Riskier Tests of the Validity of the Bifactor Model of Psychopathology

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Abstract

We advanced several “riskier tests” of the validity of bifactor models of psychopathology, which included that the general and specific psychopathology factors should be reliable and well-represented by their respective indicators, and that including a general factor should improve upon the correlated factor model’s external validity. We compared bifactor and correlated factors models of psychopathology using data from a community sample of youth (N=2498) whose parents provided ratings on psychopathology and theoretically-relevant external criteria (i.e., personality, aggression, antisociality). Bifactor models tended to yield either general or specific factors that were unstable and difficult to interpret. The general factor appeared to reflect a differentially-weighted amalgam of psychopathology rather than a liability for psychopathology broadly construed. With rare exceptions, bifactor models did not to explain additional variance in first-order psychopathology symptom dimensions or external criteria compared with correlated factors models. Together, our findings call into question the validity of bifactor models of psychopathology, and the *p-factor* more broadly.

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The lack of an empirically supported structure of psychopathology is a persistent challenge to the study and treatment of psychopathological conditions (Cuthbert, 2005; Krueger & Markon, 2006; Widiger, 2005). Contemporary taxonomies that enlist quantitative methods to yield empirically-based structures of psychopathology tend to be hierarchically organized (e.g., Kotov et al., 2017; Krueger et al., 2018; Wright et al., 2013). These models begin with signs and symptoms at the bottommost levels of the hierarchy, which are then grouped into broader dimensions based on their covariation at increasing levels of the hierarchy. In recent years, many researchers have turned their focus to the ostensible uppermost level of the hierarchy, the general factor of psychopathology (Caspi et al., 2014; Lahey et al., 2012). Hereafter, we refer to the general factor of psychopathology as the *p-factor* when discussing it as a substantive construct, and as the general factor when discussing it as a methodological construct within the context of bifactor models, so as not to conflate bifactor models with theory surrounding the *p-factor* and empirical investigations of the *p-factor* using other approaches (e.g., Forbes et al., 2017; Kim & Eaton, 2015).¹

Although the precise nature of the *p-factor* is not yet understood, it is thought to reflect a broad liability towards psychopathology. Since its “discovery” in 2011-2012, the *p-factor* literature has proliferated (see Caspi & Moffitt, 2018, for a review), and almost always emerges from bifactor models of psychopathology (cf., Forbes et al., 2017; Kim & Eaton, 2015). Notwithstanding warnings from quantitative methodologists and social scientists alike (Bagby, Taylor, Quilty, & Parker, 2007; Bonifay, Lane, & Reise, 2017; Morgan, Hodge, Wells, & Watkins, 2015; Murray & Johnson, 2013; Vanheule, Desmet, Groenvynck, Rosseel, & Fontiane, 2008),

¹ Given that most empirical investigations of the *p-factor* tend to rely on bifactor models, we acknowledge that this substantive versus methodological distinction between the *p-factor* and the general factor of psychopathology generated from a bifactor model, respectively, is nuanced and may be somewhat arbitrary.

this near exclusive reliance on bifactor models persists. Herein, we argue that much of this literature has developed incautiously, as it tends to assert the validity of bifactor models of psychopathology, and the *p-factor* that emerges from them, on the basis of preferential model fit. In this study, we offer a set of criteria that better assess the validity of the bifactor models of psychopathology, in turn placing these models and the *p-factor* at stronger theoretical risk (Meehl, 1978; Popper, 1959). Ultimately, we hope that these “riskier tests” will aid researchers in adjudicating structural models of psychopathology more broadly.

Hierarchical Models of Psychopathology

Dating back to at least the 1960s, research has organized child and adult psychopathology hierarchically, with the chief levels of the hierarchy reflecting at least two broad spectra termed externalizing and internalizing (Achenbach, 1966; Achenbach & Edelbrock, 1984; Krueger & Markon, 2006).² Externalizing represents conditions characterized by poor behavioral and emotional control, including but not limited to antisocial behavior and substance use in adults, and conduct disorder, attention-deficit/hyperactivity disorder, and oppositional defiant disorder in youth. Internalizing represents conditions characterized by elevated levels of negative emotionality, including anxiety, depression, and phobias. Subsequent research suggests that internalizing can be parsed into distress, which includes depression, anxiety, and post-traumatic stress disorder, and fears, which includes phobias and panic disorder (Clark & Watson, 2006; Krueger, 1999). Cross-sectional and longitudinal efforts to disentangle

² Much of the existing research on structural models of psychopathology has focused on externalizing and internalizing disorders, in part because they are relatively common. Consequently, other disorders tend not to be examined in these contexts given their relatively low prevalence in the population. A relatively smaller literature has accommodated other forms of psychopathology in structural models of psychopathology (Kotov et al., 2017), including but not limited to thought disorders (Caspi et al., 2014; Carragher et al., 2016), autism spectrum conditions (Noordhof et al., 2015), bipolar disorder (Kotov et al., 2011), and personality disorders (Kotov et al., 2010; Kotov et al., 2011). Despite these additions, externalizing and internalizing remain the most studied and best understood of the spectra.

externalizing and internalizing have revealed that the two are separable but substantially overlapping (meta-analytic $r = .50$; Krueger & Markon, 2006).

Many authors have adopted the *p-factor* as the explanation for this covariation. To their credit, this explanation is relatively intuitive, as the shared variance among externalizing and internalizing spectra ought to reflect what is common to psychopathology. As such, the recent psychopathology literature has proceeded by including the *p-factor* in structural models of psychopathology, where it has now been studied across most developmental periods, including childhood (e.g., Lahey et al., 2015; Olino et al., 2014; Waldman, Poore, van Hulle, Rathouz, & Lahey, 2016), adolescence (e.g., Carragher et al., 2016; Laceulle et al., 2015; Murray, Eisner, & Ribeaud, 2016; Patalay et al., 2015), and adulthood (e.g., Caspi et al., 2014; Lahey et al., 2012).

Nevertheless, there are a number of plausible broad interpretations of the *p-factor*, including that it reflects the broad constructs of maladaptivity, distress, impairment, or is an artifact of social desirability (Caspi & Moffitt, 2018; Widiger & Oltmanns, 2017a). Tacitly implied in much of the existing research is that the *p-factor* reflects a singular causal mechanism that underlies all forms of psychopathology. Researchers have posited a number of candidates for this mechanism, including poor emotional and behavioral control (Carver, Johnson, & Timpano, 2017), the tendency to experience negative emotionality (Widiger & Oltmanns, 2017b), and poor cognitive ability or disordered thought (Caspi & Moffitt, 2018). Although research has yet to converge on any one of these explanations, the majority of validation research has proceeded presupposing that the *p-factor* is substantive and meaningful, given that it has been established as a correlate of “real-world life outcomes” (p. 835, Caspi & Moffitt, 2018).

The *p-factor* has been linked to a broad swath of ostensible transdiagnostic risk factors, downstream sequelae, and other correlates, including: increased family history of psychiatric conditions (Caspi et al., 2014; Lahey et al., 2012; Martel, Pan, et al., 2017); maternal smoking

during pregnancy, harsh parental discipline, and peer delinquency (Waldman et al., 2016); self-reported physical and sexual abuse (Lahey et al., 2012); impairment (Caspi et al., 2014); neuroticism (Caspi et al., 2014; see also Widiger & Oltmanns, 2017b); impulsivity, sensation seeking, and hopelessness (Castellanos-Ryan et al., 2016); as well as to decreased intelligence, executive functioning, early life brain integrity (Caspi et al., 2014; Martel, Pan, et al., 2017; Snyder, Young, & Hankin, 2017); academic performance (Lahey et al., 2015); conscientiousness, and agreeableness (Caspi et al., 2014). Other research has demonstrated that the *p-factor* is moderately heritable (Neumann et al., 2016; Waldman et al., 2016), and that it exhibits strong homotypic continuity across time (Snyder et al., 2017).

