RUNNING HEAD: Latent variables and theory

**To wish impossible things: On the ontological status of latent variables
and the prospects for theory in psychology**

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In “Lack of theory building and testing impedes progress in the factor and network literature,” Fried (this issue) calls for more attention to theory in psychological research on individual differences, and he considers the contributions that factor and network analytic methods can make to theory. As a general topic, this is timely for multiple reasons, depending on the area of psychology in question. Personality psychology has recently seen an increased interest in theories regarding the nature and sources of personality traits (e.g., Denissen & Penke, 2008; DeYoung, 2015; Fleeson & Jayawickreme, 2015; Nettle, 2007; Roberts, 2018; Wrzus & Roberts, 2017). This pivot to theory followed several decades of atheoretical attention to the covariance structure of personality, the identification of major dimensions of covariation (most prominently the Big Five), and the investigation of those dimensions as predictors of various outcomes. Meanwhile, research on psychopathology has recently seen a shift in focus from traditional diagnostic categories, which have been thoroughly demonstrated as empirically invalid, to symptoms and putative mechanisms of psychopathology assessed dimensionally (e.g., Borsboom, 2017; DeYoung & Krueger, 2018a; Insel et al., 2010; Kotov et al., 2017; Widiger & Sankis, 2000). And researchers of cognitive abilities continue to generate a variety of interesting theories about their nature and sources (e.g., Jung & Haier, 2007; Kovacs & Conway, 2016; van der Maas et al., 2006), none of which attempt to attribute the existence of a statistical factor of general intelligence to a single causal source.

Given this widespread impetus toward theories of individual differences based on dimensional constructs, Fried’s consideration of impediments to effective theory building is clearly important for psychology. One might wonder, however, what Fried means by “lack of theory building and testing,” given the array of examples of interest in theory we just provided. What Fried has in mind by “theory” is much more specific than what many theories provide,

namely a precisely parameterized mathematical model of the causal relations that explain the phenomenon in question. In this commentary, we argue that Fried is providing a number of insights that will be very useful for psychologists, but also that he is wishing for psychology to focus on a kind of theory that it cannot generally be expected to produce at this time. As we will explain, Fried asks too much of our models and our theories. In part, this stems from a failure to consistently heed his own warnings about the limitations of statistical models, but it also reflects an overestimation of the kind of evidence and reasoning necessary to develop and test theory. We too believe that psychological research on individual differences should pay attention to theory, but we are more optimistic than Fried about the progress that can be made through a focus on the kind of theory that psychology is actually capable of producing. In order to justify our optimism, we first develop our perspective on the nature of latent variables and then consider how individual-difference researchers should approach theory that has the potential to explain them.

Fixing Problems with the Network Approach

The most salient context for Fried's article is the recent rise of network analysis as a popular approach in research on individual differences—an approach which its popularizers framed as a competitor or correction to factor analysis and other latent variable methods (Borsboom, 2017; Cramer et al., 2010, 2012). Fried goes a long way toward rectifying many of the problems with this network approach that we and others have previously identified (e.g., DeYoung & Krueger, 2018b; Forbes et al., 2017a, 2017b), and this makes his article an important advance in thinking about the role that network analysis can play in psychological research on psychological individual differences.

First and foremost, Fried shows that the hostility toward latent variables demonstrated by early articles on the network approach was unnecessary (e.g., Cramer et al., 2010, 2012). Many latent-variable and network models are fungible, meaning that they are mathematically equivalent and can model the same data equally well (Bringman & Eronen, 2018). Further, latent variables can be integrated into network models, a fact that many proponents of the network approach are beginning to acknowledge (e.g., Epskamp, Rhemtulla, & Borsboom, 2017). Any node in any network model can in principle be measured and modeled as a latent variable simply by including multiple indicators of the construct that node represents.¹ This would improve the model by removing unsystematic error from the variables of interest. Even more importantly, in any network model there may be variance that is systematically shared among more than two nodes due to some unmeasured common cause or set of causes. If these relations aren't modeled using latent variables, the structure of the network as a whole will be distorted when using typical methods of network analysis that rely on conditional dependence (Yuan, 2012).

