



From alcohol to aggression: Examining the structure and nomological network of dysregulated behaviors in a trauma-exposed community sample

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Abstract

Objective: A large body of research has shown that alcohol use, drug use, aggression, and self-harm often co-occur within the same individuals, suggesting the possibility of shared etiologies. Research has yet to determine the factor structure of these dysregulated behaviors.

Methods: Participants ($M_{\text{age}} = 40.33$; 74% women) completed self-report and interview-based measures of dysregulated behaviors (alcohol use, drug use, aggression, and self-harm), emotion dysregulation, maladaptive personality traits, and symptoms of DSM disorders (e.g., borderline personality disorder [BPD], depression).

Results: Results showed support for a bifactor model (i.e., all indicators load on a common dysregulated behavior factor and on unique alcohol, drug, aggression, and self-harm factors), which provided a better fit to the data than other models. In line with our hypotheses, the general dysregulated behavior factor was positively associated with emotion regulation difficulties, negative affect, and BPD symptoms.

Conclusions: These results have implications for several areas of psychopathology and intervention research.

KEYWORDS

binge drinking, emotional dysregulation, self-injury, violence

1 | INTRODUCTION

A large body of research has shown that behaviors such as alcohol use, drug use, aggression, and self-harm often co-occur within the same individuals (Guo et al., 2017; Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017). Research also shows that engagement in one behavior (e.g., alcohol intoxication) increases the likelihood of other behaviors (self-harm, aggression; Bresin & Mekawi, 2020; Duke et al., 2018). These findings combined with overlap in theories of drug and alcohol use, aggression, and self-harm (e.g., negative reinforcement; Baker et al., 2004; Chapman et al., 2006; Conger, 1956; Hokanson, 1974) suggests the possibility of common etiological factors. These dysregulated behaviors have serious consequences for both the individual engaging in the behaviors, those around them, and society as a whole. Thus, understanding the points of convergence and divergence across these behaviors has the potential to improve etiological models of psychopathology and inform the development of transdiagnostic interventions designed to reduce the frequency of these behaviors. Determining an empirical taxonomy of these dysregulated behaviors is necessary to guide etiological research elucidating their unique and shared functions. Therefore, the goal of this study was to test competing factor structures of a subset of dysregulated behaviors and establish the nomological network of these factors in relation to emotion dysregulation, maladaptive personality traits, and symptoms of psychopathology.

Bresin (2020; also see Selby & Joiner, 2009) defines dysregulated behaviors as active behaviors that have benefits (e.g., reduction in negative affect) in the short-term (seconds to hours) but cause serious distress or impairment to the individual and/or those around them in the long-term (days, weeks, and years). Dysregulated behaviors are proposed to be a subset of avoidance behaviors and impulsive behaviors and can be distinguished from compulsive behaviors (e.g., Guo et al., 2017) and thrill-seeking behaviors (Bresin, 2020). Although the term dysregulated behavior comes from research on borderline personality disorder (BPD; Selby & Joiner, 2009), these behaviors occur in a broader population of individuals and, therefore, represent a transdiagnostic phenomenon (Bresin, 2020).

Although it has been established that these behaviors co-occur, the specific factor structure is yet to be determined (Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017). One possibility is that there is one common vulnerability (or vulnerabilities) that underlie all dysregulated behaviors, and there are no unique vulnerabilities for specific behaviors. This would be consistent with a higher-order factor model (see Figure 1 right panel), where the covariance among factors is explained by a higher-order factor (Markon, 2019). Kingston et al. (2011) found evidence for a higher-order model of dysregulated behaviors, where a general dysregulated behavior factor was indicated by eight lower-order factors (e.g., self-harm, drug use, excessive alcohol use, and aggression). Another possibility is that there is one common vulnerability that applies to all dysregulated behaviors and separate unique vulnerabilities that apply to specific behaviors. This would be consistent with a symmetrical bifactor model (Markon, 2019), where all indicators load on a common factor and on unique factors (see Figure 1, left panel). Sadeh and Baskin-Sommers (2017) found support for a bifactor model of their 38-item Risk, Impulsive, and Self-destructive Behavior Questionnaire, where all items loaded on a general factor with eight specific factors (e.g., drug-related behavior, self-harm, aggression, and heavy alcohol use).

These preliminary factor structure studies provide some clues to the underlying nature of dysregulated behaviors; however, several questions remain. First, because one study supports each model (e.g., Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017), there is no clear evidence to support one model over the other. Second, neither study compared a higher-order factor to a bifactor model (Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017), which could provide support for one model over another. Given that the correlations between dysregulated behaviors are moderate and individuals who engage in one do not necessarily engage in all dysregulated behaviors (e.g., Kingston et al., 2011), it could be assumed that there would be common and unique factors that contributed to dysregulated behaviors. This may suggest that a bifactor model would best fit the data. Third, these studies used primarily White samples with high educational attainment, which can limit generalizability. Finally, there has been limited work elucidating the nomological network (i.e., the pattern of relations among

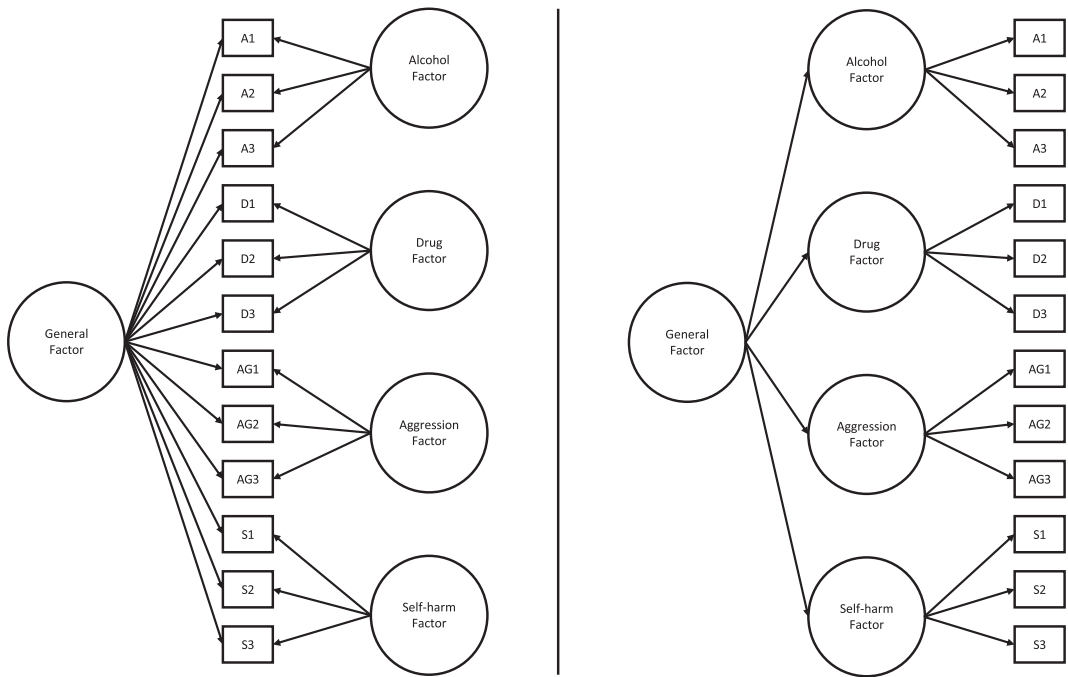


FIGURE 1 Conceptual models for the symmetric bifactor model (left panel) and higher-order model (right panel). Note: A1–A3 = Alcohol Indicator 1–3; D1–D3 = Drug Indicator 1–3; AG1–AG3 = Aggression Indicator 1–3; S1–S3 = Self-harm Indicator 1–3

antecedent, mediator, and outcome variables; Hagger et al., 2017) of the shared variance of dysregulated behaviors (see Sadeh & Baskin-Sommers, 2017 for an example).

