Quantifying inhibitory control as externalizing proneness: A cross-domain model

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Abstract:

Recent mental health initiatives have called for a shift away from purely report-based conceptualizations of psychopathology toward a biobehaviorally oriented framework. The current work illustrates a measurement-oriented approach to challenges inherent in efforts to integrate biological and behavioral indicators with psychological-report variables. Specifically, we undertook to quantify the construct of inhibitory control (inhibition-disinhibition) as the individual difference dimension tapped by self-report, task-behavioral, and brain response indicators of susceptibility to disinhibitory problems (externalizing proneness). In line with prediction, measures of each type cohered to form domain-specific factors, and these factors loaded in turn onto a cross-domain inhibitory control factor reflecting the variance in common among the domain factors. Cross-domain scores predicted behavioral-performance and brain-response criterion measures as well as clinical problems (i.e., antisocial behaviors and substance abuse). Implications of this new cross-domain model for research on neurobiological mechanisms of inhibitory control and health/performance outcomes associated with this dispositional characteristic are discussed.

Keywords: disinhibition | externalizing | inhibitory control | cross-domain | biobehavioral | psychoneurometric

Article:

Inhibitory control, the capacity to resist or modulate impulses, is a neuro-cognitive individual-difference construct with important implications for adaptive behavior and health. Deficits in this capacity as assessed by self- or other-report are associated with a wide range of adverse outcomes including reduced educational and occupational attainment, financial difficulties, mental health problems such as substance abuse and antisocial behavior, and physical ailments including metabolic abnormalities, inflammatory conditions, and sexually transmitted diseases (Moffitt et al., 2011). A strong need exists for explicating the biobehavioral bases of such deficits to clarify their role in clinical problems and determine how best to remediate them. However,

this endeavor is complicated by the complex, multifaceted nature of inhibitory control, both psychologically (e.g., Berg, Latzman, Bliwise, & Lilienfeld, 2015; Carver, Johnson, Joormann, Kim, & Nam, 2011; Fischer, Smith, & Cyders, 2008; Whiteside & Lynam, 2001) and neurobiologically (e.g., Bari & Robbins, 2013; Schall, Palmeri, & Logan, 2017).

Our aim in the current work was to establish a frame of reference for ongoing biobehavioral research on variations in inhibitory control, in the form of a measurement model encompassing indicators from domains of self-report, neurophysiology, and behavioral performance. To accomplish this, we focused on a highly specific conceptualization of inhibitory control capacity—that is, as resistance versus susceptibility to impulse control problems ("externalizing proneness")—that we expected would be amenable to a cross-domain measurement approach.

Conceptualizing Inhibitory Control as Externalizing Proneness

Our work focuses on an individual difference dimension of inhibitory control (inhibition-disinhibition) conceptualized as resistance versus proneness to problems of impulse control. Thus, we conceive of inhibitory control in trait-dispositional terms—that is, variation across individuals in the capacity to restrain behavioral impulses and affective reactions. This is in contrast to a state conceptualization, in which inhibitory control is viewed as an internal process or mechanism invoked within certain performance contexts (for further discussion of this distinction, see Patrick & Hajcak, 2016). In addition, our concept of inhibitory control is (a) biobehavioral in nature, meaning that it has measurable referents in neurophysiology and overt behavior as well as in self-reported perceptions, and (b) referenced specifically to psychopathology—emerging out of work demonstrating a shared dispositional factor underlying impulse control problems of various types (Krueger et al., 2002; Young, Stallings, Corley, Krauter, & Hewitt, 2000). In these respects, our concept of inhibitory control differs from other trait conceptions of impulsivity that emphasize separable components (facets) with differing psychological and clinical correlates (e.g., Fischer, Smith, & Cyders, 2008; Sharma, Markon, & Clark, 2014; Whiteside & Lynam, 2001).

When conceptualized as externalizing proneness, inhibitory control capacity can be presumed to have a strong biological-genetic basis: Different published studies have reported its heritability to be about 80% (Krueger et al., 2002; Young et al., 2000). As noted above, it has direct, strong relevance to clinical problems—particularly impulse-related conditions such as conduct disorder, adult antisocial personality, and substance dependence, but also (to a lesser degree) anxious-depressive (internalizing) conditions (Nelson, Strickland, Krueger, Arbisi, & Patrick, 2014; Venables et al., 2017). Importantly for the current work, inhibitory control conceptualized in this way also has well-documented neural and behavioral-performance correlates. It is for this reason that we utilized this conception in seeking to develop a cross-domain measurement model for this construct.

It bears mention that the measurement model we describe in this report is not intended to encompass all psychological facets of impulsivity or all neural mechanisms relevant to behavioral control versus disconstraint. Consistent with the developmental concept of equifinality (Cicchetti & Rogosch, 1996), evidence exists for differing developmental pathways and neurobiological mechanisms for externalizing problems. Some of these include (a)

weaknesses in cognitive-executive function (e.g., Bogg & Finn, 2010; Young et al., 2009), which we emphasize in the current work; (b) abnormalities in midbrain dopamine-mediated reactivity to rewards (e.g., Beauchaine, Zisner, & Sauder, 2017; Buckholtz et al., 2010); and (c) reduced amygdala response to distress or punishment cues (e.g., Blair, 2013). Our aim in undertaking the work reported here was to establish a model for a particular conceptualization of inhibitory control, based around reliable indicators of externalizing psychopathology from multiple measurement domains, that can serve as a referent for guiding and integrating investigative work focusing on different facets and mechanisms of this problem domain.

Cross-Domain Measurement Approach

Psychopathology and measurement theorists have long recognized that individual differences are observable across multiple domains of measurement (Campbell & Fiske, 1959) and that the process of construct validation is inherently a multivariate endeavor (Cronbach & Meehl, 1955). However, contemporary experimental psychopathology research often takes the form of mapping bivariate associations between disorder or trait-liability constructs and single neuroscientific or psychophysiological measures (cf. Patrick, Venables, et al., 2013). Criticisms of this bivariate mapping approach include concerns about the psychometric properties of lab-task data (e.g., weak or unknown reliability, limited or assumed construct validity), measurement error inherent to single measures, and problems in replicating findings from small-N studies relating biological measures to psychological traits or conditions (Lilienfeld & Treadway, 2016; Vul, Harris, Winkielman, & Pashler, 2009).

Recent scientific initiatives have advocated for the integration of behavioral and neurobiological variables into assessments of individual difference characteristics. For example, a recent report of the National Research Council (NRC, 2015) highlighted the need for incorporating data from brain and behavioral domains along with report-based data into assessments of human capabilities. Along similar lines, the National Institute of Mental Health's Research Domain Criteria initiative (RDoC; Cuthbert & Kozak, 2016; Insel et al., 2010) advocates for greater use of neuroscience methods and concepts in models of mental health and illness. Specifically, RDoC calls for an investigative focus on constructs such as acute threat, reward valuation, and response inhibition across multiple levels of analysis (ranging from genes to brain circuitry to overt behavior) to advance biobehavioral understanding and assessment of mental health problems. The focus of these initiatives is thus on characterizing individual differences in terms of cognitive, affective, regulatory, and social processes, assessed using measures from behavioral and physiological/neural domains.

However, the inclusion of behavioral and brain variables in individual difference assessments poses major challenges. Chief among these is the issue of domain-specific method variance, which complicates the interpretation of measures from particular modalities and constrains their level of association with measures from other modalities. One strategy that has been proposed for integrating measures from separate domains in assessing dispositional tendencies is the construct-network approach (Patrick, Venables, et al., 2013; see also Patrick, Durbin, & Moser, 2012; Yancey, Venables, & Patrick, 2016). This approach conceives of traits as core biobehavioral propensities that affect measurable responses in different modalities (e.g., scale report, behavioral performance, neurophysiology). Because measures from a particular response

modality contain variance *unrelated* to the common source trait (e.g., method-specific and error variance), relations between measures from different modalities tend to be systematically lower than those between measures from the same modality (Campbell & Fiske, 1959). However, by combining across measures from a given modality (e.g., scale-report or performance) that contain trait variance in common with measures from other modalities (e.g., neurophysiology), the systematic variance from each modality that reflects the shared latent trait can be accentuated—to permit aggregate measures from different modalities to cohere more strongly around a common dimension, corresponding to the underlying trait (Patrick, Venables, et al., 2013). Factor analytic techniques provide an effective means for combining sets of measures in this way and examining their associations (Kline, 2010).

The current study was undertaken to demonstrate a cross-domain approach to operationalizing variations in inhibitory control capacity (inhibition-disinhibition), through use of indicators from three response modalities: self-report (psychometric scales), overt behavior (task performance), and neurophysiological functioning (brain response). Our broader aim was to establish the foundation for a new, *biobehavioral* model of this important individual difference construct to help guide research on neurobiological mechanisms of inhibitory control and clinical problems associated with deficits in this capacity.

