Considering the appropriateness of the factor analytic operationalization of allostatic load

Citation for published version:
Crook, Z & Booth, T 2016, 'Considering the appropriateness of the factor analytic operationalization of allostatic load' Psychosomatic Medicine. DOI: 10.1097/PSY.0000000000000415

Digital Object Identifier (DOI):
10.1097/PSY.0000000000000415

Link:
Link to publication record in Edinburgh Research Explorer

Document Version:
Peer reviewed version

Published In:
Psychosomatic Medicine

General rights
Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy
The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.
Considering the Appropriateness of the Factor Analytic Operationalization of Allostatic Load

Zander Crook, MSc, Department of Psychology, The University of Edinburgh
Tom Booth, PhD, Department of Psychology, The University of Edinburgh

Corresponding author: Zander Crook, Department of Psychology, The University of Edinburgh, 7 George Square, Edinburgh, EH8 9JZ, United Kingdom. Telephone number: (+44)7791899804. E-mail address: s1368539@sms.ed.ac.uk.

Abbreviated running title: Operationalizing Allostatic Load

Keywords: allostatic load; factor analysis; multisystem physiological dysregulation; biomarkers; latent variable theory; bifactor model

Word count: 1558
Table count: 0
Figure count: 0

Conflicts of Interest and Source of Funding: Neither author has a conflict of interest to declare.

Z. C. is supported by a UK Economic and Social Research Council Advanced Quantitative Methods PhD award.
In a recent issue of *Psychosomatic Medicine*, Wiley and colleagues' (1) made a valuable contribution to the discussion of the optimal measurement of allostatic load (AL). In the most comprehensive factor analytic investigation of AL to date, they found that a bifactor model with a general AL factor and seven physiological system factors fit better than a higher-order model in which the seven system factors loaded on the general AL factor. Similar models have been applied by the author (T. B.) and others to operationalize AL (2,3,4). Here we consider the primary theoretical assumptions underlying latent variable modeling, argue that the construct of AL is inconsistent with these assumptions and propose alternate operationalizations of AL.

**Underlying Construct (Common Cause)**

A latent variable model is estimated based on the patterns of covariance in a set of variables. By including an AL general factor in a latent variable model, researchers are positing that an underlying construct is the common cause of the observed covariation in all of the modeled biological measures. Though the theoretical relation of the common cause or construct to the original variables differs in bifactor versus higher-order models, in either case we must ask: What could this common factor be? Wiley and colleagues stated that the AL factor “[captures] the notion that there is an underlying process influencing multiple physiological systems” (1: p. 4). But the observation of a general factor estimated from inter-individual summary statistics (i.e., covariances) says little about what this process may actually be.

**Independence Conditional on the Latent Trait**

A primary assumption of latent variable models is that once the effect of the latent factors has
been accounted for, the measured variables – in this case, the biological measures – are independent. This is unlikely to be the case with AL measures. Levels of different biomarkers are linked causally to each other, rather than only through the common cause latent variable(s). For example, body mass index (BMI) has previously been used as a metabolic system AL biological measure (e.g. 2,5). However, Mendelian randomization studies have found that increased BMI has a causal effect on levels of other metabolic biological measures as well as levels of AL biomarkers used to represent other physiological systems, such as blood pressure and inflammation (e.g. 6). Thus, it is most likely that the biomarkers are not conditionally independent, but are instead dynamically related in complex networks. Such networks can produce observed correlations between variables that have no common cause (7).

**Interchangeability of Indicators**

A further assumption of the latent variable model is that the definition of the latent variable does not change when different sets of indicators are used (8). This holds because the indicators are affected by, but do not affect, the latent variable. Another key finding of Wiley and colleagues was that fitting models in which the biological measures from each of the seven physiological systems were excluded caused no large changes in AL factor loadings (1). This method provides only a weak test of interchangeability. The stability of general intelligence factor loadings has long been a research focus for intelligence researchers, so AL researchers may benefit from applying their approaches to this issue (e.g. 9,10). For example, researchers could compute and correlate AL scores from different non-overlapping multisystem sets of biological measures (8). The existence of diverse causal links between AL biological measures from different
physiological systems suggests to us that the nature of what relates the biomarkers may change depending on which measures are included in the model. We predict that more thorough, more powerful tests of the stability of AL factor loadings will find that it does not hold.