Bifactor Models and their Applications to Psychopathology

Just as there are several interpretations of the *p-factor*, there are at least two possible structural representations of psychopathology that accommodate a *p-factor*: the higher-order factor model and the pervasively used bifactor model (Brunner, Nagy, & Wilhelm, 2012; Mansolf & Reise, 2017; see Markon, 2019, for a thorough review). Both of these models construe psychopathology as comprising multifaceted, hierarchically-organized constructs. Additionally, these models are more comprehensive than correlated factors models, insofar as they consider multiple levels of the psychopathology hierarchy.

In higher-order psychopathology models, the covariation among first-order symptom dimensions is influenced and explained by second-order latent factors, such as externalizing and internalizing, whose covariation is in turn influenced by a superordinate third-order latent factor, such as the *p-factor*. In this model, the effects of the *p-factor* on individual psychopathology dimensions (i.e., generalized anxiety) are *completely mediated* by lower-order, more specific factors (i.e., distress, internalizing).

In contrast, the bifactor model decomposes covariation among psychopathology into two types of factors, general and specific (Holzinger & Swineford, 1937). The general factor reflects

a single source of common variance among all indicators in the model, meaning that the general factor *directly influences* all individual psychopathology symptom dimensions. Any remaining covariation among indicators is decomposed into residual specific factors that influence and explain the covariance among subsets of psychopathology that are similar in content, such as externalizing and internalizing. In this model, the general factor is orthogonal to the specific factors, which are themselves held to be orthogonal given that all covariation among the psychopathology indicators is assumed to be captured by the general factor. In most applications, the general factor represents the central construct of interest, whereas the specific factors represent more conceptually related subdomains, or even nuisance method factors (Markon, 2019).

The lion's share of the existing literature has relied on bifactor models of psychopathology when detecting and validating the *p-factor* (c.f., Kim & Eaton, 2015; Forbes et al., 2017), which may be due to several factors. Compared with the hierarchical model, bifactor models are generally easier to specify in latent variable models and are not burdened by the same proportionality constraints required of hierarchical models (Gignac, 2016; Mansolf & Reise, 2017). Nevertheless, there are several reasons that bifactor models of psychopathology are problematic.

First, bifactor models are vulnerable to overfitting data, engendering their parameter estimates unstable (e.g., Bonifay, 2015; Bonifay et al., 2017; Reise, Kim, Mansolf, & Widaman, 2016). Second, various simulation studies have demonstrated that bifactor models are prone to fitting any possible data, including potentially invalid (Reise et al., 2016) or random response patterns (Bonifay & Cai, 2017). Third, numerous simulation studies have demonstrated that bifactor models invariably show preferential fit compared with higher-order and correlated factors models even when the population ("true") model does not follow a bifactor structure (Greene et al., in press; Maydeu-Olivares & Coffman, 2006; Morgan et al., 2015; Murray &

Johnson, 2013). Together, mounting evidence from these and other studies suggests that model fit statistics are unreliable indicators of the validity of bifactor models.

Riskier Tests of the Validity of the Bifactor Model of Psychopathology

As testament to its popularity, the seminal manuscripts on the *p-factor* and bifactor models of psychopathology (Caspi et al., 2014; Lahey et al., 2012) together have been cited well over 800 times. Others have integrated the *p-factor* into a contemporary hierarchical taxonomy of psychopathology (HiTOP; Conway et al., in press) that was previously agnostic with regards to the nature and number of highest-order psychopathology dimensions (Kotov et al., 2017). Still others have integrated the *p-factor* into structural models of allied constructs in subdisciplines of clinical psychology, including personality and personality disorders (Oltmanns, Smith, Oltmanns, & Widiger, 2017) and executive functioning (Snyder, Miyake, & Hankin, 2015).

Emerging from this literature are two general findings, namely that (1) bifactor models of psychopathology are better fitting than the widely-studied correlated factors models; and (2) the general factor in a bifactor model of psychopathology is significantly associated with a broad swath of theoretically-relevant correlates, presumably substantiating it as a valid psychopathology construct, namely the *p-factor* (Caspi & Moffitt, 2018). At the same time, the field's near exclusive reliance on bifactor model conceptualizations of the *p-factor* warrants scrutiny in light of the aforementioned criticisms and limitations associated with such models. In this manuscript, we propose a set of "riskier tests" that more closely evaluate the structural properties and validity of bifactor models of psychopathology.

The present study serves as a demonstration of these "riskier tests," using data from a large community-based sample of children and adolescents whose parents completed ratings of diagnostic criteria for common childhood disorders from the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; American Psychiatric Association, 1994). Parents also provided ratings on theoretically-relevant external criteria, including temperament, aggression,

and antisociality. After evaluating a set of alternative structural models of psychopathology using confirmatory factor analysis (CFA) and conventional fit indices, we consider each “riskier test” in turn, contrasting each model using the following set of criteria.

Test #1: *If the general factor in a bifactor model reflects broad liability for psychopathology, it should be relatively equally represented by its constituent indicators.* Presuming the *p-factor* reflects a unitary process general to all forms of psychopathology, the general factor in a bifactor model should be well-represented by its indicators, and its influence on these indicators should be relatively uniform (i.e., tau equivalent; Graham, 2006). In contrast with this principle, the general factor within a bifactor model tends to be underrepresented by some forms of psychopathology (i.e., fears) and overrepresented by others (e.g., distress, thought problems, autism spectrum disorders; Caspi & Moffitt, 2018).

Hypothesis #1: We hypothesize that the general factor within a bifactor model will not be influenced equally by all psychopathology dimensions, and instead will be defined primarily by distress (Oltmanns et al., 2018) and antagonism-imbued forms of externalizing (i.e., conduct disorder, oppositional defiant disorder). We predicted that antagonism-imbued forms of externalizing would load highly onto the general factor given that our demonstration relies on youth data, and antagonism and neuroticism tend to be more highly correlated, and thus more difficult to disentangle, in youth compared with adults (Tackett, Kushner, De Fruit, & Mervielde, 2013; Waldman et al., in press).

Test #2: *A bifactor model should produce reliable specific factors that are well-represented by their constituent indicators.* After accounting for the shared variance among psychopathology symptom dimensions captured by the general factor, a bifactor model should produce reliable specific factors that are well-represented by their constituent indicators. By reliable, we mean that a specific factor should show high convergent validity, and thus be characterized by appreciable and statistically significant factor loadings, thereby increasing the

likelihood that the specific factor will replicate across studies (Hancock & Mueller, 2001; Rodriguez, Reise, & Haviland, 2016).

Rodriguez and colleagues (2016) described a number of indices available for quantifying latent factor reliability. H , the most appropriate index for bifactor model factor reliability, conceptually reflects the extent to which a latent variable is represented by its indicators, and thus how likely it is to replicate across studies. More specifically, H is the proportion of variance explained in the indicators by the latent factor divided by the variance unexplained by the latent factor. It ranges from 0 to 1, and increases as a function of the magnitude of factor loadings and the number of indicators. Compared with coefficient omega (McDonald, 1999), another popular index of latent factor reliability, H reflects the correlation between a factor and an optimally-weighted, as opposed to unit-weighted, item composite, making it better suited for latent variable modeling (see Rodriguez et al., 2016, for a more thorough discussion and formula).

