The glue of (ab)normal mental life: Networks of interacting thoughts, feelings and behaviors
Cramer, A.O.J.

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Appendix A

Abstracts commentaries in response to Chapter 2


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 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).


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; the degree to 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.


Network analysis is a promising step forward in efforts to align psychological assessment with explanatory theory in psychological science. The implications of Cramer et al.’s analysis are quite general. Network analysis may illuminate functional relations not only among observable behaviors that comprise psychological disorders, but among cognitive and affective processes that causally contribute to everyday experience and action.

We agree with Cramer et al.’s goal of the discovery of causal relationships, but we argue that the authors’ characterization of latent variable models (as deployed for such purposes) overlooks a wealth of extant possibilities. We provide a preliminary analysis of their data, using existing algorithms for causal inference and for the specification of latent variable models.


Psychological traits and disorders are often interrelated through shared genetic influences. A combination of maximum-likelihood structural equation modeling and multidimensional scaling enables us to open a window onto the genetic architecture at the symptom level, rather than at the level of latent genetic factors. We illustrate this approach using a study of cognitive abilities involving over 5000 pairs of twins.


The network approach proposed by Cramer et al. suggests fascinating new directions of research on mental disorders. Research is needed to find evidence for the causal power of symptoms, to examine symptoms thoroughly, to investigate individual differences in edge strength, to discover etiological processes for each symptom, and to determine whether and why symptoms cohere into distinct mental disorders.


Cramer et al. make a good case for reconceptualizing comorbid psychopathologies in terms of complex network theory. We suggest the need for an extension of their network model to include reference to latent causes. We also draw attention to a neglected approach to theory appraisal that might be usefully incorporated into the methodology of network theory.


The network approach to psychiatric phenomena has the potential to clarify and enhance psychiatric diagnosis and classification. However, its generally well-justified anti-essentialism view psychiatric disorders as inevitably fuzzy and arbitrary, and overlooks the likelihood that the domain includes some latent categories. Network models misrepresent these categories, and fail to recognize that some comorbidity may represent valid co-occurrence of discrete conditions.


Cramer et al.’s account of comorbidity comes with a substantive philosophical view concerning the nature of psychological disorders. Although the network account is responsive to problems with extant approaches, it faces several practical and conceptual
challenges of its own, especially in cases where the individual differences in network structures require the analysis of intra-individual time-series data.


First, we question whether Cramer et al.’s proposed network model can provide a viable scientific foundation for investigating comorbidity without invoking latent variables in some form. Second, the authors’ claim that the network perspective is radically different from a latent variable perspective rests upon an undemonstrated premise. Without being demonstrated, we think the premise is potentially misleading.


Cramer et al. contrast two possible explanations for psychological symptoms: latent variables (i.e., specific cause) versus a network of causality between symptoms. There is a third explanation: The reason for comorbidity and the reported network structure of psychological symptoms is that the underlying biological cause is a psychoneuroimmunoendocrine information network which, when dysregulated, leads to several maladaptive psychological and somatic symptoms.


Latent variable modeling has revealed important conundrums in the DSM classification system. We agree that the network perspective has potential to inspire new insights and resolve some of these conundrums. We note, however, that alone it cannot really help us understand etiology. Etiology, not comorbidity, is the fundamental question.


Cramer et al. articulate a novel perspective on comorbidity. However, their network models must be compared with more parsimonious latent variable models before conclusions can be drawn about network models as plausible accounts of comorbidity. Latent variable models have proven generative in studying psychopathology and its external correlates, and we doubt network models will prove as useful for psychopathology research.


Cramer et al. present a thoughtful application of network analysis to symptoms, but certain questions remain open. These questions involve the intended causal interpretation, the critique of latent variables, individual variation in causal networks, Borsboom’s idea of networks as measurement models, and how well the data support the stability of the network results.
In the target article, Cramer et al. suggest that diagnostic classification is improved by modeling the relationship between manifest variables (i.e., symptoms) rather than modeling unobservable latent variables (i.e., diagnostic categories such as Generalized Anxiety Disorder). This commentary discusses whether symptoms represent manifest or latent variables and the implications of this distinction for diagnosis and treatment.