Self-report (psychometric scale) measures of inhibitory control

Propensities toward impulsive-irresponsible behavior and clinical problems such as antisocial behavior and substance use disorders are hypothesized to reflect deficits in inhibitory control capacity (Gorenstein & Newman, 1980; Iacono, Malone, & McGue, 2008; Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Patterson & Newman, 1993). On the basis of evidence for systematic covariation among various impulse-control problems and disinhibitory traits associated with them, Krueger and colleagues (2007) formulated an assessment model for this spectrum of problems/traits in the form of a self-report instrument, the 415-item Externalizing Spectrum Inventory (ESI). The ESI includes 23 facet scales that load together onto a common Disinhibition factor, with strongest loadings for scales assessing problematic impulsivity, irresponsibility/lack of dependability, thievery, and alienation. This Disinhibition factor can be operationalized effective using a brief factor-scale measure consisting of representative items from these ESI facet scales (i.e., either 20 or 30 items; Patrick, Kramer, Krueger, & Markon, 2013; Yancey, Venables, Hicks, & Patrick, 2013). Scores on this ESI-based Disinhibition (ESI-DIS) scale correlate very highly (~.7) with the factor that underlies impulse-control disorders (Yancey et al., 2013; see also Krueger et al., 2002), largely as a function of overlapping genetic influences between the two (Yancey et al., 2013). The implication is that the ESI-DIS scale indexes the heritable liability that contributes to antisocial behavior, substance dependence, and other impulse-control problems (Krueger et al., 2002; Young, et al., 2000), which in turn is theorized to reflect brain-based impairments in inhibitory control (Patrick & Bernat, 2010; Yancey et al., 2013; Young et al., 2009).

Established psychological scale measures of impulsive-antisocial tendencies exhibit robust associations with the ESI general factor. For example, Hall, Bernat, and Patrick (2007)reported that total scores from a 100-item version of the ESI (designed to index the general factor) were associated with Gough's (1960) Socialization Scale ($r = -.61$) and self-reported measures of

antisocial behavior ($r = .81$) and substance use (rs = .61 to .64). Additional efforts have sought to develop scale measures of disinhibition using items from other well-established inventories, including psychopathy questionnaires (Drislane et al., 2015; Hall et al., 2014), broad normal-range personality inventories (Brislin, Drislane, Smith, Edens, & Patrick, 2015; Brislin et al., 2017), and self-report measures of psychopathology and maladaptive personality traits (Drislane et al., 2017; Sellbom et al., 2016). Disinhibition scales from the Psychopathic Personality Inventory (PPI-DIS; Hall et al., 2014) and Multidimensional Personality Questionnaire (MPQ-DIS; Brislin et al., 2015) correlate strongly with the ESI-DIS scale (~.7) and exhibit patterns of associations with personality and clinical criterion variables that closely parallel those observed for ESI-DIS.

In sum, research has provided evidence for a latent disinhibitory liability that manifests in self-reported traits and observed clinical problems (Krueger et al., 2002). This liability factor can be assessed in the domain of self-report using the ESI-DIS scale or using existing scales such as the Socialization Scale or trait disinhibition scales composed of items from inventories such as the PPI or the MPQ. Data from other work, considered next, indicate that behavioral-performance and brain response measures from cognitive tasks may operate as indicators of this same latent disinhibitory dimension. A core premise of the current work is that lab-task measures provide coverage of aspects of this dispositional dimension that are not effectively captured by self-report.

Lab-based behavioral and neurophysiological measures of inhibitory control

Evidence that a related dimension of inhibitory control can be assessed through lab-based performance measures was provided by Young et al. (2009). These investigators reported a robust negative association between scores on a factor reflecting covariance among trait and symptom measures of disinhibitory proclivities and scores on a common executive function (EF) factor reflecting performance across three inhibitory control tasks (i.e., Stroop, Antisaccade, Stop Signal; see also Miyake et al., 2000; Miyake & Friedman, 2012). Participants in the Young et al. study consisted of twins, allowing for estimation of etiological influences on trait/symptom and performance factor scores and their overlap. The genetic correlation between scores on the two factors was substantial (–.6), indicating appreciable overlap in genetic influences contributing to higher disinhibitory tendencies and poorer inhibitory control (EF-task) performance. The implication is that EF-task performance operates as a behavioral indicator of heritable disinhibitory liability.

Regarding neurophysiological indicators, Iacono, Malone, and McGue (2003) hypothesized that reduced amplitude of the P3 event-related potential (ERP)—a positive-going brain response that occurs to salient stimuli within a series—operates as a neural indicator of the largely genetically based disinhibitory liability factor. Support for this hypothesis was provided by studies reporting reduced P3 amplitude in relation to impulse-related phenomena of various types—including impulsive and aggressive traits, child and adult antisocial deviance, and alcohol and other drug problems (Justus, Finn, & Steinmetz, 2001; Venables, Patrick, Hall, & Bernat, 2011)—as well as with general disinhibitory liability as indexed either by externalizing symptom scores (Patrick et al., 2006) or by scores on the ESI-DIS scale (Yancey et al., 2013). Other work has shown that the observed relationship between P3 amplitude and disinhibitory tendencies is largely mediated by

genetic influences (Hicks et al., 2007; Yancey et al., 2013), providing compelling support for the hypothesis that reduced P3 amplitude indexes the same heritable disinhibitory liability as EF-task performance.

In other research, Nelson, Patrick, and Bernat (2011) showed that three brain reactivity measures consisting of variants of P3 from two separate tasks along with the error-related negativity (ERN), a negative-going ERP that follows incorrect task-responses (Falkenstein, Hohnsbein, & Hoormann, 1991), formed a single common factor when analyzed together with ESI-DIS scores. Scores on this factor, interpreted as a cross-domain index of disinhibition, predicted scale-assessed externalizing symptoms and ERP criterion measures at robust levels. Extending this work, Patrick, Venables, et al. (2013) defined a joint psychometric-neurophysiological (psychoneurometric) factor using two scale measures of disinhibition and two variants of P3 response, and showed that scores on this factor predicted interview-assessed symptoms and ERP criterion measures to a similar high degree (rs ~ .6).

Current Study Aims and Hypotheses

Findings reviewed in the preceding section suggest—in line with the idea of inhibitory control as a latent biobehavioral disposition that affects responses in different domains—that Disinhibition scale scores, performance on EF tasks, and variants of P3 response operate as alternative indicators of this dispositional construct. Although work has been done to relate report-based indicators to EF-task indicators (Young et al., 2009), and report-based indicators to P3 response indicators (Patrick, Venables, et al., 2013; Yancey et al., 2013), no work has yet been done to relate variables from all three domains together in a multivariate modeling framework. The current study was undertaken to address this gap and produce a cross-domain measurement model to serve as a frame of reference for biobehavioral research on inhibitory control.

On the basis of prior research demonstrating that a cross-domain disinhibition (inhibitory control) factor can be defined using psychometric-scale and brain-response measures as joint indicators, we hypothesized that a cross-domain factor of this type could be delineated using indicators from three measurement domains, consisting of task-performance along with scale and brain response variables. Our specific study hypotheses were as follows: (a) On the basis of previous findings reviewed above, indicator variables *within* each measurement domain were expected to correlate robustly with one another; (b) correlations *across* measurement domains were expected to be higher for aggregates of indicators (i.e., domain-level factors) than for individual indicators; (c) domain factors defined by indicators from each measurement domain were expected to covary and form a higher order factor reflecting cross-domain inhibitory control; and (d) scores on the cross-domain inhibitory control factor were expected to show robust associations with criterion measures of disinhibition from each assessment domain (self-report, task performance, brain response).

Method

Participants

Study participants were 149 undergraduate students and general-community adults who met the following inclusionary criteria: no current major mental disorder (i.e., schizophrenia, bipolar I) as determined from questions pertaining to mental health history on a brief pretest questionnaire, competency in English, and lack of visual or hearing impairments. Following procedures used in prior research (e.g., Hall et al., 2007; Patrick, Venables, et al., 2013), individuals were preselected using a brief measure of disinhibitory tendencies to ensure representation of high and low scorers (top and bottom 25%, respectively, from a larger screening sample) along with representation of intermediate scorers; the portions of individuals selected from each score range (top, bottom, middle) were 52.5%, 32.6%, and 14.9% (respectively). Participants indicated willingness to be contacted for lab testing in the prescreening assessment. The mean age of study participants, of whom 43% were female, was 20.5 (SD = 3.8). The racial/ethnic composition of the sample was 69.1% White, 12.8% African American, 12.1% Hispanic, 1.3% Asian, and 4.7% other, mixed race, or unreported. Procedures for the study were approved by the Institutional Review Board of Florida State University, and all participants provided informed written consent prior to both the questionnaire screening assessment and the lab testing session.