Few studies have reported reliability indices for factors derived from bifactor models of psychopathology. In one exception reported by Martel, Pan, and colleagues (2017), only the general factor in a bifactor model with three correlated specific factors (fears, distress, and externalizing) was characterized by highly reliable variance on the basis of coefficient omega. Stated differently, there was little reliable variance in psychopathology indicators after taking into account the general factor. Although overlooked in the literature, these findings raise the possibility that bifactor models produce unreliable specific factors that are not likely to replicate across studies (see Rodriguez et al., 2016, for a broader demonstration), which is concern worthy as researchers also are interested in the nature and correlates of a bifactor model's specific factors, in addition to the general factor.

Hypothesis #2: We hypothesize that bifactor models will yield specific factors of decreased reliability relative to both the general factor in the model and to the factors yielded in

a correlated factors model. Even further, we expect that specific factors' reliability will fall below the benchmark level of acceptable reliability (Rodriguez et al., 2016).

Test #3: Including a general factor should improve upon the external validity of the correlated factors model.

As we noted earlier, much research has proceeded with validating the general factor of psychopathology in a bifactor model by examining its relations with a number of theoretically-relevant external criteria (e.g., personality, intelligence). We agree in principle that examining a bifactor model of psychopathology's correlates is a useful first step in validating the general factor, but we emphasize the virtue of comparing the external validity of the bifactor model of psychopathology with alternative structural models of psychopathology. Although it is common practice to report model fit for multiple alternative structural models of psychopathology, it is uncommon to report the external validity of multiple alternative models within the same paper (cf. Caspi et al., 2014; Martel, Pan, et al., 2017).

This practice overlooks two important possibilities, namely that a bifactor model might not explain more variance in (1) its indicators and (2) external criteria than a correlated factors model. Should bifactor and correlated factors models of psychopathology explain an equivalent amount of the variance in their constituent psychopathology indicators, it would indicate that bifactor models merely redistribute aspects of psychopathology into a greater or different number of factors. Simply redistributing variance in psychopathology renders it unlikely that bifactor models outperform correlated factors models in terms of explained variance in external criteria.

Hypothesis #3: Compared with correlated factors models, we hypothesize that bifactor models of psychopathology will neither explain more variance in psychopathology indicators, nor will they explain more variance in external criteria.

Method

Participants and Procedure

The present sample comprises 2498 individuals (51% females) aged 4 to 17 ($M_{age} = 8.6$ years, $SD = 2.9$) whose families participated in the Georgia Twin Registry and provided ratings on a battery of psychopathology and relevant external criteria for each child in the household (Waldman et al., 1998). Mothers typically completed the questionnaires (53%), and the remaining questionnaires were completed either by fathers (1%) or mothers and fathers together (46%). Seventy-one percent of the sample were twin pairs (45% MZ, 55% DZ) and 29% were siblings of twins. The racial and ethnic background of participants was 82% Caucasian, 11% African American, 1% Hispanic, and 6% other/mixed ethnicity.

Measures

Psychopathology. The Emory Combined Rating Scale (Waldman et al., 1998) assesses DSM-IV symptoms of common youth psychiatric disorders by means of ratings on a 0 to 4 scale (ranging from “not at all” to “very much” representative of the child). Items were summed into continuous symptom dimension composites. Included in this measure were anxiety disorders (i.e., agoraphobia, generalized anxiety disorder, obsessions, compulsions, panic disorder, social phobia, specific phobia), mood disorders (i.e., major depression), attention-deficit and disruptive disorders (i.e., attention-deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder), separation anxiety disorder, and vocal and motor tics. Cronbach’s alphas for all symptom dimensions ranged from .54 [Agoraphobia] to .98 [Panic disorder].

External criteria. The Antisocial Process Screening Device (APSD; Frick & Hare, 2001), the EAS Temperament Survey for Children (EAS; Buss & Plomin, 1984), and the Reactive-Proactive Aggression Scale (RPA; Dodge & Coie, 1987) were used as external criteria given that they are established correlates of childhood psychopathology (e.g., Nigg, 2006). Items were rated on a 0 to 4 scale identical to that described for the psychopathology measure.

The APSD is a 20-item scale comprising items designed to assess youth psychopathic traits and other antisocial characteristics. The APSD yields scores on three subscales: *Narcissism*, which reflects grandiosity, manipulativeness, and superficial charm; *Callous-unemotional*, which reflects lack of empathy, remorse, and guilt, as well as shallow and constricted affect; and *Impulsivity*, which reflects engagement in rash behavior and boredom susceptibility (α s were .84, .61, and .78, respectively).

The EAS comprises 20 items designed to assess four dimensions of temperament. *Emotionality* reflects a general tendency towards psychological distress, *Activity* reflects preferred levels of activity and speed of action, *Sociability* reflects a preference for the company of others as opposed to isolation, and *Shyness* reflects feelings of inhibition and awkwardness in novel social situations (α s were .84, .64, .58, and .75, respectively). Temperament data used in external validity analyses were available for 913 participants.³

The RPA comprises 12 items designed to assess proactive (instrumental, “cold-blooded”) and reactive (defensive, “hot-blooded”) forms of childhood aggression (α s were .71, and .75, respectively).

Data Analysis

We conducted all analyses using Mplus version 7.2 (Muthén & Muthén, 2014). Continuous symptom dimension scores were analyzed using the MLR estimator (maximum likelihood with robust standard errors; Yuan & Bentler, 2000) to account for their non-normality, and using the “cluster” option to account for nonindependence due to the nesting of twins and siblings within families. Factor analyses were fit to the raw data using full-information maximum

³ We tested all structural psychopathology models on the subset of data with complete psychopathology and temperament data ($N = 913$). Rank ordering of models on the basis of model fit was identical to those reported for the full sample (see Supplemental Materials). The best-fitting model was a bifactor model with three correlated specific factors, followed by a bifactor model with three uncorrelated specific factors, a bifactor model with two correlated specific factors, and a bifactor model with two uncorrelated specific factors.

likelihood given that this is an optimal way of handling missing data and yields less biased parameter estimates than do listwise or pairwise deletion (Enders & Bandalos, 2001).

Goodness-of-fit. We reported the X^2 test statistic, its associated degrees-of-freedom (df) and p value; Comparative Fit Index (CFI); Tucker-Lewis index (TLI); root mean square error of approximation (RMSEA); Bayesian Information Criterion (BIC); and standardized root mean square residual (SRMR). We did not rely on X^2 and their p values to determine adequacy of model fit because X^2 significance tests are highly sensitive to sample size, and would be virtually certain to be rejected given our large sample size. Adequacy of model fit was determined based on guidelines suggested in the literature and based on the comparison of TLI, RMSEA, SRMR, and BIC (see Loehlin, 2004, and Markon & Krueger, 2006), although we present the, χ^2 , Comparative Fit Index (CFI) and the Akaike Information Criterion (AIC) in the spirit of full disclosure.

Latent variable reliability. We reported H , which assesses construct replicability (reliability) of latent factors in a structural equation modeling context (Hancock & Mueller, 2001; Rodriguez et al., 2016). Hancock and Mueller (2001) suggested .70 as a useful benchmark for acceptable reliability. Although H is more informative than Omega and its progeny for structural equation models (Rodriguez et al., 2016), we present OmegaS, OmegaH, and Relative Omega for each factor from all psychopathology models (see Table 2; and see Rodriguez et al., 2016, for details on these reliability indices).

