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A MODEL-BASED STRATEGY FOR INTERFACING TRAITS OF THE DSM-5 AMPD WITH NEUROBIOLOGY

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The *DSM-5* alternative model for personality disorders (AMPD) groups traits into domains based on factor analyses of self-report data. AMPD traits may need to be configured differently to interface with neurobiology. Focusing on biobehavioral risk for externalizing problems in 334 adults, the authors used structural modeling to evaluate how well alternative configurations of AMPD traits, operationalized using the Personality Inventory for *DSM-5* (PID-5), interface with neural indicators of externalizing liability. A model specifying two correlated factors defined by traits within the PID-5 Disinhibition domain and brain-response indicators of externalizing proneness exhibited inadequate fit. However, a model using PID-5 traits with better coverage of biobehavioral externalizing liability evidenced good fit. Scores on this PID-5 trait factor, termed "PID-5 Externalizing Proneness," converged well with criterion measures of externalizing proneness and diverged from measures of threat sensitivity. Findings illustrate how AMPD traits can be configured for use in investigations of biobehavioral risk for psychopathology.

Keywords: psychophysiology, disinhibition, externalizing, EEG, P300, cross-domain, PID-5, AMPD

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013) provides an alternative model for personality disorders (AMPD) that characterizes these conditions in terms of scores on pathological trait dimensions as opposed to discrete categorical groupings. The Personality Inventory for *DSM-5* (PID-5; Krueger,

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Derringer, Markon, Watson, & Skodol, 2012) was developed to operationalize the trait constructs of the AMPD in the modality of self-report. The PID-5 includes 25 trait scales organized into five broad domains identified on the basis of factor analysis: Negative Affect, Detachment, Antagonism, Disinhibition, and Psychoticism.

The organization of the lower order traits of the AMPD was established empirically through factor analysis of the PID-5's 25 trait scales (Krueger et al., 2012). As report-based dimensions, the higher order factors (domains) of the PID-5 parallel higher order dimensions of personality in the five-factor model and psychopathology-spectrum dimensions within the Hierachical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017). As such, the PID-5's organizational structure is helpful for relating the AMPD system for personality pathology to influential models of normative personality and general psychopathology. However, it is also important—in light of growing interest in multimodal assessment of psychopathology-relevant characteristics (e.g., Kozak & Cuthbert, 2016; Kwako, Momenan, Litten, Koob, & Goldman, 2016; Volkow et al., 2018)—to consider how traits of the AMPD might be aggregated to connect effectively with clinically relevant *biobehavioral* dimensions such as threat sensitivity (Yancey, Venables, & Patrick, 2016), reward sensitivity (Bowyer et al., 2019), and inhibitory control (Venables et al., 2018). Doing so would facilitate efforts to incorporate neurobiological measures into AMPD-oriented assessments and establish biologically informed interventions for mental health problems (Patrick, Iacono, & Venables, 2019a; Perkins, Latzman, & Patrick, 2019).

With this in mind, the current study focused on how traits of the AMPD, assessed via the PID-5, might be configured to interface effectively—in quantitative-measurement terms—with neural indicators of biobehavioral liability for one major spectrum of psychopathology—the externalizing spectrum (see, e.g., Hicks et al., 2007; Yancey, Venables, Hicks, & Patrick, 2013). This spectrum encompasses unrestrained-erratic forms of personality pathology and other impulse-control problems (e.g., conduct disorder, attention deficit disorder, substance use disorders; Krueger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000), which are theorized to involve deficits in neurocognitive systems for inhibitory control (Patrick, Fowles, & Krueger, 2009; Young et al., 2009). Specifically, employing the Externalizing Spectrum Inventory (ESI) model (Krueger, Markon, Patrick, Benning, & Kramer, 2007) as a conceptual-empirical referent, we used confirmatory factor analysis (CFA) to evaluate the fit of alternative models seeking to account for covariance within and between indicators of externalizing proneness from modalities of self-report (i.e., PID-5 traits) and neural reactivity (i.e., variants of P3 brain response). Our aim was to identify a set of trait scales from the PID-5 that defined a coherent factor compatible with ESI externalizing proneness, and that yielded good fit when modeled alongside a correlated externalizing-proneness factor defined by neural-response indicators.

As a starting point for this work, we focused on the set of PID-5 traits most ostensibly relevant to externalizing problems—those assigned to the PID-5 Disinhibition domain. We then used CFA procedures to progress from this set of traits to a revised set that fulfilled the above-noted criteria (i.e.,

factor coherency, compatibility with ESI externalizing proneness, good fit when modeled alongside a neural dimension of externalizing proneness).

BIOBEHAVIORAL CONCEPTUALIZATION AND QUANTIFICATION OF EXTERNALIZING PRONENESS

Evidence from twin research studies has demonstrated that a strongly heritable latent dispositional factor influences both externalizing disorder symptoms and disinhibitory personality traits (Krueger et al., 2002; Young et al., 2000). Other work has shown that this externalizing liability factor exhibits systematic associations with neural (i.e., diminished P3 brain response; Nelson, Patrick, & Bernat, 2011; Patrick et al., 2006) and cognitive-performance (Brennan & Baskin-Sommers, 2018; Young et al., 2009) variables, largely as a function of shared genetic influences. P3 brain response, a positive-going event-related potential (ERP) component elicited by rare or otherwise salient stimuli, is a well-established neural indicator of proneness to substance problems (Begleiter, Porjesz, Bihari, & Kissin, 1984; Brigham, Herning, & Moss, 1995) and externalizing disorders more broadly (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). The P3 brain response's relationship with externalizing disorders reflects shared genetic variance (Hicks et al., 2007; Joyner et al., 2020; Yancey et al., 2013), and it prospectively predicts later emergence of such disorders (Berman, Whipple, Fitch, & Noble, 1993; Iacono, Carlson, Malone, & McGue, 2002). The implication is that the latent liability for externalizing problems is an individual difference characteristic that is expressed in measurement domains of neurophysiology and cognitive-task performance as well as in personality trait and clinical symptom domains.

The ESI (Krueger et al., 2007; Patrick et al., 2013a) was designed to assess, in the modality of self-report, traits and problem behaviors indicative of this latent externalizing liability. The ESI includes 23 subscales tapping various dispositional and symptomatic facets of externalizing proneness, all of which load (factor loadings refer to the degree to which a given variable is representative of the latent construct) substantially (.45 to .91) onto a broad factor. Variations along this latent factor reflect individual differences in general externalizing proneness as reflected in undependability, (lack of) planful control, maladaptive impulsivity, impatience, alienation, angry aggressiveness, dishonesty, and thievery (Krueger et al., 2007; Patrick et al., 2013a). Yancey et al. (2013) presented evidence that scores on this general externalizing factor, quantified using items from ESI subscales loading most strongly and specifically onto it, (a) correlate substantially with clinical-externalizing symptomatology, largely as a function of shared genetic variance, and (b) correlate robustly with reduced P3 brain response, entirely as a function of genetic variance in common with externalizing symptomatology. The implication is that scores on the general externalizing factor of the ESI provide an effective index of biobehavioral liability for externalizing psychopathology.

Consistent with this view, Venables et al. (2018) showed that an item-based scale measure of ESI general externalizing proneness, and other scales

harmonized with it (Brislin, Drislane, Smith, Edens, & Patrick, 2015; Hall et al., 2014; as evidence for harmonization, see Drislane & Patrick, 2017), loaded onto a latent factor that cohered with other factors defined by neural (variants of P3 response) and behavioral (task-derived measures of cognitive control) to form a multimodal externalizing proneness factor. As such, the general externalizing factor of the ESI and scale indicators of this general factor provide a useful referent for considering how best to interface traits from the *DSM-5* AMPD with neurophysiological indicators of weak inhibitory control such as reduced P3 amplitude.