1.1 | Nomological network of dysregulated behaviors

Establishing the correlates of common and unique factors of dysregulated behaviors (i.e., the nomological network) can inform theory development. For example, identifying the nomological network of the common factor would aid in the development of an etiological model common to all dysregulated behaviors. Similarly, identifying correlates of unique factors, if identified, could lead to the development of etiological theories unique to specific behaviors that could be used to develop more effective interventions for dysregulated behaviors. Although there are several candidate correlates, we focused on emotion dysregulation, personality, and symptoms of psychopathology because of their inclusion in theories of substance use, self-harm, and aggression (e.g., Chapman et al., 2006; Selby & Joiner, 2009).

1.1.1 | Emotion dysregulation

Emotion dysregulation is a factor common to theories of dysregulated behaviors (e.g., Bresin, 2020; Selby & Joiner, 2009). For example, Selby and Joiner (2009) posit that individuals engage in dysregulated behaviors to deal with uncontrollable unpleasant emotions. This suggests that emotion dysregulation may contribute to several dysregulated behaviors. Along these lines, self-reported difficulties in regulating emotions have been found to

correlate positively to individual dysregulated behaviors in separate studies (e.g., alcohol use, aggression: Grigorian et al., 2020; self-harm: Wolff et al., 2019) and within the same study (Buckholdt et al., 2015; Miller & Racine, 2020). Moreover, there is some evidence that, at least when ignoring overlap among behaviors, self-harm and alcohol use are more strongly related to different aspects of emotion dysregulation (e.g., difficulties engaging in goal-directed behaviors when upset) than drug use (Buckholdt et al., 2015; Miller & Racine, 2020). It is currently unclear whether these unique associations would be maintained when common variance across dysregulated behaviors is accounted for.

1.1.2 | Maladaptive personality traits

Research using several instruments in clinical (e.g., Samuel et al., 2010) and normal range of personality (e.g., Goldberg, 1999) has coalesced on a five-factor model of personality traits. The traits range from normal to maladaptive, with the maladaptive extremes being negative affectivity, disinhibition, antagonism, detachment, and psychoticism. Maladaptive personality traits can be linked to symptoms of psychopathology (Widiger et al., 2019). For example, the Hierarchical Taxonomy of Psychopathology (Hi-TOP; Kotov et al., 2017), a recently proposed alternative to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013)*, explains co-occurring symptoms (e.g., depression and posttraumatic stress disorder [PTSD]) by identifying higher-order factors (or spectra) that map on to the maladaptive personality traits (e.g., negative affectivity; Kotov et al., 2017).

The Hi-TOP may be used to develop several predictions about how common and unique variance in dysregulated behaviors may be associated with maladaptive personality traits. First, given that negative affectivity has been shown to positively relate to the common variance across many forms of psychopathology (e.g., Caspi et al., 2014), it could be assumed that the shared variance in dysregulated behaviors would have a positive relation with negative affectivity. Second, the Hi-TOP places substance use disorders underneath the disinhibited externalizing spectra, suggesting that substance use may be uniquely related to disinhibition. Third, the Hi-TOP predicts that disorders characterized by aggression (e.g., intermittent explosive disorder, antisocial personality disorder) are a function of the antagonistic externalizing spectra. This suggests that aggression may be uniquely related to antagonism. Finally, although self-harm is not directly modeled in the Hi-TOP, given the strong positive relation between self-harm and BPD, depression, PTSD, and eating disorders (e.g., Bentley et al., 2015; Cucchi et al., 2016), it could be posited that self-harm may be uniquely related to negative affectivity. This fits with studies showing that negative affectivity is positively related to self-harm (e.g., Brown, 2009).

1.1.3 | Symptoms of psychopathology

Within the *DSM-5*, dysregulated behaviors are most prominently represented in the symptoms of BPD. Specifically, alcohol and drug use, aggression, and self-harm are included in the symptoms of BPD. Thus, it is likely that the general dysregulated behavior factor would be strongly positively associated with symptoms of BPD. In line with this prediction, Sadeh and Baskin-Sommers (2017) found that their general factor and unique factors were positively correlated with self-reported BPD symptoms. Several other *DSM* diagnoses (e.g., major depression, PTSD, and panic disorder) have been found to have positive relations with alcohol and drug use (e.g., Cosci et al., 2007; Lai et al., 2015; Mills et al., 2006), aggression (e.g., Dutton & Karakanta, 2013; Jakupcak et al., 2007), and self-harm (e.g., Bentley et al., 2015; Fox et al., 2015; Kimbrel et al., 2016). Currently, it is unknown whether these correlations may be explained by a common dysregulated behavior factor or whether there may be unique associations between some dysregulated behaviors and symptoms of psychopathology.

1.2 | Current study

Previous research has shown that there is significant co-occurrence of dysregulated behaviors such as problematic drug and alcohol use, aggression, and self-harm (Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017); however, very few studies have examined the factor structure of dysregulated behaviors and established the common and unique correlates. Moreover, the studies that have examined the factor structure have not directly compared higher-order and bifactor models. The goal of this study was to examine the factor structure of a subset of dysregulated behaviors (i.e., those with the highest societal impact; Bresin, 2020) in a sample with a high likelihood for endorsement of dysregulated behaviors (i.e., a large urban-dwelling, trauma-exposed sample). The sample was also more racially diverse than those used in previous studies. Based on previous research showing that the correlations between dysregulated behaviors are moderate and individuals who engage in one do not necessarily engage in all dysregulated behaviors (e.g., Kingston et al., 2011), we tentatively predicted that a bifactor model would be the better fit to the data than a higher-order or correlated factor model. After determining the best fitting solution, we extracted factor scores and examined their relations with several correlates (i.e., emotion dysregulation, maladaptive personality traits, symptoms of BPD, depression, PTSD, and panic disorder) to establish construct validity. We predicted that the general dysregulated behaviors factor would be positively correlated with emotion dysregulation, BPD symptoms, and negative emotionality (Kotov et al., 2017; Selby & Joiner, 2009). We explored the relation between the general factor and symptoms of depression, PTSD, and anxiety sensitivity. In terms of unique factors, we predicted that the unique drug and alcohol use factor would be positively correlated with disinhibition, the unique aggression factor would be positively correlated with antagonism, and the unique self-harm factor would be positively correlated with negative emotionality (Kotov et al., 2017).