Fried not only emphasizes that factor and network approaches are complementary and can be integrated with each other, he also acknowledges the existence of broad common causes that influence many symptoms or traits. One of the hallmarks of the network approach, codified in the so-called “network theory” (Borsboom, 2017; Cramer et al., 2012), has been the denial that the covariance of personality traits or symptoms of psychopathology can be due to causal factors that influence many traits or symptoms, rather than to direct interactions of the traits or symptoms with each other. This denial is simply not plausible given what is known about how the brain works (DeYoung & Krueger, 2018b). For example, many of the symptoms of anxiety (e.g., physiological arousal, difficulty concentrating, and rumination) tend to co-occur in time within people not merely because of their direct causal effects on each other (although such

effects may also exist) but because they are generated by a coherent brain system in response to threatening uncertainty and motivational conflict (Corr et al., 2013; Gray & McNaughton, 2000). Plausible causal accounts of neural and psychological function involve mechanisms that carry out core functions in response to broad classes of stimuli (such as threats, rewards, and distractions) appearing in many different situations and requiring coordinated emotional, motivational, cognitive, and behavioral responses. This means that common causes are likely to exist both in the environment, as certain stimuli will influence multiple functionally related responses (Fried mentions traumas and stressors), and within the organism, as specific brain systems will influence multiple functionally related responses. The broad causal influences of such processes will often appear in psychological data as patterns of covariation that are appropriately modeled with latent variables.

Confusing Statistical Models with Causal Models

Despite our broad agreement with Fried about the compatibility of factor and network approaches, we nonetheless have some concerns about his treatment of latent variables and related models. Fried distinguishes between statistical models and theoretical models, and we agree this is a crucial distinction and the two should not be confused. Theoretical models are by definition causal and offer causal explanations. The fact that statistical models and causal models often use the same visual vocabulary of circles, boxes, and arrows contributes to their confusion. Despite this visual similarity, we assert that structural equation models are not well conceptualized as causal models. Structural equation modeling (SEM) is a statistical tool for mathematically modeling covariance relations among a set of measured variables. Only when very specific types of research design are used can SEM be in any way informative about cause, and such designs are rare in research on individual differences.

In order to avoid confusion between statistical and causal models, we advocate for reserving the term “latent variable” to refer only to statistical constructs that are or could be included in statistical latent variable models. The term should not be used to refer merely to any unmeasured construct of interest. A latent variable is *a mathematical representation of some portion of the variance of one or more indicator variables*, typically the shared variance of multiple observed variables. (Note that some latent variables have a single indicator; however, in order to be estimated, these must be specified *a priori* by fixing the loading of the indicator, often based on an estimate of the reliability of the indicator, which is effectively a guess about how strongly it should relate to other similar indicators.) A latent variable is not a causal influence on its indicators. Instead, it merely entails that some causal force(s) lead its indicators to share variance. Those causal forces could be any number of unmeasured variables or they could be direct causal interactions among the entities measured by the indicators (Bartholomew et al., 2009; van der Maas et al., 2006). A latent variable model is inherently agnostic about the nature of the causes of the covariance that it models.

Despite Fried’s attention to the difference between statistical and causal models, he sometimes blurs the distinction. For example, on p. xx he writes, “the common cause interpretation is the most sensible one given the reflective model, which features causal arrows from the latent variable to the observed indicators.” Properly discriminating between statistical and theoretical models entails acknowledging that there are never any “causal arrows” in latent variable models, only regression paths. The arrows from a latent variable to its indicators provide nothing more than estimates of the variance in each indicator that is accounted for by the latent variable, which is to say, the variance that the indicator shares with other indicators. Similarly, on p. xx, Fried refers to the “causal-reflective perspective” on latent variable models, but again,

this blurs the important distinction he made previously. There is no unified “causal-reflective” perspective, because statistical models are not causal models and “reflective” is a descriptor for a class of statistical models. In footnote 6, Fried argues that although models are necessarily simplifications of the phenomena in question, “this is no excuse for causally mis-specifying the hypothesized relation between the psychological construct and observed items.” Once again, this is contradicted by his own insight that statistically specifying a model is different from causally specifying a model. Given that a latent variable model is a statistical model only, it cannot be “causally mis-specified,” although certainly there are valid reasons to choose different types of model in different cases.