DISINHIBITION IN THE PERSONALITY INVENTORY FOR *DSM-5*

In the initial publication reporting on the development of the PID-5 (Krueger et al., 2012), the composition of its five broad trait domains was delineated by means of an exploratory factor analysis (EFA) of the inventory's 25 facet scales. This analysis resulted in the domain of Disinhibition being defined by lower order traits of irresponsibility, impulsivity, distractibility, rigid perfectionism, and risk taking (Krueger et al., 2012). Of note, the EFA results demonstrated appreciable cross-loadings for the traits of rigid perfectionism and risk taking onto factors corresponding to the PID-5's other four domains (*M* cross-loadings = |.32| and |.25|, respectively), approaching their loadings onto the factor corresponding to Disinhibition (–.38 and .31). Watters and Bagby (2018) reported findings consistent with these in a recent meta-analysis of results from 14 studies that examined the internal factor structure of the PID-5's 25 scales. Taken together, these findings suggest that rigid perfectionism and risk taking do not cohere as strongly and cleanly with traits of impulsivity, irresponsibility, and distractibility as these latter traits do with one another.

Another issue pertains specifically to the trait of risk taking. When defined, as it is in the PID-5, in terms of inclination to approach versus avoid activities/situations involving danger or risk, there is evidence that this trait relates more to biobehavioral threat sensitivity (theorized to underlie proneness to fear disorders; Yancey et al., 2016) than to biobehavioral inhibitory control (presumed to underlie proneness to externalizing problems). For example, scales indexing thrill/adventure-seeking and tolerance for uncertainty load onto a general fear/fearlessness dimension that is associated with variations in physiological threat-reactivity (Kramer, Patrick, Krueger, & Gasperi, 2012; Vaidyanathan, Patrick, & Bernat, 2009; Yancey et al., 2016); scores on this fear/fearlessness dimension relate only weakly to scores on the general externalizing factor of the ESI (see Nelson, Strickland, Krueger, Arbisi, & Patrick, 2016) and are unrelated to P3 brain response (Yancey et al., 2016). In other work, Strickland, Drislane, Lucy, Krueger, and Patrick (2013) reported that the PID-5 Risk Taking scale correlated more strongly (~.6) with a scale measure of boldness (which indexes threat sensitivity, in reverse; see Kramer et al., 2020; Patrick et al., 2019b) than it did with a measure of the ESI's general externalizing factor (~.4). These findings suggest

that a somewhat different configuration of PID-5 traits may be needed to connect the AMPD with biobehavioral externalizing liability and neural indicators of this latent liability factor.

THE CURRENT STUDY

The structure of the PID-5 Disinhibition domain has been thoroughly examined in modeling analyses employing the PID-5's 25 self-report trait scales (Watters & Bagby, 2018). However, the degree to which PID-5 Disinhibition interfaces effectively with externalizing proneness as indexed by neurophysiological (brain response) indicators (Patrick et al., 2013a; Venables et al., 2018) has not been evaluated. To address this gap, the current study utilized structural equation modeling analysis in a novel way to examine how well traits assigned to the PID-5 Disinhibition domain interface as a set with known neurophysiological indicators of proneness to externalizing problems, and whether a somewhat different set of PID-5 traits might provide a more effective interface.

Specific aims and hypotheses of the current study were as follows.

Aim 1. Use a structural-modeling approach to evaluate how well traits assigned to the PID-5 Disinhibition domain interface, as a set, with known neurophysiological indicators of biobehavioral externalizing liability (i.e., variants of P3 brain response; Hicks et al., 2007; Nelson et al., 2011; Yancey et al., 2013). In the service of this aim, we specified a correlated two-factor model that included the PID-5 Disinhibition domain scales as indicators of a trait-scale factor and variants of P3 brain response as indicators of a neurophysiological externalizing proneness factor (cf. Venables et al., 2018),[1] and employed two criteria to evaluate the model: (a) overall goodness-of-fit, as evidence for the model's ability to effectively account for covariance among indicators of the two types; and (b) balanced, robust loadings of indicators on each model factor (i.e., strong factor saturation) and high factor reliability (McNeish, 2018; Zinbarg, Revelle, Yovel, & Li, 2005), as evidence for coherence and stability of the model factors. (Factor reliability provides an index of the degree to which a latent factor is represented by its observable indicators. For example, a latent factor model where only one or two indicators exhibit very high loadings and other indicators exhibit very low loadings would produce a low factor reliability estimate.) Specific hypotheses pertaining to this aim were that: (1a) Less than adequate fit and uneven loadings would be evident for the correlated two-factor model when the five PID-5 Disinhibition domain scales were utilized as indicators of the trait-scale factor, but (1b) improved fit, more balanced loadings, and higher factor reliability would be evident when

1. We used a correlated-factor modeling approach because we sought to use the neurophysiological factor as a fixed referent (anchor) for evaluating the convergence of a self-report externalizing factor, defined by different PID-5 scale sets, with biobehavioral externalizing proneness. The variants of P3 we used as indicators of the neurophysiological factor were those shown by Venables et al. (2018) to interface effectively with previously validated scale measures of externalizing proneness.

PID-5 traits of rigid perfectionism and risk taking were omitted as indicators of the trait-scale factor.

Aim 2. Evaluate whether model fit, factor saturation, and factor reliability can be further improved by incorporating other PID-5 scales corresponding to facets of the ESI's general externalizing factor. In relation to this study aim, we hypothesized that: (2a) Addition of PID-5 Deceitfulness (as an index of dishonesty), Suspiciousness (as an index of alienation), and/or Hostility (as an index of angry aggressiveness) would improve model fit; and (2b) balanced indicator loadings (as evidenced by high factor saturation and reliability) would be evident for this revised trait-scale factor. The grounds for this hypothesis were that (a) PID-5 Impulsivity, Irresponsibility, and Distractibility scales provide effective coverage of the undependability, maladaptive impulsivity, (lack of) planful control, and impatience facets of ESI externalizing proneness, but not its dishonesty, alienation, or angry aggressiveness facets (Krueger et al., 2007; Patrick et al., 2013a), and (b) PID-5 Deceitfulness, Suspiciousness, and Hostility contain items of greatest conceptual relevance to this missing content.[2]

Aim 3. Compare associations of PID-5 Disinhibition domain scores and the trait-scale factor resulting from the foregoing analyses, termed PID-5 Externalizing Proneness, with (a) scores on an ESI-based scale measure of externalizing proneness (the ESI Disinhibition scale; Patrick et al., 2013a), and other scales developed to harmonize with it; and (b) scale measures of dispositional threat sensitivity, which are expected to be largely independent of externalizing liability (Kramer et al., 2020; Nelson et al., 2016; Venables et al., 2017). As regards this aim, our hypothesis (3) was that scores on the PID-5 Externalizing Proneness factor, compared to PID-5 Disinhibition domain scores, would correlate significantly more strongly (according to Steiger's [1980] *Z*-test statistic) with scores on previously validated scale measures of externalizing proneness, and more weakly with scales measures of dispositional threat sensitivity.

METHOD

PARTICIPANTS

Participants utilized in the current analyses were from two studies involving mixed community/college samples that employed overlapping test protocols (for details, see Bowyer et al., 2019, and Venables et al., 2018),[3] resulting in a combined *N* of 334 for analyses (46.7% female) after exclusion of 37 individuals with excessive missing data (14 with scores for fewer than two of the four brain response indicators utilized in the modeling analyses described below, 18 with scores for fewer than three of the PID-5 trait scales utilized in these

2. None of the PID-5 scales includes items pertaining specifically to stealing/theft, so it was not possible to evaluate whether inclusion of a scale indicator of this ESI externalizing facet would improve the fit of the correlated two-factor model.