2 | METHODS

2.1 | Procedure

The current study focuses on a secondary analysis of data collected from January 2006 through February 2020 as part of a larger NIH-funded study on the risk factors for the development of PTSD in an urban population with low socioeconomic resources. Participants were recruited from waiting rooms in the gynecology and primary care medical clinics at a publicly funded hospital and the emergency department waiting room of a pediatric, non-profit hospital in Atlanta, Georgia. We did not narrow recruitment to specific criteria but approached any individual in the waiting room. To be eligible for participation, participants had to be at least 18 years old and able to give informed consent. After signing the informed consent approved by the university and hospital ethics review boards, an interview was administered with questionnaires regarding trauma history and psychological variables. Trained research assistants administered this interview (approximately 45–75 min). More comprehensive assessments of psychological functioning were conducted in a separate associated study drawn from the pool of participants who completed the initial assessment. Participants were compensated for their time.

2.2 | Participants

Although the *n*'s varied from measure to measure, the current sample included 3707 adults who ranged from 18 to 65 years old ($M = 40.33$; $SD = 13.17$). The majority of the sample identified as female (74%) and non-Hispanic/Latinx (98%). The racial breakdown of the sample was as follows: 93% African American, 3% Caucasian/White, 2% mixed race, 1% Latinx, and 1% other. In terms of highest level of education achieved, 21% reported not completing a high school degree, 40% reported completing high school or GED, and 39% reported completing at least some college or

technical school. Approximately 68% of the sample indicated they were currently unemployed, and the majority reported a monthly household income of less than \$2000/month (83%). The full battery of measures was changed periodically during the 14 years of data collection; therefore, the number of participants for each measure varies.

3 | MEASURES

3.1 | Dysregulated behaviors

3.1.1 | Alcohol Use Disorders Identification Test (de Meneses-Gaya et al., 2009)

The Alcohol Use Disorders Identification Test (AUDIT) is a 10-item self-report screening instrument for problematic alcohol use occurring in the last year. Items on the AUDIT assess both consumption (e.g., "How often did you have a drink containing alcohol?") and consequences (e.g., "How often have you failed to do what was normally expected of you because of drinking?"), and responses are coded on a 0 (*never*) to 4 (*daily or almost daily*) scale. The total score ($M = 9.81$, $SD = 9.72$) was used in the current study ($\alpha = 0.88$), and data were available for $N = 3707$ participants.

3.1.2 | Drug Abuse Screening Test (Yudko et al., 2007)

The Drug Abuse Screening Test (DAST) is a 10-item self-report measure of current and lifetime substance use (excluding alcohol) and associated problems (e.g., "Have you had blackouts as a result of drug use?") which participants responded on a dichotomous scale of 0 (*no*) and 1 (*yes*). Items were summed to create a total score ($M = 3.32$, $SD = 2.82$, $\alpha = 0.77$) for lifetime use, and data were available for $N = 3621$ participants.

3.1.3 | Behavior Questionnaire-Short (Gillikin et al., 2016)

The Behavior Questionnaire-Short (BQ-S) is an internally constructed 5-item scale designed to assess aggressive behavior frequency. This measure was based on the Conflicts Tactics Scale (Straus et al., 1996), a measure commonly used to assess aggressive behavior. Items from the BQ-S ask participants how often in their lifetime they have perpetrated violent acts (e.g., punched, hit, pulled a knife or gun, stabbed, or shot at someone) using a scale of 0 (*never*) to 4 (*more times than I can count*). The sum was computed to obtain the total score ($M = 4.49$, $SD = 3.72$, $\alpha = 0.76$), and data were available for $N = 2968$ participants.

3.1.4 | Suicide and Self-Harm

The Suicide and Self-Harm (SSH) is an internally constructed, face valid 4-item scale designed to assess the frequency of suicidal behaviors. Items from the SSH ask participants how often they have engaged in various self-harm behaviors (e.g., intentionally putting oneself in harm's way, intentionally hurting oneself without suicidal intent, being hospitalized for suicidal ideation, and attempting suicide) using a scale of 0 (*never*) to 4 (*more times than I can count*). We focused on the two items more directly tied to self-harm (intentionally hurting oneself without suicidal intent and attempting suicide). The items were summed to obtain the total score ($M = 0.36$, $SD = 0.98$, $\alpha = 0.60$), and data were available for $N = 1889$ participants.

4 | MEASURES: CONSTRUCT VALIDITY

4.1 | Emotion dysregulation

4.1.1 | Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2003)

The Difficulties in Emotion Regulation Scale (DERS) is a psychometrically valid 36-item self-report measure that assesses problems with emotion regulation. Participants rated items on a scale of 1 (*almost never, 0%-10%*) to 5 (*almost always, 91%-100%*). The DERS includes six subscales, including attention to and awareness of emotions (e.g., "I pay attention to how I feel;" reverse-scored; $\alpha = 0.74$), acceptance of one's emotions (e.g., "When I'm upset, I become angry with myself for feeling that way; $\alpha = 0.89$), the ability to execute goal-directed behavior (e.g., "When I'm upset, I have difficulty focusing on other things; $\alpha = 0.83$), the ability to inhibit impulsive behavioral urges (e.g., "I experience my emotions as overwhelming and out of control," $\alpha = 0.86$), access to contextually appropriate emotion regulation strategies (e.g., "When I'm upset, it takes me a long time to feel better;" $\alpha = 0.88$) and clarity into one's emotional state (e.g., "I have no idea how I am feeling;" $\alpha = 0.79$). Items were summed to create each subscale data were collected for $N = 599-600$ participants.

4.2 | Maladaptive personality traits/(Hi-TOP spectra)

4.2.1 | Personality Inventory for DSM-5-Brief Form (Anderson et al., 2018)

The Personality Inventory for DSM-5-Brief Form (PID-5-BF) is a 25-item self-report measure of pathological personality traits included in the *DSM-5* Alternative Model of personality disorder. Participants indicated how much each item described themselves on a scale of 1 (*very false or often false*) to 4 (*very true or often true*). All five subscales of the PID-5-BF were included as follows: negative affect (e.g., "I get emotional easily, often for very little reason;" $\alpha = 0.82$), detachment (e.g., "I often feel like nothing I do really matters;" $\alpha = 0.76$), antagonism (e.g., "I use people to get what I want;" $\alpha = 0.52$), disinhibition (e.g., "I'm not good at planning ahead;" $\alpha = 0.52$) and psychoticism (e.g., "I have seen things that weren't really there;" $\alpha = 0.69$). The items were summed to obtain each subscale score and data were available for $N = 384$ participants.

