Reformulating the network theory of mental disorders: Folk psychology as a factor, not a fact

Freek Oude Maatman
Radboud University Nijmegen

Abstract
Borsboom et al. (2019) argue that the network theory of mental disorders, if correct, blocks the biological reduction of mental disorders. This is mainly argued through a partial reformulation of network theory which combines multiple realizability of symptoms with a realist interpretation of folk psychological explanations. In this article, I argue that (a) the latter is problematic and that (b) the combination of these arguments voids the previous predictive and explanatory power of network theory. I then present a novel way in which network theory could avoid biological reductionism by considering folk psychology not as a fact, but as a structuring cause of causal connections between intentional state symptoms, together with culture and time period. Drawing from this, a novel principle for network theory is proposed, which allows it to retain force against reductionism while also retaining predictive and explanatory power.

Keywords
folk psychology, mental disorders, network, psychopathology, reductionism

The network theory of mental disorders (NT) has over the last decade become a mainstay in academic discussions of psychopathology (e.g., McNally, 2019). First proposed by Denny Borsboom (2008) and further refined over the following years by what deserves to be called an “Amsterdam School of Psychopathology” (e.g., Borsboom, 2017; Borsboom & Cramer, 2013; Borsboom et al., 2019; Cramer et al., 2010; Fried & Cramer, 2017; Schmittmann et al., 2013), NT offers an alternative to the recent “biologized,” reductionist
common-cause thinking about mental disorders. Instead of understanding a specific mental disorder as arising from a single underlying disturbance of the brain, the body, or its organs due to a pathogen or dysfunction (i.e., a somatogenic interpretation), NT argues that mental disorders are networks of interacting symptoms, without a single common cause existing (Borsboom, 2017; Borsboom et al., 2019; Fried & Cramer, 2017). According to the NT, individual symptoms enter into a mereological relationship (i.e., part–whole) with the disorder; they are parts of a self-propagating feedback network that constitutes the disorder as a whole, and not consequences of a common cause.

This difference in interpretation between “somatogenicists” and network theorists at first sight seems to be serious and relevant. If we believe that mental disorders are in fact symptom networks that “cause themselves,” searching for a single underlying pathogen or cause is misguided. This position places NT in direct conflict with the commonly adhered to disease model of mental disorders, which does hypothesize the existence of unifying underlying causes for specific diagnoses.

From the broader somatogenic perspective, the network alternative is, however, not problematic at all: any higher level causal structure between symptoms must in some way be realized biologically, unless one subscribes to a dualist ontology (e.g., a Cartesian separation of mind and body as distinct substances). The disease model after all is not the only way to conceptualize a reduction to a biological basis; any causal links between symptoms are likely (if not necessarily) biologically realized as well. Such biological links could then be identified and studied, possibly allowing for a complete biological reduction to a biological causal structure instead of a single dysfunction or pathogen. By rigorously studying the biological realm, some proponents of biological reductionism even argue that it should be possible to identify which biological mechanisms are responsible for which symptoms (Insel & Cuthbert, 2015). Furthermore, they aim to reformulate existing diagnostic criteria from the bottom-up (e.g., Insel & Cuthbert, 2015; Pernu, 2019), allowing them the freedom to incorporate noncommon cause disorders. NT then is clearly not in conflict with the “somatogenic” research program; it simply draws attention to another possible way that mental disorders could be biologically realized, namely, as causal structures with inherent feedback loops.

Whilst recent biological reductionist projects have been extensively criticized in the literature (e.g., Doherty & Owen, 2014; Wakefield, 2014), the NT also holds several “aces up the sleeve” against these, which have been made explicit in Borsboom et al.’s 2019 article “Brain Disorders? Not Really: Why Network Structures Block Reductionism in Psychopathology Research.” In this article, Borsboom and colleagues target reductionist explanations of mental disorders through four arguments, besides their already mentioned attack on the common cause etiology:

1. Some symptoms of mental disorders are external to the individual and thus cannot be biologically reduced, even though they are causally relevant to the disorder.
2. The realizing biology of symptoms of mental disorders is likely to be multiply realized (i.e., be realized by different underlying biological mechanisms), especially those that refer to intentional states (i.e., mental states that are about something, such as beliefs, emotions, and desires). This would mean that the exact
biological mechanisms underlying mental disorders as a whole could be unique per individual.

3. (Inter)Relationships between intentional state symptoms can only be understood by assuming rationality and causality; in other words, by adopting an “intentional stance” towards mental disorders (Borsboom et al., 2019, p. 20; Dennett, 1987). Any attempt at biological reduction of such states would lose intentional information, making it a worse fit than the intentional stance for the goal of explanation and understanding of mental disorders, even without taking multiple realizability into account.

4. Symptom networks underlying mental disorders appear to be dependent on cultural background. This information cannot be retained in biological reductions, and adds a further layer of multiple realizability.

Whilst I am highly sympathetic to NT and recognize that, taken together, these arguments carry force against the biological reduction of mental disorders, I believe these arguments also entail a highly undesirable reformulation of NT. More specifically, in this article I will argue that the arguments proposed by Borsboom et al. (2019) reduce NT to a network interpretation of common folk psychological practice, which provides no further use to clinicians and scientists than being an alternative to biological reduction. As I believe this would amount to a waste of a highly promising theory, I then propose an additional principle for NT that allows it to retain its promise as an explanatory theory of psychopathology, as well as one that provides us with novel predictions of psychopathological ontogeny, whilst retaining the current antireductive force NT was provided with by Borsboom et al. (2019). First, however, it is necessary to discuss the older NT (as per Borsboom, 2017) and its pending reformulation as entailed by the arguments provided in Borsboom et al. (2019) in detail.

**Network theory and its pending reformulation**

As mentioned in the introduction, NT can be summarized by stating that mental disorders are constituted by symptom networks. A symptom network effectively describes a feedback loop between symptoms of a disorder, which allows them to “cause themselves.” The symptoms, then, are components that together make up the disorder; there is a mereological relationship between the two.