3. Results pertaining to associations of PID-5 traits with brain-response measures have not previously been reported.

analyses, and 5 with scores for fewer than two of the four brain indicators and fewer than three of the PID-5 trait scales). Test participants were selected from a larger screening pool administered the 20-item ESI externalizing proneness ("Disinhibition") scale (see next subsection); scores on this measure were used to ensure good representation of individuals with low, moderate, and high levels of externalizing proneness among those tested (cf. Nelson et al., 2011). Participants in the analysis sample were 20.73 years of age on average (*SD* = 4.1, range = 18–47) and self-identified as 79.6% White, 12% African American, 3.3% Asian, and 1.5% other, with 3.6% declining to specify.

Procedures for data collection were approved by the Institutional Review Board of Florida State University, and all participants provided informed written consent prior to commencement of testing. Remuneration was provided either in the form of course credit or payment of $10/hour.

INDICATORS OF THE SELF-REPORT DISINHIBITION FACTOR

The Personality Inventory for *DSM-5* (PID-5; Krueger et al., 2012) assesses maladaptive personality traits according to the dimensional system provided in Section III of the *DSM-5*. The PID-5 contains 220 items organized into 25 trait scales, grouped in turn into five broad domains: Negative Affect, Detachment, Antagonism, Disinhibition, and Psychoticism. Items of the PID-5 are answered using a 4-point response format (*False, somewhat false, somewhat true, True*), and responses were assigned numeric codes (from 0 to 3, respectively) after first reversing negatively worded items. For the current work, scores were averaged across items within each trait scale to yield a mean item score for each scale. Scores for the Impulsivity, Irresponsibility, Distractibility, Risk Taking, Rigid Perfectionism, Deceitfulness, Suspiciousness, and Hostility scales were used as indicators of PID-5 Disinhibition in a series of confirmatory factor analyses; internal consistencies (Cronbach's αs) for these trait scales ranged from .70 (Suspiciousness) to .92 (Risk Taking), with a median value of .88.

INDICATORS OF THE NEUROPHYSIOLOGICAL DISINHIBITION FACTOR

Four measures of P3 brain response were derived from three cognitive performance tasks that have been used in prior investigations of neural response differences associated with externalizing proneness (Nelson et al., 2011; Venables et al., 2018). One P3 measure was taken from a version of the rotated heads "oddball" task (Begleiter et al., 1984; Iacono et al., 1999) in which participants were presented with three types of stimuli, each for 100 ms, that differed in frequency. The most frequent of these, a simple oval shape occurring on 70% of trials, was viewed without any response. The remaining 30% of trials were evenly divided between target "head" stimuli (ovals with schematic nose and ear) requiring a button press response, and "novel" color-picture stimuli requiring no response (for further detail, see Nelson et al., 2011). The P3 response to target stimuli within this task was used in the current analyses.

Two other P3 measures were derived from a version of the flanker discrimination task (Eriksen & Eriksen, 1974) in which participants indicated,

with a left or right button press, the direction of the central arrow within a string of five arrows; on 50% of trials, the central arrow appeared in the same direction as the flanking arrows (e.g. ">>>>>"), whereas on the other 50% it appeared in the opposing direction (e.g. ">><>>"; for further details, see Weinberg, Kotov, & Proudfit, 2015). The two P3 measures from this task were the P3 response elicited by arrow-string stimuli and the P3 that followed the commission of errors. The fourth P3 measure was derived from a choice-feedback task in which participants selected one of two "door" stimuli and received feedback afterward indicating either a gain or a loss of money (for further detail, see Foti & Hajcak, 2009). The P3 response to feedback stimuli in this task was used in the current analyses.

CRITERION MEASURES

Convergent Validity Criteria. Two scale measures of the construct of externalizing proneness, conceptualized in biobehavioral terms (see Patrick et al., 2019a), were used to evaluate the convergent validity of PID-5 Disinhibition domain scores compared with scores on the PID-5 Externalizing Proneness factor resulting from our modeling analyses. One was the general Disinhibition scale of the ESI (Patrick et al., 2013a; α in current sample = .86), which consists of 20 items answered using the same 4-point response format as the PID-5. A second measure of externalizing proneness was derived from scores on the Multidimensional Personality Questionnaire (MPQ; Tellegen & Waller, 2008) in accordance with the work of Brislin et al. (2015; α = .79). Responses on the MPQ were coded as either 0 = *False* or 1 = *True*.

Discriminant Validity Criteria. Scores for a scale measure of dispositional threat sensitivity, the 44-item Trait Fear (TF-44) inventory (Kramer et al., 2020), were available for approximately two thirds of the study sample (n = 213; scale α = .96 for this set of participants). The TF-44 was developed to index the common factor underlying various trait-scale measures of fear and fearlessness that predict physiological responsiveness to threat (Kramer et al., 2012; Vaidyanthan et al., 2009). Items of the TF-44 are answered using the same 4-point response format as the ESI Disinhibition and PID-5 inventories (i.e., *False*, *somewhat false*, *somewhat true*, *True*) and are coded in the same manner (i.e., 0–43). A second measure of dispositional threat sensitivity, the MPQ-based Boldness scale (Brislin et al., 2015), was available for the full study sample. Scores on this scale, which correlate to a strong negative degree with Trait Fear scores (r = –.75; Brislin et al., 2015), were reversed for purposes of the current work to make higher scores indicative of higher threat sensitivity.

PROCEDURE

Following provision of informed written consent, participants completed questionnaire measures while sensors were placed for purposes of EEG data collection. Participants then completed the three above-mentioned cognitive tasks (oddball, flanker, choice-feedback). The sequence and timing of stimuli for each task were controlled using E-Prime software (MEL Software, Inc.), and

neurophysiological data were recorded concurrently using an EEG amplifier system and data acquisition software supplied by Neuroscan, Inc. Participants completed the study tasks while seated in a comfortable recliner at a distance of approximately 1 m from a 21-inch computer monitor on which task stimuli appeared. After completing the three tasks, participants were debriefed and compensated with their choice of either $10/hour or course credit.

PHYSIOLOGICAL RECORDING AND DATA REDUCTION

Raw EEG activity was recorded using a 128-channel elastic cap (Neuroscan Quik-Cap). Electrodes were Ag-AgCl sintered and placed according to Neuroscan's nonstandard layout (NSL) system. To allow for correction of eye-movement artifacts, vertical and horizontal electro-oculographic (VEOG, HEOG) activity were recorded from electrodes placed above and below the left eye (VEOG) and adjacent to the outer canthi of the left and right eyes (HEOG). Electrode impedances were monitored and kept below 10 kΩs. During testing, continuous EEG activity was recorded at a sampling rate of 1000 Hz using the midline central electrode site as a reference and applying a .05–200 Hz bandpass filter.

Following completion of testing, the EEG data were re-referenced to the average activity from left and right mastoid sites and parsed into epochs of –1000 to 2000 ms surrounding P3-eliciting events in each task (i.e., target stimuli in the oddball task, arrow stimuli and erroneous button presses in the flanker task, feedback stimuli in the choice-feedback task). Epochs were then corrected for eye-movement artifacts using the algorithm developed by Semlitsch, Anderer, Schuster, and Presslich (1986) and exported to Matlab (Mathworks, Inc.) for downsampling to 128 Hz using the Matlab resample command, which applies an antialiasing filter. Trials on which signal activity exceeded ±75μV during the 3000-ms epoch window were excluded from analyses.

