Spurious empirical support for the p -factor arises with the inclusion of undiagnosed cases

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Abstract

Background: The p -factor is thought to cause a positive manifold in psychopathology data, and many researchers presume that it is a substantive mechanism as opposed to a methodological artifact. Limited research suggests that including completely undiagnosed cases (i.e., cases without a single diagnosis) affects the dimensionality of psychological constructs, so we examined whether empirical support for the p -factor arises with their inclusion in structural models of psychopathology.

Methods: We drew on data from three large, nationally representative samples of US community members, the: National Epidemiologic Study on Alcohol and Related Conditions Wave 1 (N=43,093), National Comorbidity Survey (N=5,877), and National Comorbidity Survey-Replication (N=5,692). We systematically culled undiagnosed cases from the data in increments of 10% and re-estimated structural models of psychopathology.

Results: The correlation between latent externalizing and internalizing factors varied considerably as a function of the proportion of undiagnosed cases, with the correlation dropping to zero or even slightly negative when all undiagnosed cases were excluded. Also, as undiagnosed cases were removed, general factors of psychopathology explained less variance in psychopathology and weakened in terms of the extent to which it was well-represented by its indicators.

Conclusions: Including undiagnosed cases in structural models of psychopathology induces a homogeneous, unidimensional structure. Ultimately, our findings raise questions about the nature of the p -factor, including whether it reflects a methodological artifact or arises due to the inclusion of cases with absence of diagnosed psychopathology.

Spurious empirical support for the *p*-factor arises with the inclusion of undiagnosed cases

Following the 2016 U.S. presidential election, a researcher interested in patterns of media consumption among U.S. citizens found – much to their surprise – a *positive* association between watching Fox News and CNN. After reconsideration, the researcher noticed that there were a number of participants who did not watch television but were included in the analysis. Dropping those participants from the analysis resulted, as originally predicted, in a negative association between watching Fox News and CNN. Although this type of phenomenon is well recognized in other fields, such as ecology (Janson & Vegelius, 1981), its generalizability to multivariate relationships among forms of psychopathology is underappreciated.

Undiagnosed Cases and the *p*-factor

Hundreds of studies have identified two moderately correlated ($r \approx .5-.6$) spectra of psychopathology, externalizing and internalizing, that are assumed to explain much of the covariation among putatively distinct forms of psychopathology (Achenbach, 1966; Krueger & Markon, 2006). Based on their correlation (and those among higher-order psychopathology dimensions more generally), various researchers have proposed that a single dimension — most often referred to as the *p*-factor or general factor of psychopathology — gives rise to all forms of psychopathology (Caspi & Moffitt, 2018; Lahey et al., 2012; Smith et al., 2020).¹ Most research presumes the *p*-factor is substantive and causal, with proposed mechanisms including: poor emotional control (Carver et al., 2017), negative emotionality (Brandes et al., 2019; Tackett et al., 2013), and disordered thought (Caspi et al., 2014). Few researchers have considered whether a general factor of psychopathology arises from a method artifact (c.f., Caspi & Moffitt, 2018; Watts et al., 2020).

Herein, we probe one potential artifactual explanation of the *p*-factor, the inclusion of undiagnosed cases, which we define as cases that do not meet diagnostic criteria for any

¹ Note that we refer to the theoretical construct as the *p*-factor and its statistical instantiation as the general factor of psychopathology to avoid conflating theoretical and statistical models (Watts et al., 2019, 2020).

disorder. We focus on undiagnosed cases because limited research suggests that their inclusion affects the observed dimensionality of assessments of psychological constructs. For instance, Hoffman and colleagues (2019a) demonstrated that including undiagnosed cases densified network structures such that node connectivity was driven by undiagnosed cases.² Steppan and colleagues (2014) showed that the same is generally true for factor models, where the inclusion of asymptomatic cases in a factor analysis of alcohol use disorder indicators increased the covariance between all variables, in turn encouraging unidimensionality (i.e., a strong positive manifold). Thus, the structure of mental disorders and other psychological constructs may differ as a function of undiagnosed cases.

Present Study

Given that the p -factor is presumed a major source of the positive manifold in psychopathology data, the present study considers whether empirical support for it arises, in large part, with the inclusion of undiagnosed cases. The influence of undiagnosed cases is especially important to consider in the context of structural models of psychopathology given their substantial presence in epidemiologic and otherwise community-based samples, which are often used to model such structures. Large-scale epidemiologic studies find that around 70 percent of the population is undiagnosed when past year diagnoses are assessed and around 50 percent when lifetime disorders are assessed (Kessler et al., 2005).

To examine the influence of undiagnosed cases on the p -factor, we systematically culled them from the sample and re-estimated structural models of psychopathology. We conceptualized support for the p -factor in several related, but distinct, ways that run the gamut of how it might be examined empirically (Revelle & Wilt, 2013):

² This presents network models, in particular, with a major inferential challenge given that network models presumably reflect causal associations among symptoms whereby the presence of one symptom elicits another, yet causal associations in the network appeared driven by totally asymptomatic cases.

1. The correlation between latent externalizing and internalizing factors, as well as the intercorrelations among latent externalizing, distress, and fear factors (e.g., Caspi et al., 2014; Lahey et al., 2012).
2. The suitability of a general factor of psychopathology, operationalized in terms of: variance explained in psychopathology indicators (Ree et al., 2015; Zinbarg et al., 2005) and how well a general factor of psychopathology is represented by its indicators (Watts et al., 2019).
3. The degree of multidimensionality in psychopathology, operationalized in terms of model fit of a single factor solution (Forbes et al., 2020) and the magnitude of the ratio between the eigenvalues of the first and second factors from an exploratory factor analysis (Allegrini et al., 2020; Selzam et al., 2018). Though few would argue that psychopathology is unidimensional, we used the single factor model to examine whether the factor becomes less coherent (or the data became more multidimensional) as undiagnosed cases are removed from the data.