4.3 | Symptoms of psychopathology

4.3.1 | Structured Clinical Interview for DSM-IV (First & Gibbon, 2004)

The Structured Clinical Interview for DSM-IV (SCID-IV) was used to assess BPD symptoms (e.g., "Inappropriate, intense anger or difficulty controlling anger) using a semi-structured interview. Participants received a symptom score (i.e., 1 = *no evidence of symptom*, 2 = *sub-threshold*, 3 = *symptom present*) based on the extent to which they were able to provide examples of each symptom from their personal experiences. The items were summed to obtain the total score ($\alpha = 0.87$), and data were available for $N = 326$ participants.

4.3.2 | Beck Depression Inventory-II (Beck et al., 1996)

The Beck Depression Inventory-II (BDI-II) is a reliable and well-validated 21-item measure used to assess depressive symptoms. For each item, participants indicated which of four statements best described the way they had been

feeling over the past two weeks. Each statement had a corresponding score, ranging from 0 (e.g., “I do not feel sad”) to 3 (e.g., “I am so sad or unhappy that I can’t stand it”). The scores were summed to create a total depressive symptom severity score ($\alpha = 0.93$), and data were available for $N = 3246$ participants.

4.3.3 | Mini-International Neuropsychiatric Interview (Sheehan et al., 1998)

The Mini-International Neuropsychiatric Interview (MINI) for *DSM-IV-TR* is a validated and reliable structured diagnostic interview that assesses mood, anxiety, substance use, and psychotic disorders. In this study, we used the depressive disorders module, which assesses symptoms of a major depressive episode (e.g., appetite changes, changes in sleep). Participants received a symptom score (i.e., 0 = *symptom absent*, 1 = *symptom present*) based on the extent to which they were able to provide examples of each symptom occurring within a two-week period and confirm that this experience deviated from typical behavior. Data were collected from $N = 411$ for current and $N = 383$ for past.

4.3.4 | Modified Posttraumatic Stress Disorder Symptom Scale (Falsetti et al., 1993)

The Modified Posttraumatic Stress Disorder Symptom Scale (mPSS) is a reliable and well-validated 17-item measure used to assess current PTSD symptoms based on *DSM-IV-TR* (American Psychiatric Association, 2000) criteria. Participants indicated the degree to which they experienced symptoms (e.g., “persistently been making efforts to avoid thoughts or feelings associated...?”) regarding traumatic experiences on a scale of 0 (*not at all*) to 3 (*five or more times a week*). Scores were summed to create a total score ($\alpha = 0.92$), and data were collected from $N = 3168$ participants.

4.3.5 | Clinician-Administered PTSD Scale (Blake et al., 1995; Weathers et al., 2001)

The Clinician-Administered PTSD Scale (CAPS) is a psychometrically valid, standardized interviewer-administered diagnostic instrument for current PTSD based on DSM criteria. The CAPS for DSM-IV and DSM-5 were both used in this study (the adoption of CAPS-5 occurred upon its release). A sum of PTSD symptoms based on CAPS items was used as the measure of PTSD symptoms. To combine CAPS-IV and CAPS-5, all scores were weighted to balance (1) differences in the number of items (17 for CAPS-IV and 20 for CAPS-5), and (2) differences in scoring (for CAPS-IV frequency and intensity scores were separated while for CAPS-5 scoring frequency and intensity scores were combined into one severity rating on a scale of 0 (*absent*) to 4 (*extreme/incapacitating*)). The severity rating is derived based on the scores for frequency and intensity for each item, which are still scored separately for each item, and thus represent the same components of the ratings as CAPS-IV (Weathers et al., 2018). Interrater reliability (IRR) in this sample has been previously reported and has shown good IRR for the current diagnosis of PTSD ($k = 0.83$). Data were collected from $N = 839$ participants.

4.3.6 | Anxiety Sensitivity Inventory (Peterson & Heilbronner, 1987)

The Anxiety Sensitivity Inventory (ASI) is a 16-item self-report inventory of sensitivity to symptoms of anxiety. The ASI is thought to measure the fear of internal body sensations central to panic disorder (e.g., McNally, 2002). Participants indicated the degree to which they experienced anxiety sensitivity (e.g., “When I notice that my heart is

beating rapidly, I worry that I might have had a heart attack") on a scale of 0 (*very little*) to 4 (*very much*). Scores were summed to create a total score ($\alpha = 0.89$), and data were collected from $N = 3003$ participants.

5 | RESULTS

5.1 | Data analytic plan

Data analysis proceeded in two stages. In the first stage, we fit four-factor analytic structural equation models: (1) a four-factor model where alcohol use, drug use, aggression, and self-harm were separate correlated factors, (2) a three-factor model where alcohol and drug use, aggression, and self-harm, were separate correlated factors, (3) a higher-order model, where alcohol use, drug use, aggression, and self-harm were indicators of a higher-order dysregulated behavior factor, and (4) a fully symmetric bifactor model where all items indicated a general dysregulated behavior factor and four unique factors (alcohol use, drug use, aggression, and self-harm). Models were compared on their fit statistics (CFI, TFI, SRMR, and RMSEA) to determine the best fitting model. We used threshold values recommended by Hu and Bentler (1999) as indicators of fit: CFI & TFI > 0.95, SRMR < 0.10, and RMSEA < 0.06.

Because simulation studies show that fit indices are biased toward bifactor models (e.g., Greene et al., 2019), we also calculated two additional indices that have been recommended to address biases in traditional fit indices (Greene et al., 2019). The expected value of cross-validation index (ECVI; Browne & Cudeck, 1992) is a single sample index of the expected discrepancy between the calibration sample and validation sample, with smaller values indicating a better fitting model. The Vuong test for non-nested models tests which model is a closer fit to the underlying data (Merkle et al., 2016). Structural equation models were fit in the lavaan package in R (Rosseel, 2012). Missing data were addressed using Full-information Maximum Likelihood estimation. We chose Full-information Maximum Likelihood estimation over listwise deletion because listwise deletion has been shown to lead to biased results (e.g., Enders & Bandalos, 2001). We chose Full-information Maximum Likelihood estimation over multiple imputations due to several reasons (cf. Allison, 2012). Most importantly, multiple imputations introduce several choices (e.g., estimation method, number of datasets to impute) that can affect the results. Moreover, full-information Maximum Likelihood estimation has been shown to be robust to missing data that are missing completely at random or missing at random (e.g., Enders & Bandalos, 2001).¹ In our data, Little's test for missing completely at random was significant, $\chi^2(3359) = 6394.92, p < 0.001$. Therefore, we assumed the data were missing at random.

In the second stage of the analysis, the factor scores from the best fitting factor model were correlated with the construct validity measures (e.g., maladaptive personality traits). To adjust for multiple tests, we adjusted our p -values to maintain a false discovery rate of 0.05% (Benjamini & Hochberg, 1995). Tables S3–S5 also show unadjusted 99% confidence intervals for correlations, which are helpful for interpretation because the false-discovery rate adjustment only applies to the set of tests we conducted. We interpreted the size of the correlations based on Cohen's (1992) recommendations (0.1 = small, 0.3 = medium, and 0.5 = large). In addition to the main results, the Supporting Information includes exploratory correlations between the factor scores and traumatic and stressful events.