Not only does Fried slip up by attributing causal properties to factor analytic models, he does something similar when talking about network models. For example, on p. xx he writes, “from a network perspective, items that load on two factors simultaneously make for the potentially most interesting items, because they may build causal bridges between two communities of items.” Unfortunately, this follows a section of the article in which he has already pointed out that no statistical model of the covariations among cross-sectional items is in fact a causal, theoretical model, regardless of whether it involves factors or networks. *Any researcher who hopes to determine causal relations merely by fitting a statistical model that estimates regression or correlation paths in typical cross-sectional data is wishing for the impossible.* Despite making this point himself, Fried seems occasionally to confuse statistical and causal models. It is imperative for researchers to remember that paths in SEM or network models are not inherently causal and can be causally informative only with special kinds of designs (such as when one variable is directly manipulated, or in the presence of adequate instrumental variables).

Formative Latent Variables Do Not Exist

In this context, we think it is important to note that there is no such thing as a formative latent variable. The contrast that Fried draws between reflective and formative latent variable models is common but unfortunate. Because a latent variable is a representation of shared variance, it is necessarily reflective. This may seem a provocative claim, but we believe it is very clear that no one has ever fit a model that included a formative latent variable. No doubt the reader may be able to find published SEMs that claim to include formative latent variables, but any model making such a claim is mistaken. A model of a formative latent variable—that is, one in which several observed variables have regression paths pointing to a latent variable—is undefined in isolation, and hence one never sees such a model. Instead, what one sees are models in which a supposedly formative latent variable, with multiple variables predicting it, also has a regression path predicting some other variable(s). (We use “prediction” here in the regression sense of accounting for variance, not in the temporal sense.) In this situation, what is really happening in the model is that the supposedly “formative” latent variable is in fact reflective and is being indicated, and thus defined, by whatever variable(s) it is predicting. To call it “formative” is mere sleight of hand; in reality, a reflective latent variable is being predicted directly by several observed variables. This also means that when such variables are described as “formative,” they are often mislabeled because the label is typically based on the content of the observed variables that predict the latent variable rather than on the content of the variable(s) that are the actual indicators. The observed predictors are sometimes called “causal indicators,” which is a double misnomer, given that they are not indicators and the model is not a causal model. There is nothing wrong with the idea of a formative variable, but it cannot be a latent variable.

Interpreting Latent Variables

The term “latent variable” can appropriately refer either to the shared variance of indicators in a specific model or to the shared variance of an optimal set of indicators for the variable in question. The *g* factor of cognitive ability provides an excellent example of this distinction. In any particular set of data that includes sufficiently difficult cognitive tasks, a *g* factor can be created as the shared variance of performance on those tasks. Depending on the tasks involved, however, the *g* factor in those data may be a better or worse estimate of the *g* factor that exists independently of any given sample of tasks. We know that the latter *g* factor exists because, when sufficiently broad sets of indicators are used, the *g* factor is identical across different cognitive batteries (Johnson et al., 2004, 2008). This means that the *g* factor is a real psychological entity that exists independently of method variance, but remember that we have already stated that latent variables are not causal entities—so what kind of entity is the *g* factor? In short, it is a statistical entity, a real and robust pattern or regularity (Dennett, 1991), in which some people are generally better able to perform diverse difficult cognitive tasks than other people.

Not every latent variable has this kind of method-independent existence. The general factor of personality (GFP), for example, appears to be due to the presence of method variance. It exists in many datasets when one uses a single-informant method, but it does not exist when an optimal set of indicators is used to measure it—namely, a broad set of latent personality variables based on multiple raters, such that each variable serving as an indicator of the GFP represents what is shared across raters of the same construct, thereby removing the biases introduced by individual raters (DeYoung, 2006; Chang et al., 2012). The GFP, therefore, is a latent variable and a statistical entity in some data, but it is not the kind of latent variable that is method-

independent. Hence, it is not a real psychological entity and does not reflect a real tendency for all personality traits to covary. (It is important not to confuse measures and constructs; the personality traits are the actual patterns of behavior and experience, not the scores on questionnaires.)

In order to distinguish between interpretations of latent variables as statistical regularities and latent variables as causal entities, Fried (p. xx) states, “Latent variables like the *g* factor and *p* factor sometimes refer to statistical constructs, at other times to psychological constructs,” but we think that the situation is more complicated than this implies, and a further distinction needs to be made. In referring to “psychological constructs” here, Fried seems to mean a single common cause of covariance.² However, psychological constructs need not be single causes because they can instead describe tendencies to be in particular states. Such tendencies are *dispositions*. A disposition is inherently both a psychological construct and a statistical construct because it describes the likelihood of being in particular psychological states. We would argue that latent variable models, because they are not causal models, are useful precisely as ways to study dispositions. All of this entails that sometimes people use the term “latent variable” or “factor” to refer to a purely statistical construct (the shared variance of its indicators in any given model), sometimes to refer to a statistical psychological construct (the shared variance of an optimal set of indicators – a disposition), and sometimes to refer to a single common cause. Only the last of these is invalid, from our perspective.