For each task, epochs were averaged across trials, and mean activity from 200 ms prior to the event of interest (i.e., stimulus presentation or erroneous button press) through to its onset was subtracted from the epoch average. Amplitude scores were derived from the average baseline-corrected waveform for each P3 measure, using post-onset windows and electrode sites as follows: oddball target P3—peak level reached during window of 300 to 625 ms, at NSL site 66 (analogous to 10–20 site Pz); flanker stimulus P3—peak between 300 and 525 ms, at site 61 (/FCz); flanker error P3—peak between 250 and 650 ms, at site 66 (/Pz); feedback stimulus P3—peak between 300 and 450 ms, at site 64 (/CPz).

STATISTICAL ANALYSES

We began by performing a CFA to evaluate the fit of a correlated two-factor model in which the five trait scales assigned to the PID-5 Disinhibition domain (Impulsivity, Irresponsibility, Distractibility, Risk Taking, and Rigid Perfectionism) loaded onto a trait-scale factor, and the four P3 response measures loaded onto a separate neurophysiological externalizing proneness factor, with a path specified between the two factors. In what follows, we refer to this model as

the "initial model." We then performed CFAs to evaluate the fit of alternative correlated two-factor models in which (a) Rigid Perfectionism was removed as an indicator of the PID-5 trait factor, (b) both Rigid Perfectionism and Risk Taking were removed, and (c) one or another of the other trait scales deemed to be construct-relevant (i.e., Deceitfulness, Suspiciousness, or Hostility) was included as an indicator of the PID-5 trait factor, along with Impulsivity, Irresponsibility, and Distractibility.

All CFAs were performed using the *lavaan* module (Rosseel, 2012) of the *R* statistical package (*R* Core Team, 2018). To allow for inclusion of all participants ($N = 334$) in the reported CFA models, full-information maximum likelihood estimation was used to impute scores for those with partially missing PID-5 scale data (i.e., values for at least 3, but fewer than all, scale-factor indicators; $n = 6$) or partially missing neurophysiological data (i.e., values for at least 2, but fewer than all, neurophysiological-factor indicators; $n = 43$).[4] For each model, χ^2 and root mean square error of approximation (RMSEA) values were computed as measures of absolute fit, and the comparative fit index (CFI) and Tucker-Lewis index (TLI; Tucker & Lewis, 1973) were computed as measures of incremental fit. As general heuristics for model fit, RMSEA values below .05 indicate good fit, values between .05 and .08 indicate adequate fit, and values exceeding .08 indicate inadequate fit; for CFI and TLI, values exceeding .95 indicate good fit (Hu & Bentler, 1999). As a quantitative index of factor coherence/stability, we computed omega (ω) reliability coefficients (McNeish, 2018; Zinbarg et al., 2005) for the PID-5 Disinhibition factor as specified in the initial model and each alternative model. Higher values of ω reflect greater and more uniform representation of a latent factor across constituent indicators, and lesser measurement error in factor scores.

As a final step, PID-5 Disinhibition domain scores were computed by averaging scores for the five trait scales assigned to this domain, and PID-5 Externalizing Proneness was computed by averaging scores for the set of scales employed in the best-fitting correlated two-factor model. These two omnibus-score variables were then compared in terms of their convergence with criterion measures of externalizing proneness (ESI-Disinhibition, MPQ-Disinhibition) and their predicted lack of association with criterion measures of threat sensitivity (TF-44, MPQ-Boldness-reversed). Steiger's (1980) *Z*-statistic, computed using the *psych* module of *R* (Revelle, 2018), was used to test for significant differences in observed correlations of the two omnibus-score variables with these criterion measures.

RESULTS

SAMPLE DESCRIPTIVE STATISTICS AND CORRELATIONS AMONG LATENT VARIABLE INDICATORS

Table A in the Supplemental Material presents sample *N*s, means, standard deviations, and score ranges along with kurtosis and skewness values for variables used as indicators in the reported CFA analyses, and for criterion

4. Partially missing data in these cases was due to excessive EEG artifact for the pertinent response measures.

TABLE 1. Correlations Among Indicator Variables Used to Define Each Latent Factor

	1	2	3	4	5	6	7
	Neurophysiological measures (*N*s = 297–330)						
1. Rotated-Heads Target P3							
2. Flanker Stimulus P3	**.21****						
3. Flanker Error P3	**.33****	**.40****					
4. Choice-Feedback P3	**.37****	**.18***	**.16***				
	PID-5 trait scales (*N*s = 328-334)						
1. Impulsivity							
2. Irresponsibility	**.55****						
3. Distractibility	**.55****	**.54****					
4. Risk Taking	**.61****	**.36****	**.21****				
5. Rigid Perfectionism	−.06	.03	.08	**−.12***			
6. Deceitfulness	**.50****	**.66****	**.46****	**.34****	.08		
7. Hostility	**.42****	**.47****	**.47****	**.19****	**.31****	**.56****	
8. Suspiciousness	**.17***	**.32****	**.28****	.03	**.29****	**.38****	**.52****

Note. Statistically significant correlations are shown in bold. *$p < .05$. **$p < .001$.

measures used to compare convergent and discriminant validity of PID-5 Disinhibition domain scores and PID-5 Externalizing Proneness scores. All variables utilized in the analyses exhibited univariate normality as indicated by skewness and kurtosis values within ±2.

Table 1 shows correlations among variables used as indicators of latent trait-scale (PID-5) and neurophysiological (P3 response) factors in the different CFA analyses performed. On average, correlations among the five PID-5 trait scales assigned to the PID-5 Disinhibition domain were moderate in magnitude (median $r = .29$). Of note, removal of the Rigid Perfectionism scale from this domain improved the median correlation among scales to .55. In line with previous published research (Nelson et al., 2011; Patrick et al., 2013b), correlations among the brain-physiological indicators of disinhibition (i.e., variants of P3 from different tasks) were also moderate in magnitude (median $r = .27$).

MODELS SPECIFYING CORRELATED PHYSIOLOGICAL DISINHIBITION AND PID-5 DISINHIBITION FACTORS

The initial CFA model specifying a latent neurophysiological externalizing factor defined by the four variants of P3 response and a latent trait-scale factor defined by PID-5 traits of Impulsivity, Irresponsibility, Distractibility, Risk Taking, and Rigid Perfectionism is depicted in Figure 1a. This model exhibited inadequate fit: $\chi^2(26) = 100.72$, $p < .001$, RMSEA = .09, CFI = .87, TLI = .82. The estimated correlation between the PID-5 trait factor and the

neurophysiological externalizing factor in this model was significant, (ψ = –.20, p < .001), with the four P3 response indicators loading quite evenly (range = .44 to .62) onto the neurophysiological factor. By contrast, loadings of the five trait scales on the PID-5 factor of the initial model were markedly imbalanced, with Impulsivity loading close to 1 (λ = .95, p < .001), Rigid Perfectionism loading negligibly (λ = –.05, p = .35), and Irresponsibility, Distractibility, and Risk Taking exhibiting moderate-level loadings (λs = .59, .58, and .63, respectively, ps < .001; see Supplemental Table C, top row). The implication is that the PID-5 trait factor of this model and its observed association with the neurophysiological (P3) Externalizing factor largely reflected the PID-5's Impulsivity trait scale. The ω reliability coefficient for this factor was .71.

Following evaluation of this initial model, two subsequent models were specified in which the Rigid Perfectionism and then Risk Taking scales were removed as indicators of the PID-5 trait-scale factor. Fit statistics for these models are displayed in Supplemental Table B, with standardized loading estimates shown in Supplemental Table C. Removal of Rigid Perfectionism did not enhance model fit, but removal of both Rigid Perfectionism and Risk Taking resulted in improved fit of the model, to acceptable levels: $\chi^2(13) = 34.59$, p = .001, RMSEA = .07, CFI = .95, TLI = .92. Impulsivity, Irresponsibility, and Distractibility demonstrated balanced loadings on the PID-5 trait factor of this model that exceeded their loadings on the PID-5 factor of the initial model (λs = .73, .73, and .75, respectively; factor ω = .78). Loadings for the four P3 response indicators on the neurophysiological factor were comparable to those for the counterpart factor of the original model. The estimated correlation between the PID-5 and neurophysiological factors in this model was nominally lower (ψ = –.18, p = .02) than in the initial model.

