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Published in:
World psychiatry

DOI:
[10.1002/wps.20816](https://doi.org/10.1002/wps.20816)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2021

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Hartman, C. A. (2021). The important gain is that we are lumpers and splitters now; it is the splitting that needs our hard work. *World psychiatry*, 20(1), 72-73. <https://doi.org/10.1002/wps.20816>

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The important gain is that we are lumpers *and* splitters now; it is the splitting that needs our hard work

Anyone who has done a fair bit of factor analyzing broad measures of psychopathology knows about the general factor that dominates the covariance among the symptoms. He/she also knows that, when the measure includes sufficient depression and anxiety symptoms on the one hand, and aggression and conduct problem symptoms on the other, two broad factors of internalizing and externalizing psychopathology can robustly be identified, which are typically correlated at about .50, again indicative of the general factor.

Spending years on factor analyzing broad measures of psychopathology, I learned that: a) with the tentacles of the general factor so dominantly present in the covariance structure, deriving a meaningful fine-grained factor solution that replicates in the next sample, even when using the same or only a slightly different instrument, is hard; and b) while internalizing and externalizing symptoms are always well represented in broad measures of psychopathology, so that the corresponding factors easily emerge, symptoms of other problem domains (such as psychosis, autism spectrum or attention-deficit/hyperactivity) are generally less well represented, so that the corresponding factors are less robustly identified. I concluded^{1,2}, therefore, that factor analysis and its dimensional approach to psychopathology is a useful tool for psychometric analysis and scale development, but will never “carve nature at its joints”³.

Current work on the general factor of psychopathology suggests that I was partly mistaken, overlooking the obvious – i.e., this dominant general factor that is always present in broad measures of psychopathology *is* the nature of psychopathology. Its validity follows, for example, from its fit with heterotypic continuity across the lifespan⁴, partly overlapping genetic architectures⁵, or cross-diagnostic executive functioning problems⁶. Yet, the realization that the general factor captures meaningful variance provides only temporary relief. Clearly, this factor does not suffice to

understand psychopathology. While the DSM with its >200 diagnostic categories may be overly zealous on specificity, the question of which specific dimensions we can validly differentiate in psychopathology remains.

In psychiatry, we tend to find generic associations with external variables (i.e., etiological, environmental, therapeutic, prognostic)⁷. In the past, this raised the question: are our concepts in psychiatry so confounded that we do not find specificity?⁸ Now, we can follow this up by “yes”: our concepts are confounded by the general factor. Much of the generic relations we tend to find may be tied to the general factor, and whichever specific associations we are hoping to uncover are always “spoiled” by the dominant general factor which seeps right through our findings.

The paper by Lahey et al⁷ makes a strong case that we are in a better position to improve our understanding of specific associations with external variables in psychopathology if we delineate the specific factors by splitting the variance of the general and the specific factors using the bifactor model. In my view, this delineation should be pursued: only with the general factor variance removed can we have a clear window into the remaining covariance patterns among the symptoms in our measures. Only with specific measures unconfounded by the general factor can we have a clear window into specific etiological or prognostic associations.

Lahey et al should be lauded for their pioneering and persistent work in the past 10 years focused simultaneously on the general and the specific factors, following from their re-introduction of the bifactor model. Their work has, for example, suggested that the shared familial factors were associated with the general factor, while person specific influences were more likely associated with specific symptom domains⁷. While, so far, we have seen modest knowledge gains for the specific factors, relative to the “lower hanging fruits”⁴⁻⁶ easily caught by the comprehensive and currently better measured general factor, the approach advocated by

Lahey et al should be widely followed by many more research groups to get to the heart of specificity in psychopathology.

It is important to note in this context the frequent misunderstanding of the meaning of the specific factors in the hierarchical vs. the bifactor models. There is a critical difference: lower-order factors in the hierarchical model represent the dimensionality of psychopathology *within* the general factor, while the specific factors in the bifactor model represent this dimensionality *beyond* the general factor.

Empirical comparisons of the two types of specific factors to determine “the winner”, therefore, make no sense. Of course, the factor loadings of the specific factors in the bifactor model are lower, with larger standard errors, less stability over time, and so forth. This is only reflecting what we knew from our factor analytic efforts all along: free from the dominant general factor, a chaotic covariance structure of high instability often remains. This situation of poor measurement of the specific factors is a major obstacle in finding etiological or prognostic specificity.

