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Published in:
Psychotherapy and psychosomatics

DOI:
[10.1159/000453583](https://doi.org/10.1159/000453583)

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Document Version
Publisher's PDF, also known as Version of record

Publication date:
2017

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Bos, F. M., Snippe, E., de Vos, S., Hartmann, J. A., Simons, C. J. P., Krieke, van der, L., de Jonge, P., & Wichers, M. (2017). Can We Jump from Cross-Sectional to Dynamic Interpretations of Networks? Implications for the Network Perspective in Psychiatry. *Psychotherapy and psychosomatics*, 86(3), 175-177. <https://doi.org/10.1159/000453583>

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Psychother Psychosom 2017;86:175–177
DOI: 10.1159/000453583

Can We Jump from Cross-Sectional to Dynamic Interpretations of Networks? Implications for the Network Perspective in Psychiatry

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Interest in the network perspective of psychopathology is rapidly growing. This theory conceptualizes mental disorders as networks in which symptoms can trigger the presence of other symptoms [1]. It thus theorizes that symptoms actively generate other symptoms, and that this process can eventually lead to a full-blown mental disorder. If this is true, then symptom networks may be informative for clinical practice; symptoms that are more central in the network, and are thus assumed to influence many other symptoms, seem to be a logical starting point for intervention. However, in order to know how to optimally investigate the network perspective empirically, it is crucial to first establish whether different network approaches (cross-sectional vs. dynamic) result in similar conclusions.

Although the network theory described above proposes that symptoms are causally related to one other, most of the published studies use cross-sectional data on symptoms to find empirical support for this [2, 3]. These studies thereby assume, implicitly or explicitly, that group-level contemporaneous associations between symptoms reflect causal influences between symptoms over time [1, 3]. To acquire support for causality, it is important to establish a timeline, i.e., that some symptoms temporally precede other symptoms [4]. It has been questioned, however, whether concurrent, group-level associations among symptoms inform us on how symptoms follow each other over time within individuals [5, 6].

We therefore aimed to investigate to what extent a cross-sectional network yields the same conclusion as a network on the same data that includes dynamic (temporal) associations between

symptoms (i.e., dynamic networks) [7]. If both approaches result in similar conclusions, this would greatly facilitate future research and clinical applications because cross-sectional data can be more easily obtained. If not, then cross-sectional symptom networks are unlikely to reflect causal symptom dynamics, as postulated by this network theory.

To this end, we used the same experience sampling methodology (ESM) data on mood states to compare the 2 network approaches. Data came from the baseline ESM measurements of an interventional trial including 104 patients with a DSM-IV-TR diagnosis of a major depressive episode [8]. Patients rated their momentary mental states on 7-point Likert scales 10 times a day for 5 days, resulting in a maximum of 50 measurement points per individual.

Here, we focus on the 7 mood items that reflect symptoms of depression and that showed sufficient within-person variance over time, namely, sadness, irritation, loneliness, restlessness, worry, self-doubt, and anhedonia (i.e., cheerfulness reverse coded).

Given that traditional cross-sectional networks are based on (1) a single measurement point per individual, and (2) perceived symptoms that are not momentary but estimated over a somewhat longer period, we estimated 2 cross-sectional networks, each reflecting 1 of the above situations. First, we estimated partial correlations between the first observation of the first day of each participant for all 7 symptoms (Fig. 1a). Second, we estimated partial correlations between the person-means of the symptoms (Fig. 1b), i.e., the mean of all 50 measurement points of a symptom provided by 1 individual. Third, a dynamic network was estimated by examining the within-person time-lagged associations among the symptoms in 7 multilevel vector autoregressive models (Fig. 1c), including 1 of the symptoms as the dependent variable and all symptoms at a previous moment in time ($t - 1$) as fixed and random effects [7]. Variables were detrended to remove time trends (not detrending the variables yielded similar results) and person-means were centered to disaggregate within-person effects from between-person effects [9]. The analyses were conducted in STATA (v14.1) and the network graphs were made using the *igraph* package in R. For clarity, only significant associations are shown in the figures of each network (Fig. 1).

For each network, we computed node strength centrality, which is the sum of the absolute value of the strength of all associations of a given symptom with all other symptoms (also nonsignificant ones) [10]. In the dynamic network, strength can be split into “instrength” (the total weight of incoming arrows, not including self-loops) and “outstrength” (the total weight of outgoing arrows). As betweenness and closeness centrality correlated strongly with strength centrality, we decided to not additionally report these measures here.

The results showed that the network approaches not only identified different associations between symptoms, they also yielded different conclusions with regards to which symptom is the most