External validity. We examined psychopathology models' relations with external criteria by regressing each criterion onto each psychopathology factor while simultaneously estimating the psychopathology structural model, obviating the need to save factor scores. In addition to examining zero-order correlations, we conducted regressions in which subscales within measures were entered as simultaneous predictors of psychopathology dimensions to (1)

account for the significant overlap among within-measure constructs and (2) yield relations with external criteria unique to each indicator.

Results

Alternative Models of Psychopathology

We first conducted 8 tests of alternative CFA models for the underlying structure of psychopathology. The models tested were as follows: (1-2) models with correlated and uncorrelated externalizing and internalizing factors (Krueger & Markon, 2006); (3-4) bifactor counterparts to the 2 correlated factors models, with either correlated or uncorrelated externalizing and internalizing specific factors (Lacuelle et al., 2015; Olino et al., 2014); (5-6) models with correlated and uncorrelated externalizing, distress, and fears factors (Krueger, 1999); and (7-8) bifactor counterparts to the 3 correlated factors models, with either correlated or uncorrelated specific factors (Martel, Pan, et al., 2017; see Table 1 for all model fit statistics). It is important to note that we tested bifactor models that allowed for correlated specific factors, although bifactor model theorists argue that specific factors should be orthogonal (Holzinger & Swineford, 1937), based on practices observed in the psychopathology literature (Carragher et al., 2016; Caspi et al., 2014; Lacuelle et al., 2015; Martel, Pan, et al., 2017; Olino et al., 2014).

For both correlated factors and bifactor models, model fit indicated that a model that parsed internalizing into fear and distress was preferable to a model that did not despite the fact that fear and distress were highly correlated in both correlated factors model ($r = .82$, 95% CI [.74, .90]) and bifactor model with correlated specific factors ($r = .75$, 95% CI [.64, .87]). Satorra-Bentler χ^2 difference tests were significant between the (1) 2- and 3-factor oblique models ($\Delta\chi^2 = 36$, $df = 2$, $p < .001$) and (2) the bifactor models with 2 and 3 oblique specific factors ($\Delta\chi^2 = 10$, $df = 2$, $p = .007$). In addition, we could not equate fears' and distress' relations with external criteria for the 3 correlated factors model and its bifactor counterparts without a significant

decrement in model fit (ΔX^2 all significant at $p < .001$; see Supplemental Materials), indicating that fears and distress related differentially with external criteria.

Given these characteristics, we proceeded with the 3 correlated factors model and its bifactor counterparts, with either 3 correlated and uncorrelated specific factors, for our remaining riskier tests. It is worth noting, however, that all conclusions drawn from the 3-factor models hold for the 2-factor models.⁴ Results from each 3-factor model are presented with their respective factor loadings and factor correlations in Table 2 (see Supplemental Materials for complete results for the 2-factor models).

Riskier Tests

Test #1: *If the general factor in a bifactor model reflects broad liability for psychopathology, it should be relatively equally represented by its constituent indicators.* In the bifactor model with 3 correlated specific factors, psychopathology symptom dimensions' loadings on the general factor were quite variable, ranging from .06 (specific phobia) to .68 (major depression), with a median value of .22. On average, the general factor explained 9% of the variance in the first-order symptom dimensions. The general factor was characterized by small loadings from fear-related internalizing dimensions, with the exception of obsessions, and impulsivity (λ s ranged from .06 [specific phobia] to .22 [impulsivity]); moderate loadings from compulsions, generalized anxiety, inattention, and hyperactivity (λ s ranged from .25 [hyperactivity] to .40 [inattention]); and large loadings from conduct disorder, oppositional

⁴ The rare exception to this rule is that the bifactor model with 2 specific factors, models with both correlated and uncorrelated specific factors, did not explain more variance in any of its indicators or external criteria. This was generally true of the bifactor model with 3 specific factors, compared with the 3 correlated factors model, but here the bifactor models with 3 specific factors explained a significantly larger amount of the variance in major depression and proactive aggression than did the 3 correlated factors model. In our view, these findings further support our parsing distress and fears, given the increased predictive validity of the bifactor model in this instance.

defiant disorder, and major depression (λ s ranged from .55 [oppositional defiant disorder] to .68 [major depression]).

Relative to the bifactor model with 3 correlated specific factors, a model equating all symptom dimensions' loadings on the general factor fit significantly more poorly ($\Delta X^2 = 679$, $df = 14$, $p < .001$). In light of the general factor being relatively well represented by distress and externalizing but not fear symptom dimensions, we also tested a posthoc model equating externalizing and distress but not fears symptom dimensions' loadings on the general factor, which also fit the data significantly more poorly ($\Delta X^2 = 31$, $df = 6$, $p < .001$). Together, these findings indicate that the general factor was not equally represented by the first-order symptom dimensions.

In the bifactor model with 3 uncorrelated specific factors, psychopathology symptom dimensions' loadings on the general factor were quite variable, ranging from .25 (tics) to .84 (generalized anxiety), with a median value of .36. On average, the general factor explained 18% of the variance in the first-order symptom dimensions. Most notably, the loadings on the general factor were appreciably larger in magnitude relative to the bifactor model with 3 correlated factors. The general factor was characterized by moderate loadings from conduct disorder, hyperactivity, impulsivity, tics, specific phobia, compulsions, panic disorder, and social phobia (λ s ranged from .25 [tics] to .41 [inattention]), and moderate to large loadings from the remaining symptom dimensions (λ s ranged from .44 [obsessions] to .84 [generalized anxiety]), with loadings being most pronounced for major depression and generalized anxiety. Relative to the bifactor model with 3 correlated specific factors, a model equating all symptom dimensions' loadings on the general factor fit more poorly ($\Delta X^2 = 86$, $df = 14$, $p < .001$). In general, however, this model's general factor tended to be more equally represented by its indicators than did the bifactor model with 3 correlated specific factors.

To evaluate the robustness of the general factor to excluding any one psychopathology indicator, we next conducted a series of analyses in which we excluded each psychopathology dimension from the general factor in turn. This resulted in 15 models tested, with 14 loadings on the general factor freely estimated per model, for both bifactor models. Results from these analyses are displayed in two box plots. Figure 1a displays the variability of each symptom dimension's loadings on the general factor as a function of excluding each other psychopathology dimension in turn. For instance, in this panel we show the variability of inattention's loadings on the general factor when each other symptom dimension is dropped in turn. Figure 1b reorganizes these data to display variability of loadings on the general factor as a function of the symptom dimension dropped from the general factor (see Supplemental Materials for all models' factor loadings). In this panel, we show the variability of the *other* symptom dimensions' loadings on the general factor when inattention is dropped.

We then extracted factor scores for general factors from all models, the full model and the 15 models where each symptom dimension was dropped one at a time. We correlated these two sets of factor scores, specifically those (1) from the full model with those (2) from the reduced models to demonstrate the sensitivity of the general factor to the exclusion of a symptom dimension. Lower correlations indicate greater sensitivity to that symptom dimension being dropped; we viewed correlations lower than .90 as problematic (see Supplemental Materials for all convergent correlations).

Several trends can be gleaned from the box plots. First, symptom dimensions' loadings on the general factor were typically more variable in the bifactor model with correlated specific factors compared with the bifactor model with uncorrelated specific factors (Figure 1A). In addition, in the bifactor models with correlated and uncorrelated specific factors, some symptom dimensions' loadings on the general factor varied relatively little as a function of dropping each other symptom dimension from the model; this was especially true of symptom dimensions

characterized by high levels of fear. For instance, tics' loadings on the general factor ranged from -.03 (when dropping obsessions) to .14 (major depression) and .07 (oppositional defiant disorder) to .28 (major depression) in the bifactor models with correlated and uncorrelated specific factors, respectively (Supplemental Materials). Other symptom dimensions' loadings on the general factor varied considerably. For instance, conduct disorder's loadings on the general factor ranged from -.48 (obsessions) to .63 (tics) and -.45 (oppositional defiant disorder) to .63 (generalized anxiety) in the bifactor models with correlated and uncorrelated specific factors, respectively; the same was true of psychopathology symptom dimensions characterized by distress and externalizing tendencies.