To establish replicability, we drew on data from three nationally representative samples of US community members: the National Epidemiologic Study on Alcohol and Related Conditions Wave 1 (NESARCw1; Grant et al., 2003), the National Comorbidity Survey (NCS; Kessler, 1994) and the National Comorbidity Survey-Replication (NCS-R; Kessler et al., 2004). We selected these datasets because they: have been used extensively in seminal investigations of the latent, hierarchical structure of psychopathology (Kim & Eaton, 2017; Krueger, 1999; Krueger & Markon, 2006; Lahey et al., 2012); contain relatively thorough assessment of psychopathology; and are large in size, allowing for adequately powered examinations of the aims at hand.³

³ We also chose these data because NCS and NCS-R are publicly available, allowing for full reproducibility of all analyses here (NCS: <https://www.icpsr.umich.edu/web/ICPSR/studies/6693>, NCS-R: <https://www.icpsr.umich.edu/web/ICPSR/studies/20240>). The OSF project page (<https://osf.io/c9zys/>) contains a data dictionary of all indicators used in the analysis.

Methods

Data

NESARC is a two-wave national sample of adults aged 18 to 98 ($N=43093$; $M_{age}=46$, 57% female, 57% White) collected from 2001 to 2002, NCS is a national probability sample of adults aged 15 to 61 ($N=8098$; $M_{age}=34$, 53% female, 75% White) collected from 1990 and 1992, and NCS-R is an independent sample of adults aged 18 to 99 ($N=9282$; $M_{age}=45$, 55% female) collected from 2001 and 2003. NCS and NCS-R are designed such that only a subset of participants completed the full psychiatric interview, so we restricted our use of NCS ($n=5877$) and NCS-R ($n=5692$) to the subsets of participants for whom all disorders used in the present study were assessed.⁴

Psychiatric Diagnoses

Table 1 outlines disorders included in our models. We used binary lifetime diagnoses because previous studies of structural models of psychopathology in these samples have done so, and used diagnoses constructed without hierarchical exclusion rules to facilitate the examination of comorbidity (Kim & Eaton, 2017; Krueger, 1999; Krueger & Markon, 2006; Lahey et al., 2012).⁵ Given the “skip out” structure of these assessments, we could not construct continuous symptom counts. NESARC and NCS-R diagnoses were based on DSM-IV (American Psychiatric Association, 1994) and NCS diagnoses were based on DSM-III-TR (American Psychiatric Association, 1987).

We focused on externalizing and internalizing disorders given our interest in modeling the correlation between these latent dimensions and selected disorders with an eye towards

⁴ Recruitment and consent procedures for NESARC were approved by the US Census Bureau and Office of Management and Budget, and for NCS and NCS-R by the Human Subjects Committees of Harvard Medical School and the University of Michigan. The University of Missouri Institutional Review Board approved the use of NESARC; approval was not necessary for the use of NCS and NCS-R.

⁵ Models using past year diagnoses with and without hierarchical exclusion rules, as well as lifetime diagnoses with hierarchical exclusion fit the data equally well, and the latent externalizing and internalizing correlation was statistically equivalent in each (Table S2, Figure S1). The findings we report here are even more pronounced for past year diagnoses (Figure S1).

maintaining compatibility in diagnoses across samples to avoid cross-sample differences due to measurement. NESARC assessed diagnoses with the Alcohol Use Disorder and Associated Disabilities Interview Schedule—*DSM-IV* Version (Grant & Hasin, 1992), and NCS and NCS-R assessed diagnoses with a modified version of the World Health Organization Composite International Diagnostic Interview (Smeets & Dingemans, 1993); for reliability, see: Grant et al., 2003; Kessler & Üstün, 2004; Wittchen, 1994).

Model Estimation

We conducted all analyses using R version 3.6.0, the MplusAutomation R package (Hallquist & Wiley, 2018), and Mplus version 8.4 (Muthén & Muthén, 2014; see the OSF page for a complete list of R packages used). All modeling relied on the weighted least square mean and variance adjusted (WLSMV) estimator, which is most appropriate for non-normal indicators, and with cluster, stratification, and weight variables given the complex survey designs.

We estimated (Figure 1): (1) correlated factors models with (a) externalizing and internalizing factors and (b) distress, fear, and externalizing factors, as well as (2) a single factor model; and (3) bifactor models with orthogonal (a) externalizing and internalizing specific factors and (b) distress, fear, and externalizing specific factors. In NCS, the bifactor model with three specific factors often did not converge due to negative residual variances; we note where this was the case and generally limit our narrative summary of the results to the bifactor model with two specific factors (Table S3 displays model fit with all cases included). The bifactor models with three specific factors are arguably overextracted, but we included them because they are widely used in structural models of psychopathology (e.g., Lahey et al., 2012; Forbes et al., 2020).

Tests of the: (a) correlation between psychopathology dimensions (e.g., externalizing-internalizing) made use of the correlated factors models; (b) proportion of variance in psychopathology attributable to a general factor and how well the general factor was represented by its indicators made use of the bifactor model with two specific factors and its

Omega Hierarchical coefficient (Omega ranges from 0 to 1; Zinbarg et al., 2005) and standardized loadings; and (c) degree of multidimensionality in psychopathology made use of a single factor solution and its model fit indices, as well as the magnitude of the ratio between the eigenvalues of the first and second factors made use of exploratory factor analysis.

