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Scale Issues in Causality

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Abstract

Elaboration of the manner by which graphical frameworks of causality can benefit personality research is a much-needed contribution. We argue that attempts to apply these frameworks in personality will benefit from consideration of two concepts related to scale. The first is that the appropriate scale on which to evaluate causality depends upon the level of analyses on which the research is conducted. Second, the distal scale between typical expressions of personality and their possible causes limits discussion of causality to probabilistic rather than mechanistic factors.

By virtue of even attempting to integrate Judea Pearl's innovative work on causality (Pearl, 2009) into the personality psychology literature, James Lee is to be commended for initiating a conversation that is - in truth - uncomfortable for a field of "correlators" (Cronbach, 1957). But it is Lee's concise elaboration of so many nuances of the graphical framework that make the target article an invaluable contribution to the field, particularly for those unfamiliar with Pearl's work. To the extent that personality psychologists increasingly focus on the development and evaluation of predictive causal models, we consider it likely that the influence of this work by Lee will grow over time. Though many aspects of Lee's review merit further exploration, our commentary primarily focuses on two aspects of the relationship between the causal framework that Lee describes and considerations of *scale*.

The first point of note pertains to Lee's comments on the role of psychometric factors in graphical conceptualizations of psychological causality. We agree with Lee that one of the goals of personality research should be to recursively "expand a directed edge in one graph into an entirely new subgraph" (p. 47) in order to understand the mechanistic relationship between two nodes, an endeavor that would ideally result in richly detailed causal diagrams similar to those found in biology texts. Achieving this level of mechanistic detail may well resolve debates about the causal status for many psychometric factors but it would not mitigate the functional utility that factors provide.

By analogy, a graph is something like an online geographic map. Psychometric factors, like many cartographic features, are not physically observable; researchers would no more benefit from "discarding the convenient fictions of folk psychology" (p. 48) than travelers would if the town, state, and nation labels were stripped from maps. The familiar experience of "zooming" an online map to the appropriate viewing level illustrates the contextual utility of complexity, which is itself a function of scale. Practically infinite detail is *possible* in mapping of both human behavior and geography, but this would rarely be functional.

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Conceptual relationships are more easily grasped and manipulated when they are manageable in number and roughly similar in scale.

For example, it is common for trait psychologists to lament the broad imprecision of factors like the Big Five which are borne out of data reduction, yet most would agree that this is the appropriate level of analysis for evaluating topics such as the differential relationships on career outcomes of Extraversion and cognitive ability (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). More narrow organizational frameworks such as the Big Five Aspect Scales (DeYoung, Quilty, & Peterson, 2007) or the 30 NEO facets (Costa & McCrae, 1992) would be appropriate for exploring the differential relationships of various facets of Extraversion. An example of zooming in still further might include recent theoretical work relating an individual's desire to engage in prototypically extraverted behaviors to physiological measures of dopamine (Smilie, Cooper, Wilt, & Revelle, in press).

"Fictional factors" play an integral role at each of these levels of analysis in the form of latent behavioral trait constructs. Admittedly imprecise at all levels, factors enable researchers to delineate continuous streams of observable behaviors into chunks which can be reasonably well measured and organized. The speculative proposition to map each of the hypothetical relationships between low-level biology and long-term, multiply-determined outcomes such as extraverted behaviors that affect career outcomes would seem to neglect this tension between precision and efficacy. To be clear, we do not argue against the merits of model specificity, but rather for the merits of appropriate model scaling. While it is invaluable to be reminded that the factor analytic approach can only inform a subset of the questions personality psychologists hope to explore, it is also true that the parsimonious chunking of data that factoring allows will likely justify its continued application. Lee stops short of offering psychological researchers a metric for identifying and pursuing the appropriate levels of analysis.

Our second point is to emphasize that the scale on which personality unfolds requires acknowledgement of the distinction between *mechanistic* and *probabilistic* causality. Intuitively speaking, the complexity of pathway navigation is a function of proximity. To be more precise, the number of intermediate nodes is the variable that determines the number of alternative pathways, but it is effectively true that outcomes which are temporally or physically proximal to their causes are predicted with higher probability than those which are distal. In other words, the pursuit of mechanistic causality is a reasonable aspiration on a small scale because outcomes and their purported causes are relatively proximal.

Most outcomes under study by personality psychologists, however, are multiply-determined over long periods of time. This suggests that the majority of causal factors are probabilistic rather than mechanistic. Conditioning discussions of causality on this distinction is vital as the very basis for researching personality and cognitive ability would be altered if these constructs were found to be mechanistically determined by genetics. The distinction is also sobering in that it forces us to acknowledge that personality is determined by the cumulative influence of thousands of genes, a nearly infinite variety of environmental variables, and multiple types of gene by environment interplay (Johnson, 2007) (some of which may occur more commonly than the rare incident of mutation claimed by Lee as justification for general temporal restriction (p. 40)). These genetic and environmental inputs do not directly cause behavior, but are rather mediated through their effect on proteins and subsequent neural systems that lead to differential environmental sensitivities resulting in different cognitive, affective, and motivational values.

Lee acknowledges this complexity and clearly explains why the proximity of cause and outcome is less relevant for gene-trait association studies. However this does not imply mechanistic causality for the expression of the trait in any given context. Unlike the examples made of height, hair morphology and Parkinson's, personality constructs describe *typical* manners of behaving across a wide variety of contexts. We take this to mean that future knowledge regarding gene-trait associations would only allow for *probabilistic* estimates, for example, of a given individual's typical desire to attend lively parties as an opportunity to express the extraverted tendencies which result from the dopaminergic effects on their wanting system (Smilie et al., in press). While this type of knowledge will someday constitute an impressive contribution to

the field, it will not reflect "causality" as traditionally defined.

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