Finally, for the bifactor model with correlated specific factors but not the bifactor model with uncorrelated specific factors, many symptom dimensions' loadings on the general factor were either near zero or negative once symptom dimensions that defined the general factor in the full model were dropped. For instance, in the bifactor model with correlated specific factors, when oppositional defiant disorder was dropped from the general factor, conduct disorder's and obsessions' loadings on the general factor were -.45 and -.23, respectively. In general, in the bifactor model with correlated specific factors, when anxiety or oppositional defiant disorder was excluded, the general factor reflects psychological adaptivity, with negative loadings from most of the indicators that define the general factor in the full model. Finally, most of the fear-related symptom dimensions could be dropped from the general factor with little to no effect on its contents (Supplemental Materials).

These trends were also evident in the convergent correlations between the general factor in the full model and models in which one symptom dimension was dropped. For the bifactor model with correlated specific factors, convergent correlations were high ($>.90$) when the following symptom dimensions were excluded from the general factor: agoraphobia, compulsions, generalized anxiety, hyperactivity, impulsivity, panic disorder, separation anxiety,

social anxiety, separation anxiety, and tics. Convergent correlations were slightly lower for inattention (.84). In contrast, convergent correlations were essentially zero for conduct disorder (.01) and major depression (-.09) and strongly negative for obsessions (-.89) and oppositional defiant disorder (-.65).

Together, these findings indicate that the general factor in a bifactor model with correlated specific factors was highly sensitive to the inclusion of certain symptom dimensions, namely conduct disorder, depression, obsessions, and oppositional defiant disorder. In the case of the latter two symptom dimensions (i.e., obsessions, oppositional defiant disorder), the general factor was so sensitive to their inclusion that it its contents after dropping these symptom dimensions appeared to reflect psychological adaptivity, broadly construed, as opposed to maladaptivity. Consistent with the data presented in Figure 1, the general factor in a bifactor model with uncorrelated specific factors was relatively robust, with convergent correlations dropping below .90 when only two symptom dimensions were excluded: major depression (.87) and generalized anxiety (.84).

Test #2: A bifactor model should produce reliable specific factors that are well-represented by their constituent indicators. For the 3 correlated factors model, H statistics were above the .70 benchmark, indicating sufficient construct replicability (.74 for fears and distress, .84 for externalizing). Along these lines, these factors were generally well-represented by their psychopathology indicators. As shown in Table 2, standardized factor loadings on the fears factor ranged from .26 (tics) to .63 (separation anxiety), with a median value of .49. Loadings on the distress factor ranged from .64 (major depression) to .83 (generalized anxiety), with a median value of .74. Loadings on the externalizing factor ranged from .39 (conduct disorder) to .80 (hyperactivity), with a median value of .71.

For the bifactor model with 3 correlated specific factors, H statistics were above the benchmark for most fear and externalizing specific factors (.72, .80) and slightly below the

benchmark for distress (.67). Compared with the 3 correlated factors model, the fears factor was equally well-represented by its indicators, with loadings ranging from .22 (tics) to .64 (separation anxiety) and a median loading of .46. Distress tended to be less well-represented by its indicators, with loadings ranging from .35 (major depression) to .81 (generalized anxiety) and a median loading of .58, but this was primarily due to the fact that major depression was not a strong indicator of distress after accounting for the general factor (see also Tackett, Kushner, et al., 2013). Externalizing was similarly less well-represented by its indicators, with loadings ranging from .10 (conduct disorder) to .81 (hyperactivity) and a median loading of .56, but this was primarily due to antagonism-related externalizing loading less strongly on externalizing after taking into account the general factor. At the same time, specific factors' reliabilities were generally adequate, with the exception of distress, and a number of loadings on the specific factors were comparable to those from the 3 correlated factors model.

For the bifactor model with 3 uncorrelated specific factors, H statistics were sufficient for the externalizing specific factor (.77) but were poor for the fear and distress specific factors (.55, .22). Compared with both the 3 correlated factors model and the bifactor model with correlated specific factors, specific factors were generally less well-represented by their indicators. Loadings on the fear specific factor ranged from .07 (tics) to .54 (separation anxiety) with a median loading of .33. Loadings on the distress specific factor ranged from -.34 (generalized anxiety) to .36 (major depression) with a median loading of .01. Notably, generalized anxiety's loading on distress became moderately negative after accounting for the general factor. Loadings on the externalizing specific factor ranged from .20 (conduct disorder) to .79 (hyperactivity) with a median loading of .54.

Test #3: Including a general factor should improve upon the external validity of the correlated factors model. Figure 2 displays the variance explained in psychopathology symptom dimensions broken down by model. Readily apparent from inspection of this figure is

that the 3 correlated factors model and its bifactor counterparts explained a comparable, and in most cases equivalent, amount of the variance in the psychopathology symptom dimensions or external criteria. There were two exceptions to this rule. The first was that bifactor models explained more of the variance in major depression (72 and 61% for the bifactor models with uncorrelated and correlated specific factors, respectively; there was no difference in explained variance between these two models) than did the correlated factors model (28%). The second was that bifactor models explained more of the variance in proactive aggression (36% for the bifactor models with uncorrelated and correlated specific factors) than did the correlated factors model (26%).

Discussion

Bifactor model representations of psychopathology have arguably defined the contemporary zeitgeist of clinical psychology (Caspi & Moffitt, 2018). This now considerable body of research has raised the provocative notion that one's broad liability for psychopathology can be quantified by a single dimension referred to as the *p-factor*. Most of this literature relies on bifactor models and privileges model fit statistics over other criteria in adjudicating among structural models of psychopathology and establishing their validity. Such a practice is increasingly questionable in light of evidence that model fit statistics are biased in favor of detecting bifactor models, even when the true, population model follows a more parsimonious correlated factors structure (e.g., Murray & Johnson, 2013).

A simple replication and extension study of the structure of youth psychopathology using these practices would have reported the following: A bifactor model of psychopathology fit better than a correlated factors model; the *p-factor* in children appears to reflect antagonism and distress; the *p-factor*, externalizing, fears, and distress factors displayed divergent relations with external criteria; and the *p-factor* was related to increased emotionality, antisociality, and proactive aggression. As we noted earlier, several others have reported findings to this effect,

and the analyses we used to generate these findings were largely identical to those used in the existing literature. Nonetheless, based on the findings from our “riskier tests,” we worry that such conclusions are misguided.

Summary and Implications

We advanced several “riskier tests” to better adjudicate among structural models of psychopathology, including that: (1) the general factor in a bifactor model should be well-represented by its constituent indicators, (2) bifactor models of psychopathology should produce reliable specific factors that are well-represented by their constituent indicators, and (3) including a general factor should improve upon the external validity of a correlated factors model. Using our “riskier tests,” we compared the widely-adopted correlated factors model with two bifactor models increasingly used in the literature, ones with either three uncorrelated or three correlated specific factors. In turn, we discuss each of our central findings and their implications for the psychopathology literature.