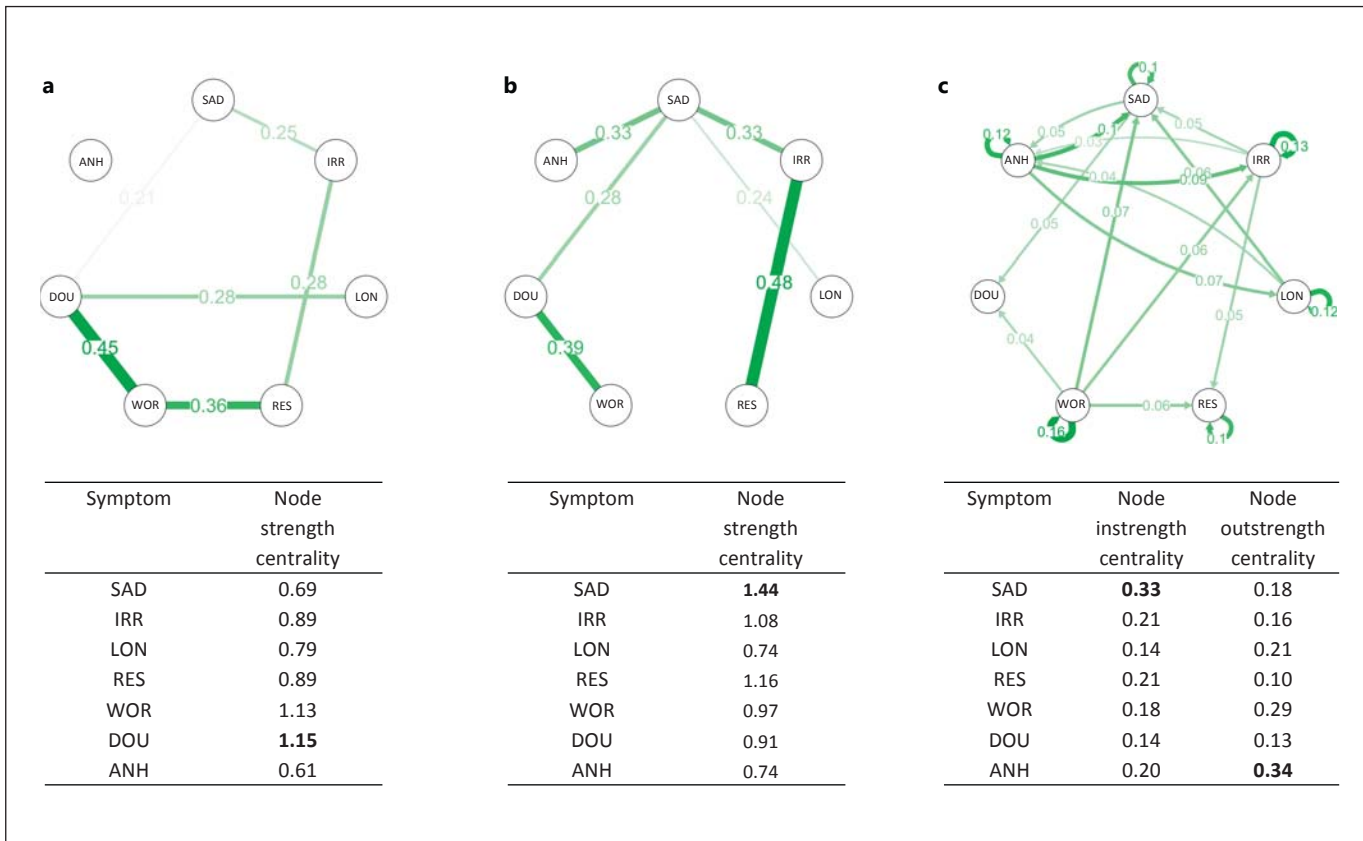


Fig. 1. The first observation network ($n = 104$) (a), the person-mean network ($n = 104$) (b), and the dynamic network ($n = 104 \times 50$) (c), including measures of node strength centrality for each approach. SAD, sadness; IRR, irritation; LON, loneliness; RES, restlessness; WOR, worry; DOU, self-doubt; ANH, anhedonia. Values in bold type denote the centrality estimates that are the most central symptoms within each network approach.

central (Fig. 1). This would have clinical implications if centrality is used to guide targets for treatment. Whereas the first observations network finds self-doubt to be the most central symptom, the person-means network indicates that sadness is the most central symptom. The dynamic network also points towards sadness, but sadness only has the highest instrength, indicating that it is mostly influenced by other symptoms. It does not have the highest outstrength, suggesting that it does not have the strongest impact on other symptoms. Anhedonia has the highest outstrength in the dynamic network, suggesting that any change here would strongly influence the occurrence of other symptoms. Thus, the cross-sectional and dynamic networks would all indicate different targets for intervention.

In terms of order of rank, the node strengths of the different networks only correlated modestly or even negatively with each other. The node strengths of the first observations network correlated negatively with the node instrengths ($r = -0.24$) and negatively with the node outstrengths ($r = -0.33$) of the dynamic network. The person-mean node strengths correlated negatively with the node outstrengths ($r = -0.43$) and positively with the node instrengths ($r = 0.62$) of the dynamic network.

Our results strongly suggest that cross-sectional networks do not reflect how symptoms trigger each other over time, and therefore may not be interpreted as such. Cross-sectional networks may, however, be useful to examine the co-occurrence of symptoms, for example, to offer an insight into patterns of current symptom comorbidity across individuals [1]. Thus, if we would like to empirically test whether causal symptom dynamics are responsible for the development of mental disorders, the use of dynamic network analysis is advised. If future studies yield support for the network theory, then dynamic network techniques may also have relevance for clinical practice in pointing towards promising targets for intervention.

Acknowledgement

We acknowledge an ERC consolidator grant to M. Wichers (project No. 681466; TRANS-ID, 2015), a Rubicon Fellowship from the Netherlands Organisation for Scientific Research to J.A. Hartmann (NWO 825.15.015), and a VICI grant from the Netherlands Organisation for Scientific Research (NWO/ZonMW No. 91812607) to P. de Jonge.

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