understand us better.

Our self-narratives may change profoundly when we experience disrupting life events (Schechtman, 2007). An example of a life event that can significantly impact our self-understanding is developing mental problems. We may have previously considered ourselves someone who can handle stressful situations well, whereas the slightest grievance now upsets us. We may doubt whether we want to keep on pursuing the career we have envisioned since childhood. We may re-evaluate the values our parents taught us. Indeed, developing mental problems can strongly influence our self-narratives (Dings & De Bruin, 2022; Mackenzie & Poltera, 2010; McConnell & Snoek, 2018).[94] Frank (1995) argues in *The Wounded Storyteller* that developing an illness

93. In **Chapter 7**, I will discuss in greater detail what "coherence" and "meaningfulness" entail in self-narration.

94. Our self-narratives are also shaped by receiving a *psychiatric diagnosis*. Indeed, Hacking (1996) argues that receiving a psychiatric diagnosis is to "change how we can think of ourselves, to change our sense of self-worth, even how we remember our own past" (p. 369). Since most people who enter clinical practice will receive a psychiatric diagnosis, their mental problems and psychiatric diagnoses will intertwine in their psychiatric narrative threads. In this chapter, however, I will focus on mental problems irrespective of their associated diagnoses for simplicity and because the use of personalized network models in clinical practice, especially when used as or in conjunction with clinical case conceptualizations, could serve as an alternative to psychiatric diagnoses.

can cause "wreckage" to our self-narration: The "present is not what the past was supposed to lead up to, and the future is scarcely thinkable" (p. 55). Something similar can be said for developing mental problems. Frank suggests that we can "repair" such "narrative wreckage" by formulating a new narrative thread that relates to, describes, and makes sense of our illness, i.e., an *illness narrative*. Similarly, developing mental problems may call for a *psychiatric narrative thread* that describes and makes sense of our mental problems, how they developed, and how they are situated in our broader self-narrative (Mackenzie & Poltera, 2010; Phillips, 2003). Formulating this narrative thread can help us make sense of our recent confusing, painful, and challenging experiences, how they relate to our past, and what they imply for our future. So, we can use psychiatric narrative threads to explain and understand our mental problems and to help others understand them better.

Depending on our personal characteristics, the specific problems we experience, and our context, this psychiatric narrative thread may be more or less tightly interwoven with the other threads in our self-narrative. Our mental problems may not always be the most central elements of our lives that we want to explain to or share with others. However, one context in which this psychiatric narrative thread strongly comes to the fore is clinical practice. In the next section, I will show how self-narratives and these psychiatric narrative threads are reflected in personalized network models in clinical practice.

6.3 Self-narratives and personalized network models: A case study

The self-narratives and psychiatric narrative threads with which clients enter clinical practice can play an important role in the therapeutic encounter. One clinical tool that actively uses clients' self-narration is *case conceptualization*. Case conceptualizations, or case formulations, are clinical tools that include descriptions of a client's current situation in treatment, hypotheses about why their psychological problems came about, and a possible plan to address them (Sim, 2005). Recently, *network-based case conceptualizations* have been developed to map and trace the development of a client's mental problems, some of which involve collecting personalized quantitative data over time.[95] Examples include *pattern diagnostics* ("patroondiagnostiek," Patterns of Life,

95. Network-based case conceptualizations are not synonymous with personalized network models, i.e., statistically estimated VAR models based on time-series data. Indeed, "network-based case conceptualization" is an umbrella term that also includes methods that do not involve the collection of quantitative personalized data over time, such as the *network intake* ("netwerk intake," PsyNet, 2020) and *perceived causal networks* (Frewen et al., 2012, 2013; Klintwall et al., 2023).

2024) and *ideographical system modeling* (Olthof et al., 2024; Schiepek et al., 2016; Van den Bergh et al., 2024). While these approaches differ in their data collection methods and the empirical patterns they focus on, the nodes and edges in these network-based case conceptualizations both reflect symptom-related and salutogenic variables relevant to a client's personal *story* and current situation. Indeed, the pattern diagnostics method explicitly mentions that these case conceptualizations are a means to "map" and "clarify" the stories that clients have about their mental problems and their ideas about recovery (Patterns of Life, 2024). So, network-based case conceptualizations are explicitly linked to clients' self-narratives and their psychiatric narrative threads.[96]

These self-narrative elements also come to the fore when we look at how personalized network models, discussed in detail in **Chapter 5**, are construed. To illustrate this, I use Von Klipstein et al. (2023) as a case study. Von Klipstein et al. (2023) examined the efficacy of integrating a personalized ESM module in psychotherapy, exploring whether this module and case conceptualizations could support each other. Von Klipstein et al. (2023) implemented this module for George, a 27-year-old man diagnosed with depression. George had been suffering from depressive symptoms since his adolescence and had tried therapeutic help on multiple instances previously; he started outpatient cognitive behavioral therapy when he was enrolled in the study. At the start of the study, the researchers got acquainted with George's life story by interviewing him about his strengths and factors contributing to his resilience and reading his intake reports, referral letters, and biography.[97] Based on this information, the researchers highlighted, amongst others, that George struggled with low self-esteem, that the loss of a loved one significantly impacted his upbringing, and that he experienced problems reconciling his faith with his homosexuality. George's life story gave the researchers a sense of what personalized variables they should include in George's ESM questionnaire (i.e., diary). The researcher brought this list of suggestions to a treatment session, where George, his therapist, and the researcher decided together on the personalized variables George's ESM diary should include. Amongst others, these personalized variables had to cover factors that maintained or attenuated George's depression. Ultimately, George's ESM diary included personalized items related to his self-esteem, acting open versus being withdrawn, feelings of losing control, engagement with faith, connectedness, and playing video games. George had to fill out his personalized ESM diary five times

96. A different, albeit related, goal of network-based case conceptualizations and personalized network models is to help clinicians guide treatment decisions and obtain information about their clients. I will touch upon the clinician's role in **Section 6.5**, but a detailed account of *clinical understanding* is beyond the scope of this thesis.

97. Von Klipstein et al. (2023) do not explicate these steps for George specifically, but describe them as part of the standard protocol.

a day for eight weeks. After two, four, and eight weeks, George received personalized feedback based on his diary via two interactive data models: (1) a visualization of the raw data showing how he answered the items over time and (2) a contemporaneous network model that showed the associations between the variables.[98] Amongst others, George's contemporaneous network model showed that his responses to items related to his self-esteem – i.e., "Since the previous beep, I felt judged by others," "Since the previous beep, I felt insecure about my capabilities," "Since the previous beep, I felt that I had no control" – were conditionally dependent on each other. In other words, George's feelings of judgment by others, insecurities about his capabilities, and lacking control frequently co-occurred.

This case study illustrates that personalized network models intimately relate to clients' self-narratives and psychiatric narrative threads specifically. Indeed, the researchers wanted to include the aspects of George's lived experience that were most relevant to his lived experience in the ESM diary; they obtained these items by interviewing George about his life and reading his biography. Hence, George's self-narrative served as input for the personalized network model. However, this case study does not tell us how the epistemic function of personalized network models relates to the epistemic function of clients' psychiatric narrative threads. In the next section, I will describe the functional and structural similarities between self-narratives and personalized network models.

6.4 A practice-oriented interpretation of self-narratives

Drawing similarities between scientific and non-scientific means to represent and inquire about the world originates in the naturalist epistemological tradition set by Quine (1969) (Antony, 2016). This tradition states that scientific and non-scientific means of inquiry may lie on a continuum, with scientific inquiry being more rigorous and systemic and requiring specific skills. Relatedly, some philosophers of science argue that the distinction between scientific and non-scientific representations is circumstantial: The only difference is that scientific representations are representations used or developed by scientists (e.g., Callender & Cohen, 2006). For instance, Knuuttila (2021b) highlights similarities between scientific models and narrative fiction, emphasizing that both are constructed and function like self-contained worlds. In line with my practice-oriented account, I claim that self-narratives can be conceptualized as *tools*, i.e., idealized, abstract representations

98. For a visual overview, see Von Klipstein et al. (2023, Appendix B). Given this chapter's focus on personalized network models, I do not discuss the observations based on the raw data visualizations.

that people can use to reason about their lived experiences. I defend this claim in this section by highlighting the structural and functional similarities between self-narratives and personalized network models. While some of these similarities may be trivial, explicating them will help – and justify – applying insights from the philosophy of modeling to psychiatric narrative threads later in this chapter.

The first similarity between self-narratives and personalized network models is that both are *abstract* and *idealized* representations of our lived experiences. Like network models, self-narratives are not direct reflections of our lived experience. The latter is too vast, complex, and multifaceted to be captured in all its detail in narrative form, and some features of our "life-world" may not even be expressible linguistically. Instead, our self-narratives omit and misrepresent our lived experience; they are "incomplete and selective by nature" (Tekin 2015, p. 190).

The second similarity is that both can be judged, in part, based on their *empirical adequacy*. Like network models, self-narratives should have a bearing on the observable world. Schechtman (1996) argues that self-narratives should meet a "reality constraint," i.e., "exhibit a fundamental grasp of what the world is like" (p. 83). Tekin (2011) states that our self-narratives should reflect different aspects of our lives and not only be a product of wishful thinking or our deprecatory attitude. Leuenberger (2020) argues that authentic self-narratives should accurately represent relevant "objective facts" or verifiable features about oneself (p. 659). However, as for personalized network models, this criterion should not be interpreted too strictly. Schechtman (1996) argues that the reality constraint does not imply "that a narrative must be totally accurate in every regard or contain no trivial mistakes" (p. 83) – such a constraint is impossible to meet. Indeed, self-narratives depend on episodic remembering, whose constructive rather than reconstructive nature is increasingly recognized (Fabry, 2023), and people are shown to confabulate explanations or reasons in their self-narration (e.g., Johansson et al., 2005; Nisbett & Wilson, 1977). So, self-narratives only have to be "weakly factive," i.e., they should conform to – or at least not contrast – our lived experience (cf. Leuenberger, 2021).

The third similarity is that both owe their *epistemic potential* not only or primarily to their epistemic adequacy but to their abstraction and idealization of our lived experiences. First, a self-narrative that is too focused on accurately describing our lived experiences is not in a good position to help us understand our thoughts, feelings, and actions, nor to help others understand them. Indeed, Schechtman (1996) states that the reality constraint has to be balanced with the "articulation constraint," i.e., "the narrator should be able to explain why he does what he does, believes what he believes,

and feels what he feels" (p. 114). So, a "good" self-narrative is a balancing act between narrating our lived experience in an empirically adequate manner and leaving out or idealizing aspects of our lived experience. Second, *how* we abstract and idealize our lived experience in our self-narratives, i.e., what specific elements and connections we highlight, influences what patterns of our lived experience become salient. Hence, the specific abstractions and idealizations of our lived experiences we incorporate in our self-narratives influence how we reason about our past, present, and future.

Finally, personalized network models and self-narratives demonstrate *reflexivity*. Self-narratives *reflect* our lived experiences but also *prescribe* how we reason about ourselves. To formulate a self-narrative, we should articulate (1) what events, experiences, and actions have happened in our life, (2) which of these were most important, and (3) how they are meaningfully connected. In the process of such articulation, we also make specific patterns salient. This can make us interpret our thoughts, feelings, and actions in ways consistent with our pre-established self-narrative. A different, albeit comparable form of reflexivity is present in personalized network models, i.e., *reactivity* or an individual changing their attitudes and behavior in response to being measured. Indeed, people may change their behavior in response to filling out daily ESM questionnaires and receiving feedback based on their personalized network models. To illustrate, let us imagine a scenario in which a person fills in a daily ESM questionnaire to explore their sleep quality.[99] Every day, they must answer the question "Did you sleep well?" using a Likert scale from 1 ("not good") to 5 ("very good"). At the start of the ESM trajectory, they say that their sleep is "average" (3) when they lay awake for two hours at night. Their reason for doing so is that they have always been poor sleepers, so to them, laying awake for two hours is just an average night. After filling out the ESM questionnaire for a couple of weeks, they discuss the results with their therapist. They are shown a personalized contemporaneous network model demonstrating a strong conditional association between their sleep quality, concentration problems, and anxiety. This prompts the therapist to discuss the role of the client's sleep on their mood. The ESM measurement, the data model, and the subsequent conversation with their therapist could have three different effects on their future behavior and ESM measurements. In scenario #1, the client takes active measures to *improve* their sleeping hygiene and, in turn, scores higher on average on the sleeping quality questionnaire. In scenario #2, the client *re-evaluates* how they grade their sleep quality: Instead of grading their sleep based on how they sleep on average, they grade their sleep based on whether they feel rested the following day. Finally, in scenario #3, the client changes what it *means* for them to sleep well. They may realize that the grade they give to their sleep

99. This example is taken from De Boer and Runhardt (2024).

quality should be determined by whether they woke up at night and went to bed later than average. This example demonstrates how, like self-narration, (the construction of) personalized network models both reflects and shapes our behavior.[100]

So, self-narratives can be conceptualized as idealized, abstract, and reflexive representations of our lived experiences that help us reason about our lives. We can also apply this characterization to our psychiatric narrative threads: They are idealized, abstract, and reflexive representations of our mental suffering that help us reason about our *mental problems*. Based on this description, we could argue that our psychiatric narrative threads and personalized network models share structural and functional similarities and similar *content*. In the next section, I will sketch how personalized network models and our psychiatric narrative threads are related.

6.5 The relationship between self-narratives and personalized network models

In this section, I discuss two ways to characterize the epistemic relationship between personalized network models and psychiatric narrative threads: *epistemic equivalence* and *epistemic complementarity*. Building on the case study described in **Section 6.3**, I explain why epistemic equivalence is an inadequate characterization and explore the complementarity relationship in greater detail.

If we claim that personalized network models and psychiatric narrative threads are *epistemically equivalent*, we assume that the former are quantitative data re-representations of the latter. In other words, they provide similar information, albeit in different ways. This epistemic equivalence interpretation can follow from

100. These scenarios could reflect different types of reactivity. For instance, Runhardt (2021) distinguishes, amongst others, between *alpha*, *beta*, and *gamma* reactivity. Scenario #1 provides an example of *alpha reactivity*, i.e., a change in the numerical outcome of a measurement due to the individual being measured, not due to a reinterpretation of the phenomenon that is measured (Golembiewski et al., 1975). Scenario #2 provides an example of *beta reactivity*, i.e., a change in the numerical outcome of a measurement due to a change in how a person interprets the scale they have been using (cf. Fabian, 2022; Golembiewski et al., 1975). Scenario #3 provides an example of *gamma reactivity*, i.e., a change in the numerical outcome of a measurement due to a change in how a person characterizes the phenomenon being measured (cf. McClimans et al., 2013). Alpha reactivity does not necessarily challenge the ecological validity of psychological measurement, but beta or gamma reactivity could: Because the client has "recalibrated" the Likert scale and/or reconceptualized the variable in question, we do not know whether the client's behavior really "improved or worsened" over time (cf. Hacking, 1996, 2007). For a more elaborate discussion on different types of reactivity, see Runhardt (2021) and Marchionni et al. (2024).

the observation that personalized network models and psychiatric narrative threads use *comparable* abstractions and idealizations. Our psychiatric narrative threads can be redescribed as collections of factors that have played an important role in the development of our mental problems – i.e., nodes – and the (causal) relations between these elements, i.e., edges. However, important differences exist in *what* and *how* personalized network models and psychiatric narrative threads represent mental problems. First, personalized network models have a more circumscribed target system than psychiatric narrative threads. We want our psychiatric narrative threads to represent factors important to developing our mental problems as temporally extended, historical beings. For instance, they may reflect a significant life event in our childhood – as was the case for George – that we believe to play an important role in the mental problems we currently experience. Our personalized network models represent a more circumscribed target system, representing those features of our lives that *currently* matter for our mental suffering. Although personalized network models can accommodate a temporal dimension, this dimension only extends to a few weeks or months – eight weeks in George's case. So, psychiatric narrative threads and personalized network models demarcate their target systems differently. Second, personalized network models and self-narratives have different representational boundaries. As stated in **Chapter 5**, the representational boundaries of personalized network models allow them to include a limited number of quantitatively measurable and non-stationary features of our lived experience. In contrast, self-narratives and their psychiatric narrative threads are much less constrained in their content. So, although psychiatric narrative threads and personalized network models share many similarities, they are not equivalent.[101] Again, this observation may not be controversial, but it is important as a starting point for conceptualizing how they differ more precisely.