We sampled without replacement from undiagnosed cases and dropped 0 to 100 percent (in increments of 10) of them in sequence. We then extracted each of the earlier-described models on each of these new datasets. To achieve stable estimates, we repeated this procedure 500 times each for 10, 20, and so on percent of undiagnosed cases. We focus on the medians and ranges of values (presented as error ribbons) from each set of 500 analyses. In sensitivity analyses, we also randomly sampled from and removed cases without: (1) externalizing diagnoses, (2) internalizing diagnoses, and (3) each diagnosis in isolation.

Results

Sixty-one, 38, and 42% of participants did not meet criteria for any lifetime disorder in NESARC, NCS, and NCS-R, respectively (Table S1).

The Externalizing-Internalizing correlation varied considerably as a function of extent of undiagnosed cases in the data (Figure 2a). With all undiagnosed cases in the data, the Externalizing-Internalizing correlation ranged from .48 and .56 across samples (see also Krueger & Markon, 2006). With undiagnosed cases excluded, the Externalizing-Internalizing correlation was essentially zero, and even slightly negative in NCS (range: -.14 to .04). Each of the factor correlations in the Externalizing-Distress-Fear model (Figure 2b) also decreased as a function of excluding undiagnosed cases, though the Distress-Fear correlation was not reduced to zero (range: .31 to .46).⁶ Dropping undiagnosed cases for externalizing or internalizing spectrum disorders only generally did not yield the same effect (Figure S2), and dropping

⁶ The three correlated factors model that excluded all undiagnosed cases in NCS-R did not converge due to the fact that specific phobia's standardized loadings on fear far exceeded 1, suggesting a negative residual variance for this indicator.

undiagnosed cases for each disorder in isolation did not have an effect on the externalizing-internalizing correlation (Figures S3-S4). Thus, latent factor correlations were generally unaffected by dropping undiagnosed cases within spectrum (e.g., internalizing) or within diagnoses (e.g., major depression).

A single, unidimensional psychopathology factor fit the data poorly regardless of the extent of undiagnosed cases in the data (Table S3), but its fit worsened with the exclusion of undiagnosed cases (Figure 3b). Also, the general factor in a bifactor model with externalizing and internalizing specific factors explained decreased variance in psychopathology diagnoses (per OmegaHierarchical; Figure 3b). When all cases were included, the general factor in the bifactor model with two specific factors explained around 78% (76-80%) of the reliable variance in psychopathology diagnoses. When all undiagnosed cases were culled from the model, the general factor explained around 41% (33-48%). Finally, as undiagnosed cases were culled from the data, we observed that the eigenvalue of the first component in a variance decomposition decreased, as did the ratio of the first to second eigenvalue, suggesting decreased variance explained in psychopathology by the first factor (Figure 3a, Table S4). More generally, the scree plot flattened, suggesting that the data became more multidimensional (Figure 3a).

Although the general factor in a bifactor model explained a relatively substantial amount of variance in psychopathology when all undiagnosed cases were excluded, it is important to note its' loadings became more varied and thus the general factor became less coherent (Tables S5-S7). Across samples, the range in loadings on the general factor was .47 to .49 when all cases were included, and 1.00 to 1.09 when undiagnosed cases were excluded. Generally speaking, with no undiagnosed cases in the model, the general factor was reasonably well-represented by internalizing diagnoses and unrepresented (or even slightly negatively represented) by externalizing diagnoses, which suggests that the "general" factor became "not so general." Interestingly, externalizing disorders, on the one hand, and internalizing disorders,

on the other, tended to cohere even as undiagnosed cases were excluded from the data, suggesting that these dimensions are relatively robust.

Discussion

As undiagnosed cases were culled from the data, support for the p -factor diminished: higher-order psychopathology dimensions became more differentiated, psychopathology data became more multidimensional, and a general factor of psychopathology explained less variance in psychopathology. In fact, when undiagnosed cases were culled completely from the data, leaving cases with at least some degree of clinically significant psychopathology, the latent correlation between externalizing and internalizing dimensions was either negligible or negative. We believe our findings raise important questions regarding the statistical and ontological status of the p -factor, the latent structure of psychopathology, and comorbidity more generally (Watts et al., 2019, 2020).

Implications

Our findings are open to a number of interpretations among which we cannot entirely adjudicate (Table 2). Some interpretations may support the p -factor's status as a substantive construct and others may support it as a methodologic artifact, but certain measurement and modeling practices make it difficult to distinguish between substantive and methodologic sources of variance.

On the methodological front, a general factor of psychopathology may arise in any given sample simply because we are using inappropriate modeling techniques. Other fields – ecology, computer science, and engineering, to name a few – have long appreciated the fact our variables' prevalence rates influence their pairwise similarities (Warrens, 2008). Ecology, for instance, relies on association measures that *exclude* the mutual absences of rare species' locations for the very reason we demonstrate here: traditional association measures (such as a correlation coefficient) erroneously indicate that two species are highly related simply because they do not occur together often (Janson & Vegelius, 1981). Neither a horse nor an alligator has

wings, but it is likely premature to conclude that they are highly similar creatures because they do not share that particular characteristic. That is, the extent of the horse and alligator's relatedness is inflated when their absence of relatedness is considered in isolation. Ultimately, there are potentially innumerable unmeasured aspects of psychopathology on which people vary. Within psychology, other statistical techniques may better take into account undiagnosed cases, including but not limited to accounting for censoring of the manifest and/or latent indicators, accounting for the count nature of continuous symptom dimensions, and two-part modeling (e.g., zero-inflated Poisson or negative binomial regression; Table 2).