Different models are useful for different purposes. A latent variable model is useful for focusing research on the disposition represented by the shared variance of its indicators. A latent variable is the thing that needs to be explained by theory; it is not an explanatory construct in and of itself. If one is using a latent variable for etiological research—trying to understand the

sources of the disposition in question by examining relevant correlates—this does suggest that one believes some (probably unspecified) number of causal processes affect all of its observed indicator variables.³ Nonetheless, the latent variable is not that cause or set of causes; it is merely the shared variance of its indicators. If that shared variance is of no interest for good theoretical reasons, because there is evidence that the indicators do not share any common cause, then it probably does not make sense to model the shared variance among those observed variables as a latent variable in etiological research. (Note that network theory has never provided good theoretical reasons for denying common causes entirely, especially given the evidence for their existence that both we and Fried have described.)

Developing Theories of Psychological Individual Differences

Latent variables can be valid estimates of dispositions, but they are not causal, explanatory entities in and of themselves. How should one go about developing theories that can explain dispositions? Fried adopts very high standards for such theories, represented paradigmatically by a theory of panic disorder that he and collaborators have self-published on the internet (Robinaugh et al., 2019). For Fried, it appears that the only good theory is a formal, computational model, in which every parameter is numerically specified and the functional form of every causal relation is hypothesized. Mathematically formalized theories are indeed “strong,” but the question for any such theory is whether it is realistic and useful.

We can contrast such theories based on computational models with a very different theory, the Cybernetic Big Five Theory (CB5T) of personality and its *cybernetic dysfunction* extension that attempts to explain psychopathology (Allen & DeYoung, 2017; DeYoung, 2015; DeYoung & Krueger, 2018a, 2020). In Fried’s terms, CB5T is a “weak” and entirely “verbal” theory, offering hypotheses mainly of the form “x is associated with y.” We are frequently asked

why CB5T does not have a computational model associated with it, given that cybernetics (the study of the principles governing goal-directed systems that self-regulate via feedback) is often used to develop computational models. As we have previously explained (DeYoung & Krueger, 2018b), computational models are not useful unless they resemble the phenomena they are modeling sufficiently accurately that one can gain new insights about those phenomena by studying the model. Too often one can gain information only about the limitations of the model. Given the truly staggering complexity of the human brain and its psychological functions, we are generally skeptical about the ability of computational models to achieve the necessary resemblance, especially when attempting to explain complex phenomena such as personality and psychopathology. Fried's vision of "testing the effect of a treatment without actually conducting a clinical trial" (p. xx) is likely to remain a pipe dream for a very long time, possibly forever.

To be clear, we are by no means denying the utility of formal models. However, such models work best in the context of relatively circumscribed psychological phenomena about which we already have a great deal of information. Typically, effective computational models focus on simulating a narrow psychological and/or neurobiological phenomenon in high detail (e.g., Pickering & Pesola, 2014). Panic disorder may be sufficiently narrow and circumscribed that the computational model of Robinaugh et al. (2019) will prove useful in advancing our understanding. Nonetheless, when considered in the context of real data, their theory immediately encounters complexities that have not been accounted for in the model. For example, panic disorder almost never presents in isolation; in a study of a sample of primary care patients, only 2% of panic disorder cases were pure cases with no comorbid diagnoses (Tilli et al., 2012). Epidemiologic data also show the rarity of "pure panic disorder" (Kessler et al., 2006). In short, panic disorder cannot be considered a distinct, categorical diagnostic entity,

given the evidence that it rarely presents in isolation and the more general fact that no common mental disorders have been demonstrated to be categorical rather than dimensional (Kotov et al., 2017), but perhaps Robinaugh et al.'s (2019) model could yield insight about panic attacks as a symptom. To our knowledge, however, the model has not yet led to any new discoveries about panic attacks.