5.2 | Stage 1: Factor analytic structural equation models

Preliminary analyses indicated high levels of endorsement for lifetime alcohol use (92% endorsement), drug use (96% endorsement), and aggression (85% endorsement) in the sample. Self-harm was less common (17% lifetime endorsement). Model fit statistics for the factor models are shown in Table 1. All fit statistics favored the bifactor

TABLE 1 Model fit statistics for factor analytic structural equation models

Model	χ^2	df	CFI	TFI	SRMR	RMSEA [90% CI]	ECVI
One-factor	3601.28	324	0.62	0.66	0.10	0.112 [0.109, 0.115]	4.614
Three-factor	5920.38	311	0.78	0.76	0.08	0.069 [0.067, 0.070]	1.642
Four-factor	3520.86	318	0.87	0.85	0.05	0.052 [0.051, 0.054]	0.997
Higher-order	3538.96	320	0.87	0.86	0.05	0.052 [0.051, 0.054]	1.001
Bifactor	2505.46	297	0.91	0.90	0.05	0.045 [0.043, 0.046]	0.734

Abbreviations: CFI, Comparative Fit Index; ECVI, Expected Value of Cross-Validation Index; RMSEA, Root Mean Square Error of Approximation; SRMR, Standardized Root Mean-square Residual; TFI, Tucker Lewis Index.

model as the best fitting model. The Vuong test comparing the bifactor to the higher-order model, $Z = 9.70$, $p < 0.001$, and four-factor model, $Z = 9.49$, $p < 0.001$, showed that the bifactor model was a significantly better fit to the data. Considering all this evidence together, we interpreted the bifactor model as the best fitting model. The Supporting Information contains additional analyses with bifactor (S-1) models (Eid et al., 2017), which provide additional evidence that the fully symmetrical bifactor model was the best fitting model.

The standardized loadings (see Table S8) on the dysregulated behavior factor were generally modest ($M = 0.37$, $SD = 0.18$). The loadings for the unique factors were much stronger: alcohol use ($M = 0.54$, $SD = 0.16$), drug use ($M = 0.21$, $SD = 0.33$), aggression ($M = 0.61$, $SD = 0.10$), self-harm ($M = 0.66$, $SD = 0.20$). All loadings were significant at $p < 0.001$. Taken together, these results may suggest that there is meaningful shared variance across dysregulated behaviors; however, there is more variance shared within specific dysregulated behaviors than across different dysregulated behaviors.

5.3 | Stage 2: Construct validity

Figure 2 displays the correlation heatmap between the factor scores and measures of emotion dysregulation. The general dysregulated behaviors factor had significant positive correlations with all the subscales of the DERS that were medium in size. Thus, the general dysregulated behaviors factor is associated with high levels of emotion dysregulation. The unique alcohol use factor was not significantly correlated to any of the subscales of the DERS. The unique drug use factor was significantly positively related to all DERS subscales, aside from lack of awareness with small effect sizes. The unique aggression factor was significantly positively related to all DERS subscales, aside from lack of awareness with medium effect sizes for goals and impulse, and small effect sizes for strategies, nonacceptance, and clarity. The unique self-harm factor was significantly positively related to the goals, impulse, strategy, and nonacceptance DERS subscales.

Taken together, there are some similarities across the unique factors. None of the unique factors were related to the lack of awareness subscale of the DERS. The effect sizes were smaller for the unique factors compared to the common factor. The one exception was the unique aggression factor had a medium-sized positive relation with the DERS impulsive control subscale. There were also some notable differences. The unique alcohol use factor was not related to difficulties regulating emotions. Aside from the unique alcohol use factor, the unique self-harm factor was the only unique factor not related to lack of emotional clarity.

Figure 3 displays the correlation heatmap among the extracted factor scores and maladaptive personality traits. The general dysregulated behaviors factor was significantly positively related to all the subscales of the PID-5-BF with small-to-medium effect sizes. This was predicted for negative affect, but not disinhibition, antagonism, detachment, or psychoticism. As predicted, the unique self-harm factor was significantly positively correlated with the

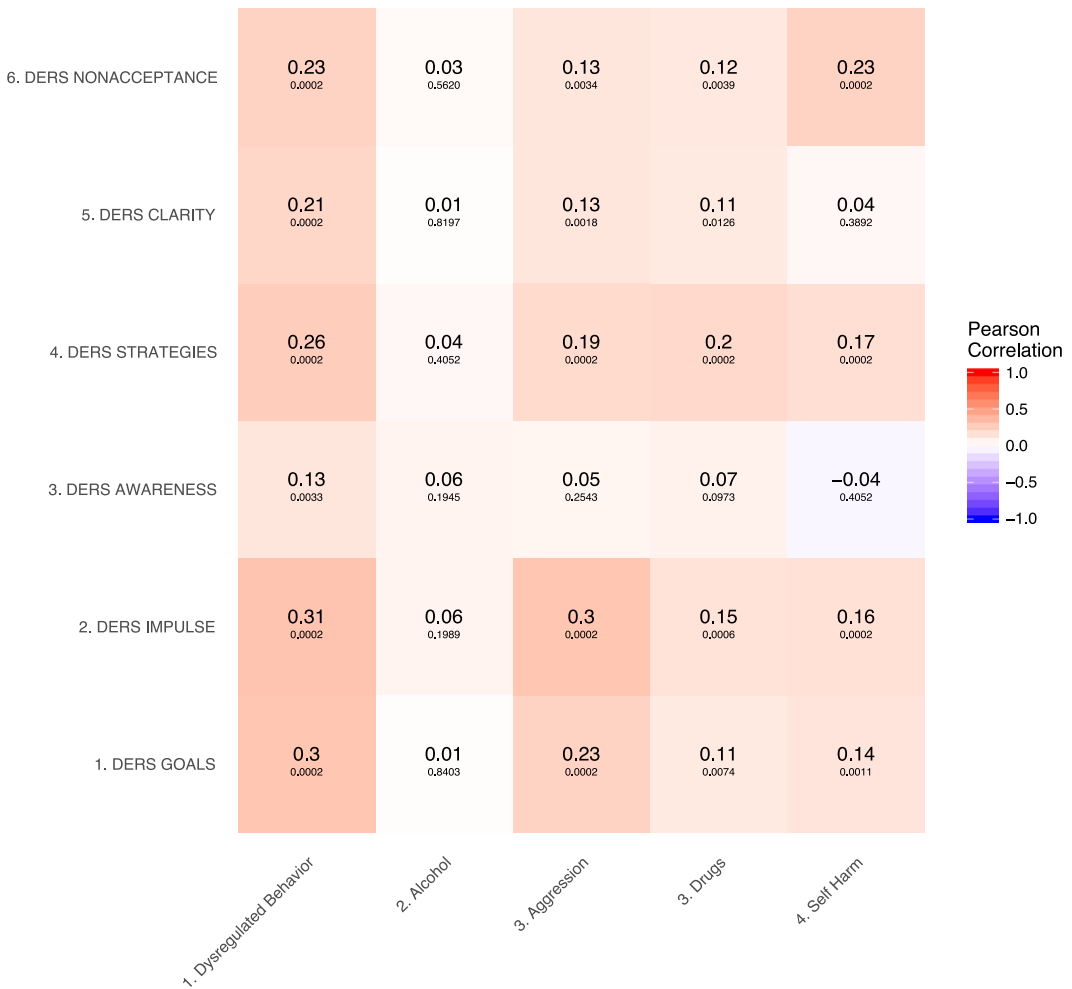


FIGURE 2 Correlations (and adjusted p -values) among the factor scores and emotion dysregulation (left panel; $N = 600$) and pathological personality traits (right panel; $N = 384$). Note: DERS=Difficulty Regulating Emotions Scale