There is more than the above to be said about NT. An important distinction needs to be made before we continue. There has been serious discussion about the possible tension between NT and latent variable models (e.g., Bringmann & Eronen, 2018), primarily due to an adversarial framing between NT and these in earlier publications (e.g., Borsboom & Cramer, 2013). This discussion, however, is primarily occupied with the psychometric properties of network modeling as opposed to latent variable modelling. What I am concerned with here is not the method but the content of the theory, namely the ontological commitments of NT and their empirical consequences (Haig & Borsboom, 2012). These ontological commitments are what follow when the theory is interpreted realistically; it is what the theory is committed to ontologically, or, in other words, the structure it hypothesizes to be the case in the real world.
The ontological commitments of NT become most clear in a 2017 summarizing article by Borsboom. There, he characterizes the theory through four core principles:

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 network structure*: The psychopathology network has a nontrivial 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, 2017, p. 7)

Principle 1 has already been implicitly discussed above, whilst principle 2 (symptom–component correspondence) is described as a pragmatic idealization in the same article (Borsboom, 2017, p. 11), which is likely to be refuted or reformulated later. This leaves principles 3 and 4 as serious commitments of NT. Of these, principle 3 (direct causal connections) has the strongest ontological commitments; it predicts “direct” causal connections between symptoms in the network. Notably, these connections can be instantiated by nonsymptom psychological processes or biological mechanisms (Borsboom, 2017, p. 7), which allows for mediation of these causal connections.

Principle 4 offers another interesting commitment, derivable from its inherent prediction. If the topology of the psychopathology network (i.e., the network of all symptoms in psychopathology) is nontrivially grouped due to symptom interrelations, mental disorders could in principle be derived from the clusters in the psychopathology network by itself (see Borsboom et al., 2011, for a demonstration). This would, however, require that symptom relationships persist over individuals and groups, committing NT to the general stability of symptom interrelationships within a network. In other words, the same relationships between symptoms should exist over individuals and groups; for example, suicidal ideation should be positively linked to sadness, but negatively to mania for all individuals, if linked to mania to begin with.

This earlier formulation of NT then appears to be relatively commitment light. To summarize, whatever symptoms are taken up in the network, these must be (a) causally connected to other symptoms in the network, which would lead to (b) similarly structured symptom networks over individuals and groups, whilst the causal connections in these networks must be (c) similar enough in direction (i.e., positive or negative) over individuals and groups for the symptom networks to crystallize into the same disorder over these. Lastly, there is the commitment of principle 1, that is, the core commitment of NT that (d) there is no common cause, as the symptom network “causes itself.”
Notably, this formulation leaves a lot of details open to be interpreted. The symptoms and their causal connections can, for example, be located on any level of explanation—be it genetic, neurological, mental, environmental, or cultural—as there is no commitment to any specific such level (Borsboom, 2017, p. 11). Furthermore, NT may in this formulation even lead to a reformulation of current diagnostic categories, if it turns out that symptoms cluster differently than was previously thought. As was mentioned in the introduction, this is also largely compatible with biological reductionism, as this 2017 version of NT does not exclude the possibility that symptom feedback loops can be completely biologized.

**Dealing with reductionism: The 2019 reformulation**

In their 2019 paper, Borsboom et al., however, add several new ontological commitments to this previous formulation in order to explicitly avoid the compatibility with biological reductionism, mainly drawing from philosophy of mind in doing so. In the introduction, arguments 1 through 4 were already mentioned. Of these, argument 1 has already partially been covered in the above discussion—it describes the fact that some current symptoms are external to the individual and thus unsusceptible to biological reductionism. This is not very problematic for biological reductionists, as such symptoms can be simply excised from future diagnostic criteria, or merely used as nonnecessary causes or consequences of the “real” underlying biological issues, as they are dependent on the organization of the external world. A similar case can be made against argument 2 (the multiple realizability of mental states) if considered by itself, on the basis of recent arguments against multiple realizability in general, as already has been pointed out by Eronen and Bringmann (2019).

Of the other arguments, argument 3 appears the strongest argument against reductionism, but also creates the most problems for NT as a whole, especially when combined with argument 2—more on which follows below. Through argument 3, Borsboom et al. (2019) draw attention to the fact that many current symptoms of mental disorders are defined as mental states with intentional content (i.e., mental states that are about something, such as beliefs and desires). Suicidal ideation, for example, can only be defined as thoughts about suicide. The attribution of such “intentional states” allows us to explain behavior in daily life; for example, we may explain my smiling after receiving ice cream by saying that I am enjoying the ice cream.

Daniel Dennett (1987) describes such explanation as “adopting the intentional stance,” which refers to adopting the folk language of beliefs, emotions, and desires (i.e., folk psychological language) to explain the behavior of an individual or system. Such talk allows us to predict and understand each other more efficiently, though less accurately, than if we were to explain each other’s behavior on a biological level. Furthermore, the predictive power of such language can also be used to describe and predict nonhuman systems in which such states are folk psychologically implausible, such as when predicting the moves of a chess computer by assuming that it “wants to win the game.” Notably, Dennett delimits this to attribution of mental states; it is not necessary that these states actually exist for their explanatory value to exist. They tap into real patterns in the world; the chess computer in the example in fact always plays such that we can describe it as
playing to win, which may or may not be caused by a desire. Using this belief–desire language simply allows us to take this fact into account without requiring too many steps or too much time.

Borsboom et al. (2019) use the above to argue that, in many cases, understanding the interrelations between intentional symptoms biologically does not make much sense; the sense-making only occurs through adoption of the intentional stance (p. 20). In doing so, they also argue that this makes sense according to the causality and rationality associated with intentional states (Slors et al., 2019); the intentional states can be said to cause each other due to leading to each other rationally. For example, someone who is afraid of contamination and believes that washing one’s hands can protect oneself from this will wash their hands compulsively. In other words, Borsboom et al. (2019) mean that intentional states that, rationally, should enter into a causal relationship (in a minimal “if X, then Y” sense), can in principle be assumed to actually have these hypothesized causal properties in rational actors.

Although there are problems with using rationality in psychopathological contexts (see Slors et al., 2019), this move also adds another ontological commitment to NT, namely, to the correctness of our common folk psychological explanatory practices. Attributing a fear of contamination and the belief that handwashing helps combat this to someone to explain their compulsive handwashing—as per the standard interpretivist interpretation (Dennett, 1987; also see Slors et al., 2019)—after all is something other than using these folk psychological explanations to ground causal relationships in a scientific, explanatory theory of psychopathology. The latter, however, are what Borsboom et al. (2019) need in order for the NT to have any force against their biological reductionist target, as well as for the NT to be useful for clinical practice, for example, in order to argue that targeting certain symptoms can resolve a mental disorder due to their high centrality in a symptom network (e.g., Robinaugh et al., 2016). Furthermore, they need this to uphold their own principle 3, NT’s commitment to direct causal connections between symptoms. In order for folk psychological explanations to factor in such a causal theory, they need to correctly describe actual causation in the world.