Given that the revised model, including Impulsivity, Irresponsibility, and Distractibility, fit the data well, with these indicators showing balanced loadings and the PID-5 trait factor exhibiting improved reliability, Risk Taking and Rigid Perfectionism were excluded as indicators of the latent PID-5 factor in subsequent models, and additional CFA models were fit to the data using Hostility, Deceitfulness, or Suspiciousness as a fourth indicator of the PID-5 trait factor; fit statistics and factor loadings for these models are displayed in Supplemental Tables B and C, respectively. The addition of the Hostility scale as an additional indicator of the PID-5 trait factor was the only configuration that resulted in improved model fit relative to the three-indicator configuration: $\chi^2(19) = 40.53$, p = .003, RMSEA = .06, CFI = .96, TLI = .94. The loading for the Hostility scale on the PID-5 factor in this model (λ = .61, p < .001) was only slightly lower than the loadings for the Impulsivity, Irresponsibility, and Distractibility scales (λs = .73, .73, and .74, respectively), and the loadings for the four P3 indicators on the neurophysiological factor remained well balanced (see Supplemental Table C, bottom row). The estimated correlation between the PID-5 and neurophysiological factors in this model (ψ = –.20, p = .01) slightly exceeded the correlation in the three-scale model without Hostility (ψ = –.18), and the factor reliability estimate also rose, to ω = .80. The model including this four-scale PID-5 trait factor, termed PID-5 Externalizing Proneness, is depicted in Figure 1b.

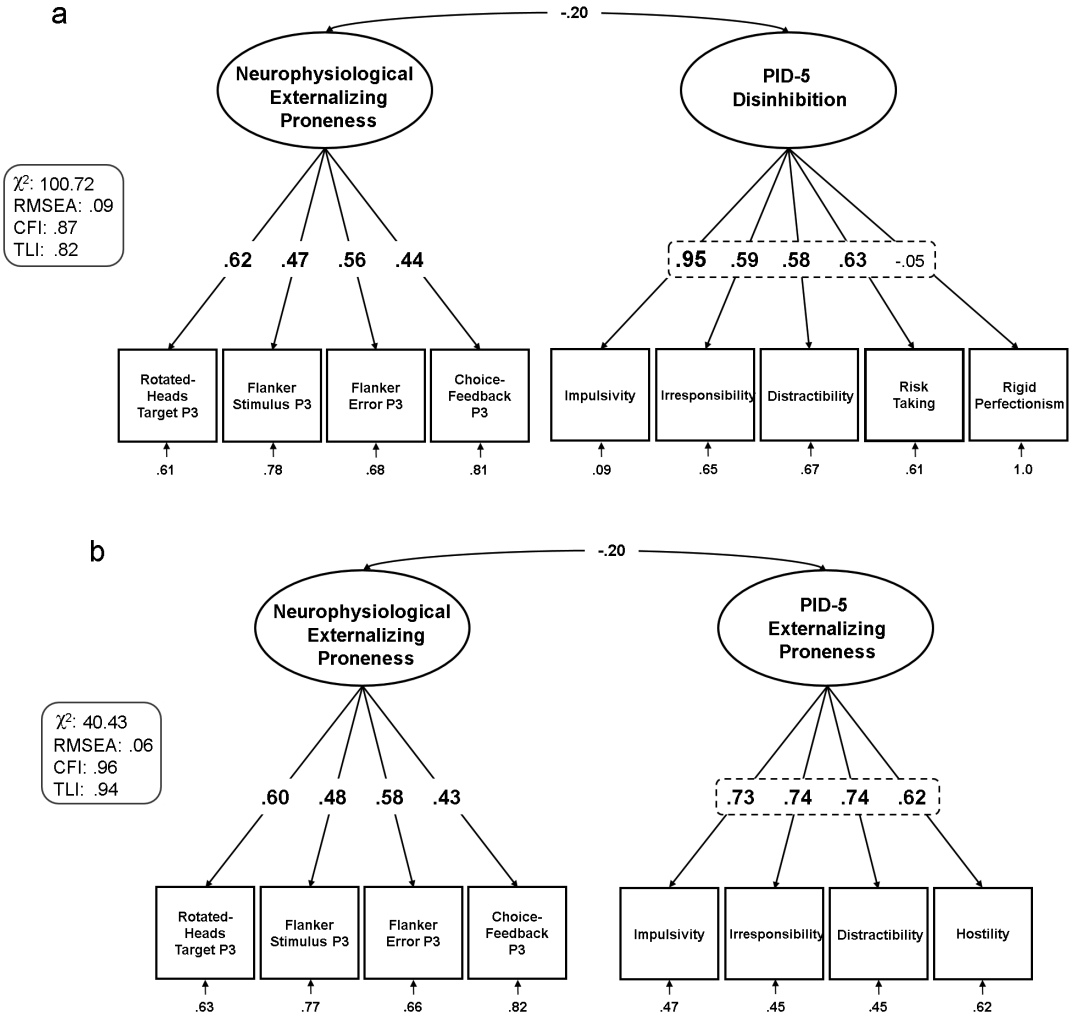

FIGURE 1. Confirmatory two-factor models specifying correlated neurophysiological and PID-5 trait factors. Numeric values within each model depiction are standardized parameter estimates (factor loadings, error terms for indicators, correlation between factors); fit statistics appear in the rounded rectangle to the left of each model. (a) Initial model, with PID-5 trait-scale factor defined by scales currently assigned to the PID-5 Disinhibition domain (i.e., Impulsivity, Irresponsibility, Distractibility, Risk Taking, Rigid Perfectionism). (b) Final, best-fitting model with the trait-scale factor defined by the PID-5's Impulsivity, Irresponsibility, Distractibility, and Hostility trait scales; this factor is labeled "PID-5 Externalizing Proneness" to reflect its effective model-fit with the neurophysiological Externalizing Proneness factor.

CONVERGENT AND DISCRIMINANT VALIDITY OF PID-5 DISINHIBITION AND RECONFIGURED PID-5 EXTERNALIZING PRONENESS SCORES

We hypothesized that a configuration of PID-5 traits that interfaced effectively, in structural-modeling terms, with neurophysiologically (P3 brain response) defined externalizing proneness would outperform PID-5 Disinhibition in terms of both convergent validity (i.e., show higher correlations with established scale measures of externalizing proneness) and discriminant validity (i.e., show lower correlations with established scale measures of a separate trait construct considered orthogonal to trait disinhibition—namely, threat sensitivity). Table 2 shows correlations for PID-5 Disinhibition, computed as the average score for the five scales included in the Disinhibition domain of the PID-5 (column 2) and the average of the four scales used to define PID-5 Externalizing Proneness in the final best-fitting model (column 3), with criterion measures of externalizing proneness and threat sensitivity. Steiger's (1980) *Z* test for dependent correlations is used to probe for significant differences in correlations for the two. The criterion measures of externalizing proneness were the Disinhibition scale of the ESI-BF (Patrick et al., 2013a), created to index the general factor of this inventory, and a scale measure of externalizing proneness composed of items from the MPQ, developed by Brislin et al. (2015) using a construct-based consensus rating approach. The criterion measures of threat sensitivity were a scale measure designed to index variations in dispositional fear versus fearlessness (the TF-44; Kramer et al., 2020) and reversed scores on an MPQ-based scale developed by Brislin et al. (2015) to assess the closely related construct of boldness.