Instead, I defend the position that personalized network models and psychiatric narrative threads are *epistemically complementary*: Psychiatric narrative threads are part of the *background knowledge* that guides and constrains how clients and clinicians reason with personalized network models. This is compatible with Currie and Sterelny (2017), who claim that narratives and formal models complement each other in historical science.[102] It also echoes my earlier claims about network models in scientific practice, i.e., that the exploratory function of network models depends

101. Another difference is that our psychiatric narrative threads have an explicit normative dimension, as I will highlight in **Chapter 7**.

102. Morgan (2001) also claims that narratives are integral to mathematical modeling but focuses on how narratives provide the questions that mathematical models can help address. In my account, psychiatric narrative threads play a more substantial role: They are actively involved in constructing and interpreting personalized network models.

on scientists' background knowledge (see **Chapter 4**) and that the hypotheses that personalized network models afford depend on their coordination with specific explanatory strategies (see **Chapter 5**). Let us go back to the case of George, whose personalized network model demonstrated the frequent co-occurrence between feeling judged by others, feeling insecure about his capabilities, and having a sense of no control. In their study protocol, Von Klipstein et al. (2023) describe how therapists could use ESM data patterns as starting points for further explorations and interpretations of the data in clinical practice, for instance, by asking their clients whether they expected these patterns or how they would explain them. So, we can imagine a scenario in which George was asked by his therapist to explain why or how the self-esteem-related dependency relationships in the network model may have come about, whether this empirical pattern reflects his lived experience, and what he wishes to do with this information. To answer these questions, George would have resorted, first and foremost, to his self-narrative and its psychiatric narrative threads more specifically.

This complementarity of personalized network models and psychiatric narrative threads shows that clients' self-narratives can determine which of the possibilities these models afford are *relevant*. In **Chapter 4**, I claimed that the possibilities for psychiatric phenomena that psychometricians can reason about are *epistemically relevant* if they are *physically conceivable*, i.e., consistent with psychometricians' available empirical and theoretical background knowledge. Similarly, we can argue that personalized network models help clients and clinicians reason about epistemically relevant possibilities for the client's mental problems if these possibilities are consistent with the client's psychiatric narrative thread.[103] Let us imagine that George would not at all recognize the co-occurrence of these self-esteem-related features in his lived experience. In that case, the possibility that these features play an explanatory role in his depression would be "physically inconceivable" to him, or, put less strongly, it would not be considered a relevant or serious possibility.

However, this analysis does not yet show how personalized network models could provide clients with new knowledge or "insights" about themselves. Indeed, I argued in **Section 6.2** that self-narratives and their psychiatric narrative threads are involved in constructing personalized network models. How can personalized network models provide clients with "new" knowledge if their self-narratives determine the construction *and* interpretation of these models? It is possible that clients only obtain "new" knowledge about their mental problems via the standardized ESM diary items,

103. Note that the empirical patterns in personalized network models that become salient to the clinician and client are also influenced by their skills in interacting with these models.

i.e., those that are not personalized. However, Von Klipstein et al. (2023) describe that George's self-esteem-related ESM findings – part of his personalized items – "led to an in-depth exploration of his thinking patterns and social interactions" during the feedback sessions (p. 1). So, this claim does not hold. Instead, I suggest three possible, not mutually exclusive, ways that personalized network models can provide "new" knowledge about clients' mental problems. First, personalized network models can make certain dependency relations between elements of our mental suffering *salient* because of their pragmatic virtues, e.g., their visualizability (see **Chapter 4**). George stated in his reflection on the module that

> [T]opics that came up were not completely new to him, but that the ESM feedback results were in line with and helpfully confirmed his thoughts about himself and his depression ('functioned like a mirror'). This helped him to communicate his thoughts and feelings to his therapist, something he had struggled with before. Overall, George became more aware of the complaints he was struggling with. (p. 8)

The pragmatic virtues of personalized network models may have contributed to this process. Second, *clinicians' background knowledge* can inform clients' reasoning with personalized network models. As highlighted in **Chapter 5** and demonstrated in the case study by Von Klipstein et al. (2023), the content and interpretation of personalized network models are shaped by a collaborative effort between the client and clinician(-scientist). So, personalized network models can function as interactive tools that allow clinicians and clients to connect and negotiate relevant background knowledge related to the client's mental problems. For instance, clinicians' background knowledge may include, amongst others, their theoretical knowledge or earlier experiences with clients facing similar mental problems. Third, personalized network models may afford possibilities that are not present in a client's self-narration but which the client nonetheless believes *could* be possible based on their *background theories*. We can imagine a scenario where George may not have been aware that he often feels judged by others at the same time when he feels that he has no control – this is not a pattern that he had registered before. However, he starts paying attention to the moments when he feels judged by others and realizes that he often feels a lack of control in those situations.[104] So, patterns in personalized network models may highlight elements of clients' lived experiences that have not come into focus thus far. Indeed, whether clients consider a possibility regarding their mental suffering as physically conceivable to them can come in degrees: Is it

104. Discussing the moral implications of this influence is beyond the scope of this thesis.

conceivable given their particular backstory, their lived experiences which they have not yet explicitly reflected on, or their beliefs about how people generally function?

So, self-narratives and their psychiatric narrative threads specifically are epistemically complementary to personalized network models: They are part of the background knowledge that guides their interpretation, alongside clinicians' background knowledge and clients' background theories. Moreover, personalized network models can make patterns in clients' lived experiences salient to them via their pragmatic virtues.

6.6 Conclusion

In this chapter, I explored the relationship between personalized network models and self-narratives, highlighting their epistemic similarities and differences. I showed that personalized network models and the psychiatric threads of clients' self-narratives both play a valuable epistemic role in clinical encounters. By casting self-narratives in practice-oriented terms, I further highlighted their similarity with personalized network models. I concluded that self-narratives and their psychiatric narrative threads specifically are part of the background knowledge that guides and constrains how clients reason with personalized network models. Personalized network models could make specific patterns of a client's mental suffering salient due to their pragmatic virtues. They could also bring together different types of background knowledge – beyond clients' self-narratives – that help guide clients' reasoning with these models.

This initial exploration of the relationship between personalized network models and self-narratives could be a starting point for future inquiries. First, it would be worthwhile to (systematically) examine whether my hypotheses about the epistemic value of personalized network models for clients' self-understanding resonate with clients' and clinicians' experiences. Moreover, this chapter can serve as a starting point for bringing together discussions in the philosophy of science – and the philosophy of modeling, specifically – and the philosophy of personal identity, both within and outside the psychiatric context.

Finally, one type of background knowledge that I did not focus on in this chapter – but which could influence our reasoning with personalized network models *and* our psychiatric narrative threads – is *psychiatric theories*. One could imagine that clinicians justify their use of personalized network models in clinical practice using psychoeducation about systemic theories of mental disorders, perhaps even about

the network theory. I have touched upon the relationship between the network theory and network models in previous chapters. This chapter has highlighted the similarities between network models and self-narratives, but focusing on the influence of psychiatric theories – including the network theory – on the psychiatric narrative threads of people with mental problems requires a separate treatment. Indeed, the relationship between psychiatric theories and self-narratives reflects an interplay between the content of psychiatric theories, their normative function, social embedding, and our self-understanding. Although clinicians may promote certain psychiatric theories in their clinical practice, the self-relating of people with mental problems goes beyond the boundaries of clinical practice and steps into the social world. Hence, in the third part of this thesis, I will focus on the final practice in which the network approach plays a role: the *daily lives* of people who experience mental problems.

Part III
Daily life

Chapter 7

Psychiatric theories and self-narratives

Abstract

In this chapter, I explore how psychiatric theories influence our psychiatric narrative threads, using the biomedical model of depression as a case study. Dominant psychiatric theories, such as the biomedical model, are comparable to *master narratives*, providing scaffolds for our psychiatric narrative threads by prefiguring causal content and influencing how these narrative threads are judged by ourselves and others. Incorporating psychiatric theories in our psychiatric narrative threads can influence their narrative coherence and their ability to help us imagine possibilities for reducing our mental suffering. To assess the normative implications of these influences, we should zoom out to the broader social context in which our self-narration occurs.

7.1 Introduction

In the third part of my thesis, I will focus on the epistemic potential of psychiatric approaches in *daily life*, i.e., how people with lived experience use psychiatric approaches to make sense of their mental problems. Specifically, I will discuss how people use *psychiatric theories* to make sense of their mental suffering. As I discussed in **Chapter 6,** developing mental problems calls for the formulation of psychiatric narrative threads as part of our self-narrative. The psychiatric theories that scientists, clinicians, or the media postulate to explain the development of our mental problems can influence these narrative threads (Tekin, 2015). The idea that psychiatric theories can influence our psychiatric narrative threads is uncontroversial in the philosophy of personal identity literature (e.g., Hassall, 2020; Leuenberger, 2021; Tekin, 2011). However, there is no systematic account of *how* psychiatric theories do so. In this chapter, I explore the influence of psychiatric theories on our psychiatric narrative threads in daily life from a practice-oriented perspective. This starting point has two implications. First, I consider psychiatric theories as *tools* that people can use to make sense of their mental suffering. This implies that psychiatric theories can fulfill specific *functions* in our psychiatric narrative threads. Second, our self-relating in daily life does not happen in a vacuum but is embedded in a *sociocultural context*. Hence, exploring the influence of psychiatric theories on our psychiatric narrative threads requires considering the social embedding and normative function of both.[105]

105. The disclaimer in **Footnote 92** also applies to the chapters in this part of my thesis. Moreover, I do not want to make the normative claim that people *should* incorporate psychiatric theories into their psychiatric narrative threads. Instead, I address the function that psychiatric theories *could* serve in our self-narratives. I focus on self-narratives rather than other forms of self-relating because psychiatric theories primarily influence our *reflexive* self-understanding, which includes our self-narration.

To illustrate my claims, I use the biomedical model of depression as a case study. As argued in **Chapter 1,** the biomedical model explains the co-occurrence of depressive symptoms by claiming that they have a shared, underlying (neuro)biological cause.[106] The rationale for focusing on the biomedical model of depression is that its relationship to self-understanding has been extensively studied, in contrast to the network theory (with Meier et al. (2022) being a notable exception). Quantitative research has explored the relationship between presenting the biomedical model in psychoeducation and the self-perception of people with a depression diagnosis (Deacon & Baird, 2009; Kemp, 2014; Lebowitz et al., 2013). Qualitative research has explored how people with a depression diagnosis incorporate the biomedical model in their psychiatric narrative threads (Kangas, 2001; Laegsgaard et al., 2010; Lafrance, 2014; Ridge & Ziebland, 2006; Schreiber & Hartrick, 2002). Conceptual research has explored the normative implications of biomedical explanations on our self-narration, both inside and outside the psychiatric context (Hassall, 2020; Leuenberger, 2021; Postan, 2016; Tekin, 2011). So, using the biomedical model of depression as a case study allows me to ground my conceptual claims empirically and embed them in a broader philosophical discussion. This analysis will serve as a basis for further analysis of the influence of the network theory on our psychiatric narrative threads in **Chapter 8**.

The chapter will be structured as follows. In **Section 7.2**, I explicate the (social) influence of "dominant" psychiatric theories on our psychiatric narrative threads by drawing an analogy between the biomedical model of depression and master narratives. Dominant psychiatric theories scaffold our psychiatric narrative threads by prefiguring their content and how they are judged by ourselves and others. In **Section 7.3**, I argue that psychiatric theories prefigure causal content, including causal elements, relations, and metaphors, for our psychiatric narrative threads. In **Section 7.4**, I explore the influence of psychiatric theories on the narrative coherence of our psychiatric narrative threads. The biomedical model can fill causal gaps in our psychiatric narrative threads but may reinforce the value of narrative coherence as a social norm and overrule more meaningful ways to provide narrative coherence. In **Section 7.5**, I explore the influence of psychiatric theories on our ability to imagine possibilities for reducing our mental suffering. Adopting the biomedical model in our psychiatric narrative threads could hamper this ability. However, the normative implications of this influence will depend on our forward- or backward-looking focus and the perceived changeability of biological factors.

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106. In this chapter, "biomedical model" refers to genetic *and* neurobiological theories of depression unless indicated otherwise. Qualitative research shows that both theories have a comparable influence on the self-understanding of people with a depression diagnosis (Laegsgaard et al., 2010).

7.2 Master narratives

Our self-narratives are personal reflections on our lived experience, but they are also inherently social: They are shaped by the people in our surroundings and the broader sociocultural context. As Flanagan (1994) claims, "our selves are multiply authored" (p. 141). This social dimension of our self-narration comes to the fore when we experience personal suffering. For instance, when we become ill, we have to share our illness narrative with clinicians to receive treatment, with our boss to explain our leave of absence, or with our family and friends to help them make sense of our experiences. Frank (1995) states that we formulate stories about our illness *because* our surroundings will ask for these stories; we shape the content of our illness narratives accordingly. Specifically, Frank (1995) argues that "[f]rom their families and friends, from the popular culture that surrounds them, and from the stories of other ill people, storytellers have learned formal structures of narrative, conventional metaphors and imagery, and standards of what is and is not appropriate to tell" (p. 3). A similar interplay between personal reflections and social influences is at play when we suffer mentally: Our psychiatric narrative threads are also inherently social. Kangas (2001) claims that the psychiatric narrative threads of people with a depression diagnosis are shaped via *cultural contextualization*, i.e., "the use and contemplation of shared cultural knowledge" about their diagnosis (p. 77). Popular, widely adopted psychiatric theories are part of the "shared cultural knowledge" that influences our psychiatric narrative threads (Tekin, 2011, 2015). To explicate the (social) influence of such "dominant" psychiatric theories on our psychiatric narrative threads, I draw an analogy between the biomedical model of depression and *master narratives*.

Master narratives are dominant narratives in a given community or society (Lindemann, 2014; McLean et al., 2023; McLean & Syed, 2015). They are widely shared, (historically) promoted by authorities such as political, scientific, medical, cultural, and religious institutions, and persist despite counterevidence (Lindemann, 2014). Master narratives exist about, amongst others, relationships, gender, normality, and the good life. An example of a master narrative is the "relationship escalator." The relationship escalator refers to the narrative dominant in Western societies that romantic relationships progress linearly from dating to the relationship becoming official, moving in together, buying a house, getting married, and having children. This narrative is widely shared within our popular culture, is (historically) promoted by political and religious institutions, and persists even though many romantic relationships do not develop this way.

Master narratives and the biomedical model have been compared previously (cf. France et al., 2007; Hoffman & Hansen, 2017; Lafrance, 2014; Tekin, 2011). Indeed, if we take the biomedical model of depression as a case study, we see that it shares many characteristics with master narratives. First, the biomedical model of depression is widely shared in our society, as discussed in **Chapter 1**. The biomedical model is part of our Western "folk psychiatry," i.e., how we generally think and talk about mental disorders (Haslam, 2003, 2005). Second, authorities, including clinicians and scientists, promote the biomedical model. Third, the biomedical model of depression persists despite counterevidence. For instance, the accuracy of the chemical imbalance hypothesis of depression (see **Chapter 1**) has been questioned for years (e.g., Hindmarch, 2002), but a recent meta-analysis that provided strong evidence against this theory still sparked a media uproar (Moncrieff et al., 2022; Moncrieff & Horowitz, 2022).