negative affect scale of the PID-5-BF. The other unique factors also had small positive correlations with negative affectivity. Inconsistent with our prediction, disinhibition was not significantly related to alcohol use but was positively related to drug use. Consistent with our prediction, antagonism was related to the unique aggression factor. All unique factors aside from alcohol use were positively correlated with the detachment, disinhibition, and psychoticism scales of the PID-5-BF with small-to-medium effect sizes. Drug use was also significantly positively related to antagonism with the small-to-medium effect size.

Figure 4 displays the correlations between the factor scores and symptoms of BPD, depression, PTSD, and anxiety sensitivity. As predicted, the general factor had a medium-sized positive correlation with symptoms of BPD. The general dysregulated behaviors factor was also significantly positively related to measures of depression, PTSD, and ASI with small-to-medium effect sizes.

The unique alcohol factor had small positive significant correlations with the BDI, PSS, and ASI. All the correlations with the interview-based measures were not significant. The unique drug use factor and aggression factor shared a similar pattern of correlations. They both had significant medium-sized correlations with BPD symptoms and small correlations with depression and PTSD symptoms. The one difference is that the unique aggression factor

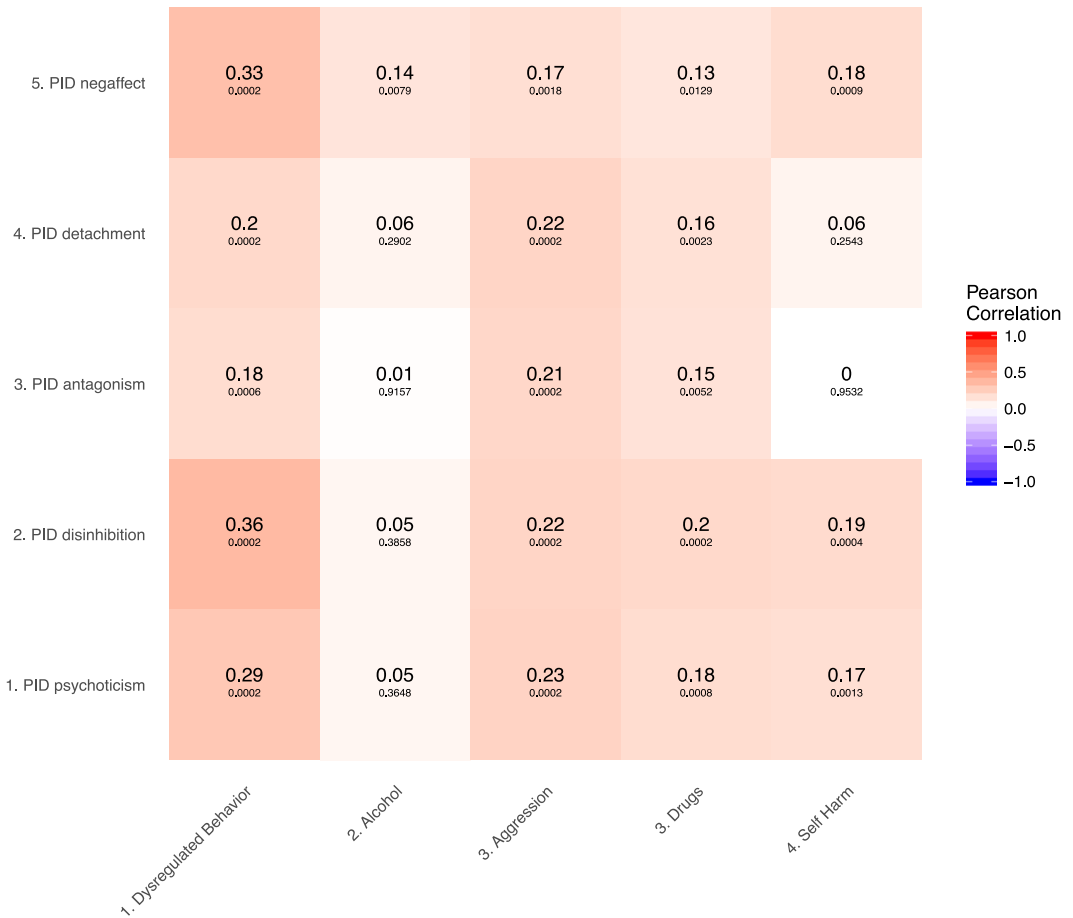


FIGURE 3 Correlations (and adjusted p-values) among the factor scores and pathological personality traits (right panel; N = 384). Note: PID=Personality Inventory for DSM-5; Negaffect=negative affectivity

was significantly positively related to anxiety sensitivity with a small effect size, whereas the unique drug factor was not. The unique self-harm factor had significant positive correlations with BPD symptoms and BDI, but not interview measures of depression. Interestingly, the unique self-harm factor was significantly positively related to PTSD symptoms assessed via self-report and interview with small-to-medium effect sizes. The unique self-harm factor was also significantly positively correlated with anxiety sensitivity with a small effect size. These results show that both the general and unique factors are generally positively related to symptoms of the DSM disorders included in our study.