Consistent with prediction, Table 2 shows that positive correlations with the two criterion measures of externalizing proneness were significantly higher, and negative correlations with the two criterion measures of threat sensitivity were significantly lower, for PID-5 Externalizing Proneness as compared to PID-5 Disinhibition (range of Steiger's *Z*s = 3.42 to 8.68, all *p*s < .001).

TABLE 2. PID-5 Disinhibition Domain Scores and PID-5 Externalizing Proneness Scores: Comparative Convergent and Discriminant Validity Coefficients

	PID-5 Disinhibition	PID-5 Externalizing Proneness	Steiger's Z
Convergent validity: *Externalizing Proneness measures*			
ESI-BF Disinhibition (n = 333)	**.52****	**.63****	3.42**
MPQ Disinhibition (n = 324)	**.57****	**.73****	5.95**
Discriminant validity: *Threat Sensitivity measures*			
Trait Fear Inventory (n = 199)	**−.49****	−.04	7.35**
MPQ Boldness–reversed (n = 324)	**−.35****	−.03	8.68**

Note. PID-5 = Personality Inventory for *DSM-5*; PID-5 Disinhibition = item sum-score for the five trait scales situated within the Disinhibition domain of the PID-5; PID-5 Externalizing Proneness = item sum-score for PID-5 Impulsivity, Irresponsibility, Distractibility, and Hostility trait scales; ESI-BF = Externalizing Spectrum Inventory–Brief Form; MPQ = Multidimensional Personality Questionnaire. Statistically significant correlations are shown in bold.
**$p < .001$.

DISCUSSION

The present study utilized structural equation modeling in a novel way to evaluate how effectively the trait scales assigned to the PID-5 Disinhibition domain interface, as a set, with neural (brain response) indicators of biobehavioral externalizing liability. We also sought to identify an alternative set of PID-5 traits that interfaced more effectively with neural indicators of externalizing proneness. To address these aims, we evaluated versions of a correlated two-factor model that included an externalizing proneness factor defined by four variants of P3 brain response along with a PID-5 trait-scale factor. The structural interface between indicators/factors from the two modalities was evaluated based on two criteria, (a) goodness of model fit, and (b) coherence and stability of the PID-5 trait factor, as evidenced by evenness of loadings of indicators on this factor and its omega reliability coefficient. When defined using trait scales assigned to the Disinhibition domain, the PID-5 factor did not interface well with the neurophysiological externalizing factor according to these criteria, but an alternative trait-scale factor defined by three scales from the PID-5's Disinhibition domain (Impulsivity, Irresponsibility, Distractibility) along with one scale from the PID-5 domain of Negative Affect (Hostility) did interface effectively. Further analyses were performed to examine convergent and discriminant validity (in relation to criterion measures of externalizing proneness and threat sensitivity, respectively) for an aggregate of these four scale indicators, termed PID-5 Externalizing Proneness, compared to an aggregate of the scales included in the PID-5's Disinhibition domain.

MODEL-BASED EVALUATION OF THE INTERFACE BETWEEN PID-5 EXTERNALIZING PRONENESS AND NEUROPHYSIOLOGICAL DISINHIBITION

Consistent with Hypothesis 1a and past reports in the literature (Watters & Bagby, 2018), inadequate fit was observed for the initial CFA model in which the trait-scale factor was defined using scales from the PID-5's Disinhibition domain (Irresponsibility, Impulsivity, Distractibility, Rigid Perfectionism, and Risk Taking) as indicators. In addition, loadings on this PID-5 trait factor were severely imbalanced; the factor was primarily defined by the Impulsivity trait scale (loading = .95), with the other trait scales showing markedly weaker loadings, resulting in low factor reliability ($\omega = .70$). Weak association of the Rigid Perfectionism trait scale with other PID-5 Disinhibition scales has been well documented (Watters & Bagby, 2018) and was evident in our initial CFA model, where this trait scale loaded negligibly (–.05) onto the latent PID-5 factor. Moreover, because the Risk Taking scale performs as an indicator of threat sensitivity (Strickland et al., 2013)—a biobehavioral trait dimension orthogonal to externalizing proneness (e.g., Kramer et al., 2020; Nelson et al., 2016; Venables et al., 2017)—inclusion of this trait scale appeared to contribute to the poor fit of the base model (i.e., model fit remained inadequate when only Rigid Perfectionism was dropped). Consistent with Hypothesis 1b, removal of both Rigid Perfectionism and Risk Taking

scales resulted in acceptable model fit, with highly similar loadings of the three remaining scale indicators (Irresponsibility, Impulsivity, and Distractibility) onto the PID-5 factor.

Consistent with study Hypothesis 2a, model fit was further improved by including one of three other PID-5 scales—namely, Hostility—as an indicator of the latent PID-5 trait factor. Inclusion of Hostility improved model fit by providing representation of angry aggressiveness, an attribute that relates strongly to externalizing proneness (Krueger, 1999; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Patrick et al., 2013a) and to neural (i.e., P3 brain response) indicators of externalizing proneness (Patrick et al., 2013b; Venables, Patrick, Hall, & Bernat, 2011). Consistent with study Hypothesis 2b, this PID-5 Externalizing Proneness factor exhibited more balanced loadings across indicators, in contrast to the initial five-indicator model, along with increased factor reliability ($\omega = .80$).

In addition, consistent with study Hypothesis 3, an observed composite score for the four traits that demarcated the Externalizing Proneness factor in this best-fitting model demonstrated significantly improved convergent and discriminant validity compared to a composite of the five scales assigned to the Disinhibition domain. Specifically, removal of Risk Taking and Rigid Perfectionism and inclusion of Hostility significantly enhanced positive correlations with the Disinhibition scale of the ESI, a well-validated measure of general externalizing proneness (Drislane, Patrick, & Arsal, 2014; Patrick et al., 2013a; Yancey et al., 2013). In addition, scores on the PID-5 Externalizing Proneness factor demonstrated significantly stronger associations than PID-5 Disinhibition scores with a harmonized measure of externalizing proneness consisting of construct-relevant items from the MPQ (Brislin et al., 2015, 2017).

Furthermore, in contrast with PID-5 Disinhibition scores, which correlated at moderately significant levels with scales measures of dispositional threat sensitivity (i.e., TF-44, MPQ-Boldness), PID-5 Externalizing Proneness demonstrated negligible associations with these measures. These reduced associations with the dimension of threat sensitivity are consistent with conceptual (e.g., Patrick et al., 2009) and empirical work (e.g., Nelson et al., 2016; Venables et al., 2017) characterizing threat sensitivity as a biobehavioral trait independent from externalizing proneness.

BIOBEHAVIORAL EXTERNALIZING LIABILITY AND THE AMPD

A central point undergirding the current work is that different dimensional organizations of lower order personality traits may be useful for different scientific and applied purposes. The PID-5 provides a dimensional system for maladaptive personality that is broadly compatible with the Five Factor Model (FFM) framework for normative personality. This compatibility affords distinct advantages that have been well documented in conceptual and empirical writings (Gore & Widiger, 2013; Krueger et al., 2012; Thomas et al., 2013). For example, normative FFM traits predict a range of important general life outcomes (Judge, Heller, & Mount, 2002; Malouff, Thorsteinsson, Schutte, Bhullar, & Rooke, 2010; Poropat, 2009) and interface effectively with clinical problems of various types (Malouff, Thorsteinsson, & Schutte, 2005; Widiger

et al., 2019). An FFM-compatible model of personality pathology provides linkages to a vast body of literature on the psychological and behavioral correlates of normative personality traits, and offers a valuable framework for further clarifying associations between normative traits and other forms of psychopathology. The organizational structure of the AMPD is also compatible with an emerging dimensional framework for psychopathology, the HiTOP model (Kotov et al., 2017), which includes higher order spectra corresponding to the AMPD's five trait domains.

