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COMMENTARY

Revising the Trait Model of the Alternative Model of Personality Disorders: Comment on Clark and Watson's Structural Review

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In their review of examination of the self-report Personality Inventory for *Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition* (*DSM–5*; PID-5) factor structure and joint factor analysis of this instrument with other self-report measures of personality and psychopathology, Clark and Watson (2022) make argument for a set of modifications to the *DSM–5* alternative model of personality disorders (AMPD)—the AMPD-5.1. In this commentary we offer opinion that their proposed modifications to the AMPD are problematic. In particular, we express concern that their modifications are based solely on research that uses a single instrument to measure the model and that this instrument employs a self-report measure methodology. We argue further that this method of assessment is vulnerable to response bias which could potentially alter the substantive structure of the model. We suggest that any proposed revised model should also be informed by other forms of measurement (e.g., informant report and structured interviews). In addition, we also argue that the best approach to delineating the universe of pathological personality traits should also include clinician input. Such input would maximize the clinical utility of any revised model. We believe that to propose a modified model that is based on self-report only and does not include other sources of information is presumptive and premature. At best, we think that Clark and Watson (2022) have only provided suggestions for a revision and expansion of the Personality Inventory for *DSM–5* (i.e., the PID-5.1).

Keywords: AMPD, structural analysis, PID-5, self-report, clinician input

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Clark and Watson (2022) in their article, "The Trait Model of the *DSM–5* Alternative Model of the *DSM–5*: A Structural Review" provide a comprehensive and detailed review of the research literature that has accumulated since the introduction of the alternative model of personality disorders (AMPD) in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM–5*; American Psychiatric Association, 2013) and the publication of the most widely used instrument designed to measure the AMPD pathological personality traits—the self-report Personality Inventory for *DSM–5* (PID-5; Krueger et al., 2012). Based on their review and synthesis of this literature, they provide a set of recommendations for modifications to this model (the AMPD 5.1) and the PID-5 (the PID-5.1; Clark & Watson, 2022; Table 3). We commend Clark and Watson (2022) for this effort, as it satisfies, and perhaps even justifies relegating the AMPD to Section III of the *DSM–5*, "Emerging Measures and Models," as a model "in need of further study" rather than including it among the chapters in the main text of *DSM–5* (now *DSM–5-TR*; i.e., Section II). The personality disorder field of researchers, particularly those interested in dimensional models of personality and psychology, responded well to this call for further research on the AMPD, producing an impressive volume of empirical publications on it. Clark and Watson (2022) distill this empirical literature and based on this distillation propose a number of recommendations for modifications to the AMPD (Clark & Watson, 2022; Table 3).

The approach used by Clark and Watson (2022) that leads to their recommendations for modification of the AMPD was twofold: (a) to review and examine stand-alone structural analyses of the PID-5–relying heavily on two previous meta-analyses of the PID-5 facet factor analytic structure—Somma et al. (2019) and Watters et al. (2019)—and (b) to conduct an examination and review of the joint structural analyses of the PID-5 with other models (and measures thereof) of personality and personality psychopathology, including the five-factor model, the Computerized Adaptive Test of Personality Disorder, and the HEXACO. This approach was meant to first identify the internal structure of a measure designed to assess the AMPD domains and its constituent facets and then to map this structure onto the broader "nomological net" of personality and psychopathology.

We have no concern with this framing of the literature or their subsequent interpretation of it. Our reservations arise with the limited nature of the information source that they frame. Clark and Watson (2022), in essence, are proposing substantial revisions to the AMPD *model* based entirely on a single *method* of measurement (i.e., a self-report of the target of observation). Although Clark and Watson (2022) do forewarn of this problem (the "vast majority