Test #1. Consistent with our hypotheses and the existing literature, we found that the general factor in both bifactor models was not well-represented by psychopathology symptom dimensions, and was instead overrepresented by distress and externalizing psychopathology and underrepresented by fears. This is broadly consistent with a handful of studies in the adult literature, including those demonstrating that depression and generalized anxiety are strong indicators of a general factor of psychopathology (e.g., Lahey et al., 2012), and others establishing neuroticism as a robust correlate of the general factor (e.g., Caspi et al., 2014). That oppositional defiant and conduct disorders also loaded substantially on our general factors developmentally extends these adult findings (see also Tackett, Lahey, et al., 2013, Waldman et al., 2011, 2018; but see Lahey et al., 2015 for inconsistent evidence) and corroborates the developmental literature in which negative affectivity and antagonism tend to intersect substantially in children (Tackett, Kushner, et al., 2013).

Moreover, our attempts to equate the contributions of all or some of the psychopathology symptom dimensions to the general factor were unsuccessful, calling into question the *p-factor* as a single, general liability for psychopathology. Such findings are more consistent, rather, with the *p-factor* reflecting an amalgam of differentially-weighted and psychologically distinct sources of variance (Krueger et al., 2018; Reise, 2012). The existing literature nevertheless suggests that the *p-factor* not only differentially weights psychopathology, but that its defining contents vary considerably across studies (Levin-Aspenson et al., 2018). Even within our own data, wherein we tested two different bifactor models of psychopathology, the contents of the *general factor* varied considerably.

Some studies do find that the general factor in a bifactor model is defined by distress (Lahey et al., 2012; Lahey et al., 2015; Olino et al., 2014; Waldman et al., 2016), but others find that the general factor is defined by externalizing (Castellanos-Ryan et al., 2016), thought disorder (Caspi et al., 2014), or even autism spectrum disorders (Martel, Pan, et al., 2017; Noordhof et al., 2015). These cross-study discrepancies have given rise to a dizzying array of interpretations of the *p-factor* (Caspi & Moffitt, 2018), but are also potentially consistent with the bifactor model's tendency to fit *any* data (Reise et al., 2016).

If our assertion is correct, cross-study comparisons of *p-factors* appear *prima facie* specious. These comparisons are especially dubious when two studies do not include identical psychopathology measurement. Although it is unlikely that depression and anxiety will be omitted from studies of the structure of psychopathology, other forms of psychopathology (e.g., bipolar disorder, schizophrenia, autism spectrum disorders) are not assessed consistently in these studies due to their relatively low base rates in the population. One study that does not assess schizophrenia or bipolar disorder, for instance, might find that the general factor is saturated with distress or negative emotionality (Lahey et al., 2012), whereas a study that assesses thought problems might find that the general factor is saturated with thought problems

(Caspi et al., 2014). We suspect that the general factor in a bifactor model pulls for pathology, viz., maladaptivity, and thus will be most strongly represented by indicators that are ostensibly “most severe.” To boot, comparisons between youth and adult *p-factors* are even more challenging, given that diagnoses of certain forms of psychopathology are limited to childhood (e.g., conduct disorder, oppositional defiant disorder, autism spectrum disorders), and others to adulthood (e.g., personality disorders). We suspect, moreover, that the content of our general factors would differ had we included other forms of psychopathology, given that general factors within bifactor models appear highly sensitive to their contents.

Test #2. We observed a general tendency for the bifactor models to yield less reliable specific factors. This was especially the case for the bifactor model with uncorrelated specific factors, where reliability indices were often well below the threshold of acceptable reliability. In the bifactor model with correlated specific factors, reliability indices were still above the threshold for acceptable reliability, but of decreased reliability relative to those for the correlated factors model. These findings accord with the scant reliability indices reported for specific factors within bifactor models of psychopathology (Martel, Pan, et al., 2017). Moreover, they accord with reliability of specific factors from bifactor models of narrower clinical constructs, including those of attention-deficit/hyperactivity disorder (Arias, Ponce, & Núñez, 2018; Willoughby, Fabiano, Schatz, Vujnovic, & Morris, 2017), disgust (Olatunji, Ebesutani, & Reise, 2015), and obsessive-compulsive disorder (Olatunji, Ebesutani, & Abramowitz, 2017).

Additionally, in our own data, specific factors tended to be underrepresented by psychopathology symptom dimensions that were strong indicators of the general factor, raising interpretability challenges in several cases. In the bifactor model with three correlated specific factors, for example, conduct disorder was not a robust indicator of externalizing, nor was major depressive disorder a robust indicator of distress (see also Lahey et al., 2012; Waldman et al., 2016). In the absence of these symptom dimensions, externalizing and distress in this model

appear to reflect narrower forms of their constructs, namely poor behavioral control and fears, respectively. In the bifactor model with uncorrelated specific factors, moreover, neither major depression nor generalized anxiety were robust indicators of the distress factor, and the sign of the latter was negative. Such idiosyncrasies have been observed for specific factors in other bifactor models of psychopathology (e.g., Caspi et al., 2014; Martel, Pan, et al., 2017). Martel, Pan, and colleagues (2017) reported, for instance, that psychosis loaded slightly negatively ($\lambda = -.10$) onto the thought disorders factor in a bifactor model with orthogonal specific factors. At minimum, these findings indicate that specific factors should be interpreted cautiously given that their contents might not be consistent with their conceptualization, and that researchers should pay close attention to what variance is extracted from a specific factor and allotted to the general factor.

Test #3. We found that our correlated factors and bifactor models of psychopathology explained a nearly identical amount of the variance in the first-order psychopathology dimensions and external criteria. The exception to this rule was that the bifactor models explained a significantly larger amount of the variance in depression and proactive aggression than did the correlated factors model, which is again consistent with our observation that our general factors were disproportionately saturated with antagonism, antisocial behavior, and distress. These findings bear important implications for the way the *p-factor's* validity is instantiated in much of the existing literature. Thus far, the psychopathology literature has proceeded by merely correlating the general factor in a bifactor model with theoretically-relevant external criteria, with the following justification: “evidence that [the *p-factor*] predicts objective, real-world life outcomes (e.g., suicide) suggests that it may be indexing something substantive, not merely something about how people behave while data are being collected” (Caspi & Moffit, 2018, p. 835). In our view, simply demonstrating that the general factor is related to some external criterion is an insufficient test of the validity of the general factor, or the *p-factor* for that

matter, without also demonstrating that the same correlates are not observed for one or more of the factors within a more parsimonious correlated factors model. It appears from these data that there is little added value in introducing the general factor into structural models of psychopathology, at least when generated by means of bifactor models, inasmuch as bifactor models do not incrementally predict relevant external criteria over and above correlated factors models. We argue for cross-model validity comparisons, given that bifactor models of psychopathology appear in large part to redistribute variance components of a correlated factors model.

A Cautionary Note

An admittedly alluring quality of bifactor models is that we can study multiple levels of the psychopathology hierarchy in tandem. To wit, bifactor models of psychopathology might be viewed as conciliatory, striking a balance between lumping and splitting approaches in psychopathology classification efforts (Greven, 2005), given that their purpose is to identify the sources, correlates, and sequelae of both the general *and* specific factors of psychopathology. Broadening our focus to multiple levels of the hierarchy presumes, however, that our model produces reliable and interpretable factors at each level. We suspect that this assumption is not met consistently for bifactor models of psychopathology.

Somewhat unexpectedly, there was a general tendency for bifactor models to yield either a reliable general factor or reliable specific factors, but not both. The bifactor model with correlated specific factors yielded a less reliable and interpretable general factor but more reliable and well-defined specific factors, whereas the opposite was true for the bifactor model with uncorrelated specific factors. In this way, bifactor models of psychopathology may be placing undue burden on the data (Bonifay, 2015; Bonifay et al., 2016; Reise et al., 2016), in turn compromising the integrity of either the general or specific factors.