Moreover, master narratives and the biomedical model of depression fulfill comparable functions in the social domain. Master narratives have a *descriptive* function: They provide general descriptions of social phenomena. For instance, the relationship escalator narrative describes how romantic relationships progress in most cases in Western society. However, they also have a *normative* function: They serve as a "ready-made option for how to construct a meaningful and productive life within a society" (McLean & Syed, 2015, p. 325). For instance, the relationship escalator narrative describes what progression of romantic relationships is considered "normal" or "desirable" in Western society and, conversely, that deviations from this narrative are "abnormal" or "undesirable." The biomedical model of depression similarly fulfills a descriptive function – providing a general explanation of depression – and a normative function – shaping how people with a depression diagnosis are perceived.

Master narratives structure how we (wish to) lead our lives, implying that they will also – implicitly or explicitly – structure the stories we hold and share about our past, present, and future lives. Indeed, master narratives *scaffold* our self-narratives (cf. Bruner, 1990). First, master narratives can prefigure *content* for our self-narratives, thereby making certain aspects of our lived experience more salient (Fabry, 2023). For instance, if we want to describe the history of our romantic relationships, we may resort to the "big events" that the relationship escalator narrative prefigures to structure our story. Second, master narratives can influence how our self-narratives are *judged* by ourselves and others. For instance, describing our romantic relationships using the relationship escalator narrative makes our self-narration more recognizable and understandable to others. Moreover, master narratives can exemplify the "ideal" self-narrative. If the story about our romantic relationships

does not match this ideal, either because our relationships did not develop this way or because we decide to focus our self-narration on other elements, this *signifies* something. So, master narratives prefigure the content of our self-narratives and how our self-narratives are judged.[107]

This observation gives us insight into how dominant psychiatric theories, such as the biomedical model of depression, could influence our psychiatric narrative threads. Dominant psychiatric theories could *scaffold* our psychiatric narrative threads. Indeed, Schreiber and Hartrick (2002) describe how difficult it is for the people with a depression diagnosis they interviewed to tell their self-narrative outside of the biomedical model narrative. Specifically, psychiatric theories can prefigure the *content* of our psychiatric narrative threads and how these narrative threads are *judged* by ourselves and others; these influences are closely intertwined. In the remainder of this chapter, I will explicate this influence in more detail. In the next section, I will focus on the specific content that psychiatric theories prefigure.

7.3 Causal information

Psychiatric theories can influence the *content* of our psychiatric narrative threads by prefiguring specific information about mental problems and thereby making certain elements related to our mental suffering salient. In this section, I show that psychiatric theories prefigure a specific type of content, i.e., *causal information*.

Causality plays an important role in narratives. Indeed, narratives show how events are connected (see **Chapter 6**), and these connections are often interpreted causally – even when causality is not explicitly asserted. For instance, Adams (1989) claims that narratives have explanatory power due to the

> assumption that the narrator uses the principle of causality to link his narrative events together, for without such a principle there is no way he could go about choosing one event rather than another from all the events

107. Note that we can form *alternative narratives* to reject and challenge master narratives if they do not align with our lived experiences or social norms (McLean et al., 2023; McLean & Syed, 2015). Rejecting a master narrative that no longer fits us can provide a sense of empowerment (McLean et al., 2023) and may be necessary for maintaining our personal identity, agency, and self-respect (Lindemann, 2014). However, it may also have adverse social consequences. Moreover, since alternative narratives are formed against the backdrop of existing master narratives, we cannot entirely escape the (normative) influence of master narratives on our lived experience.

> stored in his memory. (p. 149) So, narratives include those elements that we deem *causally relevant* to the story we want to tell.

Causality also plays an important role in our psychiatric narrative threads. Our psychiatric narrative threads consist of those events, experiences, actions, character traits, and other (autobiographically remembered) elements we deem relevant for (the development of) our mental problems; most connections between those elements are of an (implied) causal nature. Specifically, we often present our psychiatric narrative threads as *causal explanations* for our mental problems. Williams (1984) claims that the question of genesis – "What caused the illness and why did it happen to me?" – is an integral part of any illness narrative. Similarly, Kangas (2001) observed that most psychiatric narrative threads of people with a depression diagnosis share "an account of what went awry in the subjects' lives and subsequently caused depression" (p. 81). These psychiatric narrative threads were usually "organized and shaped by an explanation of the individual etiology of depression, forming a storyline" around, for instance, the shortcomings of their childhood or symptom-provoking factors in adulthood (p. 80). These examples show that our psychiatric narrative threads are, at least partly, structured to answer the question, "Why me?".

Qualitative research shows that people with lived experience integrate the causal information provided by psychiatric theories, such as the biomedical model, into their psychiatric narrative threads (e.g., Kangas, 2001; Laegsgaard et al., 2010; Lafrance, 2014; Ridge & Ziebland, 2006; Schreiber & Hartrick, 2002). For instance, a participant of Kangas (2001) states the following about her depression: "I think that it is almost like a primitive instinct. It's in the genes, this sort of way to react, for some reason, to certain kind of issues" (p. 88). Moreover, a participant of Ridge and Ziebland (2006) discusses his depression as follows:

> I mean it's chemical, you know I'm quite, you know I'm quite happy to admit there's something screwed up about my brain chemistry, you know. But you know, some people are diabetic, they take drugs, you know. And I know people say, 'Oh, it's not the same.' But I'm afraid it bloody well is. (p. 1043)

While these psychiatric narrative threads focus on the question, "Why did I develop my mental problems?," this question is not neatly separated from the question, "Why do people in general suffer from depression?". Specifically, psychiatric theories prefigure (at least) three types of causal content that people may incorporate into their psychiatric narrative threads. First, psychiatric theories prefigure *causal elements*: They present certain factors as causally important for developing our mental

problems. For instance, the biomedical model prefigures brain chemistry or genes as relevant factors to incorporate into our psychiatric narrative threads. Second, psychiatric theories prefigure *causal relations*. For instance, the biomedical model can prefigure causal relations between biological factors and our depressive symptoms as relevant relations to include in our psychiatric narrative threads. Third, psychiatric theories prefigure *metaphors* that describe the nature and causes of our mental problems. For instance, the biomedical model prefigures metaphors such as "fighting depression" or comparisons between depression and physical conditions such as diabetes (Schreiber & Hartrick, 2002).

However, focusing on the causal content that psychiatric theories prefigure is only part of the picture. First, this analysis does not help us make sense of the idiosyncratic ways people integrate psychiatric theories into their psychiatric narrative threads. For instance, people with a depression diagnosis resort to biomedical explanations in their psychiatric narrative threads alongside psychodynamic and social models of depression, sometimes switching between the three (Kangas, 2001). Some completely integrate the biomedical model into their psychiatric narrative thread, whereas others only do so partially (Schreiber & Hartrick, 2002). Moreover, descriptively focusing on the causal content that psychiatric theories prefigure does not explain *why* such content is incorporated into our psychiatric narrative threads. Goldie (2012) highlights that we pick the causal elements most *relevant* to our interests when we narrate a story. He cites Lewis (1986), who states the following regarding the multiplicity of causes of a car crash:

> If someone says that the bald tyre was the cause of the crash, another says that the driver's drunkenness was the cause, and still another says that the cause was the bad upbringing which made him so reckless, I do not think any of them disagree with me when I say that the causal history includes all three. They disagree only about which part of the causal history is most salient for the purpose of some particular inquiry. They may be looking for the most remarkable part, the most remediable or blameworthy part, the least obvious of the discoverable parts. (p. 215)

Similarly, we include causal information in our psychiatric narrative threads "for the purpose of some particular inquiry." Relatedly, this analysis does not show the *social significance* of incorporating such causal content into our psychiatric narrative threads. In what follows, I explicate two functions that psychiatric theories can fulfill in our self-narration. Note that this does not exclude the possibility that psychiatric

theories can serve other functions, too. In the next section, I will discuss the influence of psychiatric theories on the *coherence* of our psychiatric narrative threads.

7.4 Narrative coherence

The first function that psychiatric theories may serve in our psychiatric narrative threads is improving their *narrative coherence*. Causality plays an important role in narrative coherence. Goldie (2012) describes narrative coherence as revealing connections between related events through *emplotment*, following the terminology of Ricoeur (1984). A narrative is coherent if it is clear how events "happening one after another hold together in some way" (Goldie, 2012, p. 14). This description highlights two characteristics of coherent narratives: Narratives are coherent when their elements are *minimally temporally ordered* and *causally connected*. Relations between narrative elements may also be emotion- or reason-based (cf. Currie, 2006; Velleman, 2003). However, following Carroll (2001), Goldie (2012) argues that "it would be a strange story indeed if it involved no representations of causal relations whatsoever" (p. 16). Not all relationships in a narrative have to be causal, but causality nonetheless plays an important role in providing narrative coherence. This also applies to our self-narratives: Not all relationships in our self-narratives have to be causal, but we can *improve* the coherence of our self-narratives by enhancing the temporal ordering or causal integration of their elements.

The causal relations – or *explanations* – that psychiatric theories prefigure can influence the coherence of our psychiatric narrative threads.[108] Specifically, these causal explanations can improve the coherence of our psychiatric narrative threads if they fill causal gaps or strengthen our existing causal intuitions. To illustrate, let us again focus on the biomedical model of depression. The biomedical model of depression can prefigure (at least) three types of causal explanations. The model can causally explain (1) why we experience mental problems (i.e., because of a biological dysfunction), (2) why our mental problems co-occur (i.e., because of a shared, biological cause), and (3) why we recognize ourselves in or interact in specific ways with our biological parents, siblings, and children (i.e., because of our shared biology or biological predisposition). Various authors have suggested that these biomedical causal explanations improve the coherence of our psychiatric narrative threads by filling causal gaps when observable or intentional explanations are missing

108. In the context of daily life, the empirical adequacy of such explanations is less "decisive" than in, for instance, the scientific context. As I showed in **Section 7.2**, dominant psychiatric theories – such as the biomedical model of depression – demonstrate evidence resistance.

(Leuenberger, 2021; Postan, 2016). To illustrate, let us imagine the following scenario based on an example adapted from Hoffman (2015): "Before, I did not know why I did not feel as if my life was worth living, but I now believe that it was due to my serotonin transporters not working properly."[109] Hoffman (2015) argues that referring to such biomedical explanations introduces *hyponarrativity*, i.e., hampers our ability to formulate a self-narrative (cf. Sadler, 2005). However, Leuenberger (2021) argues that such a biomedical explanation *is* coherent: It introduces a causal relationship between biological factors and the onset of our depressive symptoms where none was present previously. Another example of the coherency-improving potential of incorporating biomedical explanations in our psychiatric narrative threads is presented in the study of Laegsgaard et al. (2010). These researchers interviewed people who partook in a genetic testing trial for their depression and had a strong history of depression in their families. One of their participants stated the following:

> When my brother and I participated in the research – it gave rise to discussions about it in the family – and then it appeared that the reason it is difficult to talk about in the family is that other members of the family have suffered from it – or are suffering from it – and that it has been sort of hush-hush. But now, we stand forward and say, well this is how it is – and then so many things in the family saga fall into place. If it is genetic then it explains a lot – why somebody in the older generations behaved like they did. (p. 474)

A genetic explanation of their depression helped address previously unexplained aspects of their lived experience – i.e., the behavior of the older generations – which made elements of their self-narrative "fall into place." So, incorporating explanations provided by the biomedical model into our psychiatric narrative threads could improve the narrative coherence of these threads. However, zooming out to the broader sociocultural context in which our self-narration occurs can nuance – or even problematize – this statement.

First, our desire for narrative coherence is itself socially determined. So, incorporating psychiatric theories that present clear explanations for our mental problems into our psychiatric narrative threads may *reflect* our desire for narrative coherence. Narrative coherence is usually presented as beneficial (Leuenberger, 2021; Mackenzie, 2014), especially in the context of (mental) suffering. As stated in **Chapter 6**, Frank (1995)

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109. The original example describes how, after receiving antidepressants, someone may say the following: "The reason I came to believe that the world is worth living in after all is because more of my serotonin transporters were blocked" (Hoffman, 2015, p. 319).

argues that illness narratives can help us engage in the "narrative repair" of our self-narratives following illness. Mackenzie and Poltera (2010) show how the inability to form a coherent self-narrative following a mental breakdown can be a source of suffering. Phillips (2003) argues that people with lived experience often struggle "to experience their lives as unified, coherent wholes" (p. 324) and that psychiatric narrative threads aim to bring coherence to their disordered life experiences. Finally, Dings and Glas (2020) suggest that restoring narrative coherence can help resolve the *self-illness ambiguity* that may accompany a psychiatric diagnosis, i.e., "Is it me or the disorder that makes or made me do *X*?". However, narrative *incoherence* may also be valuable. A coherent self-narrative is only valuable insofar as we want to "exist in the social world with a comfortable sense of being a good, socially proper, and stable person" (Linde, 1993, p. 3). Emphasizing the value of narrative coherence may privilege conventionality, marginalize the experiences of people who think more creatively (Hyvärinen et al., 2010), and ignore the possibility that people use self-narration as a creative study of their history and complexities (Hänninen & Koski-Jännes, 2010). Frank (1995) calls upon us to better listen to people with "chaos narratives," i.e., anti-narratives that do not have narrative order, rather than requiring their narratives to be coherent. So, improving the coherence of our psychiatric narrative threads via psychiatric theories is not necessarily beneficial.

Second, and related, psychiatric theories may play a role in determining the value we attribute to narrative coherence. The biomedical model may promote the idea that our psychiatric narrative threads *could* – and potentially even *should* – be coherent, i.e., mental problems are like puzzles that can be solved when their underlying dysfunctions are fixed. This aligns with Frank (1995), who describes the mechanistic view of illness as follows: "[T]elevisions break and require fixing, and so do bodies. The question of origin is subsumed in the puzzle of how to get the set working again" (p. 88). Laegsgaard et al. (2010) cite a participant who uses a similar puzzle analogy to describe why they wanted to participate in the genetic trial: They hoped that "the last pieces could sort of fall into place in your self-knowledge" (p. 476). So, the (dominance of the) biomedical model may play a role in the value we ascribe to narrative coherence.

Third, the causal explanations that the biomedical model prefigures may be coherent but not necessarily *meaningful* (Leuenberger, 2021). Goldie (2012) claims that narratives are (internally) meaningful if they reveal how a person's thoughts, feelings, and actions could have *made sense* from their perspective at that time point (p. 17). Self-narratives, in turn, are meaningful when they provide *reasons* for our thoughts, feelings, and (lack of) action. Indeed, we often share our psychiatric narrative

threads with others precisely to provide such reasons or justifications (Radley & Billig, 1996). To justify our thoughts, feelings, and (lack of) action, we frequently refer to folk psychological talk about our intentional psychological states (e.g., Andrews, 2009; Hutto, 2007). This implies that psychiatric theories that prefigure non-intentional, biological explanations for our psychiatric narrative threads may be less suited to justify our behavior. This aligns with Ahn et al. (2003), who show that giving an intentional explanation for deviant behavior (i.e., being stressed due to one's workload) reduces abnormality judgments, whereas giving a brain-based explanation does not. Moreover, adopting the biomedical model may *prevent* us from incorporating intentional, psychological, and possibly more meaningful relations in our psychiatric narrative threads. This is illustrated by Schreiber and Hartrick (2002), who argued the following about the psychiatric narrative threads shared by the women with depression diagnoses they interviewed: "Even though the women told stories that wove together numerous influential psychosocial threads, the stories and the threads were not given credence" (p. 101). When Schreiber and Hartrick (2002) asked their participants why they developed their depression *now* rather than earlier, their participants "would most often hesitate and then vaguely explain away the lack of connection between their story of depression and their explanatory model of depression" (p. 101). This shows how adopting the biomedical model may prevent us from incorporating *meaningful* connections in our psychiatric narrative threads.[110]

So, the biomedical model may fill causal gaps in our psychiatric narrative threads but reinforce the value of narrative coherence as a social norm and overrule other, potentially more meaningful ways to provide narrative coherence. In the next section, I will discuss the influence of psychiatric theories on our ability to imagine possibilities for reducing our mental suffering.

