**Critical View of Causal Relations and Construction Rules Regarding the Network Theory in Psychopathology**

Name: Tom Yishay

Student number: 12203424

Supervisor: Britt Veldman

Group number: 27

Number of words: 1547

Date of submission: 26th of January 2020.

Think about the last time you made dinner for your date, but to your surprise it did not work out as you expected. You followed the exact same steps as you always do, and yet you faced a shameful failure, that according to your mom, decreased the chances of a second date. Was it the stress that caused the failure or perhaps the quality of the ingredients? Did the dish truly cause the chances of a second date to drop? This familiar situation illustrates the nature of human thinking; a fundamental tendency to make sense of the world in causal explanations. In order to validate such causal inference, that will unravel the relationship between events around us- scientific research is required. In fact, research into the possibility and nature of causal relations have yielded three important principles (Campbell, Cook, & Shadish, 2002, p. 39). Firstly, the principle of priority states that for event X to constitute the cause of event Y, event X must occur before event Y. Secondly, the principle of consistency states that event Y and event X must co-vary. Namely, there is a relationship between the two events, often expressed by correlation. This is a notion that will be further discussed in the first paragraph of this essay. Lastly, the principle of exclusivity states that event X must be the sole cause of event Y. That is, there are no other plausible explanations to the occurrence of event Y, except for event X. By following such principles, causality allows scientific growth as well as practical implication to social and clinical settings, notions of utmost importance. Taken together, the use of proper and rightful methodology to infer casual relations is vital for the construction of policies, treatment, diagnoses and more. Moreover, in mental health, a recent theory in psychology seeks to explain pathology as a network; an interactive net of symptoms that together constitute mental imbalance, namely the network model of psychopathology. According to Borsboom and Cramer (2013), individuals are seen as complex systems of symptoms interacting in a causal manner. Meaning, symptoms of mental disorders are conceptualized as nodes, causally related to each other by edges. Edges are the “lines” connecting the symptoms in the system, estimated by statistical tools. Importantly, the nodes are the active and exclusive cause of one another. Nodes can also be external harmful events, as traumas for instance. To illustrate, a depressed person might suffer from sleep deprivation (first node) which leads to focus problems (second node) resulting in lower achievements in University (third node) and therefore to anxiety to drop out of school (fourth node) that finally reinforces sleep deprivation again (first node). The cause of each symptom is its connected symptom, resulting in the formation of a causal system. Vitally, as for the methodology behind the construction of such causal connections, correlation is often used to connect different symptoms as well as direct reports from clients of their perceived relations between symptoms. In this essay, I argue that network theory of psychopathology is unable to fulfill the principle of causal relations between the nodes due to insufficient methodology estimating the causation between symptoms. Firstly, I will demonstrate the inability of drawing causal relationships from mere correlations, as done in network modeling. Secondly, I will argue that the use of perceived causal relationships (PCR) as an alternative construction rule undermines the principle of causal connections between symptoms.

Correlation does not imply causality and thus network models that argue for causality can never use correlation alone to connect two nodes in a causal relationship. To begin with, correlation is simply defined as the standardized co-variance of two variables. It is the linear variation of two data sets mutually, reflecting the principle of consistency. Thus, correlation is a necessary but insufficient condition for causal inferences; causation cannot exist in the absence of correlation but vice versa this is possible. This is because correlation allows causality in three different, equally possible directions (Aldrich & John, 1995). Firstly, in the case of correlation between variable X and Y, the direction of influence is unknown. It is simply impossible, in many cases, to determine whether variable X causes variable Y or vice versa. To illustrate, a positive correlation between aggression and aggressive video games might imply that aggressive video games cause more aggression or alternatively, more aggression causes the consumption of aggressive video games. Applying this issue to network modeling, the argued causal relations based on correlations between nodes are uninformative of the direction of influence. The possibility of sad mood causing sleeping problems might be as likely as the possibility of sleeping problems causing sad mood. A second complication with using correlation to construct casual relationships is the notion of confounds (Pearl, 2018). A confound is a third, unobserved or unknown variable that causes two other variables to correlate. Namely, a common cause or result. In this case, there is a violation of the principle of exclusivity. Returning to network models, the connection of nodes based on mere correlation ignores the possibility of confounds. Perhaps a certain gene or medical condition causes or results in both mental symptoms. That is, sleeping problems and sad mood can be both caused by a medical condition that is unlikely to be discovered if only accounting for psychological properties as nodes. This is important to emphasize, as network models in psychopathology connect only “mental” symptoms and leaves out medical conditions that might constitute a confound in the system. Finally, an additional issue for causality arises given the adoption of PCR as a valid constructor of networks.