Psychometric measures of dispositional inhibition-disinhibition

The self-report scale indicators that we used contained items largely reflecting dispositional tendencies rather than specific behavioral acts (e.g., commission of crimes, use of substances). Four self-report scales were used as psychometric measures of inhibition-disinhibition: (a) the 20-item ESI-DIS scale, from the ESI Brief Form (ESI-BF; Patrick, Kramer, et al., 2013); (b) a scale consisting of 18 items from the brief form of the MPQ (Patrick, Curtin, & Tellegen, 2002), selected to index disinhibitory tendencies on the basis of content-relevance and psychometric properties (MPQ-DIS scale; Brislin et al., 2015; Brislin et al., 2017); (c) a scale consisting of 19 items from the PPI (Lilienfeld & Andrews, 1996), also selected to index disinhibitory tendencies (PPI-DIS scale; Hall et al., 2014); and (d) the Socialization Scale (SO; Gough, 1960), a 54-item measure of impulsive-antisocial tendencies known to correlate highly with general disinhibitory tendencies as assessed by the ESI (Hall et al., 2007). Descriptive statistics and internal consistency reliabilities (Cronbach's α) for these scales within the current sample are provided in the Supplemental Material available online.

Cognitive task measures of inhibition-disinhibition

Behavioral performance measures of inhibition-disinhibition were derived from lab-based inhibitory control tasks similar to those used in prior studies to assess individual differences in executive functioning (Miyake et al., 2000; Miyake & Friedman, 2012; Young et al., 2009) or attentional control (McVay & Kane, 2009, 2012). Procedural details for these tasks are described in the Supplemental Material. In brief, participants first completed a Stop Signal task (Verbruggen, Logan, & Stevens, 2008) that began with an initial block of target trials (simple geometric shapes) to assess reaction time (RT) at baseline, followed by blocks of trials in which participants were signaled at times (25% of trials) to inhibit their response to task stimuli by an auditory cue occurring after target stimulus onset. The stop signal delay (time between target onset and auditory cue) was varied on the basis of accuracy for the preceding stop trial (cf. Verbruggen et al., 2008). Participants then completed the Antisaccade task (Hallett, 1978), an oculomotor task that measures the ability to suppress prepotent responding by requiring the

inhibition of reflexive eye movement toward a visual distracter in order to correctly identify a co-occurring target stimulus. The version of the task was one that has shown associations with other performance measures of inhibitory (or attentional) control in past research (Hutchison, 2007; Kane, Bleckley, Conway, & Engle, 2001; Kane et al., 2016; McVay & Kane, 2012). Participants next completed a variant of the Stroop interference task (Stroop, 1935) in which they indicated via button presses the stimulus color of asterisk strings and color words that appeared in four different font colors. Last, participants were administered the Sustained Attention to Response Task (SART; McVay & Kane, 2009, 2012), a variant of the Go/No-Go task commonly used to measure attentional/inhibitory control. Consistent with many previous studies (e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Garavan, Ross, & Stein, 1999), the Go stimulus occurred more frequently (89% of trials) than the No-Go stimulus (remaining 11% of trials), establishing a prepotent response set that required recruitment of inhibitory control to override.

Task procedures for neurophysiological measures of inhibition-disinhibition

Participants completed three cognitive-ERP task procedures to provide neurophysiological (P3 response) indicators of inhibition-disinhibition; details of these procedures are reported in the Supplemental Material. Specifically, variants of the P3 response were derived from (a) a modified, 3-stimulus version of the "rotated-heads" visual oddball task (Begleiter, Porjesz, Bihari, & Kissin, 1984) in which picture stimuli are included as novel stimuli (see also Venables et al., 2011, for details); (b) an arrow version of the flanker task (Eriksen & Eriksen, 1974), administered in a manner consistent with previous investigations (e.g., Weinberg, Kotov, & Proudfit, 2015); and (c) a pseudo-gambling (choice-feedback) task used in a number of prior published studies (Bress, Smith, Foti, Klein, & Hajcak, 2012; Foti & Hajcak, 2009).

Criterion measures

Clinical questionnaires. Questionnaire-based measures of antisocial behavior, substance use, pathological personality tendencies, and impulsive behaviors were administered as criterion measures for evaluating the validity of factor scores from the cross-domain model of inhibition-disinhibition. The following scales were included in the present study.

The Behavior Report on Rule-Breaking (BHR; Hall et al., 2007), a 33-item self-report inventory, assesses for past instances of antisocial behaviors in adolescence and adulthood. Internal consistency reliabilities (Cronbach's α) in the current sample were .89 for the Juvenile subscale and .83 for the Adult subscale.

The Crime and Analogous Behavior Scale (CAB; Miller & Lynam, 2003) is a 55-item self-report inventory that assesses for antisocial behaviors and also substance abuse history; an abbreviated 16-item version was used in the current study. Questions on the CAB refer to specific behaviors (e.g., "Have you taken something not belonging to you worth over $50?") and are answered yes or no. Reliabilities (α) for the two subscales of this brief-form CAB (Antisocial Behavior = 9 items; Substance Abuse = 7 items) in the current study were .60 and .71, respectively.

The ESI-BF Substance Abuse scales (Patrick, Kramer, et al., 2013) are abbreviated versions of six facet scales from the ESI (Krueger et al., 2007) that assess for use of three types of substances (alcohol, marijuana, other drugs) and problems experienced in relation to each; alphas for these scales in the current sample ranged from .76 to .91 (median α = .87).

The Personality Inventory for *DSM–5* (PID–5; Krueger, Derringer, Markon, Watson, & Skodol, 2012) is a 220-item questionnaire that assesses traits specified in the alternative dimensional system for personality disorders within Section III of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–5*; American Psychiatric Association, 2013). Data for the PID–5 trait scales (αs in current sample = .63 to .96; median α = .87) were used to compute dimensional scores for the three impulsive-erratic personality disorders (PDs) represented in Section 3 of *DSM–5*—Antisocial, Borderline, and Narcissistic. Specifically, PID–5 scale scores for traits specified as relevant to each of these PDs, as follows, were standardized and averaged to form a composite for each: Antisocial PD—Manipulativeness, Deceitfulness, Callousness, Hostility, Impulsivity, Irresponsibility, and Risk Taking; Borderline PD—Emotional Lability, Anxiousness, Depressivity, Separation Insecurity, Hostility, Impulsivity, and Risk Taking; Narcissistic PD—Grandiosity and Attention Seeking. In addition, scores on a subset of 19 items from the PID–5 chosen to index disinhibitory tendencies (current sample α = .88) based on content-relevance and psychometric properties (PID–5-DIS; Drislane et al., 2017) were utilized as an additional questionnaire-based criterion measure.

The Youth Psychopathic Traits Inventory Disinhibition Scale (YPI-DIS) is a set of 14 items from the YPI (Andershed, Kerr, Stattin, & Levander, 2002) that have been shown to effectively index disinhibitory tendencies (Drislane et al., 2015). Internal consistency (α) of this scale in the current sample was .83.

The Impulsive Behavior Scale (IBS; Rossotto, Yager, & Rorty, 1994, cited in Milligan & Waller, 2001) is a 25-item inventory that asks participants to indicate how many times they have engaged in specific impulsive behaviors (e.g., reckless driving, shoplifting, unsafe sex, excessive use of drugs or alcohol); the frequency of each behavior is rated on a 5-point Likert-type scale (from 1 = *never* to 5 = *regularly*). IBS total scores (α = 87) were included as a measure of specific behaviors hypothesized to reflect inhibitory control deficits.

The Boldness scale of the Triarchic Psychopathy Measure (TriPM Boldness; Patrick, 2010) is a 19-item scale (α in current sample = .81) that indexes low versus high fearfulness as expressed in realms of social poise and assertiveness, immunity versus susceptibility to stress, and preference versus avoidance of risk. Based on prior evidence indicating that boldness-fear and inhibition-disinhibition are uncorrelated dispositions (Nelson et al., 2016; Venables et al., 2015; Venables et al., 2017), this scale measure was included for purposes of discriminant validation.

The Unlikely Virtues scale of the MPQ (Patrick et al., 2002) is a 14-item index of social desirability reflecting proneness to claim uncommon virtues and deny common weaknesses in oneself. This scale was included to evaluate whether inhibition-disinhibition assessed using indicators from multiple domains would relate differently to socially desirable responding than self-report assessed inhibition-disinhibition.