On the substantive front, our findings imply that the structure of psychopathology varies across levels of severity, or that comorbidity is moderated by extent of symptomatology (Muthén, 1989). This might mean that psychopathology becomes more multidimensional as a function of increased severity (or comorbidity, but severity and comorbidity are potentially inextricably linked; Helle et al., 2020). It might also be that psychopathology appears more unidimensional at both extremes of severity or comorbidity, consistent with a curvilinear (inverse U-shaped) association between psychopathology spectra (i.e., externalizing, internalizing) across severity levels. Such ideas can be tested with multi-group models, moderated factor analysis, heterogeneous factor analysis, or local structural equation modeling (Table 2).

Our findings might instead imply population heterogeneity, whereby there exist discrete subpopulations of people that are differentially susceptible to psychopathology. These subpopulations may even have dramatically different latent structures of psychopathology, which can be tested with factor mixture modeling. The underappreciation of population heterogeneity in the psychopathology literature may even explain the recent popularity of the bifactor model of psychopathology. Raykov and colleagues (2019) demonstrated that empirical support for the bifactor model may arise spuriously due to unaccounted for population heterogeneity (also see Kam, 2020). Indeed, although data from epidemiologic samples are often presumed to be sampled from a single, homogeneous population, parameter values might

differ across subpopulations in the presence of population heterogeneity (Muthén, 1989). Both of the prospects we raise – invariance across levels of severity and population heterogeneity – are reasonably compatible, with the critical difference between them being whether there are categorically distinct subpopulations or whether psychopathology grades along multiple distinguishable dimensions (Table 2).

These prospects are mostly speculative within the context of structural models of psychopathology, but they have been explored in the study of certain psychopathology-relevant constructs. In a previous examination of the effects of sampling on the structure of alcohol problems, Steppan and colleagues (2014) showed that the correlation between DSM-IV alcohol abuse and dependence was high when asymptomatic cases were included, and near zero when the sample was restricted to those who endorsed one or more criteria for abuse or dependence. By now, several sets of authors have also shown that the inclusion of asymptomatic cases induced a positive manifold in the covariance matrix in the context of factor and network modeling (see also Hoffman et al., 2019a).

In the intelligence literature, Molenaar (2010) showed that the structure of intelligence was potentially moderated by ability level such that the positive manifold among intelligence tasks decreased as intelligence increased (Molenaar et al., 2017; Reynolds et al., 2010). This is especially noteworthy given intelligence’s supposed superordinate dimension, g , is the conceptual cousin of the p -factor (Caspi et al., 2014). And finally, Kam (2020) recently showed that there is population heterogeneity within self-esteem such that it has different factor structures – one unidimensional and one multidimensional – depending on the subpopulation. We look forward to future examinations of the structure of psychopathology with these techniques (cf., Wright et al., 2013).

The prospect of population heterogeneity, with populations of different psychopathology structures intrigues us. Reflecting on the considerable degree of undiagnosed cases in most epidemiologic samples, it may be the case that there is a large class with a relative absence of

diagnosed psychopathology for whom psychopathology appears to conform to a unidimensional solution. Nevertheless, any *observed* latent class with minimal diagnosed psychopathology is likely composed of true and false negatives, with false negatives incorrectly undiagnosed due to a variety of methodologic factors (e.g., response bias, skip-out assessment structure), subthreshold psychopathology, and unmeasured or unmodeled psychopathology, including aspects of psychopathology not captured by dominant nosologies (e.g., attachment insecurity). Other classes may comprise reasonably symptomatic individuals for whom psychopathology appears much more multidimensional.

The theoretical subpopulation of people for whom psychopathology appears unidimensional may comprise those with an exceptionally high threshold to experience psychopathology. Schaefer and colleagues (2017) observed in the Dunedin cohort that 17% of never diagnosed with a mental disorder from birth to midlife, and they described this group as having “enduring mental health.” Diagnosed and undiagnosed groups were not differentiated by physical health, socioeconomic status, and educational attainment, but they were characterized by relative absence of family history of mental disorder and a profile of certain personality traits: emotional stability (reversed neuroticism), agreeableness, conscientiousness. In hierarchical models of personality, these three traits form a metatrait, Stability, which appears to reflect decreased risk to experience psychopathology (DeYoung & Krueger, 2018, in press). In their theory of psychopathology, DeYoung and Krueger (2018, in press) further argue that (1) Stability is either isomorphic with or strongly resembles the *p*-factor and (2) there is a true binary between having and not having psychopathology, but that its severity is dimensional. Both of these claims are highly compatible with much of our supposition.

Limitations and Potential Criticisms

We relied on binary diagnoses generated from interview assessments with “skip out” structures because we wanted to mimic common practices in the literature (Forbes et al., 2020; Gluschkoff et al., 2019; Kim & Eaton, 2017; Krueger, 1999; Lahey et al., 2012). In skip out

structures, participants with little to no clinically significant psychopathology are administered a mere handful of screening items, precluding the construction of more reliable dimensional indicators, propagating undiagnosed cases, and leading to false negatives and correlated errors among diagnoses (Hoffman et al., 2019b; Robins & Cottler, 2004). Even when binary diagnoses are not assessed with skip outs, their use poses at least two other challenges. First, they arbitrarily dichotomize continuous dimensions, resulting in indicators of compromised reliability (Vandiver & Sher, 1991). Second, they tend to assess unipolar symptom criteria (ranging from “none” to “a lot”, for instance), which limits the range of symptom continua captured, restricts the modeling to the average to above average levels of latent symptom dimensions, and yields censored indicators with floor effects. All told, binary diagnoses likely lump a heterogeneous group of people into the “absent” category, and a general factor may arise due to a number of reasons, including correlated measurement error and an excess of zeros, the latter of which we demonstrated here.