Folk psychology as a fact: Ontological issues and further criticism

Yet, committing to a realist interpretation of folk psychology is problematic for multiple reasons. First, this places serious strain on NT’s usage of interpretivism. Unlike Dennett and other interpretivists (e.g., Slors et al., 2019), Borsboom et al. (2019) need to interpret folk psychology as more than simply a theory that taps into “real patterns” for it to serve as a grounding theory for NT, that is, as a theory that correctly represents causation in the world (e.g., Haig & Borsboom, 2012). Real patterns, after all, can still be biologically reduced (Eronen & Bringmann, 2019), voiding the strength of the current move, whilst an antirealist interpretation of a folk-psychologically based NT (as proposed by Van Loo & Romeijn, 2019) carries little force against the (very realist) biological reduction program, as well as relatively weak grounds for continued usage of NT, as will become clear below.

Using folk psychology as a grounding causal theory for NT’s use of intentional symptoms and their posited interrelationships furthermore would require that our everyday
exploratory practices somehow must have hit upon the (exactly) right causal network structure for higher level mental states, and upon a correct delineation of these mental states from each other (or at least one that correctly describes this). Such a claim, however, is quite suspect; not only do we consider most previous folk psychologically posited causes as misguided (e.g., possession by the devil, witchcraft), but folk psychology is also not intended to be a scientific theory, nor has it been tested at all. In fact, folk psychology is even often informed by our current scientific knowledge, such as our current use of the word “dopamine” as a replacement for “happiness” (e.g., “I get such a dopamine rush from painting,” see Francken & Slors, 2018). Notably, psychological research and practice also have indirectly caused the current folk psychological reification of diagnostic categories such as depression (e.g., “I have less energy because I have depression.”) simply by introducing these terms to the public—leaving in the middle whether this reification is justified or not. In both aforementioned cases, sense is made of a certain relationship in a folk psychological context. However, for clinical or scientific purposes, both of the aforementioned claims are as of yet unproven, and in the latter case, a common cause explanation is even folk-psychologically acceptable (i.e., “it makes sense”).

The former two folk psychological examples (i.e., dopamine and the reification of depression) also tap into a further problem with NT’s new reliance on folk psychological explanation: folk psychological explanations are not stable. What “makes sense” in our daily practice of explaining each other is dependent on not only the knowledge we have, as was pointed out above, but is also dependent on the time period, culture, and language community we live in. Explaining someone’s depressive symptoms as being caused by the devil or idleness made sense in the Middle Ages, whilst explaining it through a melancholic personality made sense in the 19th century. Some folk psychological explanations may be language specific, such as the wide range of words the Chinese have for “happy,” possibly leading to different connotative links between symptoms. And as Borsboom et al. (2019) point out themselves when discussing argument 4, in WWII-era Japanese culture it may have made sense to have suicidal ideation after a loss of honor, whereas in Catholic culture it most certainly does not.

Lastly, there is the problem that folk psychological explanations are often not exhaustive; upon attribution of a further mental state, a different outcome may make more sense. For example, if I have a fear of contamination and think that washing my hands eliminates contamination, I might still not wash my hands due to a conflicting fear that the government put acid in the water system, unless I also believe that I am immune to acid, and so on, ad infinitum. This makes intentional state–symptom interactions heavily underdetermined by folk psychological explanations in all cases.

If the causality of the intentional parts of NT’s symptom networks is to be grounded in folk psychological explanation, adherents of the theory then have a very difficult task to perform. Given the demonstrable variety of folk psychological explanations, they then after all have the (unfortunate) task of having to decide which of folk psychology’s proposed causal relationships (and perhaps even which folk psychology) are in fact correct—for example, why do “rational” interrelationships between mental states deserve priority over say, the common-cause-like folk psychological explanations that also exist? The sheer fact that the former “make sense” to us, whilst the latter do so as well for many, amounts to little more than an intuitive preference as long as we look at the intentional
level only. When taking into account other cultures and languages with different intentional state relationships (or “sense making”), or possible future changes to folk psychology, even more issues arise. More than further folk psychological explanations as support is needed for settling this question, then.

Intuitively, we may then be inclined to turn towards the biological level to ground our folk psychological explanations. Whatever causality occurs within the individual, it must still occur through some aspects of the biological level as well, unless one adopts an antiphysicalist position. Yet, Borsboom et al. (2019) block this move through argument 2, which introduces the notion of multiple realizability to the debate. As mentioned in the introduction, multiple realizability refers to the possibility that mental states can be realized by nonidentical underlying biological mechanisms in different individuals at the same time, as well as within the same individual at different times. As an extreme but clarifying example, the biological mechanism responsible for a fear of contamination may be, say, a group of serotonergic neurons in the orbitofrontal cortex of one individual, but a group of mixed dopaminergic and serotonergic neurons in the amygdala of another individual, and so on. Not only would this be problematic for biological reductionism in general (though not for revisionist somatogenicists, see Pernu, 2019; nor for sophisticated reductionists, see Eronen & Bringmann, 2019), as this would mean that there are no clear reductions to be made from symptoms to a single underlying biological mechanism, it also makes the biological level relatively uninformative for determining which mental state someone is in—let alone for determining the causal efficacy of these mental states. Furthermore, if the folk psychological underdetermination of behavior by mental states is taken into account, the relevant multiply realized mental states for symptom networks are possibly infinite, and their combination is likely to differ over individuals as well. In the reformulation of NT proposed by Borsboom et al. (2019), the biological level then cannot be used to ground the folk psychological relations either.

This leaves us in a theoretical limbo. The only way to explain the causal relationships involving intentional symptoms, therefore, is through folk psychology, but that very folk psychology is at best highly underdetermined, as was argued before. When assuming multiple realizability, there then appears to be no way to tractably prove folk psychological explanations within NT at all; we simply need to have faith in our folk psychology “getting it right.” And it must go for all folk psychologies that NT may use to explain the interconnections of intentional states in a symptom network. Argument 4, the argument that there may be differently organized symptom networks over cultures, appears to already fully embrace this consequence, as Borsboom et al. (2019) exemplify it through the aforementioned differences between Japanese and Catholic cultures regarding suicidal ideation. In order for there to be a difference in the causal relationships between symptoms between Japanese and Catholic cultures within NT’s reformulation, this needs to be present in their respective folk psychologies, as it needs to make sense in that culture. Moreover, we also need to trust that the respective folk psychologies of these cultures both get it right for their respective populations.

