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Targeting Higher-Order Dimensions of Personality in Treatment as a Parsimonious Means to Address Co-Occurring Psychopathology

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People with borderline personality disorder (BPD) commonly have co-occurring mental health conditions that may be accounted for by higher-order factors in dimensional models of psychopathology. BPD Compass is a cognitive-behavioral treatment developed to target broad personality domains (i.e., negative affectivity, antagonism, disinhibition) associated with BPD and related conditions. The purpose of the present study was to explore the extent to which BPD Compass can serve as a transdiagnostic intervention for these comorbid conditions. Participants (N = 100; M_{age} = 28.13, 73.7% female, 79.6% White, 66% sexual minority) were assigned to either immediately begin treatment (randomized and naturalistic) or receive treatment after an 18-week waiting period. At baseline, participants met criteria for an average of 3.28 (SD = 2.02, range: 0–8) comorbid diagnoses ranging in clinical severity from 3.30 (for substance use disorder) to 4.91 (for persistent depressive disorder). Posttreatment clinical severity ratings (CSRs) for those randomized to receive BPD Compass were below clinical thresholds for all assessed conditions except premenstrual dysphoric disorder, whereas post-waitlist CSRs remained above clinical thresholds for all disorders except bipolar II, agoraphobia, and major depressive disorder. Collapsed across all patients who received BPD Compass, pre- to posttreatment improvements were significant and large in magnitude for most disorders assessed. These results suggest that BPD Compass may be an efficacious transdiagnostic intervention, though our small sample and high rate of dropout warrant further study.

Keywords: borderline personality disorder; cognitive-behavior therapy; comorbidity

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Co-occurrence of two or more mental health conditions is incredibly common (e.g., Kessler et al., 1998). For people with borderline personality disorder (BPD), comorbidity is the rule rather than the exception (Zanarini et al., 1998a; Zimmerman & Mattia, 1999). Among those with BPD in a nationally representative sample, half of respondents endorsed a co-occurring substance use disorder and/or mood disorder, and nearly two-thirds of the sample endorsed a co-occurring anxiety disorder (Grant et al., 2008).

Emerging dimensional models of psychopathology may provide an explanation for these high comorbidity rates. For example, in the Alternative Model of Personality Disorders (AMPD) included in most recent edition of the *Diagnostic and*

Statistical Manual of Mental Disorders (*DSM-5*; American Psychiatric Association, 2013), personality pathology is represented as excesses in different combinations of five transdiagnostic traits (negative affectivity, detachment, disinhibition, antagonism, and psychoticism). BPD, specifically, is characterized by negative affectivity, disinhibition, and antagonism. Other personality disorders (e.g., antisocial, avoidant, obsessive-compulsive) are also defined by combinations of these same traits, underscoring the frequent co-occurrence among them (Zanarini et al., 1998b).

Beyond comorbidity among personality disorders, dimensional models that account for the full range of psychopathology are useful for understanding the co-occurrence of BPD with "Axis I" conditions. The Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017; Cicero et al., 2024) includes six higher-order spectra (internalizing, detachment, antagonistic externalizing, disinhibited externalizing, thought disorder, and somatization) that largely correspond to the traits in the AMPD (Gore & Widiger, 2018; Michelini et al., 2021). In HiTOP, BPD is cross-listed on the internalizing spectrum, which also includes anxiety, depressive, and related disorders (e.g., eating disorders), and the externalizing spectrum, which accounts for other personality disorders and substance use disorders (Cicero et al., 2024; Kotov et al., 2017). Of note, both models (AMPD and HiTOP) demonstrate strong convergence (Michelini et al., 2021) with the well-known five-factor model of personality (FFM; Costa & McCrae, 1992) that has long been used to account for comorbidity across mental health conditions (Andrews, 1996; Trull & Sher, 1994).

Treatments that target higher-order dimensions that confer shared risk for a range of psychopathology may represent a more efficient approach to care (McHugh & Barlow, 2010; Sauer-Zavala et al., 2017). BPD Compass, which loosely stands for cognitive behavioral modules for personality symptoms, is a brief (i.e., 18 sessions) treatment that was developed to target the personality traits associated with BPD in the AMPD (Sauer-Zavala et al., 2022), as well as commonly co-occurring conditions in dimensional models of all psychopathology (e.g., HiTOP): negative affectivity, antagonism, and disinhibition.