6 | DISCUSSION

The goal of this paper was to address two limitations of the dysregulated behavior literature to advance etiological models of commonly co-occurring behaviors. First, we furthered research on the factor structure of dysregulated behaviors by comparing bifactor and higher-order models. In line with our prediction, our results showed support for a bifactor model, with a general dysregulated behavior factor and unique alcohol, drug, aggression, and self-harm factors. Second, we expanded research on the nomological network of the general and unique factors by

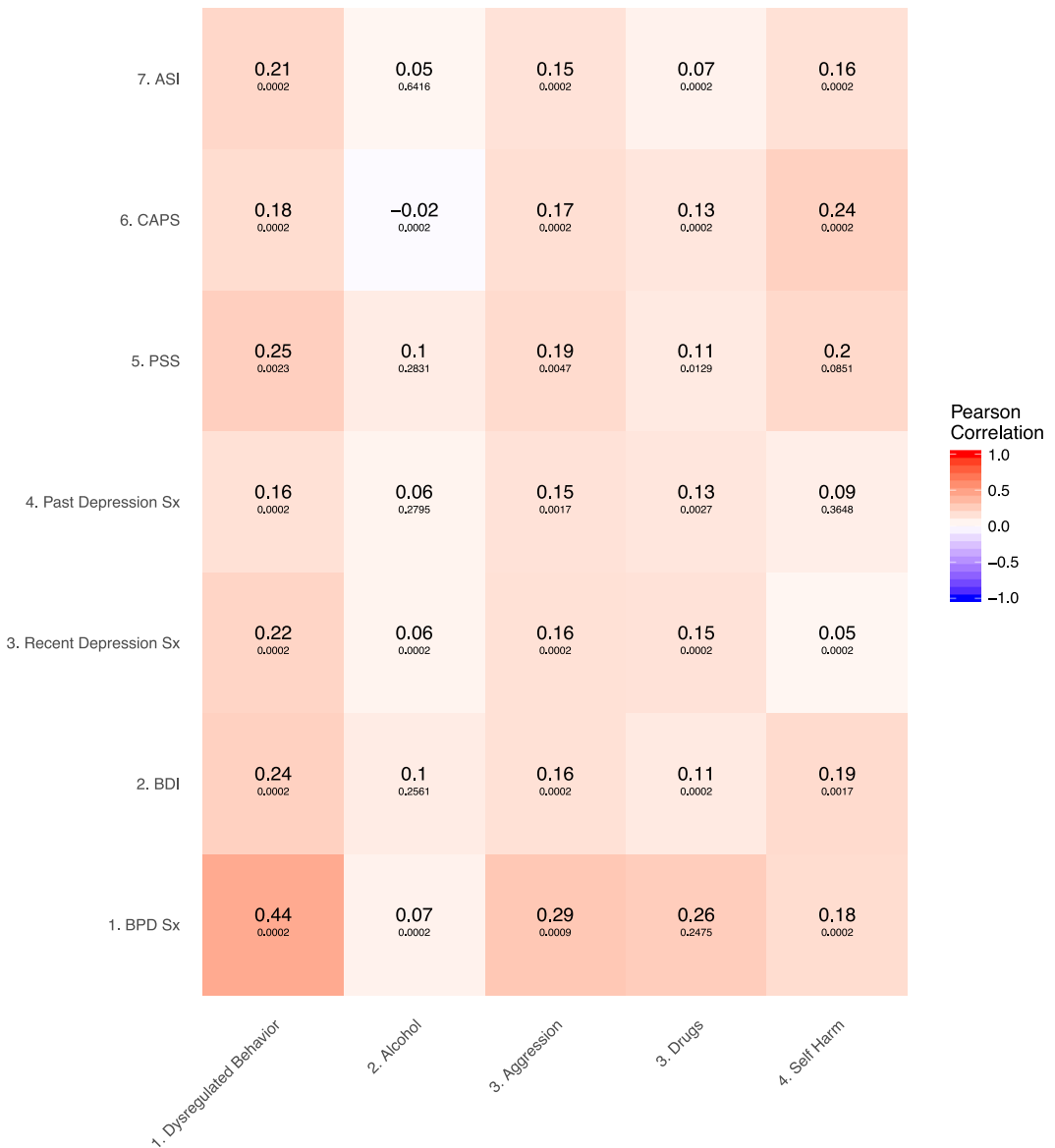


FIGURE 4 Correlations (and adjusted p -values) among the factor scores and symptoms of psychopathology. Note: BPD Sx=Borderline Personality Disorder Symptoms ($N = 326$); BDI=Beck Depression Inventory ($N = 3246$); Recent Depression Sx=Current Depression Symptoms ($N = 411$); Past Depression Sx=Past Depression Symptoms ($N = 383$); PSS=Posttraumatic Symptom Scale ($N = 3168$); CAPS=Clinician Administered Posttraumatic Disorder Scale ($N = 839$); ASI=Anxiety Sensitivity Index ($N = 3003$)

examining their relations with emotion regulation difficulties, personality traits, and symptoms of psychopathology. In line with our hypotheses, the general dysregulated behavior factor was positively associated with emotion regulation difficulties, negative affectivity, and BPD symptoms. Many of our hypotheses for the unique factors were also supported. These results add to the growing literature on dysregulated behaviors and have implications for several areas of psychopathology and intervention research.

Our results suggest that a bifactor model provides the best fit to the data. This is in line with Sadeh and Baskin-Sommers (2017), who found evidence for a bifactor model over a correlated factor model, and are different from Kingston

et al. (2011), who found evidence for a higher-order model but did not test a bifactor model. Our results add to the literature in that we directly compared the fit of a higher-order and bifactor model in a large sample of individuals at high risk for stress-related psychopathology. Thus, our results provide more direct evidence for a bifactor model where there are shared and unique vulnerabilities for dysregulated behaviors. Some caution is warranted in interpreting these results as many fit indices are biased to favor the bifactor model (Greene et al., 2019); however, we used additional measures to compare models, which are less biased and found the same results. Our results also show that these results generalize to a more racial and socioeconomically diverse sample compared to previous studies.

In line with our predictions, the general dysregulated behavior factor was positively correlated with all facets of emotion dysregulation. This indicates that part of what is shared across dysregulated behaviors is difficulty regulating emotions. This fits with Selby and Joiner (2009), who posited that individuals engage in dysregulated behaviors to regulate unpleasant emotions. Also, in line with our prediction, the general dysregulated behavior factor was positively correlated with negative affectivity. Together, this suggests that individuals who engage in dysregulated behaviors tend to experience negative affect and have difficulty regulating that affect. This is directly in line with the emotional cascade theory, which suggests that the reason people turn to dysregulated behaviors, as opposed to other methods of emotion regulation, is that they lack effective strategies to regulate their intense emotions (Selby & Joiner, 2009). We also found that the general dysregulated behavior factor was positively correlated with symptoms of BPD, which is in line with theory the emotional cascade theory of BPD (e.g., Selby & Joiner, 2009). This is also in line with Bresin's (2020) suggestion that BPD is the DSM diagnosis most directly related to dysregulated behaviors. Together, these results could indicate that negative emotionality and the inability to regulate negative emotions are critical to the development of dysregulated behaviors.