Overall, the combination of arguments in Borsboom et al. (2019) thus leaves NT severely weakened. Symptom networks that include intentional symptoms become inherently irreducible and dependent on the truth of their relevant explanatory folk psychologies, as within the reformulation of NT, there also is no constraint on causal
relationships stemming from intentional symptoms other than their “sense-making” within these folk psychologies. In effect, then, NT’s explanatory power becomes coupled to that of folk psychologies across the world, except for the exclusion of folk psychological common-cause explanations. This inflates the relatively commitment-light NT of Borsboom (2017) to a theory that is fully committed to the (correspondence) truth of all noncommon-cause folk psychological explanations regarding the symptoms of mental disorders. Or, in other words, to a network interpretation of folk psychology, as was mentioned in the introduction.

Some may find this folk psychological interpretation of NT unproblematic still. For these people, a further point can be made regarding the predictive power of this new version of NT. In its first conception, the predictive power of NT is linked to the conceived symptom network only; the theory is an alternative to common cause interpretations, and not much more than that. For the reformulation of the NT, this has changed. As mentioned above, by explicitly committing to folk psychology and intentional states, NT now is also committed to the truth of folk psychological explanations, especially if interpreted realistically, as Borsboom et al. (2019) need to and appear to do (Van Loo & Romeijn, 2019). Yet, on the 2019 reformulation, the causal power of mental states (i.e., in virtue of their rationality) can in principle never be verified without a technique to identify the intentional content of such states—even upon identification of a causal role-fulfilling brain state, we would be unable to verify whether this state has the appropriate intentional content. As the biological level by itself is to them essentially uninformative for determining what mental state someone has, the only remaining way to gauge this content then would be introspection—which is generally considered to be highly unreliable, and in this case could be considered circular as well.

Furthermore, due to this lack of verifiability coupled with adherence to intentional states, all deviation from hypothesized symptom networks could in principle be explained within the framework. If someone’s modeled intentional state relationships do not “make sense” at first sight, there could always be a further intentional state that does rationalize one’s behavior, which may not be introspectable—and certainly not measurable without advances in our knowledge of intentional content measurement. If someone’s symptom network differs greatly from that of others or the norm, this may be due to a difference in their beliefs (leading to different intentional state interrelationships), general individual differences due to development, cultural differences, and differences in the relevant external field of the individual. Even if all of these appear to be the same, a difference can still be explained by referring to the immeasurable intentional content, or to the unreliability of introspection, or, as a last-ditch effort, to irrationality.

This makes both the new NT and its symptom networks not only completely immune to falsification, but also predictively worthless; adopting multiple realizability at a cultural and individual level limits its predictive power to that which is (idiographically) empirically obtained, as it can give no grounded a priori estimate of symptom network stability. After all, there is no principled way for it to argue that symptom networks in certain populations will be relatively similar outside of existing folk psychological sense-making. Folk psychologically, however, it also makes sense that people are different. Then, however, the reformulated NT does not add anything to folk psychology; it merely adopts it and throws out all of the common causes in it. And this connection with folk
psychology is not a strength of NT itself, as proponents of NT sometimes state (e.g., Borsboom, 2017; Borsboom et al., 2019), if NT itself is essentially a formalized folk psychology without common causes—of course, outside of the biological reductions of certain symptoms that it may adopt over time.

In conclusion, the problems with Borsboom et al.’s (2019) reformulation are two-fold. By adopting folk psychology in the causal NT, the NT becomes ontologically committed to the truth of folk psychological explanations. However, due to the new NT’s reliance on multiple realizability and the resulting uninformativity of the biological level of explanation, it has no grounds (yet) to explain why or how folk psychology is correctly describing causality between mental states, let alone argue that the mental states posited by folk psychology are correctly delineated states with the correct causal powers. These claims about folk psychology, then, become mere assumptions of the theory. What NT now seems to need in order to ground itself is an argument for the correctness of folk psychological explanations, through a path that is not biological. As this is missing, but since the new NT still commits to a folk psychological explanation, it has no predictive power of its own, nor are any posited causal relationships between intentional states (or symptom networks including these) verifiable or falsifiable.

Another answer to biological reductionism: Folk psychology as a factor, not a fact

In my view, the above consequences of Borsboom et al.’s (2019) arguments are unnecessary. If one wishes to argue that mental disorders are unlikely to be fully biologically reduced at the same time as retaining the framework of the earlier NT (i.e., Borsboom, 2017), there are better options than ontologically committing to folk psychology. In this section, I will describe my preferred option for avoiding a complete biological reductionism in full. This argument forms the groundwork for my later reformulation of NT’s framework, as it also provides a nonbiological route to grounding folk psychological causation.

In short, my contention is that NT can tap into the promise of folk psychology and cultural variation as an argument against biological reductionism, without needing to accept folk psychological explanations as ontologically real or true, whilst also not dropping realist pretensions. Instead, I argue that by first interpreting folk psychology as making behavior intelligible (i.e., by providing us with explanatory reasons, but not causes) for us in daily life, followed by acknowledging that these reasons can function as structuring causes (Dretske, 1988) due to our embeddedness in a shared cultural community, it becomes possible to invert the relationship between folk psychology and mental disorders in NT from being descriptive of causality into being partially formative for symptom networks (i.e., as a factor, not a fact). After what follows, I hope this highly condensed summary has become intelligible.

Causes and reasons

Let us start by delineating between the aforementioned causes and reasons. Both causes and reasons are used in explanation, yet, generally taken, they describe two separate kinds of explanation. When something is described as a cause, the explanandum is conceived to
be a consequence of this cause. As an example, take the case of the successful swatting of a gadfly. The crushing and consequent death of the gadfly is the result of the movement of your hand; the movement of your hand caused the crushing and death of the gadfly, and not something else. When positing such a causal explanation, an ontological commitment is made to its truth (Haig & Borsboom, 2012), that is, the commitment that this is the actual interaction that occurs or has occurred in the world.

In the case of a reason, however, the explanandum is merely made intelligible (Alvarez, 2009; Sandis, 2006). Through a reason, an explanation is given of why something occurred, but this reason does not show the inevitability of the event. For example, I may close the shutters because of my delusion that the CIA is following me. This is not an inevitable event following from this single belief; if I believed that the CIA might know that I know they are following me (a further intentional state), then I may have not closed the shutters on purpose. Furthermore, I also must believe that closing the shutters somehow “protects” me from the CIA, and so on, and so on.