7.5 Imagining possibilities

The second function that psychiatric theories may serve in our psychiatric narrative threads is influencing our ability to *imagine possibilities for reducing our mental suffering*. In **Chapter 3**, I claimed that scientifically understanding psychiatric phenomena involves reasoning about possibilities for reducing mental suffering; such reasoning also plays an important role in our self-understanding.

110. As a counterargument, Postan (2016) suggests that incorporating bio-information in our self-narration may help us to "disabuse us of "unhelpful or misconceived interpretations of the causes" of our experiences (p. 140).

When we reflect on our lives, we often reflect on how our lives could have been different, how they may become different, and what could have or will bring such changes about. Thinking about such *self-related possibilities* is intimately tied to our *imaginative* abilities: Our imagination allows us to engage with things and events that are not (yet) actual and to respond to them from a different perspective (Currie, 2007). Mackenzie (2005) claims that our imagination "opens up a space within which we can try out different possibilities for ourselves – different possibilities of action, desire, emotion, and belief" (p. 289). We can imagine different pasts for ourselves (i.e., engage in "counterfactual speculation") or different futures (i.e., engage in "future-directed fantasy") (p. 289). However, if the pasts or futures we imagine for ourselves conflict with our self-narrative, this amounts to imagining a different person altogether (Mackenzie, 2008). So, our self-narratives guide and constrain the past- or future-related possibilities we can imagine for ourselves.[111]

The *perceived changeability* of the elements in our self-narratives influences the self-related possibilities we can imagine. For instance, perfectionism is an important element in my self-narrative. Based on my previous experiences and background knowledge about the stable nature of personality traits, I perceive my perfectionism as difficult to change. Hence, I have difficulties imagining how my life would have played out if I had not had perfectionistic tendencies or fantasizing about a future life where I am less perfectionistic and am not struggling with its negative consequences. If I were to perceive my perfectionism as something that – even with quite some effort – would be changeable, this would affect the possibilities I foresee for my future self. So, the perceived changeability of elements in my self-narrative guides whether I can imagine the possibility of a different past or future.

When we experience mental problems, we may try to imagine a life in which we mentally suffer less. We may try to imagine, for instance, a life in which we would not have developed our mental problems or a future life without them. Our psychiatric narrative threads shape our ability to imagine such possibilities for reducing our mental suffering, and vice versa. For instance, Ratcliffe (2015, 2016) characterizes depression as an experienced inability to imagine positive change and argues that the self-narratives of people with depressive symptoms shape and are shaped by this existential feeling. This does not mean that people with depressive symptoms are unable to imagine possibilities for reducing their mental suffering:

111. This statement echoes my earlier claims in **Chapter 6** about self-narratives being part of the background knowledge that guides clients' reasoning about self-related possibilities based on personalized network models.

> [A]lthough the capacity for self-narrative is impaired to varying degrees in depression and shaped by the kinds of existential feeling that are typical of depression, people still interpret their predicaments in ways that are, to an extent, contingent and malleable. (Ratcliffe, 2016, p. 194)

Specifically, Ratcliffe (2016) argues that causal explanations of depression can influence the possible lives that people can imagine for themselves and that it is an open question when and how they do so. Here, I formulate an initial answer to this question.

How psychiatric theories influence our ability to imagine possibilities for reducing our mental suffering depends on the perceived changeability of the elements they prefigure. The causal elements that psychiatric theories prefigure have a certain degree of *perceived malleability* (i.e., factors we believe to be changeable in principle) and *perceived controllability* (i.e., factors we believe to be within our control). For instance, empirical studies show that adopting a biomedical explanation of depression decreases the perceived malleability and perceived controllability of our depressive symptoms (e.g., Deacon & Baird, 2009; Kemp, 2014; Lebowitz et al., 2013). So, adopting the biomedical model into our psychiatric narrative threads may hamper our ability to imagine a possible life in which we do not suffer mentally. To address the normative implications of this claim, we have to zoom in on the specific possibilities imagined and zoom out to the broader sociocultural context.

First, the value we attribute to the (in)ability to imagine possibilities for reducing our mental suffering may depend on whether we engage in "future-directed fantasy" or "counterfactual speculation." If we focus on future-directed possibilities, adopting psychiatric theories whose elements are deemed non-changeable is generally considered harmful (cf. Hassall, 2020; Tekin, 2011). For instance, McConnell and Snoek (2018) suggest that recovering from mental problems may benefit from "narrative work," i.e., effortfully narrating a connection between our self-narrative and a valued future. This implies that our psychiatric narrative threads should allow us to imagine a possibility for positive change. Moreover, imagining a possible future in which we suffer less may be necessary for people to become active parts or actors in their treatment. However, if we focus on possibilities for the past, adopting psychiatric theories whose elements are deemed non-changeable may also have benefits: It may help us let go of the idea that we could have *prevented* our mental problems. To illustrate, Laegsgaard et al. (2010) cite a participant who describes how a biomedical explanation allowed them to let go of the idea that they could have prevented their depression:

> [After participating in the genetic project] it was like, you could put down some of the guilt. Before, you sort of thought about what did I do wrong. It made you feel guilt. But if it is a genetic disease – well – then it is not your fault, its just the way it is. (p. 475)

Similarly, a participant in Schreiber and Hartrick (2002) argues the following:

> It's only in the last two years that I've really realized what depression means and what it is and that it can be sort of, it's a chemical thing. Because before that you always think of it as a weakness… your idea of depression is somebody who can't quite keep it together. But now I know that's wrong. (p. 95)

Second, publicly available knowledge shapes the perceived changeability of the causal elements of psychiatric theories. In discussing the relative stability of biological factors as compared to social dynamics, Longino (2013) claims that "[i]n our brave new world of pharmacological mood and behavior management, nature may come to be perceived as the more malleable of the forces" (p. 10). Indeed, Lebowitz et al. (2013) show that emphasizing the malleability of our biology – by focusing on epigenetics – can influence the prognostic attitudes of people who hold biomedical explanations of their depression (cf. Lebowitz & Ahn, 2015). Although public ideas about the changeability of our biology develop slowly, this does not mean that they are not shifting. Thus, the influence of biomedical explanations on the possibilities for reducing our mental suffering we can imagine for ourselves may shift with changing public ideas about the changeability of our biology.[112] Finally, dominant psychiatric theories, such as the biomedical model, influence not only the possibilities for reducing our mental suffering that we can imagine for ourselves but also the possibilities that others can imagine for us.

So, adopting the biomedical model in our psychiatric narrative threads could hamper our ability to imagine possibilities for reducing our mental suffering. However, the normative implications of this influence will depend on our forward- or backward-looking focus and the (socially determined) perceived changeability of biological factors.

112. How we perceive the changeability of biological elements can also change due to personal experiences, such as the effectiveness of the medication we use.

7.6 Conclusion

In this chapter, I explored the influence of psychiatric theories on our psychiatric narrative threads in daily life from a practice-oriented stance, using the biomedical model of depression as a case study. Drawing the analogy between the biomedical model of depression and master narratives, I showed that dominant psychiatric theories can scaffold our psychiatric narrative threads by prefiguring causal content and influencing how our psychiatric narrative threads are judged. I showed that adopting psychiatric theories can influence the narrative coherence of our psychiatric narrative threads and our ability to imagine possibilities for reducing our mental suffering. We should zoom out to the broader social context to assess whether these influences are harmful or beneficial.

This chapter highlighted possibilities for future research. For instance, there is little detailed empirical research available on the value that people with mental problems attribute to narrative coherence and the ability to imagine possibilities for reducing our mental suffering. To illustrate, the frequently-used *Illness Perception Questionnaire-Revised* (IPQ-R, Moss-Morris et al., 2002) includes the following items related to "illness coherence": (1) "The symptoms of my condition are puzzling to me," (2) "My illness is a mystery to me," (3) "I don't understand my illness," (4) "My illness doesn't make any sense to me," and (5) "I have a clear picture or understanding of my condition." My analysis can be a stepping stone for more detailed questionnaire items, structured interview prompts, or thematic analyses.

Moreover, this chapter showed how focusing on the social embeddedness of psychiatric theories can inform current debates on how psychiatric theories influence self-understanding and stigma. It is important to note that not all claims related to the biomedical model of depression will translate to other psychiatric theories. Indeed, the biomedical model of depression has specific functions *by virtue of* its dominance in our folk psychiatry. However, the dominance of psychiatric theories slowly but surely shifts, even if this shift lags behind the evidence base for these theories. Related, Lafrance (2014) shows in her qualitative research that we cannot simply remove biomedical explanations from our psychiatric narrative threads without a suitable alternative. In the upcoming chapter, I will explore how the network theory could influence our psychiatric narrative threads in alternative ways.

7

Chapter 8

The network theory and self-narratives

Abstract

In this chapter, I explore how the network theory could influence our psychiatric narrative threads, focusing on the strategies it promotes for "embracing the complexity" of our mental problems. The network theory promotes two strategies for simplifying complexity: decomposition and abstraction. The self-narratives of people with a depression diagnosis reflect both strategies: They decompose their problems when describing the multifactorial causes or constituents of their depression and abstract them when describing their lived experience. Emphasizing decomposition over abstraction, or vice versa, may change the influence of the network theory on our psychiatric narrative threads. Given this analysis, the dual approaches to complexity advocated by the network theory may be complementary rather than competing in this context.

8.1 Introduction

One of the hopes behind systemic theories of mental disorders, including the network theory, is that adopting them may improve how people with mental problems *see* themselves. This postulated epistemic benefit is often tied to the *complexity* that these theories promote. For instance, in an opinion piece titled *Embracing Complexity in Psychiatric Diagnosis, Treatment, and Research*, Cohen (2016) states that acknowledging the complexity of mental disorders has social value. Related, various authors claim that promoting the complexity of mental disorders could be beneficial for people with mental problems (MacDuffie & Strauman, 2017; Olthof et al., 2023; Tekin, 2011). For instance, Olthof et al. (2023) argue that they "hope that a complex systems approach to psychopathology can contribute to a public health service and culture that de-stigmatizes psychopathology and empowers those who suffer from it" (p. 321). Moreover, Tekin (2011) argues that it is necessary for the self-understanding of people with psychiatric diagnoses "to acknowledge the complexity of [mental disorders] involving not only biological parameters but also cultural, social, and environmental factors that may escape a purely neurobiological analysis" (p. 359). However, there is no consensus on what it means to "embrace the complexity" of mental disorders, and no analysis to date on how promoting this vision could translate to epistemic benefits for people with mental problems.

In this chapter, I explore how the network theory could influence our psychiatric narrative threads, focusing on the strategies it promotes for dealing with complexity. Indeed, network representations are often used to make complexity intelligible

(Newman, 2010), and *complexity* is the first principle of the network theory. Interestingly, the principles of the network theory reflect two ways of embracing the complexity of mental disorders that could influence our psychiatric narrative threads in different ways. As in **Chapter 7**, I take a practice-oriented stance by focusing on the *function* these different strategies for handling complexity may have in our psychiatric narrative threads.

This chapter is structured as follows. In **Section 8.2**, I describe two strategies for embracing the complexity of mental disorders. *Decomposition* assumes that we can simplify the complexity of mental disorders by decomposing them into their constituent parts and interactions, while *abstraction* assumes simplification through the examination of their emergent dynamics. In **Section 8.3**, I demonstrate that the principles of the network theory reflect both strategies. In **Section 8.4**, I show that the psychiatric narrative threads of people with depressive symptoms reflect both ways of embracing complexity. Specifically, decomposition is used to reflect on the causes of depression; abstraction is used to describe the experience of depression. In **Section 8.5**, I explore how emphasizing decomposition over abstraction, or vice versa, may change the function of the network theory in our psychiatric narrative threads. Promoting decomposition may heighten the value we attribute to narrative coherence and allow us to imagine possibilities for reducing our mental suffering; promoting abstraction may heighten the value we attribute to narrative incoherence.[113]

8.2 Two strategies for embracing complexity

In everyday language, "complex" refers to things that cannot be easily understood. A phenomenon is complex when (1) we do not know why or how it came about and (2) it is caused or constituted by multiple factors. For instance, in discussing the complexity of mental disorders, Tekin (2011) argues that "[i]t is hard to identify a single cause or a single set of causes resulting in depression" and that, instead, "there is a cluster of factors, some of which are readily measurable and verifiable and others, which are not" (p. 371). Indeed, Proctor and Larson (2005) argue that the term "complexity" often "acts as a placeholder that moves among disciplines whenever they attempt to relate complicated, multifaceted, and unknown or partly unknown phenomena" (p. 1066). In this chapter, I focus on a different, albeit related, interpretation of complexity: *systemic* complexity. A systemic view of mental disorders claims that they are constituted by

113. My focus on depression as a case study may influence my argument on how adopting the network theory influences our psychiatric narrative threads. Future research should explore whether my claims extend to other conditions.

interacting parts that together produce the lived experience of our mental problems (see **Chapter 1**). But what makes mental disorders *complex* systems? In **Chapter 1**, I defined "complex systems" as systems comprised of factors from different domains that interact non-linearly over different time scales and demonstrate complex properties. However, this definition is still quite broad, and there is no consensus – even among complexity scientists – on what a complex system is and what properties all complex systems may have in common (Ladyman et al., 2013; Ladyman & Wiesner, 2020).[114] Here, I build upon **Chapter 2** to discuss two strategies for embracing the complexity of mental disorders, guided by different ideas of what complex systems are.

The first strategy for embracing the complexity of mental disorders is *decomposition*, i.e., studying (the organization of) their constitutive factors. This strategy aligns with the assumption that mental disorders are *nearly decomposable systems*. Simon (1962) describes nearly decomposable systems as follows:

> (a) in a nearly decomposable system, the short-run behavior of each of the component subsystems is approximately independent of the short-run behavior of the other components;
> (b) in the long run, the behavior of any one of the components depends in only an aggregate way on the behavior of the other components. (p. 474)

If a system is nearly decomposable, we do not have to represent all system components simultaneously; we can study each subsystem independently (Rathkopf, 2018). While component *A*'s behavior influences component *B*'s behavior, they are still considered separate entities with their own identities and properties.[115] To build on an example by Simon (1962), a nearly decomposable system is like a house with multiple rooms, where the outside walls – i.e., the system's boundaries – are well insulated, but the inside walls are not. If we assume that decomposition is a viable strategy to embrace the complexity of mental disorders, we can adopt a mechanistic explanatory strategy to study mental disorders (e.g., Craver, 2007). Moreover, decomposition justifies intervening on a mental disorder by targeting its dysfunctional parts or their connections.

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114. In line with this observation, Keller (2005) argues that the term "complexity" does not qualify as a (good) metaphor, for it does not "have a reference that is quite concrete or literal," and its meaning keeps on changing (p. 1072). Related, Stepney (2018) argues, referring to complex systems, that "[t]here is nothing quite as unhelpful as an explanation in terms of a metaphor, where the metaphor's source domain is equally ill-understood by the listener" (p. 28).