We also found several significant correlations that were not predicted. The general dysregulated behavior factor was strongly correlated with the personality trait of disinhibition. In hindsight, this makes sense because dysregulated behaviors are a subset of impulsive behaviors (Bresin, 2020), and disinhibition is an important aspect of impulsivity (e.g., Whiteside & Lynam, 2001). When combined with our a priori predictions, this suggests that high negative affect and disinhibition combined with a lack of ability to regulate emotions may be a general risk profile for dysregulated behaviors. The general dysregulated behavior factor was positively correlated with the personality traits of antagonism, detachment, and psychoticism and the symptoms of depression, PTSD, and anxiety sensitivity. The correlations between the general dysregulated behavior factor and other personality traits are somewhat surprising as psychoticism (the pathological form of openness) and detachment (the pathological form of introversion; Widiger et al., 2019) are not necessarily associated with dysregulated behaviors (e.g., Barlett & Anderson, 2012; Brown, 2009; Malouff et al., 2007). It is not surprising that the general factor was positively related to symptoms of depression, PTSD, and anxiety sensitivity because symptoms of these disorders are correlated with high levels of negative affect (Kotov et al., 2017). Regardless, given that these correlations were not predicted a priori, replication is necessary.

We had three predictions for the unique factors, all of which were generally supported. First, we found that the unique drug factor was positively correlated with disinhibition. Counter to our prediction, however, the unique alcohol factor was not significantly related to disinhibition. Second, we found that the unique aggression factor was positively correlated with antagonism. Finally, we found that the unique self-harm factor was positively correlated with negative emotionality. The finding that the common general dysregulated behavior factor and unique dysregulated behavior factors were related to the same maladaptive personality traits may be interpreted to suggest that there are common and unique pathways to specific dysregulated behaviors via the same personality traits. Specifically, it may be that the broad personality dimensions are general risk factors for all dysregulated behaviors, whereas specific facets of personality traits may be uniquely related to some dysregulated behaviors and not others. For example, some research has found that specific facets of antagonism have stronger correlations with aggression than other facets (Crowe et al., 2018). Similarly, it may be that specific negative affect states may be more strongly related to some dysregulated behaviors than others (cf., Bresin, 2020). Thus, these results represent an opportunity for future research and theory development.

Beyond our a priori predictions, there were several patterns of correlations that emerged with the unique factors. First, aside from a few small significant correlations, the unique alcohol factor was largely unrelated to the variables examined in our study, which differed from the other unique factors. Research on externalizing psychopathology suggests that the unique variance in alcohol use when accounting for overlap between alcohol and antisocial psychopathology represents sensation seeking (e.g., Patrick et al., 2013). It is also possible that when the variance shared with other dysregulated behaviors is removed, the leftover variance in alcohol use is normative social drinking. The unique drug use, aggression, and self-harm factors shared similar patterns of correlations with emotion dysregulation, personality, and symptoms of psychopathology. It is worth noting that there were no correlations with unique factors that were not also shared by the general factor. Moreover, the correlations with the general factor tended to be stronger. This may suggest that most of the vulnerability for dysregulated behaviors is shared, and thus, theories of dysregulated behaviors may have the most predictive value at the general level. Still, given the size of the correlations, there are yet to be identified sources of variance in the unique factors.

In terms of psychopathology models, our results highlight some of the limitations of examining dysregulated behaviors with the *DSM-5*. For instance, we found that the general dysregulated behavior factor was positively correlated with symptoms of several *DSM-5* disorders that are proposed to be distinct. We also found evidence for a common dysregulated behavior factor with symptoms that are part of distinct disorders (e.g., substance use, self-harm). Shared variance suggests that there may be common etiological factors across disorders. These results are in line with theory suggesting that the *DSM-5* taxonomy creates artificial distinctions between dysregulated behaviors (Bresin, 2020). Our results provide some support that the Hi-TOP model may be more effective for classifying and identifying etiological factors for dysregulated behaviors. For example, we found that the unique alcohol and substance use factors had significant positive associations with disinhibition, which is consistent with the Hi-TOP model's placement of substance use disorders under the disinhibited externalizing spectra. Similarly, we found that the unique aggression factor was correlated with antagonism, which is in line with the Hi-TOP conceptualization. Despite these results, more research on the association between dysregulated behaviors and Hi-TOP is necessary.

These results have important implications for intervention development. Our results suggest that negative affect and emotion dysregulation would be ideal treatment targets to address several dysregulated behaviors. This is consistent with interventions for dysregulated behaviors that target emotion dysregulation. For instance, Dialectical Behavior Therapy (DBT; Linehan, 1991) is a multi-modal intervention that attempts to help clients replacing unskillful behavior, which includes but is not exclusively dysregulated behaviors, with more effective behavior. DBT has empirical support for treating self-harm (DeCou et al., 2019), aggression (Frazier & Vela, 2014), and substance use (Linehan et al., 2002). There is also some preliminary evidence that improvements in emotion regulation may be a mechanism of change in DBT (Maffei et al., 2018; Neacsiu et al., 2018). Given the modest correlations between the general dysregulated behavior factor and negative affect and emotion dysregulation (as much as 90% of the variance is not shared), our results also suggest that other treatment targets are needed. For instance, increasing the ability to inhibit responding to urges (i.e., decreasing disinhibition) may be helpful. Because we found associations between the unique factors and emotion dysregulation and maladaptive personality traits, it may suggest that more specific targeted interventions may help in reducing specific dysregulated behaviors (e.g., decreasing antagonism for aggression).

6.1 | Limitations and strengths

The results of this study should be considered in light of the limitations. First, these data are cross-sectional, which preclude the ability to draw causal conclusions from these associations. Second, we had high levels of missing data for the dysregulated behaviors. Specifically, 95% of participants had data for drug use, 73% had data for aggression, and 50% of participants had data for self-harm. We assumed that these were missing at random and used statistical techniques that have been shown to be robust to missing data. Still, replication in another sample with less missing

data is necessary. Moreover, some of our model fit statistics (e.g., CFI) were below recommended thresholds, indicating that another model may fit the data better. We interpreted the best fitting model among those hypothesized. Still, a better fitting, theoretically interesting model may exist. Further replication is needed. Third, we only included a subset of dysregulated behaviors. It is possible that including other dysregulated behaviors (e.g., gambling, risky sex) would have led to a different factor structure, although Sadeh and Baskin-Sommers (2017) found support for a bifactor model with a larger set of dysregulated behaviors.

This study also has a number of strengths worth noting. First, we used a sample with a high rate of endorsement of dysregulated behaviors (a large urban-dwelling, trauma-exposed sample), whereas the two previous studies relied heavily on student and community samples (Kingston et al., 2011; Sadeh & Baskin-Sommers, 2017). Using an at-risk sample (i.e., a sample with a high rate of endorsement of dysregulated behaviors) advanced our understanding of the etiology of dysregulated behaviors in those most likely to seek and benefit from interventions. Our sample also contained a high percentage of Black participants with lower income than prior studies. This aids in generalizing the results from previous studies to other, often understudied, groups. Second, our sample size was much larger than the two previous studies, which enhances generalizability. Finally, although there are issues comparing higher-order and bifactor models (e.g., Greene et al., 2019), we used the current best methods to compare models. Taken together, our results provide support for a bifactor model of dysregulated behaviors, and identify possible common and unique correlates, which could be used for further theory and intervention development.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author.

PEER REVIEW

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ENDNOTE

- ¹ See the Supporting Information for models with listwise deletion, which show similar results for the best fitting model with missing data.

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