The work ahead is therefore clear: for progress in understanding specific associations (in as much as these exist), strong measures of the specific factors are needed, which, separate from the general factor covariance, still demonstrate high internal construct validity. The critical problem we are facing is that our existing measures of broad psychopathology have insufficient dimensionality^{1,2}, which is solvable but needs our work.

Measures that were not originally developed with a clear blueprint for specific content domains of psychopathology will not have optimal dimensionality⁹, and therefore will show low and unstable factor loadings, which becomes immediately apparent in the bifactor model. When we construct our measures using the bifactor model, we are in a better position to create, evaluate and refine this dimensionality, since we are not “fooled” by the covariance from the general factor, that overestimates the internal construct validity of our specific

measures.

The number and type of domains of psychopathology can never be clarified on the symptomatic level alone. A continuing back-and-forth validation between internal (i.e., factor structure) and external (i.e., genetic, neurobiological, cognitive, environmental, therapeutic, prognostic, and so forth) construct validity would remain. To illustrate, it has often been said that “our DNA has not read the DSM”, and this obviously holds for any conceptualization of psychopathology at the symptom level.

High-quality multidimensional measurement will not be achieved by subjecting “all existing symptoms of psychopathology” to

factor analysis. Rather, the dimensionality of our measures should be created using a top-down approach, pragmatically choosing clusters of items representing relevant conceptual domains of psychopathology. By subjecting these item clusters to the bifactor model, it will be possible to achieve a dimensional measurement that both lumps (into the general factor) and splits (into specific dimensions). Only then can we fully evaluate the specific associations of psychopathology.

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DOI:10.1002/wps.20816

Taxonomy of psychopathology: a work in progress and a call for interdisciplinary research

Taxonomy is an essential element in the process of understanding and organizing concepts that form part of any scientific discipline. This exercise of classification has its origins in the mid 1700s with Carl Linnaeus' biological taxonomy, that provided the original rank-based classification of organisms, including plants, minerals and animals. For mental health disciplines, including psychiatry and psychology, this process of classification has been made especially challenging because of issues related to both the conceptualization and the measurement of psychopathology. Some other scientific disciplines work with clearly defined sets of criteria to identify and categorize the phenomena they study. Mental health problems bring complex issues related to symptom presentation and comorbidity that have yet to be agreed on.

The usefulness and applicability of psychiatric nosology stand on at least two pillars. The first is that a taxonomy must reflect clinical reality: patients with mental health problems often present heterogeneous symptoms and comorbid disorders. The second is that a taxonomy must soundly summarize clinical information, based on appropriate statistical models, but without losing fine-grained details that are relevant for research and treatment.

Significant concerns have been raised

as to whether the current categorical classification systems of psychopathology meet either of these requirements. There is indeed extensive recognition that comorbid presentation of psychiatric disorders is the norm rather than the exception¹, and that symptoms vary across illnesses instead of being limited to individual diagnoses. A dimensional approach may be best suited to reflect this reality.

A productive debate about the appropriateness of a categorical diagnostic system is still ongoing, and concerted scientific efforts have resulted in proposals for sophisticated models as alternative approaches to psychiatric nosology, including the Hierarchical Taxonomy of Psychopathology (HiTOP)², the transdiagnostic approach³ and the Research Domain Criteria (RDoC)⁴. While a consensus has not been reached yet, there is an undeniable recognition of the pressing need to find more suitable models and methods for classifying psychopathology. Mental health research depends on it but, most importantly, clinical services rely on a suitable nosology to provide appropriate treatments to those who need it.

Lahey et al⁵ provide an overview of the hierarchical approach to psychopathology. This approach – which is strongly embedded in psychometric methods – proposes

models in which a higher-order, or general, factor (otherwise known as the p factor) captures correlated symptoms, and lower-order, or secondary, factors encapsulate specific symptoms^{6,7}.

There are valuable strengths in this approach, as it provides a concise summary of symptoms across mental health problems and retains a dimensional approach to psychopathology. However, three points deserve further considerations.

First, there is a risk that the bifactor model remains limited to a statistical representation of psychopathology. Findings reviewed by Lahey et al indicate that the p factor is genetically influenced and more stable than the secondary factors. However, this may be an artefact of statistical organization of data with, for example, secondary factors being more prone to include stochastic (i.e., randomly determined) measurement errors that are not influenced by genetic factors and are less inherently stable. These secondary factors may also, in effect, hold key information for treatment and precision medicine.

Second, the development of mental health problems is a dynamic process that changes throughout the life course and depends on social context. While there are findings supporting the validity of the p factor in samples of young children⁸, it is