115. Near decomposability stands in contrast to "full" decomposability advocated by Fodor (1983) in his "modularity of mind" thesis, where cognitive modules demonstrate "informational encapsulation," i.e., cannot access information stored in other modules.

An example of a psychiatric theory that uses decomposition as a strategy for embracing complexity is the MPC account by Kendler et al. (2011) (see **Chapter 2)**. The MPC account claims that mental disorders "are defined not in terms of essences but in terms of complex, mutually reinforcing *networks of causal mechanisms* [emphasis added]" (p. 1143). More specifically, the MPC account is motivated by a "shift from the quest for essences of psychiatric kinds among either biological and social facts about the disorders to a quest for the *complex and multi-level causal mechanisms* [emphasis added] that produce, underlie, and sustain psychiatric syndromes" (Kendler et al., 2011, p. 1146). So, the MPC account promotes the idea that mental disorders are multifactorial and multidimensional and can be simplified via decomposition.

The second strategy for embracing the complexity of mental disorders is *abstraction*, i.e., studying their dynamics or "emergent behavior." This strategy aligns with the assumption that mental disorders are *non-decomposable systems*. According to Rathkopf (2018), a system is non-decomposable when "the behavior of any given component part, even over a short time period, depends on the behavior of many other individual components" (p. 69) (see **Chapter 2**). Non-decomposability implies that we cannot meaningfully decompose a system into separate components. Instead, we can simplify a system's behavior by looking for patterns that the system as a whole demonstrates. If we assume that abstraction is a viable strategy to embrace the complexity of mental disorders, we will not resort to a mechanistic explanatory strategy to study mental disorders; instead, we can use a dynamic (Lamb & Chemero, 2014) or topological explanatory strategy (Kostić, 2020; Rathkopf, 2018). Moreover, abstraction justifies focusing our interventions on changing the dynamics of the mental disorder system as a whole.

An example of a psychiatric theory that uses abstraction as a strategy for embracing complexity is the complexity theory of mental disorders – *complexity theory*, in short – developed by Olthof et al. (2023). The complexity theory, inspired by dynamical systems theory, states that "psychopathology can be seen as a dynamic pattern that emerges from self-organized interactions between interdependent biopsychosocial processes in a complex adaptive system comprising a person in their environment" (p. 316). Olthof et al. (2023) argue that mental disorders demonstrate a variety of emergent behaviors. First, mental health and disorder can be characterized as *attractors* in our mental health system. Dynamical systems theory argues that the state of a non-decomposable system is characterized by variables whose values can change over time. For example, the state of a pendulum is characterized by the position x and velocity v of the pendulum bob. Over time – as the bob swings –, the values of these variables trace a trajectory through the state space, i.e., the

collection of all possible values these variables can take. They do not do so randomly: They evolve towards specific regions of the state space, i.e., *attractors*. The pendulum's attractor is its resting point: While the pendulum oscillates, it will eventually come to rest due to air resistance. The complexity theory characterizes mental health and disorder as attractor states that our mental health system can evolve towards.[116] Second, our mental health system can *phase transition* from a disordered to a healthy attractor or vice versa. These phase transitions reflect qualitative changes that often happen abruptly at *tipping points* and are characterized by *early warning signals*, i.e., preemptive changes in systems' behavior before a phase transition. Moreover, for a phase transition to occur, an attractor needs to *destabilize*. This can be instantiated by factors outside the system or *triggering factors*; how easily an attractor is destabilized following a perturbation depends on the system's *resilience*. Finally, our mental health system does not automatically switch back to its original attractor state when an external destabilizer is removed, i.e., *hysteresis*. That is, interventions in mental disorders should aim at *destabilizing* disordered attractors and *moving* the system to – and *supporting* – healthy attractors (Hayes et al., 2015; Olthof et al., 2023; Schiepek et al., 2016). So, complexity theory promotes the idea that mental disorders are multifactorial and multidimensional and can (only) be simplified via abstraction.

So, we can embrace the complexity of mental disorders via decomposition or abstraction. In the next section, I will show that the network theory promotes both strategies.

8.3 The network theory and complexity

The network theory wants to do justice to the complexity of mental disorders. Proponents of the network theory acknowledge that "basically every element of [a mental disorder] system is dependent on a heterogeneous set of biological and external factors" (Borsboom et al., 2019a, p. 9), and Borsboom (2017a) elaborates on the network theory's first principle – *complexity* – as follows:

> [A]ccounts of mental disorders in terms of interacting components of a complex system are not only plausible, but in a sense the only game in town. Thus, this principle encodes the consensus that mental disorders are multifactorial in constitution, etiology, and causal background, which appears overwhelmingly plausible given the current scientific record (Kendler, 2012; Nolen-Hoeksema & Watkins, 2011). (p. 7)

116. Note that complex systems may have more than two attractors, and that attractors can have different levels of intricateness.

The network theory acknowledges that mental disorders demonstrate systemic complexity but does not commit itself to a specific strategy for embracing complexity. In this section, I describe how the network theory's principles can promote decomposition *and* abstraction. Again, my claims build on **Chapter 2**.[117]

We can interpret the network theory as promoting decomposition (**Figure 8A**). First, the network theory assumes that mental disorders can be meaningfully decomposed into symptoms. In elaborating on the network theory's second principle – *symptom-component correspondence* – Borsboom (2017a) argues that psychiatric symptoms are defined at the right level of "granularity" and "successfully identify the important components in the psychopathology network" (p. 7). Second, the network theory assumes that the causal relations between symptoms are meaningful, as reflected in the third principle, i.e., *direct causal connections*. The network theory gets much of its explanatory force from emphasizing these causal relations: Borsboom (2017b) states that "the pervasiveness of symptom–symptom causation" is the network theory's "main ingredient" (p. 80). Specifically, these causal relationships are justified by referring to symptoms' meaningful, intentional content. Borsboom et al. (2019) use the following description to account for the causal association between compulsive hand washing and fear of germs mentioned in **Chapter 2**:

> To understand compulsive rituals as a response, one needs to refer to the content of the beliefs and fears involved: Because the patient has a belief *about the spreading of germs*, he or she becomes afraid of *contamination*, and because this is the content of the fearful feeling, he or she responds to it by excessive washing rituals. (p. 6, emphasis in original)

Hence, the content of the symptoms and their relationships are meaningful and can be studied in isolation. Relatedly, psychometricians emphasize the similarities between the network theory and *cognitive-behavioral theory* (Borsboom, 2017b; Bringmann et al., 2022). Cognitive-behavioral theory claims that mental disorders arise from causal connections between thoughts, feelings, and behavior (Beck, 1967), and thus assumes that mental disorders can be meaningfully decomposed into these

117. Both strategies for embracing complexity are also reflected in different *network-based case conceptualizations* (see **Chapter 6**). For instance, the *network intake* ("netwerk intake," PsyNet, 2020) promotes decomposition: The client and clinician use the case conceptualization to explore what themes – or connections – are desirable and feasible to change and what kind of interventions could facilitate this. *Ideographical system modeling* (Olthof et al., 2024; Schiepek et al., 2016; Van den Bergh et al., 2024) promotes abstraction: It "is explicitly cyclical with all components directly or indirectly influencing all other components" (Van den Bergh et al., 2024, p. 182), and the personal data model based on the case conceptualization is used to monitor the client's attractor states.

separate elements. Third, the network theory suggests that mental disorders can be treated by intervening on specific nodes or their relations (Borsboom et al., 2019a). Finally, various proponents of the network theory have highlighted its compatibility with the MPC account (Borsboom & Cramer, 2013; Robinaugh et al., 2020; see **Section 8.2**). So, the network theory promotes embracing the complexity of mental disorders via decomposition.

This strategy for embracing the complexity of mental disorders is reflected in the study by Meier et al. (2022), who examined the effects of presenting people with eating disorder symptomatology with a network theory-based psychoeducational video.[118] In the video, a clinical psychologist claims that eating disorder symptoms themselves play an important role in the development and maintenance of eating disorders: They actively cause each other. The psychologist shares how "it all starts with specific factors such as perfectionism which can trigger symptoms which again lead to the activation of other symptoms and ultimately results in the full-blown picture of an eating disorder" (supplementary materials, p. 1). She continues by describing how specific symptoms can lead to and eventually maintain each other, i.e., developing a network of symptoms. She argues that individuals may differ in what symptoms are most important or how their symptom networks are connected. The video finishes with the message that researchers have identified symptoms that function as "key players" – their way of describing central variables – and that targeting these symptoms can reduce eating pathology as a whole. Here, Meier et al. (2022) highlight the decomposability of complexity: Specific symptoms can be teased apart and separately intervened upon.

However, we can also interpret the network theory as promoting abstraction (**Figure 8B**). First, the network theory refers to the emergent behavior of mental disorders to explain their development and persistence. Indeed, the fifth principle of the network theory – *hysteresis* – refers to a phenomenon characteristic of non-decomposable systems, as mentioned in **Section 8.2**. Specifically, Borsboom (2017a) defines *mental disorder* as "the (alternative) stable state of a strongly connected network, i.e., the state of disorder that is separated from the healthy state by hysteresis," *resilience* as "the disposition of weakly connected networks to quickly return to their stable state of mental health," and *vulnerability* as "disposition of strongly connected networks to transition into a state of disorder upon a perturbation in the external field" (p. 9) – all these descriptions refer to emergent behavior. Second, psychometricians have emphasized the compatibility between the network theory and complexity theories (e.g., Borsboom, 2017b; Bringmann et al., 2022).

118. The full transcript can be found in Meier et al. (2022, supplementary materials).

Third, the network theory acknowledges that treating mental disorders concerns changing the symptom network *as a whole* (Borsboom, 2017a). So, the network theory also promotes embracing the complexity of mental disorders via abstraction. This implies that, depending on what theoretical principles are emphasized, the network theory promotes different strategies for dealing with complexity. While this may be considered a problematic inconsistency in, for instance, scientific practice, it is not necessarily an issue if we focus on how people make sense of their mental suffering in their daily lives. In the next section, I will show that decomposition *and* abstraction are reflected in how we embrace complexity in our psychiatric narrative threads.

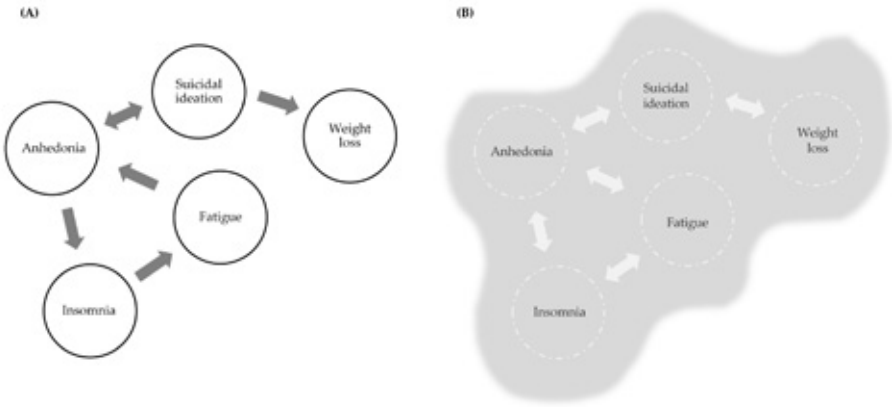

Figure 8. A nearly decomposable (**A**) and non-decomposable (**B**) interpretation of the network theory of depression

8.4 Complexity in psychiatric narrative threads

In this section, I show that our psychiatric narrative threads reflect both strategies for embracing complexity. I exemplify this using memoirs of people reflecting on their lived experience of depression (Alvarez, 2002; Plath, 1966) and qualitative research on the self-conception of people with a depression diagnosis (Kangas, 2001; Kokanovic et al., 2013; Midgley et al., 2017; Ridge & Ziebland, 2006). While conclusions based on individual testimonies may be contested, several consistent testimonies may highlight a relevant feature of psychiatric narrative threads.

First, people with a depression diagnosis may use *decomposition* to reflect on the multidimensional causes or constituents of their mental problems. Kangas (2001) concludes, based on interviews with people with a depression diagnosis, that they

experience and describe their depression as a "multidimensional" condition (p. 89). Kokanovic et al. (2013) reach a similar conclusion: Most of their participants discussed different, interconnected factors that contributed to their depressive symptoms. For instance, their participant W. states the following:

> I think there are a variety of issues. Part of it can be a chemical imbalance, part of it is environmental, and their environment when they were young, and also social pressures. Our own social mores in Western society contribute a lot to depression. I've sort of covered it globally, but when you're coming to an individual, apart from the food or alcohol issues, the situations that occur to them today can be caused by what happened to them in the past so their emotional and social environment, family, and stuff like that. (p. 118)

People may use decomposition in their psychiatric narrative threads whilst acknowledging that the "reality" of their depression may not allow for such straightforward simplifications. For instance, Ridge and Ziebland (2006) cite a 75-year-old participant who described the development of his depression as follows:

> I thought about it so much … I don't think I shall ever know (…) I suspect it was inherited. Endogenous if you like. I think that's what it was. (…) The environment as well … Because [having a mother with a mental illness] was quite… Very difficult as a 10 or 11-year-old to experience. (p. 11)

So, even when people acknowledge the limits of decomposing, they may still reflect on the possible individualizable causes or constituents of their depression in their self-narration.

Second, people who experience depressive symptoms may use *abstraction* to describe their lived experience. This is reflected in the metaphors for emergent dynamics incorporated in depression memoirs. For instance, *The Bell Jar* by Sylvia Plath (1966) describes an experience equivalent to being *stuck* in a depressed state:

> I knew I should be grateful to Mrs Guineau, only I couldn't feel a thing. If Mrs Guineau had given me a ticket to Europe, or a round-the-world cruise, it wouldn't have made one scrap of difference to me, because wherever I sat – on the deck of a ship or at a street café in Paris or Bangkok – I would be sitting under the same glass bell jar, stewing in my own sour air. (p. 178)

Relatedly, in *The Savage God: A Study of Suicide*, Al Alvarez (2002) describes experiencing severe depression as "a kind of spiritual winter, frozen, sterile, unmoving" (p. 103). Moreover, people with a depression diagnosis often refer to "spiraling" into depression (McMullen, 1999) or the spiraling of their thoughts (McCann et al., 2012; Ratcliffe, 2016) as a metaphor to describe their lived experiences.

Third, psychiatric narrative threads of people with depressive symptoms may reflect decomposition *and* abstraction simultaneously. Ellie (16), a participant of Midgley et al. (2017), describes her depressive experiences as due to

> The combination of things. I think some pressure and stress from school and just from my sleeping problems have been really bad for the past few weeks, it is just, then all of its combined, and it is just made it worse… (p. 29)

Here, Ellie describes her depressive experiences by abstracting away from the individual contributory factors, highlighting how "[t]he combination of things" combined "just made it worse." However, she simultaneously decomposes her experiences to indicate their potential causes. This is also exemplified by a participant of Kangas (2001), who describes the nature of his depression as follows:

> I think depression is to do with the person in his entirety, it is an experiential total issue. Its causes can be traced to biological factors, for instance, my mother's family has factors that predispose me to depression. (…) And then at the same time there are social relationships, your conception of life and living and the meaning you attach to yourself, they all affect your interaction. (p. 88)

Abstraction can capture the depressive *experience*, while decomposition can describe its *causes* or *constituents*. While this is only a preliminary analysis of how complexity is reflected in our psychiatric narrative threads, it does imply that both strategies for dealing with complexity are not necessarily opposed. Instead, they may serve different functions. In the next section, I will show how emphasizing decomposition over abstraction, or vice versa, may change the influence of the network theory on our psychiatric narrative threads.