However, the goal of relating clinical problems to neurobiological systems and processes may be facilitated by an alternative, but complementary approach to aggregating traits (Perkins et al., 2019)—one that focuses on dispositional constructs that are directly *informed* and *shaped* by biological data (Patrick et al., 2019a). This approach conceives of traits as psychobiological networks or structures (Allport, 1937; Eysenck, 1967) that affect measures in different modalities of response. Viewed in this way, measures from the domain of neurophysiology can serve as *indicators* of latent biobehavioral traits rather than as after-the-fact correlates of traits defined solely by report-based indicators. The use of neurobiological variables as trait indicators can give rise to an alternative scheme for organizing self-report–assessed traits, which can permit them to align better with dimensions defined in part by variables from the neurophysiological response modality (Patrick et al., 2013b, 2019a). Venables et al. (2018) provided an empirical illustration of this in the form of a multimodal measurement model for the construct of inhibitory control as related to externalizing problems, encompassing four indicators each from modalities of self-report (i.e., trait-scale measures) and neurophysiology (variants of P3 brain response), along with four others from the modality of task performance (i.e., behavioral measures from cognitive control tasks). Factors defined by indicators from these different measurement modalities loaded together onto a common higher order factor, interpretable as a cross-modal dimension of inhibitory control capacity.

Drawing on the work of Venables et al. (2018) and utilizing measures from that study along with data from a separate study, the current work provides an illustration of how lower order traits of the AMPD—operationalized using the PID-5—can be configured to interface effectively with known neural indicators of proneness to externalizing problems. Two traits assigned to the existing PID-5 Disinhibition domain had to be dropped, and one trait from a different PID-5 domain (Hostility) had to be added, to form a coherent and reliable PID-5–based Externalizing Proneness factor within the context of a well-fitting model that includes a second, correlated neurophysiological factor. From the perspective of the current work, the lower order traits of the AMPD can serve as building blocks for alternative aggregate dimensions suited to different purposes. Whereas the existing PID-5 domains interface clearly with broad trait dimensions of the FFM and disorder spectra of the HiTOP model, alternative configurations of PID-5 traits may provide a better interface with biobehavioral individual-difference constructs such as weak inhibitory control (externalizing proneness) and threat sensitivity (fearfulness vs. boldness; Kramer et al., 2012; Patrick et al., 2009, 2019b; Venables et al., 2017; Yancey et al., 2016).

What unique utility might there be to an alternative system for aggregating lower order traits of the AMPD into broader dimensions through reference to neurobiological measures? A core advantage is that trait dimensions structured in this way can be expected to covary more strongly and reliably with other measures from the neurobiological modality (for direct evidence of this, see Patrick et al., 2013b; Venables et al., 2018; Yancey et al., 2016). With regard to the current study, for example, the configuration of traits shown to interface effectively with the P3 brain response factor would be expected to correlate more robustly and consistently with other brain-based measures of externalizing proneness—including neuroimaging measures (e.g., Castellanos-Ryan et al., 2014; Foell et al., 2016) as well as other ERP measures (e.g., Hall, Bernat, & Patrick, 2007)—while also relating robustly to disinhibitory clinical conditions (see Figure 2). Improvements in the ability to identify replicable associations between psychological and neural-response measures are greatly needed to permit progress in understanding the role of neurobiological systems/processes in mental illness.

Another important feature of biobehavioral trait operationalizations is that they have proven to be effective for indexing heritable vulnerability to clinical problems. For example, using data for a twin sample, Venables et al. (2017) computed a composite index of externalizing proneness from two trait-scale measures and two P3 response measures and showed that the moderate-level phenotypic relationship between this composite index and observed substance problems was attributable almost entirely (89%) to common genetic variance. In other work, Joyner and colleagues (2020) demonstrated that the covariation between substance use problems and a multimodal (trait-scale/ERP) index of externalizing proneness was attributable mostly to shared heritable variance, whereas this was not the case for the FFM trait of Conscientiousness. Consistent with these findings, longitudinal studies have shown that P3 brain response assessed earlier in life is predictive of the later emergence of substance abuse and other externalizing problems (e.g., Berman et al., 1993; Iacono et al., 2002). These lines of evidence indicate that biobehavioral trait measures can be of value for identifying young individuals at high risk for developing clinical problems of particular types, who are most in need of early specialized assistance to prevent the emergence of such problems. From this standpoint, biobehavioral trait measures can also serve as important referents in longitudinal research studies aimed at elucidating pathways from latent risk to manifest psychopathology (Perkins et al., 2020).

Considering their utility as simultaneous referents for both observed clinical problems and relevant neurobiological mechanisms, biobehavioral trait conceptualizations can serve to bridge the methodological gap between psychophysiological research and assessment of psychopathology within clinical settings. As mentioned previously, psychophysiological measures like the P3 brain response index dispositional risk for the development of externalizing psychopathology (Berman et al., 1993; Iacono et al., 2002); however, practical measurement of such variables in a clinical setting is not always possible. Given these issues of practicality regarding psychophysiological measures in clinical settings, the current study outlines an approach for aligning existing