Nevertheless, we remain unconvinced that our findings would not replicate had we modeled continuous indicators. There is strong compatibility between structural models of psychopathology modeled with binary and continuous indicators (Kotov et al., 2017), and modeling continuous indicators also yields highly skewed and censored indicators. That is to say, we do not believe general factors of psychopathology are simply artifacts of the use of skip out structures and binary diagnoses. We look forward to future research that extends this work to dimensional and symptom-level data.

Additionally, skeptics of our findings will argue that we have invoked Berkson’s bias (or Neyman’s bias, or selection bias, or the like; Hill, 2003; Meredith, 1964) by increasingly restricting the sample to reflect higher degrees of severity (de Ron et al., 2019). To the statistically inclined, this would mean that we have artificially restricted the range of observed values, thereby decreasing the variance and covariance among diagnoses. We do not view our exercise as a form of selection bias. Though our demonstration is “artificial,” numerous factors

can affect the proportion of disorder in a sample. For instance, there is natural variation in the base rates of diagnoses across populations, including across genders, cultures, and so on. All told, in our view, population-based ascertainment bias is no less a concern than Berkson's bias, and the two reflect countervailing forces in psychopathology research. If nothing else, our findings indicate that the nature of the sample – either clinical or population-based – predetermines the covariance structure of the data, thereby predetermining the inferences we make about the empirical structure of psychopathology.

Additionally, given that correlations are attenuated when indicators have greater differences in skew, skeptics may argue that we are inducing greater varieties of skewness by culling undiagnosed cases, leading to reduced interitem correlations. In Figure S5, we have displayed histograms of each diagnosis as a function of the extent of undiagnosed cases in the data. There we see that the drop in skew as we removed undiagnosed cases appears relatively uniform across disorders, suggesting that varying degrees of skew are not responsible for the findings we report here.

Finally, accepting that the same mechanisms that operate in normal function do so in dysfunction, proponents of dimensional models of psychopathology and critics of our conclusions might argue that the class with low probability of any psychiatric diagnosis *does* inform comorbidity. Under a dimensional model, the same mechanism that gives rise to cross-dimension comorbidity may also protect against cross-dimension comorbidity if one's placement on that dimension is low. We raise several counterpoints to this argument, though it strikes us as a reasonable criticism of our work. First, comorbidity specifically refers to a *shared presence* rather than *shared absence* of two or more disorders. As we mentioned earlier, we think it is counterintuitive to define similarity based on an absence of similarity. Second, long ago, Cattell (1965) warned against examinations of dimensionality in samples of individuals who are largely unaffected by the phenomena of interest. Third, and relatedly, a dimension may be detected in an entire population, but this does not mean that it is general to the entire population.

Conclusion

The inclusion of undiagnosed cases in structural models of psychopathology creates the illusion of a more homogeneous, unidimensional structure that becomes more multidimensional when removed. Ultimately, our findings hint towards the possibility that observed dimensionality of psychopathology may depend the extent of psychopathology in the data. These findings raise questions about the nature of the *p*-factor, in particular, including whether it reflects a strictly methodological artifact. Against the backdrop of the broader literature in which there is relatively little coherence in terms of general factors of psychopathology across studies (Levin-Aspenson et al., 2020; Watts et al., 2020), we suspect general factors of psychopathology are methodological artifacts and are not general to the entire population.

A compatible possibility is that the *p*-factor is potentially a substantive mechanism that protects against the development of significant levels of psychopathology across the lifespan in *a mere subset* of the population. This means that the *p*-factor may be largely irrelevant to the subpopulation who experiences psychopathology in some significant form. Should this supposition bear out in future empirical endeavors, it would call for a significant reconceptualization of the theoretical *p*-factor construct. As yet, the *p*-factor has been described as a unitary dimension on which all humans can be placed. The higher one's placement on a general factor of psychopathology, the higher stronger one's propensity to develop several forms of psychopathology (Caspi & Moffit, 2018; Lahey et al., 2012). We found that empirical support for the *p*-factor was largely driven by people *without* diagnosed psychopathology. That is, among those with at least some degree of clinically significant psychopathology, we saw little empirical evidence of a *p*-factor.

Author Contribution Statement

We define author contribution using the Contributor Roles Taxonomy (CRediT). Watts was responsible for conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, supervision, validation, visualization, writing (original draft, editing). Meyer was responsible for data curation, formal analysis, investigation, resources, software, validation, and writing (review and editing). Greene was responsible for writing (review and editing). Wood was responsible for conceptualization, funding acquisition, and writing (review and editing). Trull was responsible for funding acquisition and writing (review and editing). Steinley was responsible for conceptualization, funding acquisition, and writing (review and editing). Sher was responsible for conceptualization, funding acquisition, and writing (review and editing).

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(a) Two correlated factors model

(b) Three correlated factors model

(c) Bifactor model with two specific factors

(d) Bifactor model with three specific factors

(e) Single factor model

Figure 1. Path diagrams for all models tested.

Figure 2. Correlations between latent psychopathology factors as a function of undiagnosed cases. Figure 1a displays the correlation between externalizing and internalizing factors and Figure 1b displays the correlations among externalizing, distress, and fear factors. The asterisks indicate that the model did not converge. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded.

