Network simulation of depression as a complex system with treatment components

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An increasing number of researchers have considered mental disorders as complex systems that are not solely caused by biological factors but also by a multifaceted interplay of various factors, including psychosocial ones (Fried, 2022). Consequently, the psychological network approach has been gaining attention, particularly in psychology and psychiatry (Robinaugh et al., 2020). Unlike the conventional view of mental disorders as latent variables producing a series of symptoms, the psychological network approach assumes that observable symptoms constitute a mental disorder system (Borsboom and Cramer, 2013). This approach is promising for studying mental disorders as a complex system, owing to its capacity to consider intricate interactions among symptoms. However, despite the recent emergence of this research methodology, an explanation of the complex phenomena of mental disorders is still lacking.

Cramer et al. (2016) conducted a series of simulations to investigate symptom changes by constructing a psychological network of depressive symptoms using empirical data. Their results showed that symptom severity escalates in tandem with the intensification of connectivity within the symptom network. Connectivity is a constant parameter that modulates the value of edges across a network. When it is high, the values of the edges across the entire network become strong, and even a weak change in a single node can have substantial repercussions on the entire network. Consequently,if the network connectivity can be attenuated through psychotherapy, the exacerbation of symptoms can be mitigated.

However, studies comparing networks before, during, and after psychotherapy implementation have had divergent findings (Höller et al., 2022). Höller et al. (2022) investigated, the structure of the symptom network in 401 outpatients at a clinic at three-time points: before, 12 weeks after, and after the implementation of cognitive behavioral therapy. They evaluated depressive symptoms using the Beck depression inventory (BDI) and found that, although the intervention significantly reduced BDI scores, network connections were strengthened rather than weakened.

At first glance, the simulations of Cramer et al. (2016) and the results of Höller et al. (2022) appear to conflict with each other. It is important to consider that the simulation of Cramer et al. (2016) solely employed a network of depressive symptoms. It is crucial to recognize that the symptom network of mental illnesses is influenced by external factors other than symptoms. It is assumed that certain symptoms can be affected by therapeutic factors that are external to the symptom network.

Based on the simulation by Cramer et al. (2016), we conducted to simulate depression by incorporating treatment components(TC). The purpose of this study was to investigate the impact of external factors on the symptom network of mental disorders using simulations of depressive symptom networks with additional TC.

Simulation

In this study, R (version 4.2.2) was used to conduct all simulations. The dataset employed to calculate the symptom network was the open dataset of symptom weights and thresholds employed by Cramer et al. (2016).

Simulation 1. Treatment components and change in depressive symptoms

A network model was employed incorporating the TC based on the hypothesis that examining only the symptom network of interest is insufficient. It was postulated that the weight and threshold of the TC would follow to a Gaussian distribution, characterized by a mean equivalent to the average of the symptom network and a standard deviation of 0.1. Furthermore, an inverse correlation was established between the TC and the symptom nodes. It is essential to emphasize that the configuration of the symptom network remained consistent with that presented by Cramer et al. (2016), and the connectivity value was maintained at 1.0.

The changes in the symptoms and TC were examined by progressively incorporating the nodes of the TC into the symptom network of Cramer et al. (2016)(Figure.1). The symptom network delineated by Cramer et al. (2016) encompasses 14 nodes pertinent to depressive symptoms. As a result, 1, 3, 6, 9, 12, 14, 15, 16, 17, and 18 TC nodes