8.5 The influence of the network theory on self-narratives

In **Chapter 7**, I claimed that psychiatric theories can function as scaffolds that prefigure causal *content* for our psychiatric narrative threads and influence how our self-narratives are *judged* by ourselves and others. Amongst others, psychiatric theories can influence our narrative coherence and our ability to imagine possibilities for reducing our mental suffering. In this section, I explore how promoting decomposition over abstraction, or vice versa, may shape the influence of the network theory on our psychiatric narrative threads. While this distinction may be somewhat artificial – in practice, both strategies are likely used – examining decomposition and abstraction separately shows an interesting feature of the network theory: It may serve different functions in our psychiatric narrative threads, depending on which strategy is emphasized. Since, to my knowledge, there is no qualitative research on or memoirs of individuals with lived experience who explicitly reflect on the network theory, this chapter is a preliminary exploration that can serve as the basis for future empirical research. Again, I use depression as a case study.

The network theory and decomposition

Let us suppose that the network theory is interpreted as promoting the decomposition of mental disorders into constitutive elements and their organization. As shown in **Section 8.2**, the second principle – *symptom-component correspondence* – and the third principle – *direct causal connections* – promote this strategy. If we center these two principles, this decomposition-based interpretation of the network theory, or *network theory*$_D$, prefigures (at least) two type of causal content for our psychiatric narrative threads. First, network theory$_D$ can prefigure symptoms and non-symptom factors in the "external field" as causal elements (see **Chapter 2**). Second, the network theory can prefigure causal relations between symptoms and between symptoms and non-symptom factors. What function could such causal information play in our psychiatric narrative threads?[119]

Adopting psychiatric theories can improve the coherence of our psychiatric narrative threads when they fill causal gaps or strengthen our existing intuitions about causal relationships between narrative elements (see **Chapter 7**). Network theory$_D$ provides various causal explanations, including why our mental problems co-occur

119. In **Chapter 6**, I argued that personalized network models and psychiatric narrative threads show structural similarities: Psychiatric narrative threads can be redescribed as collections of factors that have played an important role in the development of our psychiatric symptoms – i.e., nodes – and the (causal) relations between these elements, i.e., edges. Centering the second and third principles of the network theory also highlights the structural similarity between the network theory and psychiatric narrative threads.

(i.e., because they cause each other) and why non-symptom factors and mental problems co-occur (i.e., because non-symptom factors cause symptoms).[120] However, these causal explanations will not necessarily fill gaps in our psychiatric narrative threads. The causal explanations that network theory$_D$ provides are *non-specific* causal templates.[121] They do not address what symptoms cause each other and what "external factors" may cause our symptoms, for instance. To incorporate these non-specific causal explanations into our psychiatric narrative threads, we should relate them to symptoms and factors in the "external field" that are *relevant* to our lived experience. This implies, however, that network theory$_D$ will prefigure causal relations that we are already aware of in the first place. Whether prefiguring network theory$_D$ improves our narrative coherence thus depends on whether we previously did not "see" that these elements of our lived experience may have been causally connected, or deemed these causal connections irrelevant. In line with my claims in **Chapter 6**, our ability to "see" such causal connections may be promoted by the visual representation of network theory$_D$ (**Figure 8A**). In addition to these causal explanations, network theory$_D$ may also influence the coherence of our psychiatric narrative threads – and the perceived value of such coherence – in other ways. First, in **Chapter 7**, I suggested that the biomedical model could promote the idea that narrative coherence *is* possible and desirable by presenting our mental problems as puzzles that can be solved (Frank, 1995). Since decomposition presents a similar strategy, network theory$_D$ may promote a similar perspective on the possibility and value of narrative coherence. Second, network theory$_D$ prefigures folk psychological, intentional causal explanations. Given this, the causal explanations it prefigures may be more *meaningful* when adopted into our psychiatric narrative threads than, for instance, non-intentional, biological causal explanations (cf. Schleider & Weisz, 2018).

Adopting psychiatric theories can enhance our ability to imagine possibilities for reducing our mental suffering when we perceive the narrative elements they prefigure as changeable (see **Chapter 7**). Network theory$_D$ prefigures a variety of factors that could play a causal role in our mental suffering and, hence, prefigures many possible entry points for intervention. Moreover, network theory$_D$ predominantly focuses on the causal role of symptoms, most of which are conceptualized in psychological terms. Since behavior is generally considered more malleable when psychological rather

120. Note that I do not focus on whether these causal claims are *justified*. As I highlighted in **Footnote 108**, the empirical adequacy of psychiatric theories may play a less decisive role in daily life than in, for instance, scientific practice.

121. The biomedical model is also non-specific, for it does not tell us which specific biological factor causes our mental problems. However, the biomedical model prefigures a *non-observable* cause. Since network theory$_D$ prefigures *observable*, *intentional* causes, this critique applies more strongly to the latter.

than biological explanations are provided (Nettle et al., 2023), we can assume that network theory$_D$ prefigures elements with a high degree of perceived changeability.[122]

The network theory and abstraction

Let us suppose that the network theory is interpreted as promoting the abstraction of mental disorders, i.e., emphasizing their emergent behavior. The fifth theoretical principle – *hysteresis* – aligns with this strategy, as well as the theory's definitions of "mental disorder," "resilience," and "vulnerability" (see **Section 8.2**). If we center this principle and these definitions, this abstraction-based interpretation of the network theory, or *network theory*$_A$, prefigures various content for our psychiatric narrative threads. First, network theory$_A$ prefigures a *triggering cause* in the external field and a causal relation between this cause and the development of mental problems without specifying whether this triggering cause is, in principle, identifiable. Second, it prefigures the explanation that the change from a healthy state to a disordered state must be due to the wrong combination of a "vulnerable" symptom network and strong stressors in the external field. Third, it prefigures metaphors that can describe the state of our depression (e.g., being stuck or reaching a tipping point).

Given this analysis, network theory$_A$ may not improve the coherence of our psychiatric narrative threads. First, if we assume that the triggering cause explanation presupposes an *identifiable* cause, it faces the same criticism as that directed at network theory$_D$: Adopting this triggering cause in our psychiatric narrative threads may only enhance narrative coherence if we had not yet recognized this cause as relevant to the development of our mental problems. Second, if we assume the triggering cause is *unidentifiable*, this explanation does not prefigure causal content for our psychiatric narrative threads. Third, network theory$_A$ does not prefigure specific causal explanations that can help us make sense of what happens once we are "inside" the disordered state. Indeed, narratologists argue that the narrative format does not lend itself well to making sense of emergent behavior, for narratives cannot represent mathematical non-linearity, i.e., the presence of "multiple, simultaneous, reciprocal and recursive relations" (Walsh, 2018a, p. 16). Moreover, Abbott (2008, 2009) states that emergent phenomena are "unnarratable": "Something happens, we can see it happening, we can even chart its progress as it happens, but we cannot put in narrative form how it is caused" (2008, p. 234).

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122. A similar interpretation applies to network-based case conceptualizations that promote decomposition. For instance, a client reflects on his experience with the network intake as follows: "Because I had mapped out all my problems and created a network, it became clear that I could pick up a lot of things with my brother, even if the medication still needed tinkering" (PsyNet, 2020, translated from Dutch).

However, just as the biomedical model discussed in **Chapter 7** and network theory$_D$ may promote the value of narrative coherence, network theory$_A$ may promote the value of *narrative incoherence*, or at least challenge the importance we ascribe to coherence. In *Thinking With Systems*, Meadows (2008) argues that working with complex systems "constantly reminds me of how incomplete my mental models are (…) and how much I don't know" (p. 180). Walsh (2018b) suggests that narrative descriptions of emergent behavior may invoke *wonder*, i.e., "the affective quality attached to that region is just beyond the limits of understanding" and "the intuition of an order of things that exceeds my grasp" (p. 55). Frank (1995) argues that a good illness narrative "ends in wonder" and that "[b]eing available to yourself ultimately means having the ability to wonder at all the self can be" (p. 68). That is, illness is not a puzzle that can be solved, but a *mystery* that "cannot be solved, and while a person can seek to measure up to what a mystery presents, one cannot 'get it right' because there is no 'right' way to get it" (p. 81). By focusing on emergent behavior, network theory$_A$ may emphasize the structural limits of the narrative form and the perspectival and incomplete nature of our psychiatric narrative threads, thereby bringing about an alternative way of relating to our mental problems. So, network theory$_A$ may invoke *self-related wonder*, i.e., curiosity and humility regarding our self-narratives and their psychiatric narrative threads.

How could network theory$_A$ influence our ability to imagine possibilities for reducing our mental suffering? This depends on how changeable the elements prefigured by network theory$_A$ are perceived to be; however, the extent to which they are considered changeable remains unclear. The fifth principle – *hysteresis* – assumes that symptoms keep on activating each other after the triggering cause has disappeared. So, network theory$_A$ promotes the idea that changing our mental problems does not involve changing this triggering cause; instead, it involves other means to "destabilize" our disordered state. This can have multiple implications for our ability to imagine possibilities for change. First, since network theory$_A$ does not clearly prefigure *what* change or intervention will reduce mental suffering, it may not guide or constrain our ability to imagine possibilities for reducing our mental suffering. On an optimistic reading, this implies network theory$_A$ prefigures many possible entry points for change; on a pessimistic reading, network theory$_A$ does not prefigure such entry points. Second, our ability to imagine possibilities for reducing our mental suffering depends on how easily we believe we can destabilize our disordered state. Here, let us resort again to the complexity theory. Olthof et al. (2023) suggest that focusing on attractors can instill the belief that our mental problems are changeable. However, they also state that psychopathology attractors may attract strongly. For instance, it may take longer for individuals who are stuck in a depressed attractor state to bring

positive change to their mental problems, and the disordered attractor may itself "be more or less permanent due to person-internal constraints limiting the system's degrees of freedom in a rather fixed manner" (p. 321).

This section shows how promoting decomposition or abstraction via the network theory can serve different functions in our psychiatric narrative threads. Amongst others, network theory$_D$ may heighten the value we attribute to narrative coherence and allow us to imagine possibilities for reducing our mental suffering. Network theory$_A$ may heighten the value we attribute to narrative incoherence and have undetermined implications for our ability to imagine possibilities for reducing our mental suffering.

8.6 Conclusion

In this chapter, I addressed how the network theory could influence our psychiatric narrative threads, focusing on the strategies it promotes for dealing with complexity. The network theory promotes two means of simplifying the complexity of mental disorders, i.e., decomposition and abstraction. I showed how both strategies for embracing the complexity of mental disorders are reflected in the psychiatric narrative threads of people with depressive symptoms, highlighting that both strategies can serve different but complementary functions in our self-narration. Indeed, promoting decomposition via the network theory may heighten the value we attribute to narrative coherence and allow us to imagine possibilities for reducing our mental suffering, whereas promoting abstraction may improve the value we attribute to narrative incoherence.

I want to conclude by reflecting on the implications of my analysis. In *Thinking With Systems*, Meadows (2008) states that in our daily lives, we often experience a tension between recognizing that certain systems may not be decomposable and, nonetheless, having a desire to decompose them. On the one hand,

> [W]e have been taught to analyze, to use our rational ability, to trace direct paths from cause to effect, to look at things in small and understandable pieces, to solve problems by acting on or controlling the world around us. (p. 3)

On the other hand,

> Every person we encounter, every organization, every animal, garden, tree, and forest is a complex system. We have built up intuitively, without

> analysis, often without words, a practical understanding of how these systems work, and how to work with them. (p. 3)

The network theory reflects a similar tension between decomposing and abstracting complexity. Some may argue that this tension is a problematic feature of the network theory: Its underlying principles demonstrate internal inconsistency. However, in light of our self-relating in daily life, we could also interpret this tension optimistically. We are inherently inconsistent beings, which is reflected in how we relate to our mental suffering: We may use conflicting explanations to make sense of our mental problems or apply causal explanations inconsistently (see **Chapter 7**). Kangas (2001) concludes their study on the psychiatric narrative threads of people with a depression diagnosis by stating that "a single explanation [of depression] does not necessarily suffice in the sense-making process, even if a core explanation is found and a narrative formed accordingly. It seems that a single explanation does not structure the entirety of depression experience" (p. 89). If we translate this to the network theory, we could propose that the different explanations it provides may actually be beneficial – or even complementary – when it concerns our self-relating. To illustrate, I draw an analogy with the work of Weingart and Maasen (1997) on the metaphorical use of *chaos*. Chaos theory is a branch of dynamical systems theory that focuses on systems whose development is sensitive to the system's initial conditions. Weingart and Maasen (1997) performed a metaphorical analysis of the use of the term "chaos" in various (non-)scientific discourses. They show that different discourses are attracted to chaos as a metaphor for different reasons. In speculating on why this may be so, they argue the following:

> One reason is to be found in the opposing messages produced by the two branches, namely, that (a) chaos is a fruitful condition for order to emerge, and (b) chaotic systems seemingly repeating themselves eventually generate patterns of extreme complexity. That is to say, there is a message both for those who are threatened by the idea that stability, regularity, calculability might be lost (no order!) and for those who are afraid of the oppressive effects of uniformity (too much order!); chaos thus unites opposing (…) interests. (p. 15)

The network theory may also unite opposing interests in its embrace of complexity. Some may value the opportunities for positive change it may prefigure, while others may value its ability to challenge the need for coherence. Using the network theory to make sense of our mental problems presents a double-edged sword—this may well be a matter of principle.

Conclusion

Chapter 9

Conclusion

I started this thesis by introducing the metaphor between the two worlds of psychiatry presented by Grob (1998). Mental suffering belongs to the intractable world, whereas our psychiatric approaches belong to the idealized world. The idealized world promises knowledge that could show us the causes, constituents, and risk factors of mental disorders, aid clinical practice, and help people who suffer mentally make sense of their suffering and be empowered. However, psychiatric approaches, including the recent biomedical approach, have not met those epistemic expectations. New psychiatric reinventions attempt to fill this epistemic lacuna, one of which is the network approach. Proponents of this approach have set their hopes on this new, idealized landscape, hoping it will provide knowledge where previous approaches were lacking. However, looking at psychiatry's history and hype-disillusionment cycles provides reasons for caution. How do we avoid unrealistic expectations about the network approach without succumbing to unbridled relativism?

In this thesis, I developed a *practice-oriented* response to this question. Instead of focusing primarily on whether the network approach "accurately reflects" mental suffering, we should focus on whether and how the network approach helps us *reason* about mental suffering. This shift in perspective implies that we can judge the epistemic potential of the network approach based not only on *what* it represents but also on *how* it represents, *who* uses it, and for what *goals*. In acknowledging the inherent idealized nature of psychiatric approaches, my practice-oriented account implies that the network approach can, at most, provide knowledge on what is *possible* regarding mental suffering. We should not aim for Holy Grails in psychiatry but instead focus on the epistemic potential of psychiatric approaches, given their place in the idealized world. By reimagining psychiatry this way, we obtain a more nuanced and productive perspective on the knowledge about mental suffering that psychiatric approaches can provide.

Throughout the chapters of this thesis, I used my practice-oriented account to explore the epistemic potential of the network approach in scientific practice, clinical practice, and daily life. In this final chapter, I highlight my main findings per practice and suggest areas for future research.

9.1 Scientific practice

In **Part I** (**Chapters 2**, **3**, and **4**), I focused on the epistemic potential of the network approach in *scientific practice*. The network theory makes various causal, mechanistic, and topological explanatory claims, each requiring different assumptions to be

satisfied. Psychometricians can use the network approach to obtain scientific understanding of psychiatric phenomena by physically or mentally manipulating the nodal or topological dependency relationships demonstrated by the network theory or network models and, in this way, reason about possible ways to reduce mental suffering. The exploratory potential of network models can be characterized in terms of their ability to help psychometricians reason about epistemically relevant possibilities for psychiatric phenomena, i.e., possibilities consistent with psychometricians' available empirical and theoretical background knowledge. Psychometricians' ability to understand or explore possibilities for psychiatric phenomena using the network approach depends on various practice-independent and practice-dependent criteria. Specifically, it depends on the quantity and the empirical adequacy of the represented dependency relationships, their coordination with available background knowledge, psychometricians' training and skills, and the pragmatic virtues of the network representations. This also implies that *improving* the epistemic potential of the network approach in scientific practice, i.e., improving psychometricians' ability to use it to reason about psychiatric possibilities, depends on an interplay between these practice-independent and practice-dependent features.