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of the literature is based on the PID-5"), and consider the AMPD and the PID-5 essentially the same, stating that, "the measure *is* the model," and acknowledge that "this is not ideal," they nonetheless argue most broadly for modifications to the *model* and not specifically to the instrument designed to measure that model. One wonders if researchers and clinicians in the field of personality psychopathology are prepared to accept the large-scaled modifications to the AMPD based on an empirical literature that is composed of its measurement by single instrument, especially a self-report instrument, a method of measurement that many researchers regard as problematic when assessing psychopathology. The PID-5 has been virtually the only measure that has been used by investigators to examine the AMPD; this is no fault of Clark and Watson (2022) as they readily acknowledge in their paper—the PID-5 has been used almost exclusively to examine the AMPD. In contrast, there are very few studies that have use other methods of measurement of the AMPD, for example, the informant version of the PID-5 (PID-5-IRF; Markon et al., 2013) and various interview forms. Clark and Watson (2022) state clearly that because of this they did not include studies that use this type of assessment.

Nonetheless, and notwithstanding the arguments of Cronbach and Meehl (1955; see also Hogan & Nicholson, 1988) that measurement (scale) validation is construct validation (a camp of thought to which Clark and Watson (2022) belong and to which we also subscribe), we consider the reliance on a single method of measurement to modify a broader model of personality psychopathology more problematic than is acknowledged by Clark and Watson (2022). And we wonder if in that regard their recommendations for revision to the AMPD are premature. Again, to be fair, Clark and Watson (2022) do indicate that their focus is on structural analyses of the PID-5 and the latent domain factors and the constituent facets of each of these domains. Yet, after more than 10 years of research on the AMPD, is it sufficient to make recommendations for modifications to this model based on structural and correlational analyses of a single self-report instrument eschewing, in essence, potential contributions using other methods of assessments.

Similarly, they do not consider alternative epistemologies of conceptualization (e.g., neurobiological or cognitive/behavioral markers) and other critical sources of information (e.g., clinician input) to delineate the fundamental structure and the clinical utility of this model. It is our position that until evidence emerges from these additional sources of information, it is premature to propose substantial modifications to the AMPD, particularly because such revisions if implemented could have a large impact on the direction of future research in the field.

It is an unfortunate state in our field that the rapidly ascending predominant model of personality psychopathology is almost exclusively validated on a single self-report measure completed by the target of observation, sacrificing more broad-based examination of the AMPD constructs. Most problematic, is that self-report measure is subject to potential response bias that if left unchecked, compromises the validity and reliability of research that uses such data. Clark and Watson (2022) are correct in noting that future researchers need to assess the newly proposed model (the AMPD 5.1) with alternative forms of measurement including informant reports and interview-based assessment methods. But this cautionary advice comes *after* their model modifications are proffered. The worst-case scenario here is that the current AMPD exhibits problems in its structure and content that Clark and Watson (2022) argue "need fixing" may in fact be an epiphenomena, or a by-product of the *method of measurement* examining the structure of this model and not its *substantive* core structural problems. Because "the model is the measure", however, there is no way to parse these potential alternative possibilities. In the end, their proposed modifications are banking on a methodology that Clark and Watson (2022) readily admit is a limitation but seem to logically presume that the *measurement* method is not the limitation of the model; rather, they believe the limitations reside within the substantive core, structure, and breadth of the *model*. It may be argued, of course, that it is unlikely that self-report measurement bias could account for *all* of the observations made by Clark and Watson (2022) that lead to their proposed modifications of the AMPD. And, in fact, one of the few studies that compare the factor structure congruence between the self-report and informant-report versions of the PID-5 showed reasonable congruence, although it was by no means structurally isomorphic (Markon et al., 2013). Nonetheless, those with personality pathology have a tendency—depending on the assessment context and/or level of self-awareness of the self-reporting target—to either underreport or overreport or respond inconsistently to certain personality traits. These response bias patterns have been shown to attenuate external validity and even alter its underlying model structure (Dhillon et al., 2017; McGee Ng et al., 2016). Moreover, it has been demonstrated that mean-level discrepancies between the self-report PID-5 and its informant version (the PID-5-IRF) increased as general personality pathology severity scores also increased (Carnovale et al., 2019). It has also been shown that self-report PID-5 scale scores are strongly impacted by response bias, whereas the PID-5-IRF were not so greatly impacted (Quilty et al., 2018). The results from these studies highlight the potential perils of relying on a single source of assessment method to propose significant model modifications.