Figure 3. Dimensionality of the single factor model. Figure 2a displays scree plots for each dataset, with Eigenvalues plotted as a function of undiagnosed cases included in the analysis. Percent undiagnosed cases retained (0 through 100, in increments of 10) are each displayed with a line and are displayed as a gradient, with 0 percent of undiagnosed cases retained being the darkest line and 100 the lightest. Figure 2b displays model fit indices for a single factor (1-factor) psychopathology model as a function of undiagnosed cases included in the analysis. Dark grey lines indicate the threshold for "good" model fit in the case of CFI, TLI, RMSEA, and SRMR, and lighter grey lines (where applicable) indicate the threshold for "acceptable" fit. The dark grey line for OmegaHierarchical represents a benchmark value that reflects "essential unidimensionality." Asterisks indicate that Eigenvalues were not available for 10 and 11 components in NCS-R given that there were only 9 indicators included in the model. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded.

Table 1. *Factors that likely contribute to covariation among psychiatric phenomena, the production of a general factor in data, and interpretability challenges with the p-factor.*

Issue	Implications	How to Address It
Population invariance	There may be different subpopulations with completely different structures of psychopathology.	Test for measurement invariance with: (1) multi-group models if the moderator is categorical, or (2) moderated factor analysis, moderated nonlinear factor analysis, heterogeneous factor analysis, or local structural equation modeling if the moderator is continuous.
Population heterogeneity	There may be different populations in the data, potentially with different parameter values or structures. Factor models assume population homogeneity.	Use factor mixture modeling, including two-part mixture modeling.
Binary data	Arbitrarily dichotomizes continuously distributed traits. Cannot distinguish between whether someone has a low level on a latent trait, or whether the person is in a different class.	Use continuous indicators. Account for censoring of the manifest and/or latent indicators. Account for the count nature symptom dimensions.
Skip out assessment structure	Propagates “zero” cases, leads to false negatives and correlated errors among diagnoses, and restricts range of latent trait to upper ends.	Use two-part modeling (zero-inflated Poisson, negative binomial). Assess the full criterion set for each disorder.

Supplemental Tables and Figures

Figure S1. Correlations between latent externalizing and internalizing factors as a function of undiagnosed cases by assessment method. The asterisk indicates that the model did not converge. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded. HER = Hierarchical exclusion rule.

Figure S2. Correlations between latent externalizing and internalizing factors as a function of undiagnosed cases. The solid lines indicate that cross-spectrum undiagnosed cases (externalizing-internalizing) were culled from the sample, whereas the dashed and dotted lines culled within-spectrum cases, either externalizing or internalizing. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded. EXT = Externalizing; INT = Internalizing.

Figure S3. Correlations between latent externalizing and internalizing factors as a function of dropping undiagnosed cases for each externalizing diagnosis. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded. ALC = alcohol dependence; ASP = antisociality; CAN = cannabis dependence; CND = conduct disorder; DRG = other drug dependence; TOB = tobacco dependence.

Figure S4. Correlations between latent externalizing and internalizing factors as a function of dropping undiagnosed cases for each internalizing diagnosis. Point estimates represent median values of the 500 models for each percent undiagnosed cases retained condition and are presented with minimum and maximum values from each set of 500 models shaded. AGP = agoraphobia with panic disorder; DEP = major depression; DYS = dysthymia; GAD = generalized anxiety disorder; PTS = post-traumatic stress disorder; SOC = social anxiety disorder; SPH = specific phobia.

Figure S5. Histograms of diagnoses in NESARC as a function of percent of undiagnosed cases removed. These data were drawn from 1 randomly selected dataset of the 500 iterations.

Table S1. *Percent diagnosed cases by diagnosis and spectrum.*

Diagnosis	NESARC w1	NCS	NCS-R
<i>Externalizing</i>	23.3	33.5	14.1
Adult antisocial behavior/personality disorder	3.6	5.4	---
Alcohol dependence	12.5	14.2	5.4
Cannabis dependence	1.3	---	---
Conduct disorder	---	12.7	5.2
Other substance dependence	1.8	7.5	3.1
Tobacco dependence	17.7	---	---
<i>Internalizing</i>	26.6	46.6	53.1
<i>Distress</i>	2.1	29.0	36.6
Dysthymia	4.9	6.5	4.0
Generalized anxiety disorder	4.5	5.0	7.8
Major depressive disorder	18.2	16.9	19.2
<i>Fear</i>	12.4	31.9	32.6
Agoraphobia with panic disorder	1.1	1.3	1.4
Post-traumatic stress disorder	---	7.3	---
Social anxiety disorder	5.0	13.5	12.2
Specific phobia	9.5	11.1	12.7
<i>Any diagnosis</i>	39.2	61.9	57.9

Note. NESARC assessed antisocial personality disorder, whereas NCS and NCS-R assessed adult antisocial behavior and conduct disorder. NCS-R assessed major depressive episode and dysthymia, whereas NESARC and NCS assessed major depressive disorder and dysthymia.

Table S2. Model fit statistics for correlated externalizing and internalizing models as a function of type of diagnostic indicator.

	χ^2	df	RMSEA	CFI	TLI	SRMR	r (SE)
Correlated externalizing and internalizing factors							
Lifetime diagnoses without hierarchical exclusion rules	269	43	.030	.964	.953	.060	.481 (.027)
Lifetime diagnoses with hierarchical exclusion rules	177	43	.023	.972	.965	.057	.481 (.029)
Past year diagnoses without hierarchical exclusion rules	183	43	.024	.965	.955	.071	.468 (.033)
Past year diagnoses with hierarchical exclusion rules	142	43	.020	.967	.958	.077	.449 (.034)

Table S3. Model fit statistics for all structural psychopathology models.