**Formative Versus Reflective Indicators**

In the common factor model, the biological measures are reflective indicators—that is, they are manifested by a common cause latent variable. However, to the extent that the model assumptions are violated (discussed above), the factor model is not appropriate. Thus, it may instead be profitable to consider the biological measures as formative indicators—that is, as variables that define the construct (8). This way of thinking about how the biological measures relate to AL is consistent with any number of weighted or sum scores. It is also consistent with AL theory, in that more severe, more widespread physiological dysregulation will relate to higher AL scores.

Alternatively, the associations between AL biological measures could be modeled using each measure individually, without the need for any single latent or observed summary. This could be done with network analysis, which has been used beneficially by researchers studying symptom networks in mental disorders (11). AL indicators can also be modeled separately without consideration of their associations. Consistent with this approach, *Psychosomatic Medicine* typically provides data of separate biological measures when articles report about complex phenomena such as AL and metabolic syndrome.
Aside from any issues regarding model assumptions, two further points warrant comment about the models presented by Wiley and colleagues.

**Improved Model Fit for Bifactor Approach**

The complex causal links between biological measures from different physiological systems also help to explain why the bifactor AL model fits better than the hierarchical AL model. The hierarchical model imposes “proportionality constraints” (12: p. 115): the ratio of the AL general factor loadings to the system factor loadings is constrained to equality within the biological measures of each physiological system. Considering the diverse causal links between different AL biomarkers, both within and across systems, these proportionality constraints are likely to be violated. Further, it has been shown that when the true model contains “unmodelled complexity” (13: p. 407) in the form of small correlated residuals and cross-loadings, or even modeled complexity in the form of correlated residuals across factors, fit indices and criteria may be biased in favor of the bifactor model. Consequently, the better fit of the bifactor model follows from AL theory and research, as well as from methodological findings, for reasons other than those focused on by Wiley and colleagues (1).

**Variance Explained by Physiological Dysregulation Factors**

Statistically, a desirable property of a general factor is that it accounts for the majority of variance in the constituent indicator variables. In Wiley and colleagues’ study, the AL factor explained only approximately 11% of the variance in the AL biological measures. Some of the physiological system-specific factors were also weak. For example, the HPA axis and inflammation factors
explained only approximately 9% and 16% of the variance in their respective biological measures. Note that weak factor saturation of physiological dysregulation factors has also been an issue in other samples (2,3).

**Properties of Optimal Scores for AL**

Ideally, AL scores should be: (1) calculated using biological measures from various physiological systems; (2) consistently calculated across samples; and (3) closely related to criterion variables. Those who desire scores that are rooted in AL theory would prefer the AL scoring method that produces the scores most closely related to chronic/repeated perceived stress. For a pragmatist, the focus may not be on investigating how different physiological dysregulation scores relate to prior perceived stress, but rather on finding the scores that most strongly predict important health outcomes such as cardiovascular disease and death. It may also be advantageous to have scores which explicitly represent the accumulation of the effects of repeated environmental challenges.

Our theoretical and methodological concerns with the factor analytic operationalization of AL suggest to us that factor scores will not prove to be the optimal AL scoring method. We therefore believe that further research is required to determine the optimal operationalization(s) of AL.
REFERENCES


7 van der Maas HLJ, Dolan CV, Grasman RPPP, Wicherts JM, Huizenga HM, Raijmakers MEJ. A dynamical model of general intelligence: the positive manifold of intelligence by mutualism. Psychol Rev 2006;113;842-61.
8 Bollen KA, Bauldry S. Three Cs in measurement models: causal indicators, composite indicators, and covariates. Psychol Methods 2011;16;265-84.

9 Johnson W, Bouchard TJ Jr., Krueger RF, McGue M, Gottesman II. Just one g: consistent results from three test batteries. Intelligence 2004;32;95-107.


13 Murray AL, Johnson W. The limitations of model fit in comparing the bi-factor versus higher-order models of human cognitive ability structure. Intelligence 2013;41;407-22.