All told, our results indicate that a bifactor model of psychopathology is difficult to interpret because the general factor is not equally represented by all forms of psychopathology (Krueger et al., 2018) and is instead defined by different disorders depending on what forms of psychopathology are included in the model (Levin-Aspenson et al., 2018). Moreover, bifactor models of psychopathology tend to yield specific factors of decreased reliability and interpretability. These specific factors often exhibited decreased reliability relative to the correlated factors model, and often exhibited concerning low reliability. Lastly, introducing a general factor into a correlated factors model explains little, if any, additional variance in its constituent indicators or its correlates, but rather appears to redistribute the variance explained by the correlated factors model into more complex, and more heavily parameterized, model. These difficulties should not arise if the *p-factor* truly reflects a unitary mechanism common to all forms of psychopathology, nor should they arise if researchers wish to use bifactor models to probe multiple levels of the psychopathology hierarchy in their study (Caspi & Moffit, 2018).

Limitations

For the purpose of demonstrating the implications of our “riskier tests,” we adopted an analytic approach that is consistent with those used in the existing literature. There are a number of additional limitations with our approach that were not addressed by our “riskier tests”. First, the present demonstration relied on single-rater psychopathology data, which are vulnerable to the influence of social desirability or other response sets. In this way, it is possible that socially desirable responding inflates covariation among externalizing, fear, and distress factors. We encourage future research to adopt a multi-rater approach to modeling the structure of psychopathology but acknowledge that attempts to do so will likely be met with challenges given modest convergence between self- and other-reported psychopathology, perhaps especially in youth (Achenbach, Krukowski, Dumenci, & Ivanova, 2005; Martel, Markon, & Smith, 2017). Second, we relied on data from a representative community sample of youth in

the southeastern United States, in which there were relatively low rates of endorsement for psychopathology. This sample was also predominantly Caucasian. As such, it is possible that these findings might not generalize to those of more clinically severe or culturally-diverse samples. Third, we did not assess ostensibly more severe forms of psychopathology, including bipolar disorder, thought disorder, and autism spectrum disorder given their low base rates of diagnosis in the population writ large. Although this exclusion is common practice in the psychopathology literature, especially the child psychopathology literature, studies excluding these forms of psychopathology from their structural models likely paint an incomplete picture.

Future Directions

The “riskier tests” we generated are a starting place meant to generate increased scrutiny and discussion surrounding bifactor models of psychopathology. Moreover, although we emphasize the utility of these “riskier tests” for evaluating the validity of bifactor models of psychopathology, they can be applied to bifactor models of other constructs and can be used to adjudicate among structural models more generally. By no means are our “riskier tests” exhaustive. We encourage continued research comparing the performance of bifactor models with alternative structures of psychopathology.

Tested models could include but are not limited to correlated factors models and higher-order models. Other promising research has adopted other methods to yield hierarchical models of psychopathology. These include exploratory top-down approaches, such as bass-ackwards (or sequential principal components analysis; Forbes et al., 2017; Kim & Eaton, 2015), and bottom-up approaches, such as hierarchical agglomerative cluster analysis (Forbes et al., 2017). Additionally, further research might extend existing simulation studies (Greene et al., in press; Gignac, 2016; Maydeu-Olivares & Coffman, 2006; Murray & Johnson, 2013) by examining the conditions under which we artificially identify bifactor models on the basis of preferential model fit, as well as conditions under which we may report a more parsimonious

correlated factors model when the population model indeed follows a bifactor structure (Greene et al., in press). Lastly, given that bifactor models in many cases appear to either compromise the integrity of the general factor or the specific factors in the model, researchers might seek simpler alternatives to generating patterns of correlates with psychopathology broadly construed, or more specific aspects of psychopathology, such as externalizing and internalizing spectra (Kotov et al., 2017). Researchers might extract a single psychopathology factor if they are interested in correlates common to all forms of psychopathology (but see Smith, McCarthy, & Zapski, 2009). In contrast, researchers might enter psychopathology factors derived by means of correlated factors models simultaneously into regressions if they are interested in correlates that are unique to narrower psychopathology spectra.

Conclusion

A clear and accurate empirical structure of psychopathology is critical for scientific progress on the classification of, and ultimately the etiology and treatment of, mental illness. The prospect of rapid progress in psychiatric classification, in a field otherwise marked by slow and steady progress, is an exciting one. We must not, however, be fooled into scientific progress (Zachar, 2015) by pursuing the *p-factor* without bearing in mind the limitations associated with models used to generate it, lest we pursue a *p-factor* mirage.

To be clear, our results are not necessarily definitive, nor are our findings dispositive of an absence of the *p-factor*. Indeed, there is considerable phenotypic covariation among forms of psychopathology (Markon & Krueger, 2006), and existing evidence further suggests that there are genetic, neuroanatomical, and otherwise biological underpinnings common to many forms of psychopathology (e.g., Beauchaine & Thayer, 2015; Neumann et al., 2016; Pettersson, Larsson, & Lichtenstein, 2016; Selzam, Coleman, Caspi, Moffitt, & Plomin, 2018). Moving forward, we encourage researchers to evaluate their alternative structural models of psychopathology using these “riskier tests”, especially before adopting bifactor models on the

basis of preferential model fit and significant associations with external criteria. More broadly, based on our findings and observations from the existing literature, adopting the *p-factor* as the uppermost level of the psychopathology hierarchy (Conway et al., in press), integrating it into structural models of allied constructs (i.e., personality, personality disorders, executive functioning; Oltmanns et al., 2018; Snyder et al., 2015), and focusing on its clinical implications (Conway et al., in press; Meier & Meier, 2017) may be putting the proverbial cart before the horse.

Authorship

ALW developed the study concept. All authors contributed to study design. Data collection was performed by the laboratory of IDW. ALW performed the data analysis. All authors contributed to interpretation. ALW drafted the paper, and HEP and IDW provided critical revisions. All authors approved the final version of the paper for submission.

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Table 1. *Fit statistics for confirmatory models.*

Model Description	X^2 (df)	RMSEA (90% CI)	TLI	CFI	BIC	AIC	SRMR	N params
Two correlated factors	564*** (89)	.05 (.04, .05)	.82	.84	86468	86200	.06	46
Two uncorrelated factors	708*** (90)	.05 (.05, .06)	.76	.80	86828	86566	.12	45
Bifactor with two correlated specific factors	271*** (74)	.03 (.03, .04)	.91	.94	85741	85386	.04	61
Bifactor with two uncorrelated specific factors	297*** (75)	.03 (.03, .04)	.90	.93	85793	85443	.04	60
Three correlated factors	507*** (87)	.04 (.04, .05)	.83	.86	86307	86027	.06	48
Three uncorrelated factors	<i>NO CONVERGENCE</i>							
Bifactor with three correlated specific factors	259*** (72)	.03 (.03, .04)	.91	.94	85714	85347	.04	63
Bifactor with three uncorrelated specific factors	425*** (75)	.04 (.04, .05)	.84	.88	86055	85706	.05	60

Note. *** $p < .001$, ** $p < .01$, * $p < .05$. N params=number of free parameters.