Further, we would argue that panic attacks triggered by autonomic arousal are highly unusual in research on psychopathology in terms of the amount that is known about their specific mechanisms and the relatively simple feedback loop that they appear to entail. The chances seem low that similar models with any potential for scientific utility will be developed in the near future for many other features of psychopathology. We are concerned that Fried is succumbing to wishful thinking based on this one example of a theory targeting a feature of psychopathology that was no doubt selected for modeling precisely because of its seeming simplicity. We are even more concerned that such wishful thinking will prevent other researchers from taking seriously theories that are weaker in the formal sense but that offer good opportunities for scientific advances (of which we hope ours is one).

Given that computational models are not going to be a panacea for psychological theory, what can we get from verbal theories, like CB5T, that are the norm in psychology? We believe they have several things to offer. First, as Fried notes, theories provide sets of “axioms,” often in the form of definitions of terms. Such definitional axioms are not intended nor able to be justified empirically; rather they are justified through philosophical arguments about the coherence and utility of the definitions in question. By providing rigorous, logical, verbal arguments, broad theories like CB5T that attempt to provide comprehensive, synthetic accounts

of psychological phenomena can be useful for clarifying the objects of study and the relevant theoretical and empirical questions.

Such theories can also provide conceptual frameworks in which to integrate existing empirical knowledge. Attempting to build formal theories without knowing enough about the processes being modeled is unlikely to lead to progress, but comprehending a body of empirical knowledge prior to a formal theory nonetheless requires an organizing framework. A piecemeal list of empirical observations without any conceptual organization is intractable. Verbal theories are the way out of this Catch-22. Scientific theory is primarily the product of *abduction*, inference to the best explanation (Haig, 2005; Niiniluoto, 1999),⁴ and verbal theories are a necessary, initial, abductive step toward more complete formal theories. They can allow some degree of explanation and prediction and even potentially some degree of control of phenomena, if they are sufficiently well-specified and have sufficient validity.

Before formal theories are possible, psychologists can nonetheless take steps to improve the specification of their verbal theories. Fried (p. xx) describes weak theories as those that “do not spell out the functional form in which two variables relate to each other, the conditions under which a hypothesized effect should occur, or the magnitude of a proposed effect.” Attention to this list can make psychological theory stronger without demanding the impossible from it.

As Fried notes, dense longitudinal studies of states within individuals are necessary to test much of what is posited by network theory. In contrast, many of the most obvious hypotheses that can be generated from CB5T posit that a disposition toward some feature of personality or psychopathology will be associated, in between-person analysis, with variation in some measure of psychological or neurobiological function. (To be informative, the latter must usually involve a modality different from the one used to measure the disposition in question,

such as behavioral tasks or neuroimaging; linking questionnaires to questionnaires provides little traction, given the prevalence of method variance.) As a default for testing such hypotheses, we recommend assuming that the associations posited by CB5T (and other comparable theories) are of a magnitude in the range of $r \approx .1$ to $.3$. Rather than unrealistically wishing for a point-prediction of the magnitude of individual effects when not enough is known to generate such predictions, we suggest this generic range-prediction based on two observations: First, the average effect size in research on individual differences (as in psychology more generally) is around $r = .2$, and very few effects are larger than about $r = .35$ when the variables involved do not share method (Funder & Ozer, 2019; Gignac & Szoderai, 2016; Hemphill, 2003). Second, the maximum possible effect size is a function of the number of causal forces that influence the disposition in question (Ahadi & Diener, 1989; Strube, 1991). Given that any dimension of psychological individual differences is likely to be extensively multiply-determined, we should never expect to find correlations larger than about $.3$, and we should ideally ensure that we have power to detect correlations closer to $.1$.

Relying on a minimum correlation of interest, such as $.1$, helps to allay the concern that any association, no matter how small, will be statistically significant given a large enough sample. Meehl (1990, p. 108) asserted that a ubiquitous “crud factor” invalidated the strategy of relying on mere statistical significance as evidence because “everything is somewhat correlated with everything.” Relying on a range-prediction instead of a merely directional prediction helps to avoid this issue. Additionally, we suspect that Meehl’s intuitions overestimated the magnitude of the crud factor because he was most conversant with data from questionnaires, in which pervasive method factors increase all correlations. In our experience, correlations between measures from different modalities (such as questionnaires, behavioral tasks, and biological

assessments) often head toward zero in large samples. Therefore, we think it is reasonable to pay attention to significant correlations larger than .1.