As proposed earlier, we also thought the proposed modifications to the AMPD to be problematic without including direct clinician feedback; such participation was incorporated in the initial development of the AMPD and played a critical role in enhancing the clinical utility of the model (Skodol et al., 2014).. Clark and Watson (2022) propose including a number of additional facet traits to the revised AMPD model to shore up the factor structure of the PID-5 and to make the "AMPD more comprehensive and useful clinically." These additions may indeed increase model comprehensiveness, but the foundation of any claim of clinical usefulness should at least in part be based on clinician input. Examining the structure of the PID-5 on its own is of course an important step toward informing modifications to the AMPD, but for the revised model to maximize its clinical utility, we believe it should be more than a statistical exercise of moving facet traits from one domain to another to improve mathematical fit. Neither do we think that facet traits, not previously a part of the AMPD, now be added in order "to enhance comprehensiveness" based only on evidence that such traits were incorporated in other personality models (also measured with self-report). Rather, we think it important that the added clinical value to the restructuring of the model and additions to it require direct clinician input (Morey et al., 2013). We believe that to propose a model without such input could potentially compromise the clinical utility of such instrument.

Clark and Watson (2022) indicate at the start of their article that there are a number of topics that they do not cover in their review, including not giving any consideration to the pathological traits that form the remaining six personality disorders (PDs) in the AMPD. Why not? These PDs were retained in the AMPD presumably in part

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because they had at least some scientific merit and most certainly some clinical utility. Despite the hope of many personality disorder researchers, the prospect of a purely dimensional model remains mostly aspirational at least for the foreseeable future (e.g., witness the failure of the American Psychiatric Association to include the AMPD or otherwise to elevate dimensional models to the main text of the recently released *DSM–5-TR*). One can examine the collective unity of these PD trait constellations and their clinical relevance to a revised alternative structure of the AMPD and not use the mostly flawed categorical diagnostic method to diagnose PDs. Some attention to them, we believe, serves the purpose of engaging clinicians to the familiar and further enhances clinical utility.

Clark and Watson (2022) also indicate that they did not consider neurobiomarkers in their proposed revisions due to a lack of research in the field; again, the lack of research here is no fault of their own. Although we agree that there is a dearth of research on the AMPD traits (see, however, Hyatt et al., 2021), we believe that some consideration be given to the fact that some of these phenotypic traits in the AMPD may have potentially unique neurobiological (or cognitive/behavioral) markers. To this end, we think it important to examine the association between the phenotypic traits included in the AMPD and their possible association with neurobiological markers. We believe this is especially important in the context of the current neuroscience zeitgeist and would perhaps enhance the perception of the scientific integrity of the model. In streamlining the AMPD with their modifications, Clark and Watson (2022) may have unwittingly provided a potentially improved and perhaps more veridical representation of various psychopathological trait domains that could afford a clearer path, if one exists, for potential markers for these domains.

It could be argued that examining neurobiological correlates or other methods of assessment can be done after the PID- 5.1, and the newly proposed universe of domain and assigned facet traits are examined and tested; but again, why propose a new model based on a self-report measurement alone without any consideration to potential neurobiological/neuro-cognitive components that may underlie these modified domains and facets? And, why propose a new model if certain components of it have not been vetted by those clinicians who would use it?

In sum, Clark and Watson (2022) have provided a set of proposed modifications to the AMPD based on the PID-5 internal structure and it's correlates with traits measured by other models of personality. They present reasonable argument and structural model evidence upon which they base their model modifications, proposing a reconfiguration of the AMPD and its underlying structure. They also propose an expansion of the overall domain of personality psychopathology to the AMPD. This is all done, however, within the narrow confines of self-report measurement. We believe that it is premature to propose a revised model that could influence the direction in the field to a considerable degree based exclusively on the accumulated analyses of a single self-report instrument and its correlates. At best, they have offered a basis for a revised and expanded PID-5.

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