Behavioral and neurophysiological criterion measures. As described in the Data Scoring section, a subset of variables from the inhibitory-performance tasks and the cognitive-ERP tasks were reserved as behavioral and neurophysiological criterion measures for validating factor scores reflecting the general dimension of the cross-domain model of inhibition-disinhibition.

Procedure

After providing informed written consent, participants completed the four behavioral inhibitory-control tasks (Antisaccade, Stop Signal, Stroop, and SART). These tasks were administered using E-Prime presentation software (MEL Software, Inc.), with button-press responses recorded via a serial response box (Psychology Software Tools Inc., Model 200A). Participants viewed stimuli for these tasks (and for cognitive-ERP tasks as well) on a 21-inch computer monitor (situated ~ 1 m away, at eye level) while seated in a comfortable recliner. Questionnaires were administered during breaks between the behavioral tasks, and afterward during attachment of sensors for the cognitive-ERP tasks (novelty-oddball, flanker, pseudo-gambling). Neurophysiological data were collected using two computers, one equipped with E-Prime presentation software for stimulus delivery and the other with Neuroscan Acquire software for physiological data acquisition. After completing this set of tasks, participants filled out any remaining questionnaires and were then debriefed. Student participants were compensated with course credit and/or payment of $15 per hour; community participants received $15 per hour as compensation.

Data scoring

Procedures used to derive behavioral and physiological dependent measures for reported analyses are described in detail in the Supplemental Material along with information about participants missing data for certain measures. In brief, behavioral measures as follows were derived from the four inhibitory control tasks: Antisaccade task—target identification accuracy and RT variability (i.e., within-subject *SD* for each participant's distribution of RTs); Stroop task—accuracy and RT for incongruent trials; Stop Signal task—proactive inhibition (mean RT for "go" trials of main task minus mean RT for initial baseline block; cf. Verbruggen & Logan, 2009);[1] and SART task—signal detection sensitivity (d') and RT variability. The variables from among these that were used as behavioral indicators in modeling analyses of inhibition-disinhibition were Antisaccade task accuracy, Stroop task RT, Stop Signal task proactive inhibition, and SART task RT variability; the remaining variables (Antisaccade RT variability, Stroop accuracy, SART d') were reserved as criterion measures for evaluating the validity of factors from the resultant cross-domain model. Task-behavioral variables used in the structural modeling analyses were ones previously shown to operate most effectively together as indicators of an inhibitory control dimension (Miyake et al., 2000; Miyake & Friedman, 2012; see also McVay & Kane, 2012; Young et al., 2009). If the model indicator-variable from a given task consisted of an RT-score measure (e.g., Stroop RT), an accuracy-score measure from that task (e.g., overall Stroop color-naming accuracy) was used as a criterion in validation analyses—or vice versa.

Procedures for recording of electroencephalographic (EEG) activity, processing of the EEG data, and derivation of ERP measures followed our prior published work (Patrick, Venables, et al.,

2013; Venables et al., 2011; Yancey et al., 2013); details are provided in the Supplemental Material. The neurophysiological variables that served as indicators of disinhibition in the reported analyses consisted of P3 response to target stimuli in the novelty-oddball task, measured at the midline parietal (Pz) scalp site; P3 response to target stimuli and error-P3 (P3e) following incorrect responses in the arrow-flanker task, assessed at midline parietal (Pz) and midline frontocentral (FCz) scalp sites, respectively; and P3 response to feedback stimuli in the pseudo-gambling task, measured at the centroparietal (CPz) scalp site.[2] One other ERP variable, P3 response to novel (picture) stimuli in the oddball task, derived from activity at scalp site Pz, was used as a neurophysiological criterion measure.[3]

In addition, scores for the following clinical questionnaire measures were used as criteria in validation analyses: BHR Adolescent and Adult scales; CAB Antisocial and Substance Use scales; ESI-BF Substance Use and Problem scales, and an ESI Substance composite computed as the mean of standardized scores for these six scales; an externalizing composite, computed as the mean of standardized scores for the two BHR scales and the ESI Substance composite (cf. Krueger, 1999); PID–5 PD-trait composites, as described above; PID–5 and YPI Disinhibition scales; IBS inventory; and TriPM Boldness scale.

Statistical analyses

As an initial analytic step, zero-order correlations for indicators of inhibition-disinhibition within each measurement domain (scale-report, behavioral performance, brain response) were computed. Next, principal axis exploratory factor analyses (EFAs) were conducted for indicators of each type to test the hypothesis that measures from each domain would cohere together around a single common factor, reflecting inhibitory control capacity as expressed in that domain. Following this, confirmatory factor analysis (CFA) was used to evaluate the fit of alternative cross-domain models of the data, consisting of (a) a one-factor model, in which indicators from the three measurement domains were specified as loading directly on a single cross-domain factor, and (b) a higher order (correlated-factor) model, in which three subfactors demarcated by indicators from the differing measurement domains were specified as loading in turn on a general, cross-domain factor. CFA analyses were performed using Mplus (Version 7.3; Muthén & Muthén, 1998–2015), with full-information maximum likelihood estimation to accommodate missing data for individual indicators, allowing for estimation of factor scores for all participants ($N = 149$).

The two candidate models were each tested for absolute fit, and compared as well for fit against one another. Absolute fit was assessed using the traditional chi-square (χ^2) method, which yields lower values for better fitting-models, and also (given notable limitations of χ^2; Hu & Bentler, 1999) other absolute fit indices as follows: root mean square error of approximation (RMSEA), standardized root mean square residual (SRMR), Tucker-Lewis index (TLI), and comparative fit index (CFI). For RMSEA and SRMR, values below .05 indicate good fit, values from .05 to .08 indicate acceptable fit, and values above .08 indicate inadequate fit; for CFI and TLI, values of .95 or higher indicate good fit (Hu & Bentler, 1999). The two candidate models were evaluated against one another using two comparative fit indices, the chi-square difference statistic ($\Delta\chi^2$) and the Bayesian information criterion (BIC). Significant values of $\Delta\chi^2$ indicate better fit for a

model with more constraints over one with fewer constraints, and BIC values above 10 indicate a significant improvement in the fit of one model over another (Kass & Raftery, 1995).

Last, correlational analyses were performed to evaluate associations between factors from the best-fitting cross-domain model of inhibition-disinhibition and the various criterion measures described above. Estimated factor scores were computed for the full participant sample ($N = 149$); however, because of missing data for certain criterion measures, the Ns for validity coefficients ranged from 143 to 149 for self-reported clinical problem measures, and from 128 to 149 for lab-task criterion measures.

Results

Disinhibition indicators within measurement domains

Descriptive statistics and intercorrelations. The Supplemental Material presents descriptive statistics and internal consistency reliabilities for indicators of disinhibition within psychometric scale, behavioral performance, and neurophysiological measurement domains. Skewness and kurtosis values were acceptable, except for the SART RT variability measure, which showed a strongly leptokurtic ($\kappa = 9.7$) score distribution. For this variable, scores beyond 3 SDs from the mean were winsorized (Dixon, 1960) to values of 3 SDs, resulting in acceptable kurtosis (see Table S1). Internal consistency reliabilities (α) for psychometric scale indicators were good (.78 to .85). Reliabilities for behavioral and neurophysiological indicators were computed using split-half (odd/even) correlations adjusted by the Spearman-Brown prophecy formula. As seen in the online material, reliabilities for behavioral performance (82 to .98) and neurophysiological indicators (.84 to .98) were quite high.

Table 1. Correlations Among Indicators of Disinhibition Within Each Measurement Domain

Indicator	1	2	3
Psychometric scale ($n = 146–149$)			
1. ESI-DIS	—		
2. MPQ-DIS	.70**	—	
3. PPI-DIS	.66**	.72**	—
4. SO Scale	−.67**	−.53**	−.51**
Behavioral performance ($n = 118–145$)			
1. Antisaccade accuracy	—		
2. Stop Signal proactive inhibition	.17	—	
3. Stroop RT	−.25**	−.07	—
4. SART RT variability	−.24**	−.19*	.27**
Neurophysiological (ERP; $n = 112–132$)			
1. Target P3	—		
2. Flanker Response P3e	.44*	—	
3. Flanker Stimulus P3	.18*	.35**	—
4. Gambling Feedback P3	.32**	.35**	.11

Note: ESI-DIS = Disinhibition Scale of the Externalizing Spectrum Inventory Brief Form (Patrick, Kramer, et al., 2013); MPQ-DIS = Disinhibition Scale of the Multidimensional Personality Questionnaire (Brislin, Drislane, Smith, Edens, & Patrick, 2015); PPI-DIS = Disinhibition scale of the Psychopathic Personality Inventory (Hall et al., 2014); SO Scale = Gough's Socialization Scale (Gough, 1960); SART = Sustained Attention to Response Task; RT = reaction time; P3 = amplitude of P3 event-related potential (ERP) response; P3e = amplitude of P3 ERP following incorrect responses to task stimuli. *$p < .05$. **$p \leq .01$.