Table 2. *Fit statistics for bifactor models that equated psychopathology symptom dimensions' loadings on general and specific factors.*

Model Description	χ^2 (df)	RMSEA (90% CI)	TLI	CFI	BIC	AIC	SRMR	N params	$\Delta\chi^2$ (df)
Bifactor with two correlated specific factors	271*** (74)	.03 (.03, .04)	.91	.94	85741	85386	.04	61	
Equating all loadings on general	785*** (88)	.06** (.05, .06)	.73	.77	86441	86167	.06	47	679*** (14)
Equating all but fears loadings on general	326*** (80)	.04 (.03, .04)	.89	.92	85923	85603	.05	55	31*** (6)
Equating all loadings on externalizing specific	466*** (78)	.05 (.04, .05)	.87	.83	85923	86255	.05	57	106*** (4)
Equating all loadings on internalizing specific	361*** (83)	.04 (.03, .04)	.91	.88	85755	86058	.06	52	58*** (9)
Bifactor with two uncorrelated specific factors	297*** (75)	.03 (.03, .04)	.90	.93	85793	85443	.04	60	
Equating all loadings on general	538*** (89)	.05 (.04, .05)	.83	.85	86433	86165	.07	46	172*** (14)
Equating all but fears loadings on general	323*** (81)	.04 (.03, .04)	.90	.92	85917	85602	.05	54	27*** (7)
Equating all loadings on externalizing specific	471*** (79)	.05 (.04, .05)	.83	.87	86313	85986	.05	56	72*** (4)
Equating all loadings on internalizing specific	396*** (84)	.04 (.04, .04)	.87	.90	86118	85821	.06	51	65*** (9)
Bifactor with three correlated specific factors	259*** (72)	.03 (.03, .04)	.91	.94	85714	85347	.04	63	
Equating all loadings on general	445*** (86)	.04 (.04, .05)	.86	.88	86215	85930	.06	49	131*** (14)
Equating all but fears loadings on general	304*** (78)	.03 (.03, .04)	.90	.93	85881	85549	.04	57	29*** (7)
Equating all loadings on distress specific	321*** (73)	.04 (.03, .04)	.88	.92	85847	85486	.04	62	344*** (1)
Equating all loadings on fear specific	295*** (79)	.03 (.03, .04)	.91	.93	85877	85551	.05	56	21*** (7)
Equating all loadings on externalizing specific	353*** (76)	.04 (.03, .04)	.87	.91	85917	85573	.05	59	84*** (4)
Bifactor with three uncorrelated specific factors	425*** (75)	.04 (.04, .05)	.84	.88	86055	85706	.05	60	
Equating all loadings on general	517*** (89)	.04 (.04, .05)	.83	.86	86478	86210	.08	46	86*** (14)
Equating all but fears loadings on general	481*** (81)	.04 (.04, .05)	.83	.87	86319	86005	.07	54	42*** (6)
Equating all loadings on distress specific	442*** (76)	.04 (.04, .05)	.83	.88	86070	85727	.05	59	58*** (1)
Equating all loadings on fear specific	450*** (82)	.04 (.04, .05)	.84	.88	86206	85897	.05	53	11*** (4)
Equating all loadings on externalizing specific	574*** (79)	.05 (.05, .05)	.78	.84	86418	86092	.06	56	33*** (7)

Note. *** $p < .001$, ** $p < .01$, * $p < .05$.

N params=number of free parameters. Satorra-Bentler chi-squared difference tests were conducted on full bifactor models and their constrained counterparts. Constrained models are indented below the full model.

Table 3. Factor loadings, intercorrelations, and reliability indices for confirmatory factor analytic models.

	3CFs			Bifactor with 3CSFs				Bifactor with 3UNCsFs			
	FEAR	DIST	EXT	GEN	FEAR	DIST	EXT	GEN	FEAR	DIST	EXT
Inattention			.70	.40			.56	.41			.54
Hyperactivity			.80	.25			.81	.32			.79
Impulsivity			.74	.22			.74	.31			.70
Oppositional defiant disorder			.71	.55			.49	.49			.49
Conduct disorder			.39	.61			.10	.36			.20
Major depression		.64		.68		.35		.77		.36	
Generalized anxiety		.83		.37		.81		.84		-.34	
Panic	.53			.16	.51			.36	.42		
Social anxiety	.42			.09	.42			.36	.18		
Separation anxiety	.63			.14	.64			.42	.54		
Agoraphobia	.62			.18	.60			.46	.40		
Obsessions	.51			.38	.39			.44	.25		
Compulsions	.45			.17	.41			.36	.24		
Tics	.26			.14	.22			.25	.07		
Specific phobia	.46			.06	.49			.28	.44		
Factor intercorrelations	FEAR-DIST: .82				FEAR-DIST: .75				FEAR-DIST: .00		
	FEAR-EXT: .39				FEAR-EXT: .27				FEAR-EXT: .00		
	DIST-EXT: .54				DIST-EXT: .30				DIST-EXT: .00		
H index	.74	.74	.84	.73	.72	.67	.80	.86	.55	.22	.77
OmegaS	-	-	-	.82	.72	.80	.83	.86	.72	.85	.81
OmegaH	-	-	-	.38	.64	.44	.53	.64	.31	.00	.55
Relative Omega	-	-	-	.47	.89	.55	.64	.75	.43	.00	.67

Note. Bolded are $p < .001$; italicized are $p < .01$.

CSFs = Correlated factors; UNCsfS = Uncorrelated specific factors; DIST= Distress; EXT = Externalizing; FEAR = Fears; GEN = General.

Figure 1A. Variability in each symptom dimensions' loadings on the general factor when each other symptom dimension is dropped from the general factor.

Figure 1B. Variability in loadings on the general factor when each symptom dimension is dropped from the general factor.

Note. Figure 1A displays the variability of each symptom dimension's (x-axis) loading on the general factor (y-axis) as a function of dropping each other symptom dimension from the general factor one at a time across the 15 models tested. Figure 1B displays loadings on the general factor (y-axis) when each symptom dimension is dropped from the general factor one at a time (x-axis).

Bi3CSF = Bifactor model with 3 correlated specific factors; Bi3UNCSF = Bifactor model with 3 uncorrelated specific factors; INA = Inattention; HYP = Hyperactivity; IMP = Impulsivity; ODD = Oppositional defiant disorder; CND = Conduct disorder; DEP = Major depression; GAD = Generalized anxiety; PAN = Panic disorder; SOC = Social anxiety; SAD = Separation anxiety; AGO = Agoraphobia; OBS = Obsessions; COM = Compulsions; TIC = Tics; SPH = Specific phobia; None = Full model with no symptom dimensions dropped.

Figure 2. Explained variance in psychopathology and external criteria by psychopathology model.

Note. Explained variance = R^2 . All error bars reflect 95 percent confidence intervals.

3CF = 3 correlated factors; Bi3CSF = Bifactor model with 3 correlated specific factors; Bi3UNCSF = Bifactor model with 3 uncorrelated specific factors; INA = Inattention; HYP = Hyperactivity; IMP = Impulsivity; ODD = Oppositional defiant disorder; CND = Conduct disorder; DEP = Major depression; GAD = Generalized anxiety; PAN = Panic disorder; SOC = Social anxiety; SAD = Separation anxiety; AGO = Agoraphobia; OBS = Obsessions; COM = Compulsions; TIC = Tics; SPH = Specific phobia; EAS EMO = EAS Emotionality; EAS ACT = EAS Activity; EAS SOC = EAS Sociability; EAS SHY = EAS Shyness; APSD CU = APSD Callous-unemotional; APSD NARC = APSD Narcissism; APSD IMP = APSD Impulsivity; RPA PROACT = RPA Proactive Aggression; RPA REACT = RPA Reactive Aggression.