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All Models Are Wrong, but the p Factor Model Is Useful: Reply to Widiger and Oltmanns (2017) and Bonifay, Lane, and Reise (2017)

Hannah R. Snyder¹ and Benjamin L. Hankin²

¹Department of Psychology, *Brandeis University*

²Department of Psychology, *University of Illinois-Urbana Champaign*

We welcome the commentators' viewpoints, which add to the vigorous debate critical for advancing research using bifactor models in general, and the p factor model in particular. They conclude such models have potential, but that problems remain in interpreting and using them. We agree, to the extent that all models, constructs and methods have both strengths and limitations, especially relatively new ones such as the p factor model. It is important to remember the adage "all models are wrong, but some are useful" (Box & Draper, 1987). "All models are wrong" in that they are simplifications that capture only part of a complex picture. However, we believe there is growing evidence that the p factor model is useful, as the p factor, internalizing and externalizing latent factors are related to theoretically and practically important risk factors and outcomes, and thus hold promise to advance clinical science.

First, the p factor model is important for clarifying comorbidity patterns—long-standing questions in psychiatric epidemiology and developmental psychopathology. Second, it has the potential to greatly clarify complex and overlapping patterns of risk factors for, and outcomes of, different forms of psychopathology. For example, childhood maltreatment and poverty (Caspi et al., 2014), negative emotionality (Caspi et al., 2014; Hankin, Davis, Snyder, Young, & Glynn, 2016), poor cognitive control (Caspi et al., 2014; Hankin, Davis et al., 2016), and reduced prefrontal grey matter (Snyder, Hankin, Sandman, Head, & Davis, 2016) are all associated with the p factor, and thus may contribute to broad comorbidity (Hankin, Snyder et al., 2016). It is for these reasons, not model fit alone, that we are optimistic about the p factor model. Indeed, we agree with Bonifay et al. that model fit is a necessary but not sufficient reason for preferring a model.

Our target paper built on this literature, which has robustly replicated the p factor model across different populations, measures and methods (e.g., Caspi et al., 2014; Laceulle, Vollebergh, & Ormel, 2015; Lahey et al., 2012; 2015; Patalay et al., 2015; Tackett et al., 2013). Thus, we did not focus on validating this model, per se, but rather addressed new questions—what is the longitudinal stability of these latent psychopathology factors, and how specifically are they related to one another over time? We found strong homotypic continuity between latent psychopathology factors, advancing understanding of youth psychopathology risk trajectories.

While it was not the focus of our target paper, we agree with both commentaries that more work is needed to understand the nature of the p factor—a rapidly advancing research area. There is growing evidence that common (multi-genetic) risk may substantially underlie the p factor (Rhee, Lahey, & Waldman, 2015). Not mutually exclusive, and parallel to Widiger and Oltmanns' interpretation of the common personality factor, compromised cognitive control also explains significant variance in common psychopathology (Beauchaine & Thayer, 2015; Caspi et al., 2014; Hankin, Davis et al., 2016).

That said, we agree with Bonifay et al. that common factors are not necessarily generated by single latent traits. All latent factors are simply reflections of statistical covariance among indicators, and are agnostic regarding causes of that covariance. The p factor is compatible with more complex structural models of psychopathology, such as mechanistic property clusters (Kendler, Zachar, & Craver, 2011). Property clusters do not have deterministic essences or single causal factors—rather, they are generated by multiple causal mechanisms (Kendler et al., 2011). Consistent with this model, mounting evidence indicates that latent psychopathology dimensions are not defined by any single construct or causal driver, but rather are related to multiple, potentially interacting, risk factors (e.g., Hankin, Snyder et al., 2016; Beauchaine & McNulty, 2013). This certainly makes the p factor a more complex construct. Rather than diminishing its usefulness however, it suggests important new directions as the field moves beyond research focused on singular, DSM-defined disorders towards untangling interactions among multiple transdiagnostic dimensions.

Each commentary brings up specific challenges and suggestions for future research. Bonifay et al. state that cognitive neuroscience will likely play a more important role than fit statistics in validating the p factor model. We agree that cognitive neuroscience approaches are one promising avenue, and such research is underway (reduced grey matter volume in critical neural regions, Snyder et al., 2016; neurovascular integrity, Caspi et al., 2014; autonomic function, Beauchaine & Thayer, 2015). However, we caution against any single approach (neural, genetic, other) suggested to singularly underlie, or function as a sole causal mechanism to, latent psychopathology dimensions (c.f., Miller, 2010).

Widiger and Oltmanns discuss conceptual problems with interpretation of the common personality factor. We note that using multiple reporters, with method factors and random intercepts, as we did in our target article, helps to address concerns about social desirability and other reporting biases that they raise. We agree with Widiger and Oltmanns that the hypothesis that general factors in both personality and psychopathology may be capturing dysfunction/impairment, consistent with Caspi et al.'s (2014) interpretation of the p factor as overall psychopathology severity, is worthy of further research.

We anticipate that research to better understand the constructs in the p factor model and the causal mechanisms involved will continue at a rapid pace. The p factor model should, and we trust will, be modified as this research yields new insights. Meanwhile, we agree with both commentaries that it is important to be suitably cautious in interpreting the meaning of these factors, and not prematurely reify them. In our target article, we refrained from making strong claims about the nature of the latent psychopathology factors, and have instead provided evidence that they are capturing stable individual differences in youth. These

findings have important implications for models of psychopathology development and suggest the need for future research to understand the processes contributing to this strong stability in latent psychopathology factors (in contrast with traditional DSM-based “episodic” views of psychopathology). We are optimistic that the p factor model will continue as an important organizing structure that can be used to better understand the strong stability and comorbidity of psychopathology.

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