Table 1 shows correlations among indicators of inhibition-disinhibition within each measurement domain. Psychometric scale indicators were strongly intercorrelated as expected (median $r = |.67|$; Table 2, top section), whereas correlations among behavioral performance indicators were more modest (median $r = |.22|$; Table 2, middle section), consistent with prior research (McVay & Kane, 2012; Miyake et al., 2000; Miyake & Friedman, 2012). Correlations among neurophysiological (P3) indicators were intermediate in magnitude (median $r = |.34|$; Table 1, bottom section).[4]

Table 2. Validity of Factor Scores From the Higher-Order Cross-Domain Model of Inhibition-Disinhibition: Correlations With Self-Report, Behavioral, and Physiological Criterion Measures

Criterion measure	*N*	Scale factor	Behavioral factor	Neuro (ERP) factor	Cross-domain factor
Antisocial behavior					
BHR juvenile	148	.59**	−.04	−.20**	.26**
BHR adult	148	.52**	−.04	−.29**	.25**
CAB Antisocial Behavior	148	.40**	−.01	−.23**	.23**
Antisocial Behavior Composite	148	.57**	−.03	−.23**	.28**
Substance use/problems					
CAB substance use	148	.54**	−.10	−.15	.24**
ESI marijuana use	149	.48**	−.12	−.16	.24**
ESI marijuana problems	149	.53**	−.17	−.11	.23**
ESI drug use	149	.55**	−.10	−.17	.25**
ESI drug problems	149	.53**	−.13	−.12	.22**
ESI alcohol use	149	.44**	−.05	.15	.21**
ESI alcohol problems	149	.54**	−.09	−.22**	.29**
ESI substance composite	149	.64**	−.14	−.21**	.31**
Externalizing composite	148	.64**	−.07	−.22**	.30**
DSM-5 Section III Cluster B personality disorders					
PID-5 antisocial PD	143	.75**	−.14	−.24**	.35**
PID-5 borderline PD	143	.68**	−.19*	−.27**	.37**
PID-5 narcissistic PD	143	.44**	−.01	−15	.19*
Other disinhibition scales					
PID-5 disinhibition	144	.81**	−.23**	−.32**	.45**
YPI disinhibition	145	.77**	−.26**	−.26**	.41**
Impulsive behaviors	148	.64**	−.01	.14	.22**
TriPM boldness scale	147	.02	.17*	.03	−.07
MPQ unlikely virtues	149	−.26**	−.02	−.05	−.10
Behavioral/physiological criteria					
Antisaccade RT variability	149	.15	.37**	−.24**	.30**
Stroop accuracy	144	−.10	.38**	.21**	−.28**
SART *d′*	145	.08	.40**	.08	−.16
Novel P3	128	−.11	.38**	.63**	−.57**

Note: ERP = event-related potential; BHR = Behavior Report on Rule-Breaking; CAB = Crime and Analogous Behavior Scale; ESI = Externalizing Spectrum Inventory; Externalizing Composite = aggregate of substance use and antisocial behavior problems; PID-5 = Personality Inventory for *DSM-5*; YPI = Youth Psychopathic Traits Inventory; TriPM = Triarchic Psychopathy Measure; MPQ = Multidimensional Personality Questionnaire; Antisaccade RT variability = within-subject reaction time *SD*; Stroop Accuracy = proportion correct responses; SART = Sustained Attention to Response Task; Novel P3 = amplitude of P3 ERP to novel stimuli during novelty-oddball task.
*$p < .05$. **$p \leq .01$.

Exploratory factor analyses. Principal axis EFAs were used to evaluate whether indicators within each measurement domain cohered around a common dimension. The factor analysis for

the four psychometric scale indicators (ESI-DIS, MPQ-DIS, PPI-DIS, SO scale; $N = 146$) revealed one dominant factor, evident from scree plot inspection and confirmed by parallel analysis (Horn, 1965), accounting for 72.4% of the total among these measures. Figure 1 shows the scree plot for this analysis (upper left) along with loadings (lower left) of scale indicators on the common factor (range = |.67–.88|, median = .81). The factor analysis of scores for the behavioral performance indicators (Antisaccade accuracy, Stop Signal proactive inhibition, Stroop RT, SART RT variability; $N = 115$) likewise revealed one dominant factor (Fig. 1, upper middle), accounting for 40.1% of the total variance among these indicators (range of loadings = |.27–.59|, median = .48; see Fig. 1, lower middle). The factor analysis of scores for the four neurophysiological indicators (Oddball target P3, Flanker response P3e, Flanker stimulus P3, Gambling feedback P3) also revealed one dominant factor (Fig. 1, upper right), accounting for 46.5% of the variance among these indicators (range of loadings = |.37–.70|, median = .54; see Fig. 1, lower right).

| Measure	Factor Loadings	Measure	Factor Loadings	Measure	Factor Loadings
ESI-DIS	.88	Antisaccade	−.49	Target P3	.70
MPQ-DIS	.83	Stop Signal	−.27	Flanker Response P3e	.68
PPI-DIS	.79	Stroop	.59	Flanker Stimulus P3	.37
SO Scale	−.67	SART	.45	Gambling Feedback P3	.39
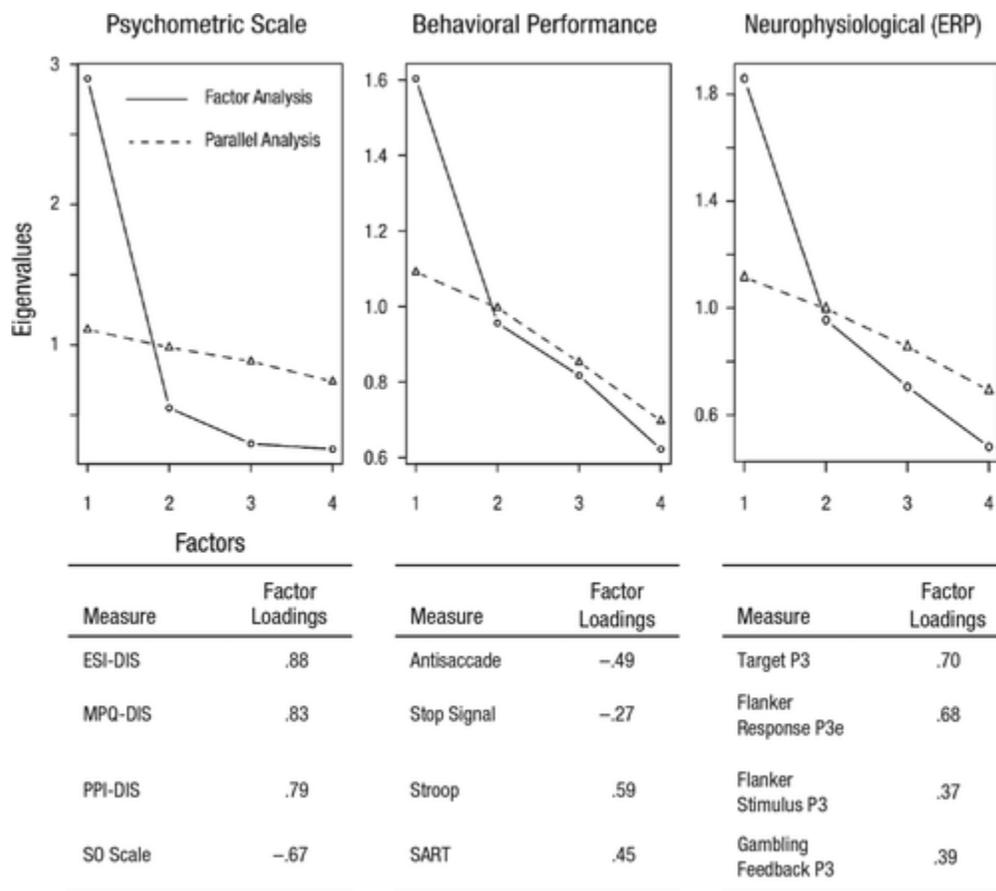
Fig. 1. Scree plots and variable loadings for factor analyses conducted on measures of disinhibition as assessed by differing measurement domains. In each case, a one-factor solution for psychometric scale indicators, behavioral performance variables, and neurophysiological indices was evident by visual inspection of the scree plots and by parallel analysis, a quantitative technique for determining the number of factors to extract by comparing observed eigenvalues of the analysis with those of a randomly generated data set (Horn, 1965). Observed eigenvalues for each analysis are depicted by solid lines, whereas eigenvalues estimated from a parallel analysis based on 100 random samples are denoted by dashed lines.

