

GOAL-DIRECTED APPROACH TO PSYCHOPATHOLOGY

The goal-directed model as an alternative to reductionist and network approaches of psychopathology

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Abstract

As an alternative to biological reductionist and network approaches to psychopathology, we propose a non-reductionist mental-mechanistic approach. To illustrate this approach, we work out the implications of the goal-directed framework of Moors, Boddez, and De Houwer [1], which has the potential to explain the heterogeneous manifestations of psychopathology with a restricted set of broad theoretical principles.

Keywords: latent variable model; network model; reductionism; psychopathology; goal-directed

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A current debate in psychopathology centers around two contrasting approaches: the latent variable approach and the network approach. The latent variable approach is a mechanistic approach, characterized as reductionist, in which symptoms of a mental disorders are understood as caused by a single common cause—often a neural deficit, situated on the brain level of analysis. For instance, abnormal amygdala morphology has been related to anxiety symptoms in autism spectrum disorder [2]. The network approach, on the other hand, proposes that a mental disorder emerges from and is constituted by the causal relations among the symptoms of the disorder (and external factors) and not by an underlying common cause [3]. Proponents of the network approach criticized the latent variable approach because of a lack of direct and indirect evidence for many of the purported neural deficits. Direct evidence is evidence that identifies the neural deficits themselves. Indirect evidence (at least one type of it) is evidence for the coherence of the symptoms belonging to a mental disorder within and across individuals. This is at odds with the heterogeneity in symptom profiles that is typically observed for mental disorders (like depression, anxiety disorder, obsessive-compulsive disorder, and schizophrenia). Whereas the latent variable approach has to invoke extra factors that moderate the relation between the common cause and the symptoms to explain heterogeneity, the network approach only deals with symptoms and is therefore naturally equipped to account for heterogeneity. Another selling point of the network approach is that the nodes in the network have Intentional¹ content (i.e., aboutness; e.g., sadness about job loss). This makes mental disorders meaningful (even if not therefore rational), something that is beyond the reach of reductionist explanations in terms of neural deficits [3].

¹ Intentionality is used here in the philosophical sense to indicate the property of mental representations that they can be directed at or about objects outside of themselves. Following Searle [23], we capitalize the term to mark the distinction with intentionality in the ordinary sense, which is applied to actions and processes and indicates that these are caused by intentions.

While we acknowledge the problems of the latent variable approach, we are not convinced that rejection of this approach necessarily drives us in the arms of the network approach. One set of arguments cautions against the hasty rejection of the mechanistic approach resting on a narrow understanding of this approach as reductionist [4,5]. Leading philosophers of science have convincingly argued that a genuine mechanistic approach is not reductionist but *multi-level* [6,7]. In psychology, it is useful to distinguish between an observable level, a mental level, and a brain level. The observable level describes relations between observable stimuli and observable behavior. Phenomena (e.g., symptoms) described at the observable level can be explained by mechanisms situated at the mental level and the brain level. In addition, the mental level can play an important role in bridging the observable level and the brain level [5]. The type of approach that we propagate specifies these three levels and their relations is thus without risk of “explaining away” one of the levels. Furthermore, the mechanistic approach is not committed to the assumption that all symptoms situated at the observable level are generated by a single latent variable, whether mental or neural. This is because mechanisms are typically *componential*, comprised of several latent variables that are causally connected [8]. Things can go wrong in different components for different individuals, and this can account for the heterogeneity of symptoms across people (see below). In sum, the mechanistic approach is not reductionist but multi-level and does not strive for a single common cause but rather a componential mechanism.

Another set of arguments points at shortcomings of *existing* network models². A first problem is that the nodes in these models, which belong to different levels of analysis (e.g., avoidance behavior on the observable level, fear of spiders on the mental level, amygdala activity on the neural level) are treated as if they belong to the same level of analysis [9]. This is apparent from the fact that the relations between them are treated as causal and not as

² Not all of these shortcomings are principled shortcomings of the network approach.

constitutive. There seems to be no room in *existing* network models of psychopathology for constitutive relations, except for the relation between symptoms and the emergent disorder.

A second problem is the lack of theoretical principles that guide the choice of the nodes in the network [10,11]. Existing work with the network approach is bottom-up and exploratory. Researchers pick a set of symptoms (and perhaps other factors) with the aim of discovering their causal connections in empirical research, but the selection of symptoms is often based on folk theory [11]. Researchers not only take the content of the nodes at face value, but also the causal relations among them. Relations between symptoms are considered valid because they “make sense” [3, p. 5]. However, this type of intuitive reasoning is notoriously unreliable [11]. For example, the causal relation between acute sleep deprivation and euphoria can hardly be considered as making intuitive sense [12]. Also contrary to popular belief, feelings of fear and avoidance often diverge, both in the clinic [13] and in the laboratory [14].

