The Network Perspective Will Help, But Is Comorbidity the Question?

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Commentary to Cramer, Waldorp, van der Maas, and Borsboom

ABSTRACT

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.

It has become popular in the last few years to model large sets of inter-correlated variables as networks. There are probably two reasons for this. First, and perhaps trivially, as was the case about 25 years ago with latent variable modeling, the statistical techniques and tools to do this tractably and readily are relatively recently available. That is, researchers now can. This is, however, not *a priori* a reason to take whatever it offers us as the best answer to our scientific questions (Gigerenzer, 1991). But second, again as did latent variable modeling about 25 years ago, the network perspective offers exciting prospects for fresh understanding of dynamic systems ranging from power grids to epidemic spread through a population to the development of chronic disease such as diabetes in the individual. Psychopathology, with its common occurrence and multifaceted manifestations seems a particularly apt target for the network perspective, and we are pleased to see Cramer, Waldorp, van der Maas, and Borsboom (CWVB) take some concrete steps towards applying it.

At this point, problems with the Diagnostic and Statistical Manual IV system of psychopathological diagnostic system are widely acknowledged. Despite CWVB's criticism of it, much of the credit for revealing those problems, perhaps especially comorbidity, should rest with latent variable modeling (e.g., Krueger, Markon, Patrick, Benning, and Kramer, 2007). Latent variable modeling might also be credited with something else: revealing the tension between trying to use the same diagnostic system for purposes of systems of administration and treatment and for purposes of understanding development and etiology. The former takes place and requires description at the level of the population, while the latter takes place and requires description at the level of the individual. Researchers can count on latent variable modeling to tell us something about the former, but whatever it says may not apply to the latter (e.g., Cervone, 2004; Molenaar, 2004).

As CWVB note, the reason latent variable modeling may not tell us about development and etiology involves the assumption of local independence underlying it. From the latent variable perspective, pervasive comorbidity is the chronic symptom of this trouble. If two disorders share the same symptom, how can there be local independence of symptoms? And if two disorders share the same symptom, it should be no surprise to find comorbidity. But it is the strong causal attributions of latent variable models that carry with them the need for assumptions like local independence, not the structural equations that define the parameters to be estimated themselves. The appropriateness of latent variable models is assessed by how well those structural equations can replicate the data, not by any direct test of the appropriateness of the causal attributions. Completely different patterns of causal attribution can be described by the same sets of structural equations, and these different causal models will fit the data equivalently. For example, it is probably just as likely that depression emerges from a constellation of symptoms as it is that depression is the underlying latent cause of those symptoms, and the two models would fit the data identically well (Borsboom, Mellenburgh, & van Heerden, 2003). If depression does emerge in this way from a constellation of symptoms, the co-emergence of some other disorder that shares those symptoms is no problem at all, as, for example, when obesity contributes to the emergence of both heart disease and diabetes. Thus comorbidity is a problem not because of the structural models that have been used but because of the causal attributions associated with latent variables. Administration systems rely on accurate description and need not rely on causal understanding at all. In fact, many of the most effective treatment protocols to date have not relied on causal understanding. But ultimately understanding psychopathology will rely critically on understanding development and etiology. For that, researchers only get in trouble when they assume what they should be trying to test. Thus the critical problem with latent variable models is not really the comorbidity they have helped to identify, but the causal attributions they entail.

CWVB have demonstrated that the network perspective offers potential to develop important insights into the patterns of association among DSM IV symptoms, particularly through the possibilities it offers to include estimates of parameters that express the relative frequencies of nodes and the extent to which they are interconnected with other nodes. More importantly, even when the causal assumptions in latent variable modeling are relaxed, their structural models can be accurate descriptions at the level of

the population, but may not be accurate at the level of the individual, which is of necessity also the level at which function must be understood. Network models cannot guarantee consistency between the intra- and inter-individual levels either, but they may be more likely to show it, though this remains to be tested. It might also turn out that a combination of transactional processes (as can be modeled in networks) and latent causal factors provides the best description of the development of some traits (Fraley & Roberts, 2005). Perhaps most importantly of all, however, use of new statistical tool frees such as network modeling us to think about etiology in new ways. Network models cannot tell us directly about causation either, especially if we are not even sure that we have the optimal symptom designations for the disorders, as CWVB hint and we would emphasize. Nor can they help us fix the DSM if we do not have the optimal symptom designations. But they can open our minds to new ideas about etiology that can be tested in other ways, especially if we go beyond the basic cross-sectional data CVWB used for illustration here. And that's what we need to be thinking about.

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