Most hypotheses from CB5T and other verbal theories are first tested in terms of linear association. Assuming a linear functional form may sometimes be an oversimplification, but it is often a reasonable place to start. Linear effects are probably more likely in between-person than within-person associations. If a linear effect is nonsignificant, there is no reason not to investigate other forms of association as well, as long as one makes adequate corrections for multiple testing and is committed to replication as part of the process for confirming discoveries. Of course, if one has a reason to hypothesize a nonlinear effect, this should be tested immediately, but given how little we know about any of the causal processes involved in most individual differences, this will rarely be the case.

What might all of this look like in practice? As an example, we will briefly describe one element of CB5T that has been tested empirically, regarding the sources of the personality trait conscientiousness (C). Despite C's well-demonstrated ability to predict academic and workplace performance as well as physical and mental health (Roberts et al., 2014), compared to other personality traits C has a surprisingly small number of known correlates that seem etiologically relevant (in other words, correlates that are neural parameters or behavioral measures of putative underlying psychological processes). Based on the few existing findings, CB5T proposed that a major neural correlate and causal contributor to variation in C might be one particular large-scale brain network (Allen & DeYoung, 2017; DeYoung, 2015).

This is a good example of abduction. Multiple studies had found associations of C with volume of regions in the dorsolateral prefrontal cortex (DLPFC), a brain structure believed to support deliberation, planning, and maintenance of goals, all of which are conceptually related to

C. However, previous research had found that C is unrelated or even slightly negatively related to variation in working memory and intelligence, which are the psychological functions most often linked empirically to DLPFC. A map of the brain's large scale functional networks showed that a network believed to be involved in directing attention toward goal-relevant stimuli had a small node in DLPFC, surrounded by a different network that is typically associated with working memory and intelligence (Yeo et al., 2011). This new observation suggested the abductive inference that the best explanation for previous piecemeal findings regarding C might be that it was associated with the functioning of this network, which we subsequently labeled the *goal priority network* (GPN; Rueter et al., 2018) because, in Yeo et al.'s map, it did not correspond to any single well-studied network but rather to a combination of two such networks (the *ventral attention* and *saliency* networks) plus a region of DLPFC.

After developing this theoretical account, we tested the hypothesis that variation in functional connectivity (i.e., temporal synchrony of neural activity) within the GPN would be positively associated with C, while participants were at rest in a brain scanner (Rueter et al., 2018). (The theory implies that neural activity and connectivity during relevant tasks should also be correlated with C, but resting brain scans are a reasonable place to start, given the availability of such data.) We found that the internal connectivity or *coherence* of one component of this network was positively correlated with C. We also found that connectivity between this component and other components of the GPN was positively correlated with C. Finally, we showed that connectivity in the GPN was not correlated with the other four traits in the Big Five, nor was activity in other networks with nodes in lateral PFC correlated with C. This sort of specificity helps to constrain and strengthen theory.

One limitation of this study is that it had 80% power to detect an effect of about $r = .2$ rather than $.1$. Another limitation is that the method used to identify relevant brain areas is specific to a given sample, and thus a complete replication would be difficult. Nonetheless, let us consider what should happen if a slightly different hypothesis designed to test the same theory about C in a different sample were not supported. Would we therefore have good reason to abandon the C–GPN theory, or would we be tempted to blame what Fried (p. xx) calls “auxiliary scape goats”? Here again we think that Fried has the right idea but may be asking for too much. Fried acknowledges that strict Popperian falsificationism is an unrealistic approach to science and that theories always depend not only on core propositions and related hypotheses but also on a variety of auxiliary theories and hypotheses having to do with one’s measurement tools and various contextual or moderating variables (Meehl, 1990). As long as one’s core theory continues to offer better abductive explanations about the phenomenon in question than competing theories, then adjusting auxiliary hypotheses need not be seen as mere scape-goating. Indeed such *post hoc* modifications are common and necessary in all branches of science, even though the resulting theory is certainly weaker than if potential modifications had been anticipated ahead of time. If one needs to modify auxiliary hypotheses that protect the “hard core” of one’s psychological theories (Meehl, 1990), one should attempt to make sure that those modifications are themselves well-specified and testable. As Fried notes, handwaving in the direction of “hidden moderators” will not do.