Finally, the Intentional content of network nodes (e.g., sadness about job loss) may create a third problem. Different content requires a different network model, so that a plurality of network models is needed [11]. A person whose social anxiety leads to staying home ends up with a different network structure than a person whose fear about job loss leads to working overtime. Yet a proliferation of models is at odds with the aim of nomothetic science to explain the heterogeneous manifestations of psychopathology with a restricted set of broad theoretical principles (combining scope and parsimony), and it may obfuscate that these persons may benefit from a similar type of treatment.

Taken together, we argue that there is merit in a theory-driven mechanistic approach situated at the mental level of analysis that makes abstraction of Intentional content. The promise of this approach is to reveal that various mental disorders follow a single logic despite their apparent heterogeneity and to reveal new treatment directions. We illustrate this

by analyzing psychopathology through the goggles of the goal-directed model of behavior causation by Moors et al. [1].

This model proposes a cycle starting with the detection of a discrepancy between a first goal (i.e., the representation of a valued outcome) and a (current or anticipated) stimulus, which can be reduced via the broad strategies of acting (i.e., assimilation), replacing the first goal by a different goal (i.e., accommodation), or biasing the interpretation of the stimulus (i.e., immunization). In the case of assimilation, people select the behavior option from their repertoire with the highest expected utility (i.e., the highest expectancy of reducing the discrepancy and reaching the valued outcome). This activates the corresponding action tendency, which translates in overt behavior and produces an outcome. This outcome is fed back as the stimulus input to the next cycle. The cycle is repeated until the discrepancy is resolved (see Figure 1).

The suboptimal behavior (and affect) in mental disorders can be understood as problems in one or more steps of this cycle [15,16]. Aggression (as in impulsive disorders) may be selected as a strategy (assimilation) to solve the discrepancy with a personal goal (e.g., winning an argument), but people may fail to detect that this behavior is itself discrepant with another goal (e.g., to keep their relationship intact). Even if they do detect the discrepancy, they may lack an action option in their repertoire (e.g., negotiating calmly) to reach both goals, or they may underestimate the expectancy of such an action option. Crucially, these principles can be abstractly formulated without making reference to a specific goal (which could be to win an argument, but also other goals such as the goal to maintain high social status), contributing in this way to combining scope and parsimony. Another example is relentless yearning for a deceased partner (as in pathological grief [17]). This could be understood as persisting in fruitless approach behavior (i.e., a failure to accommodate). The incapacity to let go may not only pertain to deceased persons (e.g., grief

over a broken relationship), or not even to persons at all (e.g., melancholic fixation on the old days). A further example is apathy (as in depression), which could be understood as forsaking to reduce the discrepancy between stimuli and goals altogether, because of previous failures to reduce such discrepancies via behavior (i.e., failure to assimilate [18]) and because the goals are too fundamental to be replaced by other ones (i.e., failure to accommodate). Again, the goals can range from obtaining meaningful relationships with others to obtaining a sense of competence on a professional level. Finally, hallucinations or delusions (as in schizophrenia) can be understood as a form of immunization. This would hold for delusions about infatuated others, but also for delusions about being a brilliant scientist. So here as well, the hypothesis makes abstraction of content and therefore provides a ground for generalization between heterogeneous manifestations of psychopathology.

So, while a network approach takes phenomenological variety at face value and accepts heterogeneity, a mechanistic approach holds the promise of accounting for heterogeneity by use of broad theoretical principles, which could further lead to a reorganization of the landscape of mental disorders (e.g., according to failures in various steps in the goal-directed cycle). As illustrated above, the type of mechanistic approach that we propagate specifies the relation between the abstract mental level (e.g., assimilation) and the observable level (e.g., staying at home because of the goal to remain disease-free) and is thus without risk of explaining away the latter (i.e., reductionism).

The goal-directed model suggests a therapeutic approach in which problems are analyzed with the aim of uncovering and remediating problems in one or more steps of the goal-directed cycle: facilitating discrepancy detection, extending the behavioral repertoire with behavior options that satisfy multiple goals, and changing values (e.g., of family life vs. immediate gratification in addiction) and/or expectancies (e.g., of being able to reach certain

goals in depression). In fact, several existing therapies already fit in this approach, but recognizing that they do has the benefit of uniting them and of revealing blind spots.

In sum, we are not convinced that the network approach provides a replacement for theoretical and experimental work on the mechanisms of psychopathology. Some have argued that network models can accommodate any type of content, on all levels of abstraction, and all levels of analysis [19,20]. But even if they would, it remains unclear what the network approach would then add over and above the causal relations that are already part of existing non-reductive, mental-level mechanistic theories. The network approach could, in our opinion, still be ancillary to such a mechanistic approach, for instance, by offering a mathematical toolbox [21] for organising the (causal) relations among the entities at various levels [22].

References

[1] Herrington JD, Maddox BB, Kerns CM, Rump K, Worley JA, Bush JC, ... & Miller JS: Amygdala volume differences in autism spectrum disorder are related to anxiety. *J Autism and Developmental Disorders* 2017, 47: 3682-3691.

** [2] Borsboom D, Cramer AO, Kalis A: **Brain disorders? Not really: Why network structures block reductionism in psychopathology research.** *Behav Brain Sci* 2019a, 42:1-11. <https://doi.org/10.1017/S0140525X17002266>

The authors criticize the reductionist approach in psychopathology and contrast it with the network approach, highlighting three features: they preclude a common, biological cause, they exemplify intentionality, and they can easily accommodate contextual variety.