To address neuroticism, BPD Compass adopts Barlow and colleagues' functional model of neuroticism (Barlow, Ellard, et al., 2014; Barlow, Sauer-Zavala, et al., 2014). This model suggests that aversive reactions to frequently occurring negative emotions prompt the use of emotionally avoidant coping strategies (e.g., self-injurious behaviors, binge eating, substance use) that paradoxically result in more frequent and intense negative emotions (i.e., exacerbating and maintaining negative affectivity; Bullis et al., 2019). By contrast, sustained decreases in the frequency of negative emotions, achieved by targeting aversive/avoidant responses to emotions, may constitute decreases in negative affectivity. Transdiagnostic behavioral interventions (see Farchione et al., 2024) targeting aversive reactivity are associated with reductions in emotional disorder (anxiety, depressive, and related disorders; Barlow et al., 2017) and BPD (Sauer-Zavala et al., 2016) symptoms, along with significantly larger decreases in neuroticism than symptom-focused protocols (Sauer-Zavala, Fournier, et al., 2020). Moreover, there is emerging evidence that improvements in negative affectivity in transdiagnostic treatments may even predict symptom reduction (Stumpp et al., 2024).

Antagonism, characterized by distrust, manipulativeness, and oppositionality (Mullins-Sweatt et al., 2012), is a risk factor for developing externalizing psychopathology (Anderson et al., 2007; Kotov et al., 2010; Miller et al., 2003). Higher levels of antagonism are associated with insecure attachments to childhood caregivers, which can manifest in adulthood as behaviors that function to protect a person in interpersonal contexts perceived as threatening (Young et al., 2006). Here, attachment insecurity represents an actionable functional mechanism linking the personality trait of antagonism to externalizing symptoms, akin to the role of aversive reactivity in the relation between neuroticism and internalizing symptoms. Emerging research suggests that improving patients' ability to consider others' perspectives, along with challenging negative schemas about oneself and others, improves attachment security in adults (Levy et al., 2006; Vogt & Norman, 2019), though there is limited data (if any) of reducing antagonism through treatment.

Finally, disinhibition, or trait impulsivity, is characterized by sensation-seeking (the tendency to seek out novel and thrilling experiences), lack of deliberation (the tendency to act without thinking), lack of persistence (an inability to remain focused on a task), and urgency (the tendency to act rashly in response to positive and negative emotional experiences; Cyders et al., 2007; Whiteside et al., 2005). Roberts and colleagues have published several theoretical accounts of how to alter this trait in treatment (Magidson

et al., 2014; Roberts et al., 2017). They suggest that individuals' expectancies about their performance on certain tasks, along with how much they value these actions, predict conscientious behaviors (e.g., paying bills on time, subjugating impulses that would be gratifying in the short-term; Eccles, 2009). Others have suggested that trait impulsivity is maintained by high reward orientation such that disinhibited individuals will continue to pursue rewards (e.g., relief from negative emotions, substance-related highs) despite negative consequences (Carver & White, 1994; Gray, 1987). Thus, intervention strategies that focus on values, provide immediate (reinforcing) feedback on progress, and engage performance expectancies have been suggested to address this trait (Magidson et al., 2014) and have been included in BPD Compass.

BPD Compass is associated with large, significant decreases in BPD symptoms (Sauer-Zavala et al., 2023a) that are comparable to gold-standard approaches for this condition (Cristea et al., 2017). After an 18-week window, BPD symptoms were significantly less severe in people who received BPD Compass relative to those assigned to the waitlist control condition (Sauer-Zavala et al., 2023a). Additionally, BPD Compass is associated with large reductions in neuroticism, moderate reductions in BPD-relevant facets of antagonism (i.e., mistrust, manipulativeness), and small reductions in disinhibition that were all statistically significant (Sauer-Zavala, 2024).

Given that BPD Compass was developed to target broad personality domains that have been prospectively linked to conditions that commonly co-occur with BPD, this intervention may be particularly adept at addressing comorbid psychopathology. The goal of the present study, a secondary analysis of Sauer-Zavala et al. (2023a), is to examine the degree to which BPD Compass can serve as a transdiagnostic intervention for BPD and related conditions. Our first aim was to characterize patterns of comorbidity in the sample, including the range and average number of comorbid conditions exhibited by patients, as well as the frequency of each comorbid condition. In our second aim, we explored whether the clinical severity of comorbid conditions improved (a) across 18 sessions of BPD Compass compared to an 18-week waitlist condition (WLC) and (b) from pre- to posttreatment across all participants. We hypothesized that BPD and comorbid disorder symptoms would be less severe at posttreatment (a) for patients who received BPD Compass compared to those in a waitlist condition and (b) at posttreatment compared to pretreatment.

Method

PARTICIPANTS

A sample of adults seeking outpatient treatment was recruited from Kentucky. Participants were eligible if they met *DSM-5* criteria for BPD, which was assessed using a clinician-rated structured clinical interview (see Measures). Eligible participants also agreed not to take part in concurrent behavioral interventions, and, if applicable, to remain on a stable dose of psychotropic medication during their study participation. Individuals were excluded if they had symptoms or diagnoses in which alternative treatment is the standard of care, including severe substance use disorders, uncontrolled bipolar I disorder (i.e., mania within the past year), psychotic features, or acute suicide risk (i.e., imminent intent).