Fried (p. xx) suggests that theories should provide “specifications of the core findings (what variables matter), auxiliary hypotheses (what variables can we vary that do not matter), and mechanisms or processes that lead to the observed effect in an experiment (manipulations specifically of mechanism A, but not of other mechanisms, explain the observed effect).” We

appreciate all three items on this list, in principle, but in practice the latter two are difficult or impossible to achieve given the current state of knowledge in psychology and the nature of our subject matter. *Post hoc* modification of auxiliary hypotheses cannot be forbidden, although we can demand rigorous specification and testing of them, and proposed mechanisms and processes must be allowed to apply to correlational as well as experimental research. Developing theories to explain psychological individual differences is particularly difficult because we cannot manipulate the system at will (for example, we cannot artificially adjust people's trait levels of conscientiousness for the purposes of an etiological experiment; at best we could try to get them to act temporarily more or less conscientious). Nonetheless, we must continue our attempts at theory.

Conclusion

We applaud Fried's exhortation to embrace complexity in psychological theorizing, as well as his point that this should entail integrating factor and network analytic statistical models and acknowledging the existence of both broad and narrow causes of individual differences. However, we caution researchers against wishing for the impossible: statistical models never inherently provide causal evidence, and the phenomena of interest in psychology are too complex to allow us to wait for the development of effective computational models before researching them. With the relatively weak theories that are currently possible, we can nonetheless make progress in understanding individual differences and in treating psychopathology and more generally enhancing human well-being. The perfect is the enemy of the good, and the cure for this problem, in psychological research, is to embrace guidelines that will prevent our theories from being too weak while recognizing that our theories will rarely be as strong as we would like them to be.

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Footnotes

1. On p. xx, Fried states, “Network theory ... suggests that correlations among items ... stem from causal interactions between items rather than from one shared origin.” Surely this must be a very loose use of the word “item,” given that it is the entity or construct that each item is intended to measure that can interact with other such entities (the item, of course, is merely a string of words with a numerically coded response). This clarification highlights the possibility of using latent variables to model each node in a network, even when those nodes are conceived as very narrow constructs, because one could simply use multiple items to measure each of them.

2. There is some ambiguity about what Fried means by “psychological construct.” Often it is clear that he is referring to the flawed assumption made by other people that a psychological construct must be a single common cause or “*core driving force* of relations among items” (p. xx). In contrast, Fried himself is clearly willing to consider that psychological constructs may not be causally unitary, as when he states, “Network theory is based on the guiding principle that psychological constructs are emergent properties that arise out of the interactions of constituent elements” (p. xx). However, in some places, including the passage quoted in our main text to which this footnote refers, it seems that he is accepting the definition of “psychological construct” as common cause. Otherwise, how could one makes sense of the following sentence? “A well-fitting factor model cannot be taken as evidence or proof that a psychological construct such as the *p* factor or *g* factor exists as a psychological construct” (p. xx). If something is a psychological construct, how could it not exist as a psychological construct? The only way we can read this sentence cogently is to assume that the first use of “psychological construct” refers to something different from the second use, with the first meaning any kind of psychological

construct, including a statistical regularity such as the method-independent *g* factor, and the second meaning a psychological construct understood only as a common cause.

3. Note that such a belief does not contradict the further belief that there are additional, more specific causes contributing to some but not all of the associations between indicators. As Fried notes, common and specific causes can easily coexist. In fact, because of the hierarchical nature of trait constructs, there will almost always be some valid unique variance in an indicator of a latent variable that is not mere error variance (DeYoung, 2015; Möttus et al., 2017). Fried states that the standard latent variable model “disregards variance that is not shared among indicators as measurement error,” but this is not strictly true. The variance that is disregarded in a latent variable is simply variance unique to each indicator, some of which is error but some of which may be valid variance not shared with other indicators. This is why the so-called “error-terms” in latent variable models are alternatively labeled “uniquenesses” or “disturbances.” They account for any source of unshared variance in the indicator, whether random error or systematic force.

4. Because statistical models are not causal models, we disagree with Haig (2005) that exploratory factor analysis is an abductive method. Rather it is an inductive method, simply detecting patterns in data. Haig refers to latent variables as if they were explanatory, causal entities, which is contradicted by our literal interpretation of them as purely statistical constructs. What requires abduction is not identifying the existence of a latent variable but rather explaining the sources of a latent variable, which may be of any number.