So far, reasons may not seem to be very different from causes. They both explain why something occurs. Yet, both serve very different roles in explanation. The following example may be helpful here, which I have lifted from Stoutland (1998): while driving my car, I stop for a stop sign. The cause of my stopping, in the actual sense, is a chain of events: light from the stop sign hitting my eye, some resulting neural activations, my foot pushing down the brake pedal of my car and the components of my car then making the car stop. Yet, this does not explain why I stopped at the stop sign; it does not make my act intelligible for others. For this we need reasons; I may firmly believe in following traffic rules, or want to avoid causing accidents, or like to look at stop signs, or simply recognize the traffic rules regarding stop signs, or want to avoid being ticketed, or a combination of these (or more).

Normally, we take such reasons to also be part of the causal chain, which is also what Borsboom et al. (2019) do in order to use folk psychology in their causal explanation of psychopathology. However, as described before, such an interpretation leads to complicated ontological scenarios, in which all of my relevant beliefs and desires need to be consulted before I, say, stop at a stop sign. This also leaves us with commitments to the existence of beliefs and desires at some level of reality and to their causal power for behavior, which are the subject of decades (if not centuries) of philosophical infighting. In the case of the reformulated NT, it left us with a commitment to the causal correctness of folk psychology.

To avoid such consequences, I propose that we interpret the “rational,” semantic relationships between intentional states that Borsboom et al. (2019) discuss and our attribution of such states to others as reasons. The “rationality” merely refers to our folk psychology making the relationships between intentional state symptoms intelligible; we use it to explain our actions to each other, but this does not have to commit us to the truth of folk psychology all together.

Moving from reasons to causes: Structuring causation

This move at first appears to play right into the hands of biological reductionists. Intentional state symptoms then, after all, do not describe what is “really there,” such as
“real causes” do—that is, the biological causes underlying mental disorders. Drawing this conclusion is premature, however. As we already noticed when discussing Dennett (1987), our reasons do give us explanatory power; by attributing a certain “reason” for behavior, we can predict future behavior more easily than by exhaustively listing the biological causal chain. Furthermore, in some cases, the actual causal chain itself appears to be underdetermined, for example, why does a loss of honor lead to suicidal ideation for a given WWII-era Japanese person, but not for a Catholic?

We cannot make sense of this difference purely at the biological level; the answer necessarily is that the Japanese individual’s brain is wired to react as such to the loss of honor, and that the Catholic’s is not. And, notably, given that this difference indeed is the case, such an explanation would be necessarily correct. The problem, however, is that this gives us very little predictive or explanatory power at the biological level only; we cannot know what someone does until we either know how their brain is wired (which may run into multiple realizability), or until we know what they actually do—which we can then explain by stating that they were wired that way. Notably, this is similar to the reformulated NT’s problems with predictive power. Only in this case, we have trouble gaining a prediction to begin with, instead of having issues with finding the correct one. And once more, information from another level is needed to make the theory informative again.

In the example of the WWII-era Japanese and Catholic reactions to losses of honor, it is the cultural level of explanation that is needed. WWII-era Japanese individuals considered losses of honor reasons to consider and/or commit suicide because they learned this association. The link between a loss of honor and suicide was ingrained in their culture, in their belief system, and in their folk psychology; honor is important, and the only way to regain honor after a significant loss of it is by committing suicide. For the devout Catholic, exactly the opposite relationship has been internalized; committing suicide at any given point condemns one to hell, and therefore should be avoided at all costs. Notably, however, these are reasons: when explaining themselves to others or deciding upon a course of action, Japanese and Catholics can cite these reasons—and similarly, they may be asked why they did (or did not) act in reason-conforming ways within their respective cultures. These reasons then also play a role in the culture-specific folk psychologies as attributable beliefs, and are considered rational against the larger background of norms, values, knowledge, and institutions of their respective cultures.

This leads to a conundrum. The biological level—which must instantiate the actual causation—offers too little information to be informative, but it appears that the cultural level only supplies us with reasons for the supposed causal relationship. Yet, by taking the concept of structuring causation (i.e., the cause why \( X \) causes \( Y \)) and further cues from the work of Fred Dretske (1988), I believe there to be a way out.

My account starts by acknowledging that the actual causes remain the neural states underlying intentional states. However, in cases such as the WWII-era Japanese honor-suicide relationship, these neural states have only acquired their causality in virtue of reasons, that therefore function as causes. I will use the aforementioned WWII-era Japanese honor-suicide relationship as an example. During their development, a WWII-era Japanese child would have to learn to recognize “losses of honor” due to their relative importance in the Japanese culture of the time. Some neural state \( I \), activation of which
is indicative of loss of honor situations, will then have to be constituted or recruited through neural plasticity. Yet, because in this Japanese culture losses of honor are also reasons for suicidal ideation, the activation of neural state $I$ will, given proper acculturation of this individual, also become a cause of the neural state $S$ that underlies the mental state of suicidal ideation, simply due to the learning and internalizing of this rational relationship. In other words, the fact that $I$ is an indicator of losses of honor then leads it to be recruited to also cause activation of $S$, due to the embeddedness of the individual in this cultural environment, which requires and reinforces this connection through normative enforcement (i.e., acting in accordance with the culture is rewarded, deviance is “punished”).

In this sense, the loss of honor is not only a reason for, but also a cause of the suicidal ideation. This is the case because the semantics of losses of honor within the WWII-era Japanese culture are why $I$ gains the causal power regarding $S$, and not $I$’s biological properties; given that an individual grew up in a different culture (or time period) without the semantic/rational relationship between losses of honor and suicidal ideation (e.g., a Catholic culture), $I$ should not have causal bearing on $S$ simply because this link is not learned. We are then not committed to a perceived loss of honor being a cause in virtue of its intentional content alone nor because of the direct causal power of “losses of honor” regarding suicidal ideation; instead we are committed to it being a cause of the suicidal ideation symptom in virtue of this semantic relationship occurring in the particular folk psychology and culture that a WWII-era Japanese person’s brain is acculturated to (see Note 1). On Dretske’s account, the Japanese folk psychology and culture then function as a structuring cause. More specifically, the (structuring) cause of activation of $I$ being a cause of activation of $S$ is the enculturation of the individual to the WWII-era Japanese culture and related folk psychology, within which losses of honor rationally lead to suicidal ideation.

It is important to note that this analysis works even if we want to differentiate between automatic, direct connections between intentional states/their underlying neural states (e.g., a perceived slight immediately causing anger about it; $I$ directly causing $S$), which works as just described, and causal connections that arise through the mediation of active, conscious self-reflection. Once more, let us use the WWII-era Japanese honor–suicide relationship as an example. In this latter case, the loss of honor is actively recognized by the individual instead of passively, leading (perhaps from $I$) to a derivative state $I'$ which realizes verbal thought about this fact (e.g., “I just lost my honor!”). Given proper acculturation and ensuing acceptance of the internal logic of the culture, $I'$ should cause another verbal thought realizing state $S'$ (e.g., “I should consider suicide”) that causes $S$—or simply cause $S$ directly, given that $S$ indicates active reflection about suicide—through more or less intermediate steps, as the rationality of this relation within the culture should be encoded in the verbal system especially.