Next to these general conclusions, I presented additional claims about the epistemic potential of the network approach in scientific practice in these chapters. First, I defended the position that, given the relative instability of psychiatric phenomena and the difficulty of identifying clear difference-makers, all psychiatric theories – including the network theory – provide how-possibly explanations. This does not mean that anything goes – they can still be evaluated in terms of their empirical adequacy – but it does imply that we should not expect them to provide "completely accurate" accounts of our mental suffering.

Second, I showed that the network theory's (extended) explanatory claims benefit from additional justification. There is renewed interest in theory development in psychology, and much of this centers around the network approach or uses it as an example (Borsboom, Van der Maas, et al., 2021; Eronen & Bringmann, 2021; Eronen & Romeijn, 2020; Fried, 2020; Oude Maatman, 2024). My overview of the assumptions that should be met to make causal, mechanistic, or topological claims could help fine-tune the network theory.

Third, and related, I highlighted that *coordinated* (background) knowledge is important for the epistemic potential of the network approach (Eronen & Romeijn, 2020). Roughly sketched, if data models and theories are coordinated, then data models can provide evidence for the possible existence of the phenomena that

theories attempt to explain, and theories can aid in formulating epistemically relevant hypotheses based on the data models. Such coordination applies to the network theory, network models, *and* other types of background knowledge. Psychometricians should evaluate whether, in reasoning with the network theory or network models, the background knowledge they use to guide their reasoning can actually *speak* to the dependency relationships that the network theory or network models afford. Moreover, in line with Oude Maatman (2024), I recommend that psychometricians be aware of the background knowledge that drives their reasoning with the network theory or network models and ideally make this knowledge explicit to ensure that its coordination suffices.

Fifth, I discussed the importance of skills and pragmatic virtues in determining the epistemic potential of the network approach. In my discussion, I focused on how network models' visualizability and functional transparency allow psychometricians to "see" epistemically salient patterns and manipulate these models. Future research should explicate in more detail what other pragmatic virtues are involved in the network approach (e.g., analogical reasoning, Borsboom, Van der Maas, et al., 2021) and how they drive the epistemic potential of the network approach.

Finally, I reflected on the relative epistemic potential of a "multilayer network approach." Compared to the network theory that conceptualizes mental disorders as "monolayer networks," a network theory that conceptualizes them as multilayer networks may have more explanatory breadth. Similarly, multilayer brain-symptom network models may have more exploratory breadth than standard network models. However, I also highlighted the epistemic limitations of a multilayer network approach and suggested various practice-dependent and practice-independent developments to improve its epistemic potential. Alongside ascertaining the approach's empirical adequacy, its epistemic potential would improve by explicating – and testing – a multilayer theory and improving the functional transparency of multilayer network models.

9.2 Clinical practice

In **Part II** (**Chapters 5** and **6**), I focused on the epistemic potential of the network approach in *clinical practice*, specifically on the use of personalized network models. Starting from my practice-oriented position that clinicians and clients use personalized network models to reason about possibilities for clients' mental problems, these chapters showed how the interplay of practice-independent and

practice-dependent factors determines the *interpretation* and *content* of these models. Indeed, deciding what variables (not) to include in personalized network models, which influences the dependency relationships these models afford, is influenced by a variety of practice-independent constraints (i.e., representational and explanatory boundaries) and practice-dependent constraints (i.e., clients' goals, values, and stories about their mental problems). Focusing on the interplay between personalized network models and clients' psychiatric narrative threads, I showed how the latter simultaneously influence what dependency relationships personalized network models demonstrate and guide clients' reasoning with these models.

These chapters also presented additional insight into the epistemic potential of the network approach in clinical practice that could inform future research. First, I discussed the epistemic potential of clinical ESM data over traditional psychological data from a philosophy of measurement perspective. Since this data type is personal and contextualized, it overcomes some standard critiques of using quantitative data in the social domain. However, as I briefly mentioned, using ESM data may also introduce new epistemic and moral questions about, for instance, reactivity and the status of data in clinical practice. Addressing these questions is important, for personalized data will most likely become increasingly prominent in clinical practice. For instance, recent developments allow for the collection and integration of ESM data alongside data from sensors that can measure physical activity or sleep quality and digital phenotyping (Roefs et al., 2022). Future research should explore the epistemic (and moral) potential and limitations of ESM data and these other types of personalized data.

Second, I showed that it is possible and epistemically valuable to reflect in a principled manner on the variables we wish to include in personalized network models and the *boundaries* of these models. This also extends to other network-based clinical tools, such as network-based case conceptualizations. Given developments in multisource data collection and modeling, such as those mentioned above, questions about model demarcation may become even more salient. I presented three ways of looking at model demarcation, but these options could be fine-tuned and elaborated on in future research.

Third, my practice-oriented account showed that the epistemic value of personalized network models in clinical practice depends on clinicians' and clients' ability to *interact* with these models, which depends on their psychometric and digital *skills*. The epistemic value of such skills is important to keep in mind in light of future developments in *blended psychiatry*. To illustrate, the innovation network *Redesigning Psychiatry* presents a provisionary list with "vacancies of 2030" for clinical practice,

which includes a "data lab technician," whose power is to "get associations on the table that are difficult to see or experience in daily life" (De Boer et al., 2016, translated from Dutch). When implementing data-based clinical tools, it is important to ensure that all participants in the clinical encounter have the skills to interact and reason with them. Related to this, my account shows that the epistemic potential of network-based tools in clinical practice depends on their pragmatic values; future research should explore what pragmatic virtues are important in clinical practice and how these can be promoted in innovations.

Finally, I discussed how the stories that clients tell about their mental problems can be conceptualized as idealized, abstracted, and reflexive tools. Promoting this perspective in clinical practice might have interesting implications for clients' self-relating. By highlighting that there is a "distance" between our mental suffering and our psychiatric narrative threads, we can more critically reflect on the content of our self-narration and the value it should play in our lives. Further philosophical exploration of this relationship between narratives and tools can help explicate their similarities and differences in more detail.

9.3 Daily life

In **Part III** (**Chapters 7** and **8**), I focused on the epistemic potential of psychiatric theories in *daily life*. I explored how people who experience mental problems use the biomedical model and could use the network theory to make sense of their mental problems, focusing on the *function* these theories may play in their psychiatric narrative threads. I showed that psychiatric theories can prefigure causal content for our psychiatric narrative threads and influence how we and others judge these threads. Incorporating psychiatric theories into our psychiatric narrative threads can influence their narrative coherence and our ability to imagine possibilities for reducing our mental suffering. Taking the biomedical model as an example showed that psychiatric theories may promote narrative values and that background knowledge could determine how psychiatric theories are interpreted. Translating this to the network theory, I showed how it promotes the idea that mental disorders are "complex" and could promote different strategies to deal with such complexity, each serving a different function in our psychiatric narrative threads.

Again, these chapters presented additional claims that open up avenues for future research on the epistemic potential of psychiatric approaches – and the network approach specifically – in daily life. First, I drew an analogy between psychiatric

theories and master narratives. Psychiatric theories do more than provide (how-possibly) explanations of our mental suffering: They influence how we judge our thoughts, feelings, and actions, whether we accept our mental suffering, whether we are hopeful about the future or pessimistic about our possibilities for change, amongst others. The normative function of psychiatric theories has been recognized before. However, the analogy with master narratives demonstrates similarities between how psychiatric theories influence our psychiatric narrative threads and how other social narratives related to, for instance, gender and relationships influence our self-narration. This analogy highlights an important feature of psychiatric theories that has thus far been underexposed in the philosophy of personal identity: We can reject and replace dominant psychiatric theories if we disagree with the values they present or the functions they fulfill in our self-narration. Exploring this dynamic between dominant and alternative psychiatric theories in more detail would be an interesting future area of research.

Second, I highlighted that the psychiatric theories we adopt may influence what we deem valuable about our psychiatric narrative threads. Whereas the biomedical model and the decomposition interpretation of the network theory may promote the value of narrative coherence, emphasizing emergent behavior and abstraction may promote the value of narrative incoherence. The idea that incoherent self-narratives may be valuable has been stated before (Frank, 1995; Hyvärinen et al., 2010; Linde, 1993), but the idea that psychiatric theories could *promote* narrative incoherence has, to my knowledge, not been addressed in the academic literature. Future research could explicate this influence in more detail, specifically in light of psychiatric theories promoting abstraction, such as the network theory or complexity theory.

Third, I discussed how the function of psychiatric theories in our psychiatric narrative threads depends, in part, on how the causal information they provide is *perceived*. This perception can be influenced in various ways, for instance, by our personal experiences or sociocultural context. If we take a brief detour from daily life to clinical practice, this observation highlights that clinicians should be mindful of such background knowledge that guides how clients interpret the psychiatric theories presented in psychoeducation.

Fourth, I showed that both "complexity" and "embracing complexity" can be interpreted in different ways. This could affect how people adopt complexity-driven psychiatric theories, such as the network theory, into their psychiatric narrative threads. If we again analyze this from a clinical perspective, this observation highlights the importance of making these strategies for embracing complexity

explicit in psychoeducation. Whilst I suggested that both strategies for embracing complexity may have fruitful implications, being explicit about one's strategies can prevent miscommunication.

Finally, by discussing the biomedical model and network theory, these chapters highlighted a more general claim: My practice-oriented account is not tied to the network approach alone but can also be used to evaluate the epistemic potential of other psychiatric approaches. With this realization, I will finish this thesis with some final thoughts.

9.4 Final thoughts

In his address, Grob (1998) argued that with every new psychiatric approach, its proponents insisted that psychiatry "stood on the threshold of fundamental breakthroughs that would revolutionize the ways in which mental disorders were understood and treated" (p. 217). More than twenty-five years later, we see that new psychiatric approaches are still introduced with similar gusto. This makes sense: Mental suffering is confusing, frightening, and isolating, and we want to be able to alleviate it. My thesis suggests, however, that psychiatric approaches may have been judged and promoted under unrealistic ideas about what it means for them to contribute knowledge about mental suffering. Psychiatric approaches can help us reason about mental suffering but will not give us "true" insight into the intractable world of mental suffering. This requires humility from those who work with and disseminate psychiatric approaches, whether scientists, clinicians, or media outlets. They can argue for the value of psychiatric approaches while acknowledging this limitation.

Since I started writing this thesis, I have observed that discussions of the network approach have changed from "unbridled optimism" to a more nuanced and productive reading of its possibilities and limitations. With this thesis, I hope to contribute to this development alongside the important empirical and conceptual work done by psychometricians, clinicians, and others. Although an academic thesis has limited impact, I hope some of my conclusions reach beyond the walls of philosophical departments to other practices.

Most of all, I hope my thesis can show people who experience mental suffering an alternative way of relating to the idealized world that scientists, clinicians, and popular culture present. From personal experience, I know how necessary it can feel to hold on to the idealized world in an attempt to make sense of one's mental

suffering. Meadows (2008) argues that the realization prompted by systems thinking that "no paradigm is 'true'" can "be the basis for radical empowerment. If no paradigm is right, you can choose whatever one will help to achieve your purpose" (p. 164). Although I would phrase this with more nuance, there is some truth to the statement. Recognizing that the idealized world of psychiatry is never a complete reflection of our mental suffering – and that we have some freedom in choosing a world that works for us – can indeed provide a sense of empowerment. Reimagining psychiatry opens up a new space of possibilities, not only for the network approach but also for those wandering the intractable world.

Appendices

Samenvatting (NL)
Summary (EN)
Acknowledgements
Bibliography
About the author

Samenvatting (NL)

Introductie

Wanneer we met psychisch lijden te maken hebben, bewegen we ons tussen twee werelden. Enerzijds is er de “ondoorgrondelijke” en ongrijpbare wereld van het psychisch lijden zelf dat vaak moeilijk te begrijpen en verklaren is. Anderzijds is er de "geïdealiseerde" wereld waarin we psychisch lijden conceptualiseren en bestuderen. In deze geïdealiseerde wereld generaliseren en vereenvoudigen we de geleefde ervaring van psychisch lijden om verklaringen, patronen of aanknopingspunten voor behandelingen te zoeken. Door de geschiedenis heen is deze geïdealiseerde wereld steeds opnieuw geschetst; om de zoveel decennia ontstaat er een nieuwe psychiatrische benadering die nieuwe manieren behelst om mentale stoornissen te conceptualiseren en bestuderen. Momenteel staat de psychiatrie aan de vooravond van wederom een heruitvinding. De biomedische benadering van mentale stoornissen die de afgelopen decennia domineerde, heeft niet geleid tot de beloofde doorbraken. Als reactie hierop zijn er systemische alternatieven ontstaan, zoals de *netwerkbenadering*. De netwerkbenadering stelt dat mentale stoornissen netwerken zijn van elkaar instandhoudende symptomen (de *netwerktheorie*) en pleit voor het gebruik van *netwerkanalyse* om mentale stoornissen te bestuderen. Voorstanders van de netwerkbenadering hopen dat deze benadering kennis zal opleveren waar de biomedische benadering tekortschoot. De geschiedenis van de psychiatrie leert ons echter dat elke psychiatrische heruitvinding kennisdoorbraken belooft die nooit volledig zijn waargemaakt. Hoe kunnen we realistisch kijken naar het “epistemisch potentieel” van de netwerkbenadering, ofwel de mate waarin deze benadering kennis over psychisch lijden kan verschaffen?

In dit proefschrift evalueer ik het epistemisch potentieel van de netwerkbenadering vanuit een *praktijkgericht perspectief*. Hierbij onderzoek ik hoe de netwerkbenadering wordt gebruikt in de wetenschappelijke praktijk, de klinische praktijk en het dagelijks leven om kennis te vergaren over mogelijkheden met betrekking tot psychisch lijden. In plaats van de netwerkbenadering voornamelijk te beoordelen op hoe goed ze de realiteit van psychisch lijden weerspiegelt – zoals vaak impliciet wordt gedaan –, kijkt mijn praktijkgericht perspectief ook naar *hoe* de netwerkbenadering psychisch lijden representeert, *wie* de netwerkbenadering gebruikt, en met welk *doel* ze wordt gebruikt.

Wetenschappelijke praktijk

In het eerste deel van dit proefschrift onderzoek ik hoe psychometrici (wetenschappers die psychologische fenomenen kwantitatief bestuderen) de netwerktheorie

en netwerkmodellen gebruiken in de *wetenschappelijke praktijk* om generaliseerbare kennis over psychiatrische fenomenen te verkrijgen.

In **Hoofdstuk 2** richt ik mij op de manieren waarop de netwerktheorie het ontstaan en het in stand houden van mentale stoornissen *verklaart*, met speciale aandacht voor de veronderstelde invloed van omgevingsfactoren. Ik analyseer wat voor verklaringen de netwerktheorie biedt en toets deze aan inzichten uit de wetenschapsfilosofie. De netwerktheorie biedt oorzakelijke, mechanistische en topologische verklaringen voor psychiatrische fenomenen; elk type verklaring vereist dat specifieke assumpties worden vervuld. Als de netwerktheorie mentale stoornissen zou beschrijven als *meerlagige* netwerken die verschillende domeinen omvatten, dan kan ze mogelijk meer verschillende soorten verklaringen bieden en daarmee haar verklarend potentieel verhogen.