[3] Elbau IG, Binder EB, Spormaker VI: **Symptoms are not the solution but the problem: Why psychiatric research should focus on processes rather than symptoms.** *Behav Brain Sci* 2019, 42:16-17. <https://doi.org/10.1017/S0140525X18001000>

[4] Hur J, Tillman RM, Fox AS, Shackman AJ: **The value of clinical and translational neuroscience approaches to psychiatric illness.** *Behav Brain Sci* 2019, 42:20-22.
doi: [10.1017/S0140525X18001036](https://doi.org/10.1017/S0140525X18001036)

[5] Bechtel W: *Mental mechanisms: Philosophical perspectives on cognitive neuroscience.* Taylor & Francis; 2008.

[6] Craver CF: **When mechanistic models explain.** *Synthese* 2006, 153:355-376.
<https://doi.org/10.1007/s11229-006-9097-x>

[7] Craver CF, Bechtel W: **Top-down causation without top-down causes.** *Biol and Philos* 2007, 22:547-563. doi 10.1007/s10539-006-9028-8

[8] Oller DK: **Evolutionary-developmental modeling of neurodiversity and psychopathology.** *Behav Brain Sci* 2019, 42:29-30.
<https://doi.org/10.1017/S0140525X18001103>

[9] Fried EI: Lack of theory building and testing impedes progress in the factor and network literature. 2020 <https://doi.org/10.31234/osf.io/zg84s>

[10] Ward T, Fischer R: **Families of network structures—we need both phenomenal and explanatory models. *Behav Brain Sci* 2019, 42:42-44.

<https://doi.org/10.1017/S0140525X1800122X>

The authors criticize symptom network models, arguing that they are phenomenal models in that they identify patterns of symptoms only, which is only one phase of a scientific enquiry. What is more urgently needed are etiological and constitutive mechanistic models to explain these patterns.

[11] Giedke H, Schwärzler F (2002). **Therapeutic use of sleep deprivation in depression.** *Sleep Med Rev* 2002, 6:361-377. <https://doi.org/10.1053/smrv.2002.0235>

[12] Rachman S, Hodgson R (1974) I. **Synchrony and desynchrony in fear and avoidance.** *Behav Res and Ther*, 12:311-318. [https://doi.org/10.1016/0005-7967\(74\)90005-9](https://doi.org/10.1016/0005-7967(74)90005-9)

[13] Mineka S: **The role of fear in theories of avoidance learning, flooding, and extinction.** *Psychol Bull* 1979, 86:985-1010. <https://doi.org/10.1037/0033-2909.86.5.985>

** [14] Moors A, Boddez Y, De Houwer J: **The power of goal-directed processes in the causation of emotional and other actions.** *Emot Rev* 2017, 9:310-318.

<https://doi.org/10.1177/1754073916669595>.

The authors criticize traditional dual-process models with a default-interventionist architecture regarding the interplay between stimulus-driven and goal-directed processes and proposes an alternative dual process model with a parallel-competitive architecture and in which goal-directed processes determine the lion share of emotional and other actions.

[15] Moors A: **Towards a goal-directed account of weak-willed behavior.**

2019 <http://dx.doi.org/10.23668/psycharchives.3126>

[16] Köster M, Moors A, De Houwer J, Ross-Hellauer R, Verbruggen F: **Behavioral reluctance in adopting open access publishing: Insights from a goal-directed perspective.**

Manuscript submitted for publication.

[17] Boddez Y: **The presence of your absence: A conditioning theory of grief.** *Behav Res and Ther* 2018, 106:18-27. <https://doi.org/10.1016/j.brat.2018.04.006>

Craver CF: **The explanatory power of network models.** *Philos of Sci* 2016, 83:698-709.

[18] Seligman MEP, Maier SF: Failure to escape traumatic shock. *J Exp Psychol* 1967, 74:1-9. <https://doi.org/10.1037/h0024514>

[19] Borsboom D, Cramer A O, Kalis A: **Author's response: Reductionism in retreat.** *Behav Brain Sci* 2019b, 42:44-61. <https://doi.org/10.1017/S0140525X18002091>

[20] Hyland ME: **Functional disorders can also be explained through a non-reductionist application of network theory.** *Behav Brain Sci* 2019, 42:22-23.

<https://doi.org/10.1017/S0140525X18001048>

[21] Epskamp S, Fried EI: **A tutorial on regularized partial correlation networks.** *Psychol Methods* 2018, 23:617–634. <https://doi.org/10.1037/met0000167>

[22] Redish AD, Kazinka R, Herman AB: **Taking an engineer's view: Implications of network analysis for computational psychiatry.** *Behav Brain Sci* 2019, 42:35-36.

<https://doi.org/10.1017/S0140525X18001152>

[23] Searle JR: *Intentionality: An essay in the philosophy of mind.* Cambridge University Press; 1983.