The foregoing analyses demonstrated common factors for each measurement domain reflecting variance shared among indicators within each. Consistent with the hypothesis, scores on these three domain factors were significantly correlated with one another (r for scale domain factor with behavioral domain factor = .21, p = .03; r for scale factor with neurophysiological domain factor = –.28, p < .01; and r for behavioral factor with neurophysiological factor = –.27, p < .01), at levels exceeding cross-domain correlations for individual indicators (see Table 2 in the Supplemental Material).
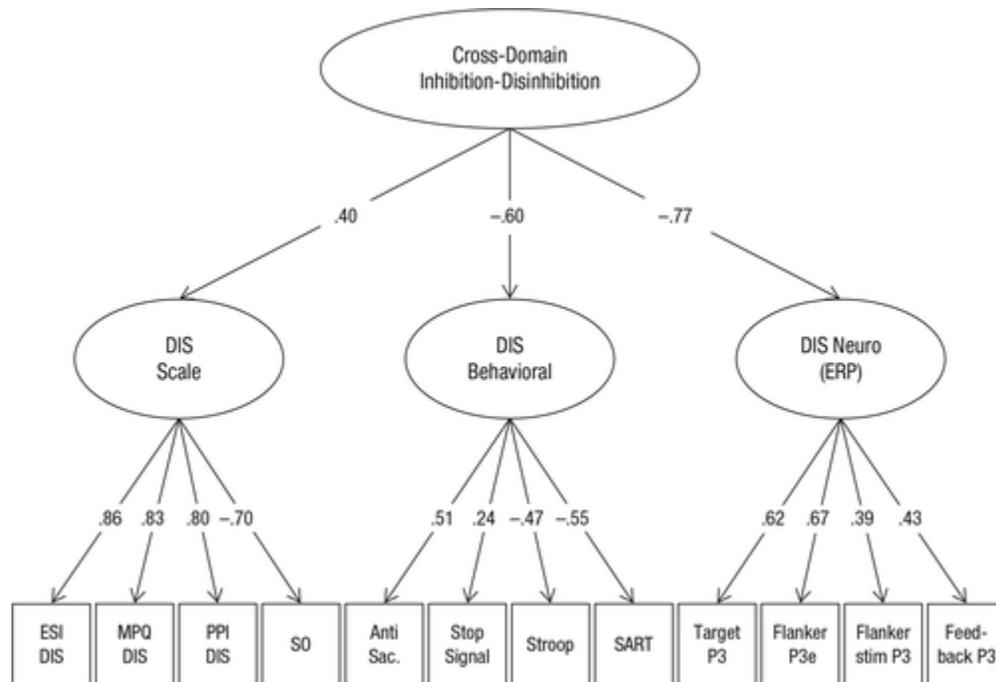
Fig. 2. Depiction of the best-fitting higher-order confirmatory factor model of inhibitory control (inhibition-disinhibition), with standardized parameter estimates. The model includes three lower-order factors reflecting covariance among indicators within each measurement domain, and a single higher-order factor reflecting variance in common across domains. ESI-DIS = Externalizing Spectrum Inventory Disinhibition scale; MPQ-DIS = Multidimensional Personality Questionnaire Disinhibition scale; PPI-DIS = Psychopathic Personality Inventory Disinhibition scale; SO = Gough's Socialization Scale; AntiSac. = Antisaccade accuracy; Stop Signal = proactive inhibition from the Stop Signal task; Stroop = reaction time to incongruent stimuli from the Stroop task; SART = reaction time variability from the sustained attention to response task; ERP = event-related potential; Target P3 = amplitude of P3 ERP to targets in the Oddball task; Flanker P3e = amplitude of P3 ERP following errors in the Flanker task; Flanker stim. P3 = amplitude of P3 ERP to arrow stimuli in the Flanker task; and Feedback P3 = amplitude of P3 ERP to feedback cues in the pseudo-gambling task.

Cross-domain model of scale, behavioral, and neurophysiological indicators

Based on hypotheses as described earlier, two CFA models were evaluated. The first model specified all indicators from the three measurement domains as loading directly onto a single common factor; the second model specified three lower order factors corresponding to

psychometric scale, behavioral performance, and neurophysiological domains that loaded in turn on a higher order *cross-domain* factor reflecting covariance among the lower order domain factors. Results from the one-factor model indicated inadequate fit to the data: $\chi^2(54) =$ 144.81, $p < .001$, RMSEA = .11, 90% CI = [.086, .127], SRMR = .105, CFI = .77, TLI = .72. By contrast, the alternative three-subfactor/one higher order factor model fit the data well: $\chi^2(51) =$ 68.68, $p = .05$, RMSEA = .048, 90% CI = [.001, .076], SRMR = .059, CFI = .96, TLI = .94. In addition, comparative fit indices revealed significantly better fit for the latter model compared with the former, ΔBIC = 61.1 and $\Delta\chi^2 = 76.1$ ($\Delta df = 3$, $p < .001$). Figure 2 depicts the model specifying three domain factors and one higher order factor, with standardized factor loadings.[5]

The pattern of loadings for individual indicators on the domain factors in the higher order model largely paralleled those observed in the separately conducted EFAs. As for loadings on the higher order factor of the model, the neurophysiological domain factor showed the strongest loading (–.77), followed by the behavioral domain factor (–.60) and then the scale domain factor (.40). All factor loadings were statistically significant ($p \leq .01$). The higher order factor of this model was labeled as the Inhibition-Disinhibition Cross-Domain factor given that it reflects variation in inhibitory control as operationalized across measurement domains, with individuals scoring high on this factor exhibiting deficient inhibitory control (disinhibition) in terms of brain and behavioral response as well as self-reported tendencies.

Relations of cross-domain factor with external criterion measures

Scores on the cross-domain factor of the higher order model were estimated using least squares regression (in Mplus) and evaluated for convergent and discriminant associations with scale, behavioral, and neurophysiological criterion measures as described earlier. In addition, scores for the three domain factors of the model were estimated in the same manner, and their relations with criterion measures of each type were evaluated in comparison with those for the cross-domain factor.[6]

Table 2 shows that scores on the cross-domain factor of the model, reflecting the common construct indexed by measures from the three assessment domains, showed moderate-level associations with all clinical criterion measures including antisocial behavior (rs = .23 to .28), substance use/abuse (rs = .21 to .31), impulsive-erratic PD dimensions (rs = .19 to .37), and other scale measures of impulsivity and disinhibition (rs = .22 to .45). The scale domain factor showed systematically higher correlations with self-report based criterion variables (rs = .44 to .81; median r = .55), but this factor evidenced weak associations with the behavioral and neurophysiological criterion measures (rs = |.08–.15|; median r = .11). The cross-domain factor exhibited a strong significant correlation with the neurophysiological criterion measure (P3 response to novel stimuli in the Oddball task; r = –.57) and moderate-level associations with two of the three behavioral criterion measures (Antisaccade RT variability, Stroop accuracy; rs ~ .3), with the correlation for the third behavioral criterion (SART d') weak but near significant (r = – .16, p = .05). Reciprocal to results for the scale domain factor, the behavioral and neurophysiological domain factors showed strong significant correlations with same-domain criterion measures (rs = .37–.40 and .63, respectively) and uniformly weaker associations with scale-assessed clinical criteria (rs = |.01–.32|; median r = .15). The behavioral domain factor showed a moderately strong association with the neurophysiological (P3) criterion variable (r =

.40), whereas correlations for the neurophysiological criterion with the behavioral criterion variables were more modest (median $r = .21$).

The cross-domain factor showed discriminant validity in terms of a negligible association ($r = .07$), as expected (e.g., Nelson et al., 2016), with dispositional fearlessness/fear as indexed by the Boldness scale of the TriPM. Similar null relationships with TriPM Boldness were evident for the scale domain factor and the neurophysiology domain factor ($rs = .02$ and $.03$, respectively). It is interesting that the behavioral domain factor showed a trend-level ($p = .038$) positive association with TriPM Boldness. Also of note, the scale domain factor showed a robust negative association with scores on the MPQ Unlikely Virtues scale ($r = -.26, p < .01$), indicating a component of low social desirable responding to reported disinhibitory tendencies, whereas the behavioral, neurophysiological, and cross-domain factors were unrelated to Unlikely Virtues scale scores ($rs = |.05–.01|, ps > .51$).

