

Common Factors and Interpretation of the p Factor of Psychopathology



To the Editor:

One of the most discussed recent topics in psychopathology research is the p factor of mental illness. This single dimension is understood to measure “a person’s liability to mental disorder, comorbidity among disorders, persistence of disorders over time, and severity of symptoms.”¹ A recent paper by Constantinou *et al.*² published in the *Journal* investigated the external validity of the p factor. We commend the authors for the contribution to the literature and want to highlight two points: (1) the interpretation of p as a causal entity, and (2) selection of bifactor models over alternative models for reasons of superior fit.

THE p FACTOR AS A CAUSAL ENTITY

Much of the p factor literature seems to interpret p as a causal entity that gives rise to correlations in psychopathology data and reflects an underlying essence shared by all symptoms. This interpretation is consistent with the statistical approach used in the literature to model the p factor: reflective latent variable models where the latent variable p is the shared origin of the observed variables. Caspi and Moffitt¹ have explicated this most clearly: “latent variable analysis posits that the positive correlations between symptoms (as well as disorders) arise from a g -like causal factor.” As explained in detail elsewhere, a causal reading of the reflective model is indeed the only one that can rationalize the fact that these models discount all unique item variances as measurement error.³ Although we agree that there is considerable evidence that current p factor models summarize the data well, psychopathology data feature a *positive manifold* with positively interrelated items. Such data can *always* be described well by a p factor model, even when correlations among items were generated by a different true model than a reflective model.⁴ This means that the causal-reflective interpretation is not substantiated by the data itself: it is *assumed*.

Making this assumption explicit is important for three reasons. First, fitting a reflective model can lead to bias if the true model is not a reflective model.⁵ Second, if p is understood as a summary of the data and not its shared origin,⁶ we do not know the extent to which p generalizes across datasets with different items. The generalizability of p needs to be

tested; simply assuming it by combining evidence for P from different datasets (eg, Kotov *et al.*⁷) requires a causal-reflective interpretation of p where all items are interchangeable indicators. Finally, if p reflects no shared essence but is simply the statistical result of the positive manifold,⁴ it does not *explain* the covariation among items. This might limit the utility of the p factor in the search for mechanisms that can be used to improve treatment. After reading the manuscript by Constantinou *et al.*,² we are curious to know how the authors interpret the p factor, as they refer to p both as an explanation in their abstract and as a summary of the relations among items in their introduction.

BIFACTOR MODELS AND MODEL FIT

We also have questions about the modeling decisions made by the authors, as the explanation they provide seems circular to us. Specifically, the authors fit a correlated factors model and a bifactor model to the data; the former shows somewhat better fit. This small difference might be meaningful, given that bifactor models tend to fit data better than correlated factors models due to their complexity, to the degree that bifactor models show better fit even when data are simulated under correlated factors models.⁸ Constantinou *et al.*² acknowledge the complexity of bifactor models but rely on fit indices that only penalize complexity based on the number of freely estimated parameters (AIC and BIC); for other fit indices that may be more appropriate, see Greene *et al.*⁸ The authors then adapt the bifactor model to improve its fit but do not further adjust the correlated factors model. Finally, they justify the decision to opt for the adapted bifactor model as the final model based on a superior model fit over the correlated factors model (p. 780). We were hoping the authors could elaborate on these modeling decisions.

CONCLUSION

Although causal-reflective interpretations of p are common in the literature, this inference is theoretical and does not follow from fitting reflective latent variable models to correlations among a set of items. One can plausibly hypothesize that p *explains* relations among items, but fundamentally different data-generating mechanisms can give rise to the same correlation pattern among items.⁴ In this case, p would merely be a necessary result arising from

the positive manifold; or, as Caspi and Moffitt¹ put it: “statistical tomfoolery” (p. 833). Similarly, although many fit indices show that bifactor models describe the data better than competing data generating mechanisms such as correlated factors models, this does not imply that the data come from a bifactor model.

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Accepted September 5, 2019.

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The authors have reported no funding for this work.

The authors would like to thank Riet van Bork, PhD, of the University of Amsterdam, and Ashley L. Greene, BA, of Stony Brook University, for their helpful comments in the preparation of this manuscript.

Disclosure: Dr. Fried and Mr. Aristodemou have reported no biomedical financial interests or potential conflicts of interest.