	χ^2	df	RMSEA	CFI	TLI	SRMR	r (SE)
One factor							
NESARC Wave 1	3412	44	.042	.882	.853	.120	-
NCS	1187	44	.066	.816	.770	.131	-
NCS-R	488	27	.055	.884	.845	.120	-
Correlated externalizing and internalizing factors							
NESARC Wave 1	713	43	.019	.977	.970	.052	E-I: .562 (.012)
NCS	269	43	.030	.964	.953	.060	E-I: .481 (.027)
NCS-R	174	26	.032	.963	.948	.071	E-I: .529 (.031)
Correlated externalizing, fear, and distress factors							
NESARC Wave 1	241	41	.011	.993	.991	.030	D-E: .542 (.013) D-F: .719 (.014) E-F: .503 (.017)
NCS	190	41	.025	.976	.968	.055	D-E: .426 (.031) D-F: .765 (.026) E-F: .480 (.032)
NCS-R	49	24	.014	.994	.990	.042	D-E: .458 (.032) D-F: .697 (.019) E-F: .523 (.035)
Bifactor model with externalizing and internalizing specific factors							
NESARC Wave 1	82	33	.006	.998	.997	.017	-
NCS	105	33	.019	.988	.981	.041	-
NCS-R	237	75	.020	.975	.964	.059	-
Bifactor model with externalizing, fear, and distress specific factors							
NESARC Wave 1	162	33	.010	.995	.992	.024	-
NCS	93	33	.018	.990	.984	.041	-
NCS-R	27	18	.009	.998	.995	.030	-

Note. These models were conducted on the full samples prior to undiagnosed cases being dropped.

Table S4. *Eigenvalues as a function of undiagnosed cases.*

% undiagnosed cases retained	Component number											Ratio of 1 st to 2 nd eigenvalues
	1	2	3	4	5	6	7	8	9	10	11	
NESARC												
100	5.37	1.75	.91	.57	.49	.44	.39	.37	.27	.24	.21	3.08
90	5.25	1.78	.93	.59	.50	.45	.39	.38	.27	.25	.22	2.96
80	5.12	1.81	.95	.60	.51	.46	.40	.39	.28	.25	.23	2.83
70	4.97	1.85	.98	.61	.52	.48	.41	.40	.28	.26	.24	2.69
60	4.81	1.89	1.01	.63	.54	.50	.41	.41	.29	.27	.25	2.55
50	4.62	1.94	1.04	.65	.56	.52	.42	.41	.30	.27	.26	2.39
40	4.41	1.99	1.08	.67	.58	.54	.44	.41	.31	.28	.27	2.21
30	4.16	2.06	1.13	.70	.61	.57	.46	.41	.33	.29	.29	2.02
20	3.86	2.14	1.19	.74	.64	.61	.48	.40	.34	.30	.29	1.81
10	3.52	2.23	1.27	.80	.68	.65	.51	.40	.37	.31	.26	1.57
0	3.12	2.35	1.38	.90	.72	.69	.55	.43	.39	.33	.15	1.33
NCS												
100	4.67	1.91	.98	.72	.61	.49	.42	.38	.31	.25	.25	2.45
90	4.54	1.94	1.00	.73	.62	.51	.43	.39	.32	.26	.25	2.34
80	4.39	1.98	1.02	.74	.64	.52	.44	.41	.34	.27	.26	2.22
70	4.23	2.02	1.05	.76	.65	.54	.45	.42	.35	.28	.26	2.10
60	4.05	2.06	1.07	.77	.67	.55	.47	.43	.36	.29	.27	1.97
50	3.85	2.10	1.11	.79	.69	.57	.48	.45	.38	.30	.28	1.83
40	3.62	2.16	1.14	.81	.71	.59	.50	.47	.40	.31	.29	1.68
30	3.36	2.21	1.18	.84	.74	.62	.52	.49	.42	.33	.29	1.52
20	3.06	2.27	1.23	.86	.77	.65	.54	.51	.44	.35	.30	1.34
10	2.74	2.32	1.29	.89	.81	.68	.56	.55	.47	.37	.31	1.18
0	2.58	2.19	1.35	.93	.87	.73	.59	.58	.50	.40	.29	1.18
NCS-R												
100	4.21	1.53	1.04	.60	.50	.43	.32	.22	.16	---	---	2.75
90	4.10	1.55	1.06	.61	.51	.44	.33	.22	.17	---	---	2.64
80	3.98	1.58	1.09	.62	.53	.46	.34	.23	.18	---	---	2.51
70	3.84	1.61	1.12	.64	.55	.47	.35	.24	.18	---	---	2.38
60	3.68	1.65	1.15	.65	.57	.49	.37	.25	.19	---	---	2.23
50	3.50	1.69	1.19	.67	.60	.51	.39	.26	.19	---	---	2.07
40	3.28	1.73	1.24	.69	.63	.54	.41	.27	.20	---	---	1.89
30	3.02	1.79	1.30	.71	.67	.57	.43	.29	.21	---	---	1.69
20	2.71	1.85	1.38	.75	.72	.61	.47	.30	.21	---	---	1.46
10	2.34	1.92	1.48	.82	.75	.66	.51	.32	.20	---	---	1.22
0	2.08	1.85	1.60	.93	.80	.71	.57	.34	.12	---	---	1.12

Table S5. *Loadings on the general factor in the bifactor model with 2 specific factors as a function of undiagnosed cases in NESARC.*