Discussion

Our broad aim in the work reported here was to develop a cross domain measurement model for the construct of inhibitory control (inhibition-disinhibition) using variables from different assessment domains that have been shown to relate to general disinhibitory liability, or externalizing proneness—namely self-report scale measures of trait disinhibition, behavioral performance variables from inhibitory control tasks, and indices of brain response from cognitive control tasks. Our findings serve to bridge differing lines of previously published work (e.g., Iacono et al., 2003; Krueger et al., 2002; Yancey et al., 2013; Young et al., 2009) and establish a useful frame of reference for continuing research on facets and mechanisms of inhibitory control as related to externalizing psychopathology and other clinical conditions as well as adaptive performance.

Prior published work has demonstrated relationships for cognitive-ERP (Nelson et al., 2011; Yancey et al., 2013) or cognitive-performance measures (Friedman & Miyake, 2004; Young et al., 2009) with scale measures of trait disinhibition. In a prior article (Patrick, Venables, et al., 2013), for example, we presented evidence for covariation between two scale measures of disinhibition and two variants of P3, and showed how this covariation could be represented in a simple, one-factor EFA model. However, the work reported here is the first to document and model relations of multiple indicators from both brain-ERP and task-performance domains with trait-scale measures. Thus, the current research is innovative but linked to a solid foundation of prior work by different investigative teams. Its connection to prior published research is critically important in light of concerns that have been raised about the replicability of findings in clinical and general psychological science research (Lilienfeld, 2017; Open Science Collaboration, 2015; Tackett et al., 2017) and the reproducibility of findings from genetic and neuroscientific studies of psychopathology phenotypes specifically (Button et al., 2013; Iacono, Vaidyanathan, Vrieze, & Malone, 2014; Ioannidis, 2011; Patrick, 2014). Given its reliance on variables shown to function as reliable indicators of trait disinhibition or externalizing proneness in prior published work, the model reported here can be expected to replicate and thus serve as a dependable anchor for continuing research on correlates and mechanisms of inhibitory control capacity.

Cross-domain model of inhibitory control: Criterion validity of domain and general factors

In line with our study hypotheses, and essential to our modeling aims, variables within each measurement domain cohered together in reliable ways. As expected from recent work demonstrating strong convergence among alternative scale measures of trait disinhibition (Drislane & Patrick, 2017), self-report indicators in the present study were robustly interrelated and formed a single common factor. Scores on this scale domain factor showed expected strong correlations with clinical problem criteria assessed via self-report, including antisocial behavior, substance abuse, and impulsive-erratic PD dimensions (rs = .40 to .75). Also in line with prediction based on prior work (e.g., Miyake et al., 2000), behavioral performance indices from response inhibition tasks were moderately intercorrelated and formed a coherent factor that exhibited modest validity in terms of relations with clinical criterion measures. Replicating prior research (Nelson et al., 2011; Patrick, Venables, et al., 2013), we also found that variants of P3 brain response from different tasks (and task conditions, in the case of stimulus P3 and error P3 from the flanker task) were moderately intercorrelated (median r = .34). Scores on the neurophysiological factor defined by these P3 variants showed convergent validity in relation to clinical criterion measures, albeit expectedly lower than for the scale factor, given the difference in measurement modality (Campbell & Fiske, 1959; Patrick, Venables, et al., 2013). Reciprocally, the neurophysiological factor correlated to a markedly higher degree (r = .63) with the neurophysiological criterion variable (P3 to novel stimuli in the visual oddball task) than the scale factor (r = –.11).[7]

A novel and important finding of the current study was that task-performance indicators of inhibitory control correlated with variants of P3 brain response, consistent with the hypothesis that variability in P3 reflects, in part, inhibitory control processes related to EF (Begleiter & Porjesz, 1999; Iacono et al., 2003; Patrick, Foell, Venables, & Worthy, 2015). This finding was expected given evidence from separate prior studies demonstrating a strong heritable basis for correlations of both P3 response (Yancey et al., 2013) and EF task-performance variables (Young et al., 2009) with disinhibitory traits/problems—indicating that both P3 and EF-performance index heritable liability for externalizing problems. However, the current study is the first to evaluate whether these nonreport variables correlate with one another and overlap in their associations with trait disinhibition. The finding that the first-order domain factors all loaded appreciably on the higher order, cross-domain factor indicates that P3-brain and EF-task variables share variance in common with scale-assessed disinhibition. At the same time, though, the brain and task-performance factors cohered more closely with one another than with the scale factor, causing the cross-domain disinhibition factor to be defined more strongly by these two domain factors (loadings on higher order factor = –.77 and –.60, respectively) than by the scale domain factor (loading = .40). This finding has important conceptual and empirical implications, as discussed in the next subsection.

Implications for assessment of inhibitory control in clinical-applied and research contexts

Beyond the specific aim of bridging between literatures on neurophysiological and task-behavioral indicators of trait disinhibition (externalizing proneness), a broad goal of the present study was to establish a conceptual framework for research directed at understanding the contribution of trait variations in inhibitory control to behavioral outcomes of differing types.

Calls have arisen from different areas of the field for increased integration of behavioral and neurobiological variables into assessments of individual difference characteristics. Some of these calls focus on mental health problems (e.g., Kozak & Cuthbert, 2016), others on adaptive performance in real-world contexts (e.g., NRC, 2015). However, these proposals do not articulate a systematic strategy for integrating data across different modalities into assessments of target characteristics. In the current study we sought to address this need by illustrating how expectably modest bivariate relations among indicators of a trait construct from different measurement domains can be synthesized using quantitative modeling methods to form an effective cross-domain index of the target construct. This integration strategy capitalizes on the ability of higher order latent variable modeling to capture the systematic (i.e., error-free) covariance among indicators *within* specific measurement domains and, in turn, to demarcate a general factor reflecting the overlap among common-variance components *across* domains.

Returning to a point made in the preceding section, a notable feature of our cross-domain model of inhibitory control is that the general factor was weighted more toward the behavioral-performance and neurophysiological domains than the self-report domain. As a function of this, scores on this cross-domain index of disinhibition, unlike scores on the self-report domain factor, were unrelated to socially desirable responding as indexed by the MPQ Unlikely Virtues scale. However, the cross-domain factor still contained sufficient representation of reported dispositional tendencies to demonstrate robust predictive relations with reported externalizing symptoms. Thus, this cross-domain factor holds strong promise as a target phenotype for studies seeking to identify differences in brain structure, function, and circuitry related to variations in inhibitory control that connect in turn to psychopathology. For example, it can be expected that functional neuroimaging investigations of neural processes related to externalizing proneness would yield larger effect sizes for samples characterized in terms of scores on this cross-domain disinhibition factor versus scores on report-based trait or diagnostic measures alone.

However, a basic principle of assessment to be borne in mind is that measurement devices (tests) have utility in relation to particular intended purposes. Thus, although stronger weightings for brain and task-behavioral indicators may be desirable in assessments conducted for purposes of research on neural mechanisms, increased weighting of report-based indicators is likely to be beneficial for other assessment purposes (e.g., predicting clinical outcomes that relate strongly to psychological characteristics such as perceptions, attitudes, and beliefs). As an illustration of this, an alternative three-factor higher order model, in which the three domain-specific factors were constrained to load equally on the higher order factor, was fit to the current data (for analytic details, see the Supplemental Material). Results indicated that adding the constraints did not significantly decrease model fit, ΔBIC = −6.9, $\Delta\chi^2$ = 3.12, Δdf = 2, p > .21. It is important, though, that scores on the cross-domain factor from this constrained model showed increased associations with reported impulse control problems (median r = .38) relative to those for the unconstrained model (median r = .27). Results from this supplemental analysis highlight the fact that choices regarding selection and weighting of indicator variables in an assessment protocol are importantly dependent on the purpose(s) of the assessment.

Limitations and future directions

Some limitations of the current work are important to acknowledge. First, although multiple indicators of disinhibition from different measurement domains were included in the structural model, the criteria used to validate the factors of the model consisted largely of self-report measures of clinical problems,[7] along with a small number of behavioral performance (i.e., three) and neurophysiological measures (one). It will be important in future work of this kind to include non-questionnaire-based clinical measures (e.g., interviewer or informant ratings of psychopathology) along with a wider array of behavioral and brain measures derived from separate lab tasks. In particular, it will be informative to examine how general and specific-domain factors of the model relate to cognitive functions and brain processes apart from those included in the model—for example, working memory capacity and subcortical brain activation during anticipation of reward, both of which have shown associations with externalizing proneness in prior work (e.g., Bogg & Finn, 2010, and Buckholtz et al., 2010, respectively).

A second limitation of the current study is the relatively modest size of the participant sample, which did not allow for systematic evaluation of possible moderating effects of variables such as gender, race/ethnicity, and general intelligence. Another is that data regarding income, education, or socioeconomic status were not collected. In addition, the results of the present study are limited by the selection of behavioral and brain indicators. It remains to be seen whether a similar model would emerge from analyses utilizing other indicators from these assessment domains—such as behavioral measures from more realistic performance protocols (e.g., responding within virtual environments) and brain activations in functional neuroimaging tasks. Given these limitations, the current work is a significant step toward developing a cross-domain model of inhibitory control capacity, but should viewed as an important extension of prior work (e.g., Patrick, Venables, et al., 2013; Young et al., 2009) rather than as a finished product.