Cramer et al. present an original and interesting network perspective on comorbidity and contrast this perspective with a more traditional interpretation of comorbidity in terms of latent variable theory. My commentary focuses on the relationship between the two perspectives; that is, it aims to qualify the presumed contrast between interpretations in terms of networks and latent variables.


Cramer et al. persuasively conceptualize major depressive disorder (MDD) and generalized anxiety disorder (GAD) as network disorders, rejecting latent variable accounts. But how does their radical picture generalize across the suite of mental and personality disorders? Addictions are Axis I disorders that may be better characterized by latent variables. Their comorbidity relationships could be captured by inserting them as nodes in a super-network of Axis I conditions.


In developmental psychopathology, differentiating between the coexistence and the clinical entity of two problem areas is of utmost importance. So far, logistic regression analysis has already provided helpful answers, as shown in studies on comorbidity of tic disorders. While the concept of *bridging symptoms* may be investigated adequately by both logistic regression and the network approach, the former (latent variable) seems to be of advantage with regard to the problems of multiple comorbidities and development.


We agree with Cramer et al. that pure cases of behavioral disorders with no symptom overlaps are rare. However, we argue that disorders do exist and the network idea is limited and limiting. Networks of symptoms are observed mainly at behavioral levels. The core deficit is commonly at the cognitive or brain levels, and there the story is completely different.

Staniloiu, A., & Markowitsch, H. J. (2010). Looking at comorbidity through
the glasses of neuroscientific memory research: A brain-network perspective. *Behavioral and Brain Sciences, 33,* 170-171.

As psychiatric illnesses have correlates in the brain, it is surprising that Cramer et al. make almost no reference to the brain’s network character when proposing a network approach to comorbidity of psychiatric diseases. We illustrate how data from combined neuropsychological and functional and structural brain-imaging investigations could inform theoretical models about the role played by overlapping symptoms in the etiology of psychiatric comorbidity and the pathways from one disorder to another.


We suggest that the network approach to comorbidity (Cramer et al.) is best examined by using longitudinal, multi-measurement, intra-individual data. Employment of time-series analysis to the examination of the generalized anxiety and major depressive disorder comorbidity enables a detailed appreciation of fluctuations and causal trajectories in terms of both symptoms and cognitive vulnerability.


Cramer et al.’s proposal to view mental disorders as the outcome of network dynamics among symptoms obviates the need to invoke latent traits to explain co-occurrence of symptoms and syndromes. This commentary considers the consequences of such a network view for genetic association studies.


Cramer et al.’s article is an example of the fruitful application of complex dynamic systems theory. We extend their approach with examples from our own work on development and developmental psychopathology and address three issues: (1) the level of aggregation of the network, (2) the required research methodology, and (3) the clinical and educational application of dynamic network thinking.


We welcome network theory as a tool for modeling the multidimensional interactions that characterize disease. However, we feel that Cramer et al. have neglected one important aspect: how diseases change over developmental time. We discuss principles such as fan in, fan out, bottlenecks, and common pathways, argue that modeling these developmental aspects can be vital, particularly in deriving properly targeted treatments.

Cramer et al.’s network approach reconceptualizes mental comorbidity on the basis of symptom space originating from psychometric signatures. We argue that the advantages of this approach need to be regarded in the context of the multi-level functional organization of the neural substrate, ranging from neurogenetic to psychometric. Neuroelectric oscillations are proposed as a level-integrating principle.


Cramer et al.’s critique of latent variables implicitly advocates a type of scientific anti-realism which can be extended to many dispositional constructs in scientific psychology. However, generalizing Cramer et al.’s network model in this way raises concerns about its applicability to psychopathology. The model could be improved by articulating why a given cluster of symptoms should be considered disordered.