The use of perceived causal relationship (PCR) as an estimation of causal relationship between two symptoms (nodes), is inadequate and biased, and thus suffers from low validity. Validity, in this essay, refers to whether a procedure measures what it purports to measure (Kelley, 1927). PCR is a method in which the client reports the nature and type of symptoms, according to their subjective view. The client connects the symptoms in casual manner, based on their self-perception. For example, a patient might report that a recent drop in sleep quality caused him to feel increased sad mood lately. The client is the ultimate source of data from which the psychologist draws causal inferences. The validity of the self-report rests on the grounds of memory and perception of the client as the absolute source of network construction, a capacity known to be biased, by the availability bias for example (Esgate & Groome, 2005). Availability bias is defined as the reliance on immediate examples that pop into one’s mind when evaluating a topic or a concept. To illustrate, if during the week before the interview a client barely experienced any sad mood but only repetitive sleeping issues (even though sad mood might have been the main cause over a long period of time), he might not mention sad mood at all. Taken together, the ability of PCR to validly capture the casual relationships between symptoms is questionable due to the possibility of biased conclusions. Moreover, all three mentioned principles of causality are violated when using the PCR method (Shadish, Cook & Cambell, 2001, p. 39). It is rather improbable, for the client to know whether a certain symptom preceded another (priority), was the exclusive cause of it (exclusivity) and clearly the client is unable to report standardized co-variance (consistency) between the symptoms. This issue becomes even more problematic when considering abstract symptoms such as thoughts, desires and/or feelings, that are inevitably a part of psychological networks. To demonstrate, a client that is asked to report whether he first experienced extremely sad mood and then suicidal thoughts or the vice versa, is required to make an inaccessible distinction. Thus, reports that rely on memory and perception suffer from low validity. As the PCR is one of such self-reports, it is an invalid as a sole method for the estimation of causal connection between symptoms in a psychological network.

To conclude, the implantation of network models in psychopathology involves methodological complications regarding the causality of symptoms and events, often violating three important principles of causation. Firstly, the use of correlations to estimate causal connection between nodes, has shown to be problematic because of interchangeable causal inferences regarding the same data set. Secondly, the PCR method, utilizing subjective reports to construct networks, lacks sufficient validity due to cognitive biases. Perhaps by accounting for biases in self report and supporting correlations with mechanistic and theoretical backgrounds- the theory of network models in psychology can be dramatically improved. To demonstrate, self-report can be concretized by adding reports from the social environment of the individual, such as close friends and family. This simple act may already increase the validity of the PCR method. Additionally, the use of statistical tools should be supported by laboratory, experimental designs that shed light on the interaction of life events, symptoms and their causation. Lastly, devoting more attention into the three guidelines of causation will enhance the possibility of causation effectively. This essay asks to remark the significance of unconditional critical view. Practically, as new perspectives to mental and physical illnesses evolve and offer fresh and unique viewpoints, it is vital for the researcher to regard these with critical thinking, promising positive and vital developments in the field of health and wellbeing.

**References**

Aldrich, J. (1995). [Correlations genuine and spurious in pearson and yule](https://web.archive.org/web/20060219042545/http%3A/www.economics.soton.ac.uk/staff/aldrich/spurious.pdf). *Statistical Science, 10*, 4, 364–376. [doi](https://en.wikipedia.org/wiki/Digital_object_identifier):[10.1214/ss/1177009870](https://doi.org/10.1214/ss/1177009870).

Borsboom, D., & Cramer, A. O. J. (2013). Network analysis: an integrative approach to the structure of psychopathology. *Annual Review of Clinical Psychology, 9,* 91-121. Doi: 10.1146/annurev-clinpsy-050212-185608.

Campbell, D. T., Cook, T. D., & Shadish, W. R. (2002). Experimental and quasi-experimental designs for generalized causal inference. *Houghton Mifflin Company*. ISBN: 0-395-61556-9.

Esgate, A. & Groome, D. (2005). [An introduction to applied cognitive psychology](https://books.google.com/books?id=B4agXAUYv6QC)*. Psychology Press.*[ISBN](https://en.wikipedia.org/wiki/International_Standard_Book_Number):[978-1-84169-318-7](https://en.wikipedia.org/wiki/Special%3ABookSources/978-1-84169-318-7)*.*

Kelley, L. (1927). Interpretation of educational measurements. *World Book Company.*

Mackenzie, D., & Pearl, J. (2018). “The book of why”. *Basic Books*. ISBN: 9780465097609.