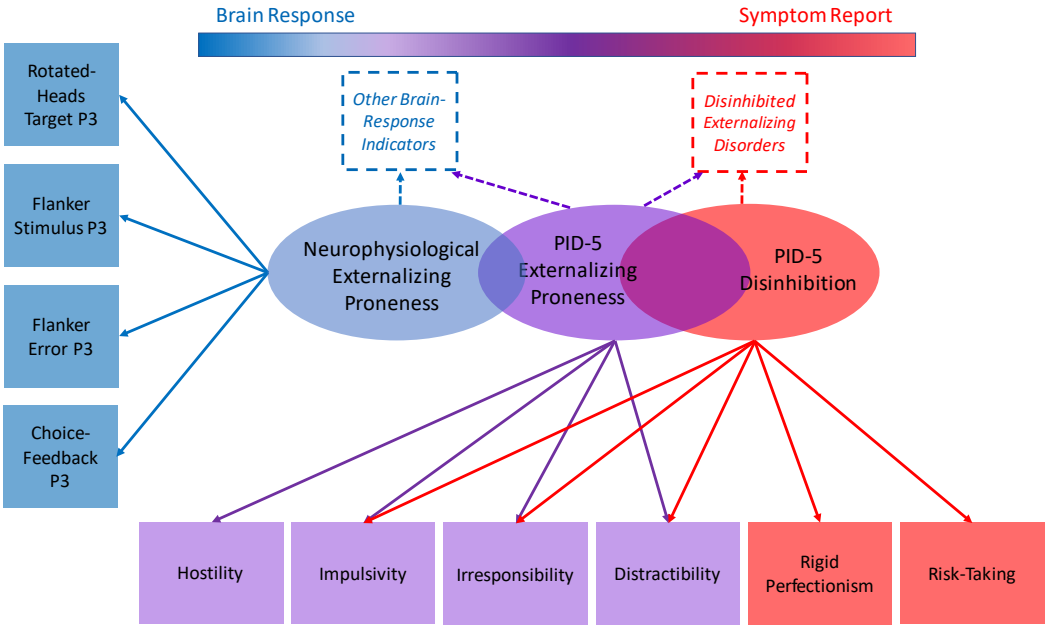

FIGURE 2. Depiction of PID-5 Disinhibition factor, PID-5 Externalizing Proneness factor, and neurophysiological (P3 brain response) Externalizing Proneness factor (represented by ovals); indicators used to model each in the current work (represented by solid rectangles); and their expected relations with disinhibited externalizing disorders and other brain response indicators of externalizing proneness (represented by dashed rectangles). The PID-5 Disinhibition and PID-5 Externalizing factors overlap substantially, reflecting their high interrelationship; the PID-5 Externalizing and Neurophysiological Externalizing factors overlap to a lesser extent, reflecting their lower interrelationship. Solid arrows extend from factors to their respective indicators; PID-5 Impulsivity, Irresponsibility, and Distractibility are indicators of both PID-5 Disinhibition and PID-5 Externalizing, whereas Rigid Perfectionism and Risk Taking are indicators of PID-5 Disinhibition only, and Hostility is an indicator of PID-5 Externalizing only. Dashed arrows extend from factors to variables of other types predicted to correlate with each. Other brain response indicators of externalizing proneness (e.g., error-related negativity [Hall et al., 2007]; affective-anticipatory brain response [Foell et al., 2016]) are expected to correlate with the neurophysiological Externalizing factor, and also with the PID-5 Externalizing factor, given its association with the neurophysiological factor. Disinhibited externalizing disorders (e.g., conduct disorder, antisocial personality, alcohol and drug dependence) are expected to correlate with the PID-5 Disinhibition factor, and also with the PID-5 Externalizing factor, given its association with PID-5 Disinhibition.

scale measures of psychopathology with relevant dimensions of neurobiology to eventually improve existing assessment tools and allow for more adequate measurement of dispositional risk. Systematic efforts to interface the AMPD trait framework with emerging multimodal measurement models for disinhibition and other biobehavioral traits (e.g., threat sensitivity, reward sensitivity,

affiliative capacity; Patrick et al., 2019a), as illustrated in the current work, can enhance the scientific and applied value of both.

LIMITATIONS AND FUTURE DIRECTIONS

Some limitations of the current study warrant mention. One is that an iterative approach—involving the testing of successive models—was used to reconfigure PID-5 Disinhibition to interface effectively with neurophysiological indicators of disinhibition. It will be important to reevaluate the models reported here using data for other samples in order to establish the replicability of the current results. Another is that the participant sample, while balanced in terms of gender, was quite homogeneous with respect to other demographics. Further work of this kind is needed using samples with greater diversity in terms of age and race/ethnicity.

In addition, our work was limited by the use of neural indicators of only one type (i.e., variants of P3 brain response) known to be associated with biobehavioral externalizing proneness. It will be valuable in future work to incorporate neural indicators of other types (e.g., error-related negativity [Hall et al., 2007]; neuroimaging measures of affective-anticipatory response [Foell et al., 2016]) into a multimodal measurement model for this biobehavioral liability construct. Future efforts to interface traits of the AMPD with neurobiology would also benefit from consideration of neural measures that relate selectively to callous-aggressiveness ("meanness"; Patrick et al., 2009), the biobehavioral counterpart to the Antagonism domain of the AMPD trait model. Neural measures of callous-aggressiveness include reduced amygdala activation and reduced early brain-ERP (N170, P200) response to affective face stimuli (fearful faces in particular; Brislin et al., 2018; Brislin & Patrick, 2019; Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008). Work of this kind would help to advance understanding of biobehavioral pathways to distinct forms of externalizing psychopathology marked by deficient inhibitory control versus predatory exploitativeness.

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Supplemental Material

Contents

Table A. Descriptive Statistics for Study Measures

Measure	*n*	Mean	*SD*	Skewness	Kurtosis
PID-5 Trait Indicators					
Impulsivity	329	.91	.75	.65	–.51
Irresponsibility	333	.45	.47	1.29	1.56
Distractibility	334	1.09	.67	.32	–.79
Risk Taking	334	1.46	.64	.12	–.50
Rigid Perfectionism	334	1.11	.64	.24	–.55
Deceitfulness	334	.74	.58	.96	.70
Hostility	334	.88	.59	.75	.22
Suspiciousness	334	.89	.54	.51	–.30
Neurophysiological Externalizing Indicators					
Rotated-Heads Target P3	319	18.67	7.52	.69	.66
Flanker Stimulus P3	330	7.53	7.60	.59	1.43
Flanker Error P3	312	8.7	4.38	.92	1.59
Choice-Feedback P3	325	20.61	6.53	–.09	.64
Convergent Validity Criteria					
ESI Disinhibition	333	.69	.47	1.16	1.62
MPQ Disinhibition	324	.35	.21	.32	–.57
Discriminant Validity Criteria					
Trait Fear Inventory	199	1.29	.57	.16	–.72
MPQ Boldness (reversed)	324	.53	.21	–.16	–.72

ESI: Externalizing Spectrum Inventory; MPQ: Multidimensional Personality Questionnaire.

Table B. Model Fit Statistics for Initial CFA Model and Alternative Correlated Two-Factor Models

Indicator(s) dropped	Indicator(s) added	χ^2	CFI	TLI	RMSEA
—	—	100.72	.87	.82	.09
Rigid Perfectionism	—	89.03	.88	.82	.11
Rigid Perfectionism, Risk Taking	—	34.59	.95	.92	.07
Rigid Perfectionism, Risk Taking	Deceitfulness	50.80	.95	.92	.07
Rigid Perfectionism, Risk Taking	Suspiciousness	67.36	.90	.85	.09
Rigid Perfectionism, Risk Taking	Hostility	40.43	.96	.94	.06

Note. Row 1 in the table shows fit statistics for the initial CFA model, which employed PID-5 trait scales of Impulsivity, Irresponsibility, Distractibility, Risk Taking, and Rigid Perfectionism as indicators of the PID-5 trait factor. Subsequent rows show fit statistics for alternative models in which Rigid Perfectionism and then Risk Taking were removed as indicators, and other scales were individually added.

Table C. Standardized Factor Loadings and Factor Correlations for Initial Model and Alternative Correlated Two-Factor Models

			Trait Scale Indicators of PID-5 Disinhibition Factor (Standardized Loadings)						P3 Response Indicators of Neurophysiological Disinhibition Factor (Standardized Loadings)			
Indicator(s) Dropped	Indicator(s) Added	Correlation Between Model Factors (Neurophys, PID-5)	Impulsivity	Irresponsibility	Distractibility	Risk Taking	Rigid Perfectionism	Added Indicator	Rotated-Heads Target P3	Flanker Stimulus P3	Flanker Error P3	Choice-Feedback P3
—	—	–.20*	.95**	.59**	.58**	.63**	-.05	—	.62**	.47**	.56**	.44**
Rigid Perfectionism	—	–.20*	.95**	.59**	.58**	.63**	—	—	.62**	.47**	.56**	.44**
Rigid Perfectionism, Risk Taking	—	–.18*	.75**	.73**	.73**	—	—	—	.60**	.49**	.58**	.42**
Rigid Perfectionism, Risk Taking	Deceitfulness	–.19*	.69**	.83**	.66**	—	—	.75**	.60**	.49**	.59**	.42**
Rigid Perfectionism, Risk Taking	Suspiciousness	–.21*	.72**	.75**	.73**	—	—	.36**	.60**	.48**	.58**	.42**
Rigid Perfectionism, Risk Taking	Hostility	–.20*	.73**	.74**	.74**	—	—	.62**	.61**	.48**	.58**	.43**

Note. Row 1 in the table shows—for the initial CFA model employing PID-5 Impulsivity, Irresponsibility, Distractibility, Risk Taking, and Rigid Perfectionism scales as indicators of the PID-5 trait factor—loadings of indicator variables for the model on each factor and the correlation between the two factors. Subsequent rows show loadings and factor correlations for alternative models in which Rigid Perfectionism and then Risk Taking were dropped as indicators, and other scales were individually added. *$p < .05$. **$p < .001$.