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Open Peer Commentary

Latent variables and the network perspective

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Abstract: We discuss the latent variables construct, particularly in regard to the following: that latent variables are considered as the sole explanatory factor of a disorder; that pragmatic concerns are ignored; and that the relationship of these variables to biological markers is not addressed. Further, we comment on the relationship between bridge symptoms and causality, and discuss the proposal in relationship to other constructs (endophenotypes, connectionist-inspired networks).

Since the early stages of the discipline of psychiatry, the construct of psychiatric semiology and nosography has been indissociable from the etiological conceptualization of observed phenomena. Nevertheless, it is widely admitted that psychiatric disorders are multifactorial and etiologically complex, and explanatory models should refer mostly to explanatory pluralism rather than to biological reductionism. Our knowledge about psychiatric disorders remains incomplete, and we can only hope to get “small explanations, from a variety of explanatory perspectives, each addressing part of the complex etiological process leading to disorder,” and try to understand “how these many different small explanations all fit together,” etiological pathways being considered “complex and interacting more like networks than individual pathways” (Kendler 2005, p. 435). Our current categorical classifications of mental disorders in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; American Psychological Association 1994) and in the World Health Organization’s International Statistical Classification of Diseases, 10th revision (ICD-10) have been conceptualized on assumptions of more global and simple hypothetical explanations.
In that context, the clinical assessment of psychiatric conditions has been addressed in reference to the “latent trait hypothesis,” which considers each observed symptom or cluster of symptoms to be related to a specific latent cause. Any attempt to go beyond the usual categorical construct of current mental disorders classifications could constitute a valuable epistemological contribution in view of the upcoming new version of mental disorders classifications (DSM-V), as it takes an important step toward a less categorical, and rather dimensional conception of mental disorders. We have the following specific comments to make on Cramer et al.’s discussion of latent variables in the target article.

1. The target article makes a restrictive interpretation of the latent variable models. Along the article’s lines, latent variable models are represented as unidirectional trees, the “latent variable” (the common cause) being the root. In this representation, the authors assume that all links have the same importance. Yet, by definition, a latent variable is only non-observable, and is not necessarily causally central. Cramer et al. are probably right in criticizing the assumption (implicit in psychiatry) that all symptoms should be related to a central latent variable, but they mistakenly underestimate the potential role of accessory latent variables. Getting rid of all latent variables would be tantamount to assuming that everything is known about the observed phenomenon. Moreover, there is no reason why the flexibility they claim for their network approach (multi-directionality, different link strength) should not be allowed within the context of a latent variable model.

2. Besides, a heuristically good reason to suppose the existence of a latent variable is mainly therapeutic rather than methodological. This kind of hidden variable is often seen as a therapeutic target rather than an etiological node; that is, not something to find that would explain everything, but something to act upon that would dissolve everything. If a match is considered the cause of a fire in a building, rather than oxygen in the air, which is less required to start a fire, it is because the match seems the most appropriate factor to act upon. Mackie (1974), Hesslow (1984), Garnett (1999), and Magnus (1992), among others, have shown the importance of pragmatic concerns in the search for a single target which might be called the cause of a disease (it is called the problem of causal selection). This kind of pragmatic interpretation of a latent variable as “what we have to act upon” may justify the otherwise objectionable assumption that there is actually a latent variable which explains and causes everything. There is, however, a question as to how the network approach is to be translated into the definition of therapeutic targets. For instance, while such a definition is obviously easy on the basis of the target article’s Figure 1, one might ask what could be proposed on the basis of Figure 4.

3. It would also be interesting to discuss this model, as well as the latent variable model, with regard to the biological markers of these diseases. Indeed, particular markers of the disorder could be related to specific biological alterations. For example, anhedonia could be related to a deficit in nucleus accumbers processing, or a defect in stress reactivity to a dysregulated neuroendocrine axis.

4. Beyond that, in the case of two comorbid disorders, do the authors propose that each symptomatic node be related to a specific biological dysfunction that would be common to the two comorbid pathologies? In this case, a given biological marker defect underlying pathology A would also be altered in the comorbid pathology B. If there is no latent variable underlying the different symptomatic features, what is the explanation as to why these symptoms often co-occur? Moreover, if two comorbid disorders have a common epiphenomenal symptom, should this be regarded as a bridge symptom? For example, if decreased eating occurs in an anxiety disorder as well as in depression, but does not induce (or is unrelated to) any of the other symptoms of depression or anxiety, might it not be considered a bridge symptom underlying comorbidity? How can symptoms be distinguished from “non-symptom causal processes” (sect. 2, para. 9) or from the “external effects” (sect. 5, para. 6) if the boundaries of the disorders are “fuzzy” (sect. 6, para. 6)?

5. It would be interesting to compare the network model described by Cramer et al. with the psychopathological endophenotype approach that has been developed to dissect major depression into different independent entities (see, e.g., Hasler et al. 2004), or with other constructs used in the field of psychiatry, such as connectionist-inspired ones (e.g., Tanti & Belzung 2010).

The rocky road from Axis I to Axis II: Extending the network model of diagnostic comorbidity to personality pathology

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Abstract: Although the network model represents a promising new approach to conceptualizing comorbidity in psychiatric diagnosis, the model applies most directly to Axis I symptom disorders, for which the model generalizes to Axis II disorders remains open to question. This commentary addresses that issue, discussing opportunities and challenges in applying the network model to DSM-diagnosed personality pathology.

Cramer et al.’s network model represents a promising new approach for conceptualizing and quantifying comorbidity in psychiatric diagnosis, helping avoid the thorny challenge of operationalizing latent constructs, and shifting the focus of comorbidity research from syndrome to symptom. Scrutiny of Cramer et al.’s analysis reveals that the theoretical underpinnings and empirical evidence bearing on this model apply most directly to Axis I symptom disorders (e.g., major depression, generalized anxiety). Because Axis II personality disorders differ in myriad ways from Axis I symptom disorders, the degree to which the network comorbidity model generalizes to Axis II disorders remains open to question. This commentary addresses that issue, discussing issues that arise in applying the network model to DSM-diagnosed personality pathology (i.e., the personality disorder [PD] diagnoses offered in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition or DSM-IV; American Psychiatric Association 1994).

As Cramer et al. have noted, diagnostic comorbidity evidence involving DSM-IV Axis I disorders can yield ambiguous, confusing patterns. Diagnostic comorbidity evidence bearing on DSM-IV Axis II is far worse. Consider: The number of differential diagnoses per DSM-IV PD ranges from 3 (dependent, obsessive-compulsive) to 7 (paranoid), with the mean number of differential diagnoses per PD being 4.5. Thus, on average each DSM-IV PD shows substantial overlap with 50% of the remaining PDs. When Eksehuls et al. (1994) calculated correlations among interview-derived scores for PDs in a heterogeneous sample of psychiatric patients and nonclinical participants, they obtained a mean interclass correlation (r) of .41, and statistically significant interscale correlations in 41 of 45 comparisons (91%). Subsequent comorbidity studies have confirmed these results (Bornstein 1998; 2005).

Given these patterns, extending the network comorbidity model to Axis II presents some unique challenges, but it also involves some unique opportunities to gain new perspective on