If this analysis is accepted, the fact that this individual was embedded in a specific culture and folk psychology is a cause of this causal connection between intentional states, as well as a cause of the underlying neurological realization of $I$’s connection to $S$. Importantly, this also flips the role of folk psychology; the causal connection between these two intentional/neural states is not described by, but in fact caused by their culture-specific folk psychology.
Folk psychology as a factor: Consequences of structuring causation in network theory

Now it is time to move back to the NT. Given that folk psychology and culture can function not only as reasons but also as a cause of causal connections between intentional/neural states in virtue of these connections being “rational” or enforced within the specific folk psychology or culture, it is an easy step up to say that the same goes for culturally or folk psychologically “rational” connections between intentional state symptoms in symptom networks, as well as the causal connections from such intentional state symptoms to behaviors and other (nonintentional) symptoms. Folk psychological explanation, therefore, is not relevant for NT because it correctly describes causality within symptom networks or the brain, but because it is (partially) formative for the intentional state symptom connections in symptom networks. In other words, I am arguing for an inversion of Borsboom and colleagues’ (2019) argument: folk psychology does not correctly describe causation in the brain, but the brain “mirrors” our folk psychology and culture. Folk psychology then should not be considered a fact, but a factor.

This step solves most of the issues with the reformulation of NT that were pointed out in the first section. First, we do not have to assume a realist interpretation of folk psychology, nor do we need to assume that the causal connections between intentional state symptoms depend on rationality alone. Second, the predictive usefulness of folk psychologies is not explained by them having somehow hit upon the correct delineation of mental states, but instead by them being internalized during development within a specific culture. Third, the problem that folk psychology is both unstable over time and (sometimes) includes common cause explanations, can be explained by it being a cultural phenomenon, the structure of which can be influenced by dominant, time-specific cultural institutions (e.g., Bushido in WWII-era Japan and the Bible in Catholicism, but also science in our current culture).

However, the discussed underdetermination of symptom networks by folk psychology remains, and in fact is also applicable to culture; any individual may deviate from the cultural or folk psychological standard for any number of reasons, including the aforementioned further mental states or an explicit disagreement with dominant cultural values. Yet, this problem can be partially remediated by recognizing the aforementioned distinction between direct connections between intentional states (i.e., \(I\) directly causes \(S\)) and self-interpretation mediated connections between intentional states (e.g., \(I\) causes \(I'\), which causes \(S'\), which causes \(S\)). The former connection is established through conditioning; a continuous process of internalization occurs due to embeddedness in the (cultural) environment during development, where deviation is (socially) punished and conformity is rewarded (directly or indirectly). The latter type of connection relies on the following of culture/folk psychology-specific rationality, and therefore allows for mediation by any other type of semantically relevant belief or desire.

We can derive two predictions from this distinction. First, the difference in supposed causal chain entails that these two types of intentional state symptom connections might exist independently within an individual, and therefore also that they might differ within that individual. For example, the Dutch culture that I grew up in may have enforced an automatic connection between a perceived lack of work performance \((P)\) and stress \((Q)\)
due to the dominant value of “industriousness,” even though I might actively believe that I do not need to work all the time \((P'\) does not entail \(Q'\) or \(Q\)). Second, it entails that the underdetermination of intentional state symptom causation by folk psychology likely applies primarily to the latter, self-interpretative type of connection. Direct causal connections between any \(I\) and \(S\) are normatively conditioned over a long period of time, and should therefore be relatively impervious to short-term fluctuations in intentional states, due to the hypothesized difference in underlying causal chain. Even though this distinction does not solve the underdetermination problem entirely, it does reduce the possible variance in intentional state symptom connections within and across cultures that one might expect, especially if these can be argued to function through a direct connection.

Besides these solutions to the issues introduced in the first section, my proposed position also entails several further derivable predictions that add to NT’s falsifiability, and explanatory and predictive power, some of which I will outline in the remainder of this section. First of all, the above solution to underdetermination can be reframed as a prediction of greater similarity of intentional state symptom interconnections between individuals with the same cultural background. More specifically put, my position entails that the network of causal connections between intentional states should be more similar between individuals sharing the same cultural and folk psychological background.

The proposed position also predicts that a change in culture can (over time) cause a shift in the connections between intentional state symptoms, even within already acculturated individuals. For example, when the Japanese person from our running example would become a Catholic priest, this could lead to a disconnection of “loss of honor” and “suicidal ideation.” Notably, this prediction does not only apply to changes of the culture a person adheres to, but also to changes in the culture itself; such a change should (over time) also be reflected within the brains of the individuals acculturated to this culture, which might influence which connections between and with intentional state symptoms might be realized. The principle then predicts that, for example, generational differences will be reflected in (future) symptom networks, as long as these differences influence or change the to-be-realized symptom interrelationships. Extending from this latter point, this principle then also might allow the NT to causally explain the historicity of mental disorders (i.e., their embeddedness in specific time periods, as well as their possible transience; e.g., Hacking, 2006); perhaps a certain type of psychopathological symptom network is only possible within a certain (time-bound) culture due to the realization of an intentional state symptom interconnection that is necessary for its stability.

Drawing from the above, my position possibly also allows NT to explain the varying symptomatology and expression of disorders over cultures themselves (e.g., Agbayani-Siewert et al., 1999; Crafa & Nagel, 2019; Luhrmann et al., 2015). Different cultures entail different biologically realized intentional state symptom interconnections, which even in the case of mental disorders with a possible common-causal basis (e.g., schizophrenia) can influence the expression of the disorder—such as unique types of psychosis. Differing self-evaluations of auditory hallucinations (Luhrmann et al., 2015) also imply a difference in symptom connections between the “auditory hallucination” node and other intentional state symptoms. When combining this with the above explanation of time-bound symptom networks, NT can possibly also explain culture-bound disorders by referring to structuring causation; the culture (and other relevant factors) causes the
development of a neural network in which a relatively unique loop between symptoms is possible—for example through adding a symptom interconnection that is nonexistent in other cultures, yet which allows for a new type of stable, self-reinforcing psychopathological symptom network.