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<https://doi.org/10.1016/j.jaac.2019.07.953>

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Drs. Constantinou and Fonagy Reply:



We thank Aristodemou and Fried¹ for their thoughtful comments on our article about changes in the psychopathology bifactor dimensions over a psychosocial intervention.² Aristodemou and Fried expressed concerns over our interpretation of the *p* factor and our selection of the bifactor model over alternative models. We will address each concern and highlight the issues they raise for the field more broadly.

HOW DO WE INTERPRET THE *p* FACTOR?

Aristodemou and Fried¹ discuss how the widespread interpretation of the *p* factor as a causal entity is problematic because factors rely on an unproven assumption of causality. We agree that authors (including ourselves) could be more mindful of these assumptions in their writing. It might be helpful to distinguish between two distinct but related entities: general psychopathology, the construct hypothesized to explain the positive co-occurrences among mental health problems, and the *p* factor, a statistical representation of these positive co-occurrences that is no more real than any dispersion statistic for representing individual differences. Unless we validate the *p* factor against external criteria (as we attempted) or, better still, against prospective measures of causal mechanisms, we are at risk of making interpretative leaps beyond the data.

The *p* factor is first and foremost a statistical re-expression of the covariance among psychopathology variables. If this covariance accurately reflects co-occurrences in people’s experiences of mental health problems, then the *p* factor will, statistically speaking, represent covariation in mental health problems and its underlying construct, ie, general psychopathology. Like any measure, the *p* factor is influenced by the methods used to estimate it,³ but the target construct remains constant. This is not to say that general psychopathology is unidimensional; there are various risk factors for psychopathology that interact in complex ways for each individual.⁴ However, the *p* factor provides a tool for isolating these broad influences and investigating their treatment targets. Contrary to Aristodemou and Fried’s suggestion that an artifactual *p* factor could limit its utility in studying mechanisms for improving treatment, specific factors and their treatment targets can still be investigated free from the common method variance (which is conflated in other models).

WHY DID WE CHOOSE THE REVISED BIFACTOR MODEL?

We chose a revised bifactor model with cross-loadings over a standard bifactor model without cross-loadings and a correlated factors model. As Aristodemou and Fried¹ point out, the correlated factors model fits slightly better than the standard bifactor model, which is surprising given the bias toward bifactor models in model comparisons.⁵ However, this was likely a result of placing overly stringent constraints on the shared variance beyond the general factor in the standard bifactor model, and was hence resolved by freeing the cross-loadings among specific factors in the revised bifactor model. On a related note, we did not revise the correlated factors model because the cross-loadings were a

result of shared variance beyond the p factor and hence not present in an exploratory correlated factors model, which conflates the general and specific variance.

Nonetheless, our modeling decisions raise two issues with the current practice of bifactor modeling. The first, discussed by Aristodemou and Fried,¹ is choosing a model using model fit indices alone. Researchers (including ourselves) can be criticized for prioritizing the superior fit of the bifactor model—which can occur for nonsubstantive reasons⁵—without justifying its theoretical basis.⁶ The notion of a severity dimension that is distinct from styles of symptomatic expression appears to have emerged a posteriori with the resurgence of bifactor models, but it has a history in personality research and clinical practice. Still, we would argue that theoretical justification is not the only reason why a model might be preferred over another. Models might be selected because they are practically useful, even if they do not represent the true data-generating mechanism. Total and subscale scores in psychopathology measures tend to be underpinned by a single dimension, even if they sample a diverse range of problems.³ The bifactor model allows us to capture variation in responding that we partially impose with the design of our measures; clinical outcomes look rather different when we take this into consideration.²

The second issue concerns shared variance beyond the p factor. Bifactor models with cross-loadings or specific factor correlations are becoming popular, but there is a danger in freeing these covariances to improve model fit without considering the consequences. Estimating shared variance threatens the interpretation of specific factors and implies model mis-specification owing to unmodeled factors.⁶ Yet not estimating the shared variance can also lead to model mis-specification and inflate general factor loadings.⁷ Justifying the inclusion of shared variance beyond the p factor with past research (as we attempted) is

important to avoid capitalizing on sample-specific error, but further work is needed to identify its methodological and theoretical impact.

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Accepted November 15, 2019.

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The authors have reported no funding for this work.

Disclosure: Please see the disclosure statement in the original article published in the August 2019 issue.

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<https://doi.org/10.1016/j.jaac.2019.11.026>

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