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Symptom network models in depression research

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CHAPTER 7

BETWEEN- VERSUS WITHIN-SUBJECTS ANALYSIS

Based on:

Van Borkulo, C. D., Borsboom, & Schoevers, R. A. (2016). Group-Level Symptom Networks in Depression — Reply. (2016). *JAMA Psychiatry*, 73 (4); DOI: 10.1001/jamapsychiatry.2015.3157.

Based on the paper described in Chapter 6 (Van Borkulo et al., 2015), E. H. Bos and Wanders (2016) wrote a comment. In this Chapter, you can find a summary of their comment, and our reply.

7.1 Summary of comment

The their comment, E. H. Bos and Wanders (2016, henceforth BW) state that drawing inferences from cross-sectional (i.e., group-level analyses) on the level of an individual is unwarranted. They note that network models are conceptualized as dynamic, temporal interactions between symptoms (Borsboom, 2008), but that most studies on networks are performed at the group-level. However, as BW state, co-occurring symptoms at group-level do not imply that they influence each other over time within individuals. This is because associations at the group-level may differ radically from associations at the level of an individual. This phenomenon is known as Simpson's paradox (Robinson, 1950). Therefore, BW state that drawing inferences from cross-sectional, group-level analyses is not informative of processes within individuals, and "will obscure scientific reasoning" (E. H. Bos & Wanders, 2016).

7.2 Reply

In our publication in JAMA Psychiatry (see Chapter 6, Van Borkulo et al., 2015), we reported that the structure of symptom networks is related to the course of depression. Our findings are based on a between-patients design. Although we agree with BW that this has implications for the interpretation of our results, we do not think their conclusions are warranted.

BW correctly point out that, in theory, associations identified through group-level analyses may differ radically across individuals (Simpson's paradox). However, we think that this is not very likely for the reported associations between depression symptoms in our study. First, it is hard to imagine that some patients become less depressed as a result of feeling worthless or get alert and focused when they feel slowed down. Associations between symptoms plausibly differ in degree, but not in kind, so that radical heterogeneity should not be expected for depression symptom networks. Second, our network parameters are partial correlations, not zero-order correlations: thus, each symptom-symptom connection

in the network is already controlled for individual differences in all remaining symptoms, so that Simpson's paradox is ruled out with respect to these symptoms (and strong correlates of them). Third, recent research, which used intraindividual analyses for network estimation, showed that patients with depression had a more densely connected intraindividual network of negative mood states than healthy control individuals (Pe et al., 2015), which parallels our result and suggests a positive answer to Bos and Wanders' question of whether our results generalize to the individual level.

BW further argue that the reported associations between symptoms could be the result of a common cause instead of causal associations between symptoms; they find it "suggestive" that the difference in network connectivity largely disappeared in certain analyses. However, we think this is merely the result of a loss of power due to a decrease in sample size (after matching on severity, the overall sample decreases from 515 to 344) and the strong regularization penalty; both networks lose almost all of their connections and, in that trivial sense, become more alike. As shown in our article (Van Borkulo et al., 2015), when using procedures that have less effect on power (like partialling out general level of functioning or weakening the regularization parameter), differences between groups become more, rather than less, pronounced.

Although we believe that it is not very likely that the associations between symptoms are substantially different for individual patients, intraindividual analyses are needed to test this. In addition, intraindividual analyses are warranted to determine whether symptoms are associated over time within patients. Therefore, we gladly reveal that the Netherlands Study of Depression and Anxiety (Penninx et al., 2008), from which we drew our sample, recently started a new wave of measures in which 400 of its nearly 3000 participants are studied with Ecological Momentary Assessment (aan het Rot et al., 2012) over 2 weeks. The aim of this study is to provide more insight into the association between intraindividual and interindividual differences, which will lead to an increased understanding of how nomothetic and idiographic analyses are related.