Notably, a full biological reduction of intentional state symptoms and their connections is still not possible in my variant of NT; the relevant symptoms, their interconnections, and the biological realizations underlying these can only be identified in virtue of the folk psychology of the specific culture that is studied, or of the internalized relevant values of the culture (or some further nonneural constraint). The resulting biological realizations are likely to be highly idiographic due to not only biological constraints, but due to the difference in developmental trajectory amongst individuals as well. This, however, does not necessarily mean that a fully idiographical psychiatry or clinical psychology is desirable. Individuals of the same culture, sharing the same folk psychology and similar enough relevant knowledge are likely to (self-)interpret in similar ways (see also Zawidzki, 2013), and are likely to share a similar set of possible (psychopathological) brain states.

**From fact to factor: Summarizing**

My proposal then boils down to this. There is no genetic, biochemical, or neural problem underlying intentional state symptoms and their interrelations per se; the mechanisms underlying intentional state symptoms (and their causal connections) exist on a network level in the brain, realized in the setup of the brain’s neural network itself, just like other learned knowledge and behaviors. Given similarities in learned folk psychology, knowledge, or culture, a functionally similar neural network setup must be present—and it is this very network level that is informative for the interconnections between intentional state symptoms in symptom networks. Culture and folk psychology code for what is biologically realized through learning processes; thereby, the way they are organized also causes the possibility of different symptom networks in the person. As learned adaptations of the brain, however, we can argue—like Borsboom et al. (2019)—that any symptom network involving intentional states is likely multiply realized on the biological level. In that sense, the structuring causal power of culture and folk psychology is formative for symptom networks in individual brains, and can be used to make predictions regarding the symptom networks of groups that share the relevant cultures, folk psychologies, or other relevant types of background.

It must be noted that the above does not mean that the biological level itself is irrelevant or uninformative. As mentioned above, it is likely there are biological constraints on the possible adaptations that may occur within the brain (i.e., not every type of neural architecture is biologically possible for a human being that needs or is able to interact with our world). The constraints of biology, culture, and other background factors bias the eventual “space of possible symptom networks” one may enter during development together.

Besides the above, there are also likely specific biological constraints regarding specific psychopathologically relevant adaptations and interconnections, as well as for the possibility for such adaptations to enter into a self-reinforcing feedback loop, or to attain
hysteresis, as proposed by NT (Borsboom, 2017). For example, psychopathologically relevant conscious thought patterns such as suicidal ideation or rumination are likely to be realized in language-related brain areas. Such areas may have clinically interesting macroproperties relevant to feedback loops, independent of the actual realizing structures of such symptoms within them. In this sense, the biological level of explanation may still provide valuable information regarding such intentional symptoms, besides its possible explanatory value for “clearly” biological symptom interconnections (e.g., lack of sleep → fatigue). Yet, if my contention is correct, we also cannot eliminate the aforementioned “structuring” constraints from our study of mental disorders if we are to understand those mental disorders that include intentional state symptoms, especially those whose expression may vary over cultures and folk psychologies. The cultural and folk psychological background must also be taken into account, as it determines the form of automatic intentional state connections as well as that which self-interpretations (and thus self-interpretation mediated intentional state symptom interconnections) may take, through determining the learned content and thereby acting as a structuring cause.

**A new principle for network theory: Structuring causal influence**

Having formulated a more adequate way for NT to defend against biological reductionism, I am now left with the task of integrating my contention with the earlier formulation of NT by Borsboom (2017). In order to do so, I propose to add another principle to the aforementioned four core principles of NT:

**Principle 5. Structuring causal influence on symptom networks:** Some symptoms and/or their interrelationships with other symptoms are underdetermined by the biological level, instead being structurally caused by the (time-dependent) culture and culture-specific folk psychology of the individual. These factors influence the underlying biological realization of the symptom and its interrelationships with other symptoms through learning, and thereby the shape of symptom networks that include such symptoms.

With this addition, NT retains its earlier explanatory power, as opposed to when we accept the reformulation proposed by Borsboom et al. (2019). Principle 4’s commitment to stability of mental disorders over groups becomes restricted to those groups sharing the same culture and folk psychology if intentional symptoms are included, but no further restrictions are made. Furthermore, if this new principle is accepted, NT can also make predictions about the ontogeny of certain mental disorders as (partially) arising from culture or folk psychology, by describing causally how such influence is possible—namely, through learning and enculturating processes acting on the brain through neural plasticity. These predictions also entail a return of falsifiability for the theory, and allow NT to deal with the possibility of folk psychological common-cause explanations, as opposed to the arguments proposed by Borsboom et al. (2019). Furthermore, my proposal continues to allow for a realist interpretation of NT due to not exclusively relying on the folk psychological level, whilst—as far as I can tell—also being immune to sophisticated reductionism (e.g., Eronen & Bringmann, 2019; Pernu, 2019) due to a lack of reliance on multiple realizability alone.
Conclusion
In this article, I argued that Borsboom et al.’s (2019) arguments led NT to a scientific “dead end,” through committing it to the truth of folk psychology and losing its predictive and explanatory power. Instead, I proposed that biological reductionism could be better avoided through recognizing that external factors such as culture, but also folk psychology itself, can exert a structuring causal influence on intentional state symptoms and their interconnections within symptom networks, due to the (learned) adaptations of individuals’ brains to these factors. Drawing from this, I proposed to add a new principle to the NT that describes this process, and avoids the aforementioned problems with predictive and explanatory power.

Acknowledgements
I wish to thank Prof. Dr. Jan Bransen, Dr. Fred Hasselman, Sander Bisselink and Merlijn Olthof for our productive discussions on this topic, as well as their helpful comments on drafts of this article. I also want to thank Prof. Dr. Anna Bosman, Prof. Dr. Marc Slors, the BSI’s Complex Systems Group, and Radboud Theoretical Psychology labgroup for their support.

Declaration of Conflicting Interests
The author declares that there is no conflict of interest.

Funding
The author received no financial support for the research, authorship, and/or publication of this article.

ORCID iD
Freek Oude Maatman https://orcid.org/0000-0002-4795-435X

Notes
1. Notably, this diverges from Dretske (1988) by not using the indicator state’s intentional content or information as a structuring cause, but instead the external culture and/or folk psychology.
2. This claim also relates to, and in fact could be made far stronger by, the mindshaping hypothesis of Zawidzki (2013), which states that we accommodate our society’s folk psychology and norms during development in order to facilitate mindreading practices. This leads to homogeneity, which in turn creates the success of our folk psychology.
3. My proposal could be considered related to the concept of “local cultural invitations” in the “social kindling” hypothesis discussed in Luhrmann et al. (2015). As a contrast, they discuss the influence of the local culture as a cognitive interpretational bias for the interpretation of hallucinations, whereas I discuss culture as formative in general.