A further limitation of present study is its cross-sectional design. Although studies of this type are important for development and initial validation of cross-domain measurement models, etiologically informative (twin and/or longitudinal) designs (Vaidyanathan, Vrieze, & Iacono, 2015) will be needed to test the hypothesis that cross-domain assessments reflect liabilities or neurobiological manifestations of psychopathology (see, e.g., Venables et al., 2017). However, given the costs and logistical challenges associated with large-scale twin and multiwave longitudinal designs, smaller N cross-sectional studies like the current one are important for (a) developing hypotheses, testing candidate indicators, and establishing procedures for participant selection in larger N, etiologically informative investigations; and (b) connecting new variables of interest to already existing etiologically informative data sets through collection of overlapping measures that can serve as "metric bridges" (Friedman, Kern, Hampson, & Duckworth, 2014).

It is important to reiterate that the model we report here reflects a highly specific conceptualization of inhibitory control—that is, as resistance versus susceptibility to disinhibitory problems (externalizing proneness). As noted at the outset, inhibitory control is a complex construct, encompassing multiple psychological facets (e.g., Berg et al., 2015; Carver et al., 2011; Fischer et al., 2008; Whiteside & Lynam, 2001) and differing neural circuits/processes (e.g., Bari & Robbins, 2013; Schall et al., 2017). The current model is not intended to encompass all psychological and neurobiological aspects of inhibition-disinhibition or related constructs such as trait impulsivity (Beauchaine et al., 2017) or constraint (Tellegen & Waller, 2008).

Rather, it is intended to serve as a concrete and dependable referent for interconnecting variables from different measurement domains that relate to this biobehavioral construct, conceptualized in dispositional terms.

In addition to further evaluating the effectiveness of indicators included in our model and ones that were omitted (SSRT, ERN), it will be important in future work to examine how measures of psychological attributes or neural processes not represented directly in the model relate to variables included in the model. For example, some facets of trait impulsivity may connect with self-report scale variables in our model but not with brain-ERP (P3) variables, and some neural-systems measures of inhibitory control may connect with brain-ERP or task-performance variables in the model but not with scale-report variables. Through systematic work of this kind, our multivariate, multidomain model for the construct of externalizing proneness can be interfaced with measures of other facets of inhibitory control or trait impulsivity—including facets that connect with disorders outside the externalizing spectrum (e.g., Fischer et al., 2008).

In summary, the current study represents an important advance in conceptualization and assessment of inhibitory control capacity—an individual difference attribute that plays a critical role in adaptive performance and maladaptive behavior. Findings from this work serve to link separate lines of research on behavioral performance and brain response indicators of inhibitory control as related to externalizing problems. Results from the current work also highlight the need for developing multidomain indices of core neurobehavioral constructs to effectively assess liabilities for mental health problems. The strategy used to integrate measures of inhibitory control across different domains in the current work can be readily applied to other neurobehavioral trait constructs such as threat sensitivity (Weinberg et al., 2016; Yancey et al., 2016) and reward responsiveness (Hajcak Proudfit, 2015). Continuing work of this kind can serve the dual role of advancing multilevel understanding of mental health/illness and progressing toward a new, integrated approach to assessment of disorder liability and adaptive potential.

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Author Contributions
N. C. Venables and C. J. Patrick developed the study concept. All authors contributed to the study design. M. J. Kane and R. W. Engle provided behavioral task procedures and input to N. C.

Venables and C. J. Patrick on derivation of behavioral dependent measures. Testing and data collection were coordinated by N. C. Venables, J. Foell, J. R. Yancey, and C. J. Patrick. N. C. Venables performed the data analysis under the supervision of C. J. Patrick and with input by all authors. N. C. Venables drafted the manuscript and all other authors provided critical feedback. All the authors approved the final version of the manuscript for submission.

Notes

1. Stop signal reaction time (SSRT) was also computed from this task (as mean RT for "go" trials minus mean delay time for "stop" trials), but was not used as a behavioral indicator in analyses because it showed near-zero correlations with other task-performance indicators (rs = –.006 to .03, ps > .78); the proactive inhibition variable from this task, which correlated as expected with Antisaccade accuracy and Sustained Attention to Response Task (SART) RT variability (rs = .17 and –.24, respectively; see Table 2), though only weakly with Stroop RT (r = –.07), was included.

2. Amplitude of the error-related negativity (ERN; Falkenstein, Hohnsbein, & Hoormann, 1991) was also derived from the arrow flanker task, but was not used as a neurophysiological indicator in modeling analyses because of very weak correlations with other brain response indicators (rs = |.07–.18|; median r = .13).

3. Of note, two candidate indicators from the domains of task performance and brain response— SSRT and brain ERN—were excluded from current analyses. SSRT, a variable previously found to relate to self-reported impulsivity (Logan, Schachar, & Tannock, 1997), did not correlate reliably with other performance indicators of cognitive control in the current study sample. By contrast, the proactive inhibition score from the Stop Signal task, which reflects strategic adjustment of behavior to meet anticipated control demands of the task (Verbruggen & Logan, 2009), did covary with other performance indices that covaried in turn with brain and trait-scale indicators. The implication is this index of strategic adaptation taps into general inhibitory control capacity more so than SSRT. Relevant to this, Barch, Braver, Carter, Poldrack, and Robbins (2009) pointed out that "strategic adaptations that result in slowing of go response times" in the Stop Signal task may affect the validity of SSRT as an index of dynamic adjustments in cognitive control. Also in contrast with prior findings (Nelson et al., 2011), ERN response from the flanker task was largely unrelated to P3 indicators, whereas the error-positivity response from this same task (i.e., P3e) did emerge as an indicator of the neurophysiological

domain. Although ERN amplitude has been characterized as an indicator of disinhibitory traits and problems (e.g., Hall, Bernat, & Patrick, 2007; Olvet & Hajcak, 2008), it has also been proposed as an index of *sustained threat*, an RDoC construct related to anxiety (Weinberg et al., 2016; see also Olvet & Hajcak, 2008). Furthermore, ERN amplitude has recently been shown to relate in complex configural ways to personality traits including negative affectivity, conscientiousness, and impulsivity (Hill, Samuel, & Foti, 2016). These findings together with results from the current study highlight the need for further research into ERN as an index of inhibitory control capacity.

4. Correlations between indicators from different domains are included in the Supplemental Material.

5. We fit an alternative three subfactor/one higher order factor model that included two variables (SSRT and ERN) considered as potential indicators for the behavioral-performance and neurophysiological subfactors, respectively, but omitted because of low rs with other indicators from these domains. This alternative model exhibited less than adequate fit, $\chi^2(74) = 145.29$, $p < .001$, RMSEA = .08, 90% CI = [.061, .10], SRMR = .083, CFI = .84, TLI = .81, because of very weak loadings of the two added indicators on their respective subfactors (−.12 for SSRT and −.06 for ERN, ps > .36). In addition, comparative fit indices revealed significantly weaker fit for this model compared with the model omitting these two indicators, ΔBIC = 2204, $\Delta\chi^2 = 76.62$, $\Delta df = 23$, $p < .001$.

6. Simple means tests for gender differences in scores on the factors of the model revealed a trend-level difference only for the psychometric scale factor, with women scoring lower on average than men, Ms = −.065 and .048, respectively; $t(147) = 1.99$, $p = .05$. Male and female participants did not differ significantly in scores on the behavioral performance ($p > .20$), neurophysiological ($p > .07$), or cross-domain factors ($p > .20$). In addition, regression analyses were used to test for moderating effects of gender on correlations for factors of the model with the various criterion measures. These analyses revealed no significant moderating effects (all ps > .10).

7. It warrants mention that two of the self-report measures included in our model (ESI-DIS scale, SO scale) contain a small number of items (four each) pertaining to illicit acts (e.g., theft) or excessive drinking. To address whether these particular items might have amplified correlations for the self-report domain factor of the model with criterion measures of antisocial behavior and substance abuse, we computed scores for these two scales after omitting these items, and respecified our CFA model with these revised scale variables included (along with PPI-DIS and MPQ-DIS) as indicators. The loadings of the four scales on the self-report domain factor changed very little, and scores for this revised scale-domain factor correlated almost perfectly ($r = .99$) with scores for the original-model factor—consistent with the idea that the self-report factor of the original model mainly reflects trait-dispositional tendencies that are related in turn to specific externalizing behaviors.

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