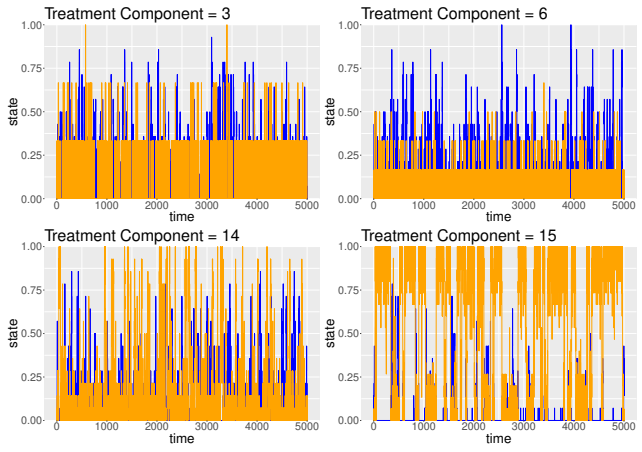

Figure 1: Changes in treatment effects and symptoms

were incorporated. Specific symptoms upon which the TC would exert influence were randomly assigned. In the Figure.1, the blue lines depict depressive symptoms, and the orange lines depict treatment effects. The simulation results demonstrated a gradual and steady continuation of the treatment effect as the number of TC increased, accompanied by a decline in depressive symptoms. A significant increase in the treatment effect was observed when the number of TC increased from 14 to 15, which was accompanied by a marked reduction in depressive symptoms. The simulation involved a stochastic computational process. Therefore, 5000 calculations were conducted for each additional TC, and the necessary number of TC was estimated based on the average value. These results suggest that 14 or 15 therapeutic components may be necessary to alleviate depressive symptoms when using 14 symptoms to treat depression.

Simulation 2. Estimation of the number of treatment components by differences in the number of symptom nodes

In Simulation 1, we found that incorporating TC into the depressive symptom network could reduce symptoms independent of network connectivity when the number of TC matched the number of symptom networks. Consequently, to explore the relationship between the number of symptoms and TC, a simulation was conducted to determine the number of TC that would be necessary if the number of symptoms were reduced. Simulation 1 involved 14 symptoms, and the corresponding number of TC required was 14 or 15. Thus, when the weights and thresholds of the TC and symptoms are comparable, it is necessary to match the number of TC with the number of symptoms to alleviate them.

The purpose of Simulation 2 was to ascertain the requisite quantity of TC by reducing the number of symptoms. The basic structure remained analogous to that in Simulation 1. Symptoms were incrementally reduced from 13 to 3, with one symptom excised during each iteration. For example, when only 3 symptoms were present, 5 TC proved necessary, whereas 12 TC were required when there 7 symptoms. In these conditions, this investigation revealed a discrepancy between the number of symptoms and the essential quantity of TC, indicating that a greater number of TC were required compared with the number of symptoms.

Simulation 3. Relationship between the strength of connectivity and treatment components

Simulation 3 was conducted to explore the relationship between the TC and network connectivity. The fundamental configuration was similar to that used in Simulation 2. In Simulation 3, the network connectivity was altered to explore its impact on the number of TC, consistent with the work of Cramer et al. (2016). The connectivity varied from 0.8, indicating weak connections, to 2.0, indicating strong connections, in increments of 0.05.

Initially, the severity of depression deteriorated with an increase in connectivity. For the number of TC required, it was found that, for low connectivity levels (approximately 0.8 to 1.0), more than 14 TC were necessary. However, once the connectivity exceeded 1.0, approximately 14 TC were required. When the connectivity became excessively large, the symptoms displayed unstable behavior, repeatedly rising and falling higher and lower, even with an increase in the number of TC. Specifically, when connectivity surpassed 1.3, increasing the number of TC beyond 14 resulted in alternating rapid activation and suppression of symptoms (for instance, 14 TC decreased symptoms, 15 TC increased symptoms, and 16 TC decreased symptoms). These findings suggest that interventions aimed solely at increasing the number of TC may not necessarily be effective in patients with strong network connectivity.

Conclusions

Our simulations suggest that a symptom-focused network may not adequately capture mental disorders as complex systems. Our simulations included TC nodes but could benefit from incorporating non-symptomatic nodes, such as environmental stressors. Understanding each TC's impact is crucial, and reconciling discrepancies between simulation outcomes and clinical data is necessary for successful implementation.

Advancements in understanding mental disorders through simulations, such as those in this study, have the potential to deepen the understanding of how these disorders are developed, maintained, and treated. Progress in understanding mental disorders may also benefit artificial life research, contributing to the development of more realistic and more accurate models of complex systems and making more precise research possible.

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