References
Agbayani-Siewert, P., Takeuchi, D. T., & Pangan, R. W. (1999). Mental illness in a multicultural context. In C. S. Aneshensel & J. C. Phelan (Eds.), Handbook of the sociology of mental health. Handbooks of sociology and social research (pp. 19–36). Springer.
Alvarez, M. (2009). How many kinds of reasons? *Philosophical Explorations, 12*(2), 181–193. https://doi.org/10.1080/13869790902838514

Borsboom, D. (2008). Psychometric perspectives on diagnostic systems. *Journal of Clinical Psychology, 64*(9), 1089–1108. https://doi.org/10.1002/jclp.20503

Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry, 16*(1), 5–13. https://doi.org/10.1010/wps.20375

Borsboom, D., & Cramer, A. O. J. (2013). Network analysis: An integrative approach to the structure of psychopathology. *Annual Review of Clinical Psychology, 9*, 91–121. https://doi.org/10.1146/annurev-clinpsy-050212-185608

Borsboom, D., Cramer, A. O. J., & Kalis, A. (2019). Brain disorders? Not really: Why network structures block reductionism in psychopathology research. *Behavioral and Brain Sciences, 42*, Article e2. https://doi.org/10.1017/S0140525X17002266

Borsboom, D., Cramer, A. O. J., Schmittmann, V. D., Epskamp, S., & Waldorp, L. J. (2011). The small world of psychopathology. *PLOS ONE, 6*(11), Article e27407. https://doi.org/10.1371/journal.pone.0027407

Bringmann, L. F., & Eronen, M. I. (2018). Don’t blame the model: Reconsidering the network approach to psychopathology. *Psychological Review, 125*(4), 606–615. https://doi.org/10.1037/rev0000108

Crafa, D., & Nagel, S. (2019). The adaptive self: Culture and social flexibility in feedback networks. *Behavioral and Brain Sciences, 42*, Article E5. https://doi.org/10.1017/S0140525X18001255

Cramer, A. O. J., Waldorp, L. J., Van der Maas, H. L. J., & Borsboom, D. (2010). Comorbidity: A network perspective. *Behavioural and Brain Sciences, 33*(2–3), 137–150. https://doi.org/10.1017/S0140525X09991567

Dennett, D. C. (1987). *The intentional stance*. MIT Press.

Doherty, J. L., & Owen, M. J. (2014). The research domain criteria: Moving the goalposts to change the game. *The British Journal of Psychiatry, 204*(3), 171–173. https://doi.org/10.1192/bjp.bp.113.133330

Dretske, F. (1988). *Explaining behavior: Reasons in a world of causes*. MIT Press.

Eronen, M. I., & Bringmann, L. F. (2019). Networks, intentionality and multiple realizability: Not enough to block reductionism. *Behavioral and Brain Sciences, 42*, Article e8. https://doi.org/10.1017/S0140525X18001012

Francken, J. C., & Slors, M. (2018). Neuroscience and everyday life: Facing the translation problem. *Brain and Cognition, 120*, 67–74. https://doi.org/10.1016/j.bandc.2017.09.004

Fried, E., & Cramer, A. O. J. (2017). Moving forward: Challenges and directions for psychopathological network theory and methodology. *Perspectives on Psychological Science, 12*(6), 999–1020. https://doi.org/10.1177/1745691617705892

Hacking, I. (2006). Making up people: Clinical classifications. *London Review of Books, 28*(16), 23–26.

Haig, B. D., & Borsboom, D. (2012). Truth, science, and psychology. *Theory & Psychology, 22*(3), 272–289. https://doi.org/10.1177/0959354311430442

Insel, T. R., & Cuthbert, B. (2015). Brain disorders? Precisely. *Science*, 348(6234), 499–500. https://doi.org/10.1126/science.aab2358

Luhrmann, T. M., Padmavati, R., Tharoor, H., & Osei, A. (2015). Hearing voices in different cultures: A social kindling hypothesis. *Topics in Cognitive Science, 7*(4), 646–663. https://doi.org/10.1111/tops.12158

McNally, R. (2019). The network takeover reaches psychopathology. *Behavioral and Brain Sciences, 42*, Article e15. https://doi.org/10.1017/S0140525X18001073

Pernu, T. (2019). Elimination, not reduction: Lessons from the research domain criteria (RDoC) and multiple realisation. *Behavioral and Brain Sciences, 42*, Article e22. https://doi.org/10.1017/S0140525X18001139
Robinaugh, D. J., Millner, A. J., & McNally, R. J. (2016). Identifying highly influential nodes in the complicated grief network. *Journal of Abnormal Psychology, 125*(6), 747–757. https://doi.org/10.1037/abn0000181

Sandis, C. (2006). The explanation of action in history. *Essays in Philosophy, 7*(2), Article 12.

Schmittmann, V. D., Cramer, A. O. J., Waldorp, L. J., Epskamp, S., Kievit, R. A., & Borsboom, D. (2013). Deconstructing the construct: A network perspective on psychological phenomena. *New Ideas in Psychology, 31*, 43–53. https://doi.org/10.1016/j.newideapsych.2011.02.007

Slors, M., Francken, J., & Strijbos, D. (2019). Intentional content in psychopathologies requires an expanded interpretivism. *Behavioral and Brain Sciences, 42*, Article e26. https://doi.org/10.1017/S0140525X18001176

Stoutland, F. (1998). The real reasons. In J. Bransen & S. E. Cuypers (Eds.), *Human action, deliberation and causation* (pp. 43–66). Springer.

Van Loo, H., & Romeijn, J. (2019). What’s in a model? Network models as tools instead of representations of what psychiatric disorders really are. *Behavioral and Brain Sciences, 42*, Article e30. https://doi.org/10.1017/S0140525X18001206

Wakefield, J. C. (2014). Wittgenstein’s nightmare: Why the RDoC grid needs a conceptual dimension. *World Psychiatry, 13*(1), 38–40. https://doi.org/10.1002/wps.20097

Zawidzki, T. (2013). *Mindshaping: A new framework for understanding human social cognition*. MIT Press.

**Author biography**

**Freek Oude Maatman** is an external PhD student and junior lecturer at Radboud University Nijmegen. His main research interest is the evaluation of the methodology and theory of Psychology, especially those of social and clinical psychology.