Diagnosis	Percent undiagnosed cases retained											Mean	Median	SD	Range
	100	90	80	70	60	50	40	30	20	10	0				
AGP	.77	.76	.76	.76	.76	.75	.75	.74	.75	.77	.74	.75	.76	.01	.03
ALC	.38	.36	.34	.31	.28	.25	.20	.15	.08	-.02	-.14	.20	.25	.17	.53
ASP	.43	.41	.40	.38	.36	.34	.31	.28	.24	.18	.10	.31	.34	.10	.32
CAN	.48	.47	.46	.45	.44	.42	.40	.38	.35	.30	.22	.40	.42	.08	.27
DEP	.86	.85	.84	.83	.82	.80	.78	.75	.72	.68	.73	.79	.80	.06	.18
DRG	.51	.50	.49	.48	.46	.45	.43	.40	.37	.33	.25	.42	.45	.08	.26
DYS	.81	.80	.80	.80	.80	.80	.80	.79	.78	.73	.63	.78	.80	.05	.17
GAD	.77	.76	.76	.75	.75	.74	.73	.72	.71	.69	.65	.73	.74	.04	.12
SOC	.68	.67	.66	.65	.64	.62	.60	.58	.56	.54	.50	.61	.62	.06	.18
SPH	.61	.60	.58	.56	.54	.51	.48	.44	.40	.38	.34	.49	.51	.09	.27
TOB	.41	.38	.36	.33	.29	.25	.20	.13	.04	-.08	-.26	.18	.25	.21	.67
Range in loadings	.48	.49	.50	.52	.53	.55	.60	.67	.74	.85	1.00				

Note. Loadings are the median of the 500 iterations. AGP = agoraphobia with panic disorder; ALC = alcohol dependence; ASP = antisociality; CAN = cannabis dependence; DEP = major depression; DRG = other drug dependence; DYS = dysthymia; GAD = generalized anxiety disorder; SOC = social anxiety disorder; SPH = specific phobia; TOB = tobacco dependence.

Table S6. *Loadings on the general factor in the bifactor model with 2 specific factors as a function of undiagnosed cases in NCS.*

Diagnosis	Percent undiagnosed cases retained											Mean	Median	SD	Range
	100	90	80	70	60	50	40	30	20	10	0				
ALC	.39	.37	.35	.32	.70	.68	.21	.15	.08	-.02	-.17	.28	.32	.27	.88
AGP	.69	.68	.68	.68	.22	.20	.65	.64	.63	.69	.67	.58	.67	.19	.49
ASP	.38	.37	.35	.33	.72	.70	.25	.21	.15	.08	-.03	.32	.33	.23	.75
CND	.28	.26	.23	.20	.44	.37	.08	.02	-.06	-.17	-.33	.12	.20	.24	.77
DEP	.77	.76	.75	.74	.27	.24	.71	.69	.67	.56	.41	.60	.69	.20	.53
DRG	.43	.42	.40	.38	.84	.84	.30	.26	.21	.14	.03	.38	.38	.26	.82
DYS	.73	.73	.73	.73	.27	.25	.74	.75	.75	.58	.39	.60	.73	.20	.50
GAD	.71	.71	.71	.70	.29	.27	.69	.69	.68	.62	.49	.60	.69	.17	.44
PTSD	.6	.59	.57	.56	.34	.32	.50	.46	.42	.34	.21	.45	.46	.13	.39
SOC	.64	.63	.60	.58	.25	.22	.45	.38	.31	.39	.42	.44	.42	.15	.43
SPH	.66	.65	.64	.62	.21	.18	.53	.47	.42	.56	.60	.50	.56	.17	.49
Range in loadings	.49	.51	.52	.54	.63	.66	.66	.74	.81	.86	1.00				

Note. Loadings are the median of the 500 iterations. AGP = agoraphobia with panic disorder; ALC = alcohol dependence; ASP = antisociality; CAN = cannabis dependence; CND = conduct disorder; DEP = major depression; DRG = other drug dependence; DYS = dysthymia; GAD = generalized anxiety disorder; SOC = social anxiety disorder; SPH = specific phobia; TOB = tobacco dependence.

Table S7. *Median loadings on the general factor in the bifactor model with 2 specific factors as a function of undiagnosed cases in NCS-R.*

Diagnosis	Percent undiagnosed cases retained											Mean	Median	SD	Range
	100	90	80	70	60	50	40	30	20	10	0				
AGP	.66	.66	.65	.65	.65	.64	.64	.64	.63	.59	.54	.63	.64	.04	.12
ALC	.45	.44	.42	.40	.37	.34	.30	.26	.19	.11	-.04	.30	.34	.15	.49
CND	.31	.29	.27	.24	.21	.17	.12	.06	-.03	-.16	-.39	.10	.17	.22	.70
DEP	.79	.78	.76	.74	.73	.70	.67	.63	.59	.55	.54	.68	.70	.09	.25
DRG	.49	.48	.47	.45	.43	.40	.37	.33	.28	.20	.06	.36	.40	.13	.43
DYS	.75	.75	.75	.74	.74	.73	.73	.74	.76	.80	.70	.74	.74	.02	.10
GAD	.66	.65	.64	.62	.61	.59	.57	.54	.50	.46	.36	.56	.59	.09	.30
SOC	.76	.75	.73	.72	.70	.68	.64	.60	.52	.41	.28	.62	.68	.15	.47
SPH	.65	.64	.62	.60	.58	.55	.52	.47	.41	.31	.23	.51	.55	.14	.42
Range in loadings	.47	.48	.49	.50	.53	.56	.61	.68	.79	.96	1.09				

Note. Loadings are the median of the 500 iterations. AGP = agoraphobia with panic disorder; ALC = alcohol dependence; CND = conduct disorder; DEP = major depression; DRG = other drug dependence; DYS = dysthymia; GAD = generalized anxiety disorder; SOC = social anxiety disorder; SPH = specific phobia.