In **Hoofdstuk 3** onderzoek ik hoe psychometrici de netwerkbenadering gebruiken om mentale stoornissen *wetenschappelijk* te *begrijpen*. Psychometrici kunnen psychiatrische fenomenen wetenschappelijk begrijpen als ze aan de hand van de netwerkbenadering kunnen nadenken over mogelijke manieren om het psychisch lijden geassocieerd met deze fenomenen te verminderen. Dit doen ze door afhankelijkheidsrelaties in de netwerktheorie of netwerkmodellen te manipuleren, bijvoorbeeld door elementen in de theorie of het model (in gedachten) te verwijderen, toe te voegen of te veranderen. De mate waarin de netwerkbenadering wetenschappelijk begrip kan verschaffen hangt daarmee af van de specifieke afhankelijkheidsrelaties in de netwerktheorie of netwerkmodellen, hun mate van coördinatie, de vaardigheden van psychometrici en de *pragmatische waarden* van netwerkrepresentaties (de kenmerken van deze representaties die psychometrici helpen ze te gebruiken).

In **Hoofdstuk 4** karakteriseer ik de *verkennende* functie van netwerkmodellen. Psychometrici kunnen netwerkmodellen gebruiken als een startpunt om zich mogelijke verklaringen voor psychiatrische fenomenen voor te stellen. De mogelijkheden die psychometrici zich kunnen voorstellen, zijn *epistemisch relevant* als deze in overeenstemming zijn met de beschikbare empirische en theoretische achtergrondkennis van psychometrici. Ook moet dergelijke achtergrondkennis zijn afgestemd op de afhankelijkheidsrelaties die het netwerkmodel laat zien en "empirisch adequaat" zijn, oftewel, overeenkomen met empirisch bewijs of wat we waarnemen in de observeerbare wereld. Daarnaast hangt de verkennende functie van netwerkmodellen af van hun pragmatische waarden, waaronder hun visualiseerbaarheid en transparantie (het kunnen weten hoe de input van het model vertaald wordt naar de output). Aan de hand van deze criteria duid ik het "verkennend potentieel" van een voorgestelde innovatie binnen de netwerkbenadering: het gebruik

van meerlagige brein-symptoom netwerkmodellen. Ik laat zien dat meerlagige brein-symptoom netwerkmodellen meer afhankelijkheidsrelaties tonen dan standaard netwerkmodellen, maar niet per se meer verkennende waarde hebben.

Klinische praktijk

In het tweede deel van dit proefschrift bekijk ik hoe clinici en cliënten de netwerkbenadering gebruiken in de *klinische praktijk* om na te denken over de psychische problemen van cliënten. Ik focus hierbij op het gebruik van *gepersonaliseerde netwerkmodellen*.

In **Hoofdstuk 5** onderzoek ik het epistemisch potentieel van gepersonaliseerde netwerkmodellen in de persoonsgerichte geestelijke gezondheidszorg. Gepersonaliseerde netwerkmodellen kunnen nuttige hulpmiddelen zijn voor persoonsgerichte zorg omdat ze worden geschat op basis van persoonlijke data en gepersonaliseerde variabelen kunnen bevatten. Omdat deze persoonlijke data, verzameld met *experience sampling methods*, contextgevoelig is en niet wordt gemiddeld over groepen, kan het deels de gangbare kritiek op het gebruik van kwantitatieve data voor het bestuderen van mentale stoornissen en andere sociale fenomenen omzeilen. Het personaliseren van netwerkvariabelen kan echter ook *afbakeningsproblemen* opleveren: Hoe bepaal je welke factoren wel of niet in het model moeten worden opgenomen? Ik laat zien dat een combinatie van praktijkonafhankelijke factoren (de representatieve en verklarende grenzen van persoonlijke netwerkmodellen) en praktijkafhankelijke factoren (de doelen en waarden van cliënten) kunnen helpen bij deze afbakening.

In **Hoofdstuk 6** onderzoek ik de relatie tussen gepersonaliseerde netwerkmodellen en zelfnarratieven. Wanneer we psychische problemen ontwikkelen, kunnen we een "psychiatrische verhaallijn" in ons zelfnarratief formuleren dat onze klachten omschrijft en begrijpelijk maakt voor onszelf en anderen. Deze verhaallijnen spelen een belangrijke rol in de constructie van gepersonaliseerde netwerkmodellen in de klinische context. Zowel gepersonaliseerde netwerkmodellen als zelfnarratieven kunnen worden gezien als geïdealiseerde, abstracte en reflexieve representaties van onze geleefde ervaringen die ons helpen na te denken over onze psychische problemen. Ze zijn echter niet *epistemisch gelijkwaardig*, maar *epistemisch complementair*: Psychiatrische verhaallijnen maken deel uit van de achtergrondkennis die beïnvloedt hoe cliënten en clinici redeneren met gepersonaliseerde netwerkmodellen.

Dagelijks leven

In het derde deel van dit proefschrift richt ik mij op het *dagelijks leven*, en specifiek op hoe mensen met geleefde ervaring psychiatrische theorieën gebruiken om hun psychische problemen te begrijpen.

In **Hoofdstuk 7** onderzoek ik hoe psychiatrische theorieën onze psychiatrische verhaallijn kunnen beïnvloeden; hiervoor gebruik ik het biomedische model van depressie als casus. Dit model, en andere dominante psychiatrische theorieën, zijn vergelijkbaar met *master narratives*. Enerzijds presenteren deze theorieën oorzaken voor psychisch lijden die we in onze psychiatrische verhaallijnen kunnen verwerken; anderzijds kunnen deze theorieën beïnvloeden hoe wij en anderen deze verhaallijnen beoordelen. Hiermee hebben ze invloed op de coherentie van onze psychiatrische verhaallijnen en op de mate waarin deze verhaallijnen ons in staat stellen om een vermindering van ons psychisch lijden voor te stellen. Om de normatieve implicaties van deze invloeden te beoordelen, moeten we kijken naar de bredere sociale context waarin ons zelfnarratief is gesitueerd.

In **Hoofdstuk 8** analyseer ik hoe de netwerktheorie onze psychiatrische verhaallijnen kan beïnvoeden, waarbij ik focus op de strategieën die de theorie promoot om de complexiteit van onze psychische problemen te omarmen. De netwerktheorie presenteert twee manieren om de complexiteit van mentale stoornissen te vereenvoudigen: *decompositie* (het opdelen in losse onderdelen) en *abstractie* (het focussen op de dynamiek van het geheel). De psychiatrische verhaallijnen van mensen met een depressie diagnose reflecteren beide strategieën: Ze delen hun problemen op in losse delen wanneer ze de multifactoriële oorzaken van hun depressie beschrijven, en abstraheren naar het grote geheel ze wanneer ze hun geleefde ervaring omschrijven. Dit betekent dat, afhankelijk van welke strategie de nadruk krijgt, de netwerktheorie onze psychiatrische verhaallijnen op verschillende manieren kan beïnvloeden. In plaats van tegenstrijdig te zijn, kunnen deze strategieën elkaar in deze context juist versterken.

Conclusie

Mijn praktijkgericht perspectief biedt een alternatieve kijk op de waarde van de netwerkbenadering en draagt daarnaast bij aan discussies over de geïdealiseerde wereld van de psychiatrie in het algemeen. Het vraagt enerzijds om bescheidenheid van professionals die psychiatrische benaderingen gebruiken; anderzijds biedt het een nieuwe manier om ons te verhouden tot de geïdealiseerde wereld die de psychiatrische wetenschap, de klinische praktijk en de populaire cultuur ons voorspiegelen.

Summary (EN)

Introduction

When dealing with mental suffering, we navigate between two worlds. The "intractable" world of mental suffering itself is often difficult to understand and explain; the ways we conceptualize and study mental suffering belong to the "idealized" world. In this latter world, we generalize and simplify the lived experience of mental suffering to seek patterns, explanations, or entry points for therapy. Throughout history, the landscape of this idealized world has shifted; every few decades, a new psychiatric approach emerges that encompasses new ways of conceptualizing and studying mental disorders. Currently, psychiatry is entering another reinvention: The biomedical approach to mental disorders that has dominated the past few decades has not led to its promised breakthroughs. In response, systemic alternatives have emerged, such as the *network approach*. The network approach states that mental disorders are (stable sets of) causally interacting symptoms (the *network theory*) and advocates the use of *network analysis* to study mental disorders. Proponents of the network approach hope that this approach will provide knowledge where the biomedical approach is lacking. However, psychiatry's history teaches us that every psychiatric reinvention promises knowledge breakthroughs that have never fully materialized. How can we realistically evaluate the "epistemic potential" of the network approach, that is, its ability to provide knowledge of mental suffering?

In this thesis, I evaluate the epistemic potential of the network approach using a *practice-oriented perspective*. I examine how the network approach is used in scientific practice, clinical practice, and daily life to obtain knowledge about possibilities regarding mental suffering. Rather than evaluating the network approach only in terms of how well it reflects the reality of mental suffering – as is often implied –, my practice-oriented account also considers *how* it represents mental suffering, *who* uses it, and for what *purpose*.

Scientific practice

In the first part of this thesis, I examine how psychometricians (scientists who study psychological phenomena quantitatively) use the network theory and network models in *scientific practice* to obtain generalizable knowledge about psychiatric phenomena.

In **Chapter 2**, I focus on how the network theory *explains* the development and maintenance of mental disorders, with special attention to the proposed influence of environmental factors. I analyze which explanations the network theory offers and test them against insights from the philosophy of science. The network theory offers

causal, mechanistic, and topological explanations for psychiatric phenomena; each type of explanation requires that specific assumptions are met. If the network theory described mental disorders as *multilayer* networks encompassing different domains, it might provide a wider variety of explanations, improving its explanatory potential.

In **Chapter 3**, I explore how psychometricians use the network approach to *scientifically understand* mental disorders. Psychometricians can understand psychiatric phenomena scientifically based on the network approach by reasoning about possible ways to reduce the mental suffering associated with these phenomena. They do so by manipulating the dependency relationships in the network theory or network models, for instance, by (mentally) removing, adding, or changing elements in the theory or model. The network approach's ability to provide scientific understanding thus depends on the specific dependency relationships that the network theory or network models demonstrate, their coordination, the psychometricians' skills, and the pragmatic values of network representations (the features that help psychometricians use them).

In **Chapter 4**, I characterize the *exploratory* function of network models. Psychometricians can use network models as a starting point for reasoning about possible explanations for psychiatric phenomena. The possibilities that psychometricians can imagine are *epistemically relevant* if they are consistent with psychometricians' available empirical and theoretical background knowledge. Such background knowledge must also be coordinated with the dependency relationships demonstrated by the network model and be "empirically adequate," meaning it conforms to the observable world or empirical evidence. Moreover, the exploratory function of network models depends on their pragmatic values, including their visualizability and transparency (the ability to know how the model's input is translated into its output). I use these criteria to examine the "exploratory potential" of a proposed innovation in the network approach: the use of multilayer brain-symptom network models. I demonstrate that these multilayer network models show more dependency relationships than standard network models but do not necessarily have more exploratory value.

Clinical practice

In the second part of this thesis, I examine how clinicians and clients use the network approach in *clinical practice* to reflect on clients' mental problems, focusing on the use of *personalized network models*.

In **Chapter 5**, I explore the epistemic potential of personalized network models in person-centered psychiatric care. Personalized network models can be useful

tools for person-centered care because they are estimated based on personal data and can include personalized variables. Because this personal data, obtained with *experience sampling methods*, is context-sensitive and not averaged over groups, it can circumvent some standard critiques of using quantitative data to study mental disorders and other social phenomena. However, personalizing network variables can also introduce *boundary problems*: How do we determine which factors should (not) be included in the model? I show that a combination of practice-independent factors (the representational and explanatory boundaries of personalized network models) and practice-dependent factors (clients' goals and values) can help with this demarcation.

In **Chapter 6**, I examine the relationship between personalized network models and self-narratives. When we develop mental problems, we may formulate a "psychiatric narrative thread" in our self-narrative that describes our mental problems and makes them understandable to ourselves and others. These narrative threads play an important role in the construction of personalized network models in the clinical context. Both personalized network models and self-narratives can be conceptualized as idealized, abstract, and reflexive representations of our lived experiences that help us reason about our mental problems. However, they are not *epistemically equivalent* but *epistemically complementary*: Psychiatric narrative threads are part of the background knowledge that influences how clients and clinicians reason with personalized network models.

Daily life

In the third part of this thesis, I focus on *daily life* and specifically on how people with lived experience use psychiatric theories to make sense of their mental problems.

In **Chapter 7**, I explore how psychiatric theories can influence our psychiatric narrative threads; to do so, I use the biomedical model of depression as a case study. Dominant psychiatric theories, such as the biomedical model, are comparable to *master narratives*: They present causes for mental suffering that we can incorporate into our psychiatric narrative threads, and they influence how we and others judge these narratives. In doing so, these theories can influence the coherence of our psychiatric narrative threads and their ability to help us imagine possibilities for reducing our mental suffering. To assess the normative implications of these influences, we should look at the broader social context in which our self-narration is situated.

In **Chapter 8**, I explore how network theory can influence our psychiatric narrative threads, focusing on the strategies the theory promotes for embracing the complexity of our mental problems. The network theory presents two ways to simplify the

complexity of mental disorders: *decomposition* (breaking them down into separate parts) and *abstraction* (focusing on their overall dynamics). The psychiatric narrative threads of people with a depression diagnosis reflect both strategies: They divide their problems into separate parts when describing the multifactorial causes or constituents of their depression and abstract when describing their lived experience. This implies that, depending on which strategy is emphasized, the network theory can influence our psychiatric narrative threads in different ways. Rather than being contradictory, these strategies may actually complement each other in this context.

Conclusion

My practice-oriented account offers an alternative view of the value of the network approach and contributes to discussions about the idealized world of psychiatry in general. It calls for humility from professionals who work with and disseminate psychiatric approaches while offering a new way of relating to the idealized world presented to us by psychiatric science, clinical practice, and popular culture.

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About the author

Nina S. de Boer (1996, Rheden, The Netherlands) obtained her Bachelor of Science in Liberal Arts and Sciences (summa cum laude) at Amsterdam University College. She completed her Master's in Philosophy of Neuroscience and Research Master's in Neurosciences (cum laude) at the Vrije Universiteit in 2019 and 2020. She worked as a research assistant at the Amsterdam Institute for Addiction Research at the Amsterdam University Medical Center (location AMC). In 2020, she started working as a PhD candidate in philosophy at Radboud University within the Center for Cognition, Culture, and Language. Her research focused on the epistemic potential of the network approach to mental disorders. She was a visiting researcher at the University of Edinburgh (Edinburgh, UK) and the Egenis Research Centre (Exeter, UK). Additionally, Nina has taught various philosophy courses to bachelor's and master's students. Nina currently works as a postdoctoral researcher within the Center for Cognition, Culture, and Language. Next to her academic work, she worked as a moderator and speaker at popular scientific events.

As the biomedical approach to mental disorders has fallen short of its promised breakthroughs in our knowledge of mental suffering, systemic alternatives – like the *network approach* – are gaining traction. But given psychiatry's history of promoting reinventions with big promises and unrealistic expectations, how should we evaluate the *epistemic potential* of the network approach?

In this thesis, Nina de Boer formulates a practice-oriented response to this question, focusing not just on how well the network approach reflects the reality of mental suffering but also on *how* it represents mental suffering, *who* uses it, and for what *purpose*.

By examining its use in scientific practice, clinical practice, and daily life, De Boer offers a new perspective on the value of the network approach and contributes to broader debates about the epistemic potential of psychiatric reinventions.

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