A total of 150 people consented to participate and completed the initial eligibility assessment (Figure 1). Of these, 50 people were withdrawn prior to randomization due to endorsing one or more of the exclusion criteria listed above. Thus, 100 participants were eligible to participate in the parent trial following the baseline assessment. The average age of the sample was 28.13 (SD = 8.80) and a majority of participants identified as female (n = 73; 73.7%) and White (n = 88; 88.9%; Table 1). A quarter of our sample (n = 25; 25.3%) identified as an ethnic or racial minority and nearly a fifth identified as a gender minority (i.e., transgender/nonbinary; n = 17; 17.2%).[1] Finally, the majority of our sample (n = 64; 64.6%) identified as sexual minorities.

PROCEDURES

All study procedures were approved by the University of Kentucky Institutional Review Board and registered at clinicaltrials.gov (NCT04587518). Participants were recruited via advertisements posted on various social media platforms, participant recruitment websites, and university listservs. People who were interested in participating first completed a phone screen, which included the McLean Screening Instrument for BPD (MSI-BPD; Zanarini et al., 2003) and questions to assess for exclusion criteria.

People considered likely eligible then completed a semistructured diagnostic assessment via telehealth and a battery of self-report questionnaires. The first 51 (51%) patients who met inclusion criteria were randomized (1:1) to either the BPD Compass condition (BPD Compass-Randomized;

[1] Participants could select more than one race/ethnicity (e.g., White and Indigenous) and gender (e.g., female and transgender).

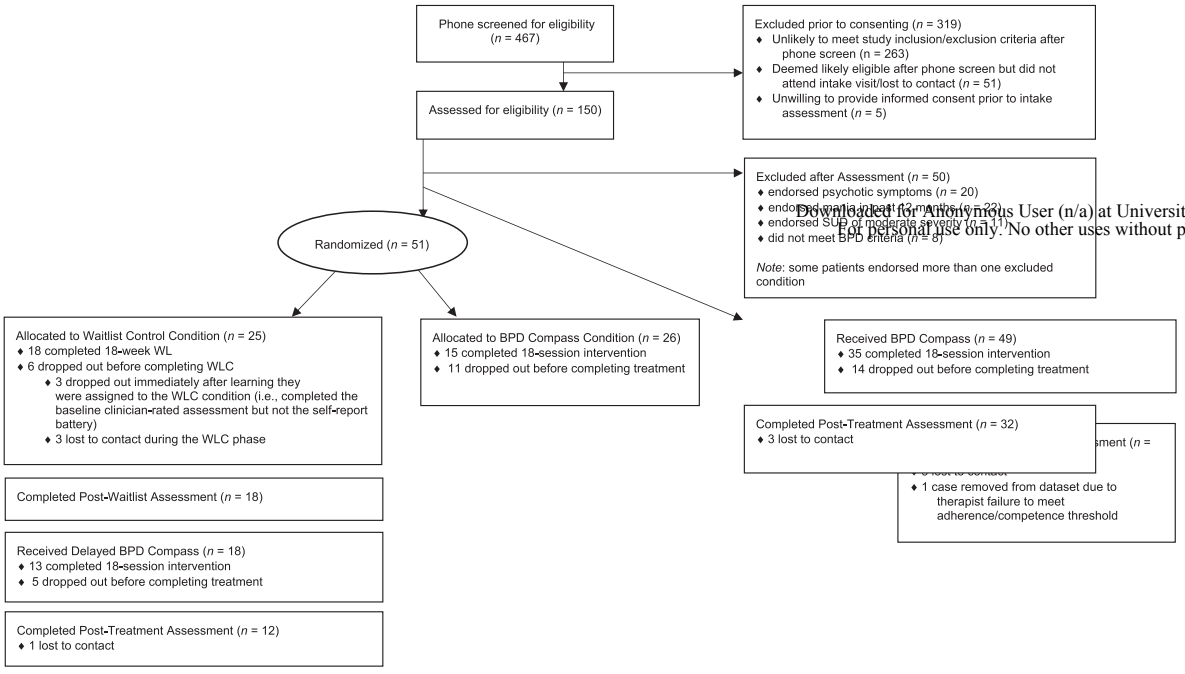

FIGURE 1 CONSORT Diagram of study flow for BPD Compass-R, Waitlist, and BPD Compass-NR patients

n = 26; 51%) or the WLC (WLC; *n* = 25; 49%). We employed a sequential analysis design (Lakens, 2014) in which we stopped randomizing after we were powered to detect a large effect on the parent trial's primary outcome of interest (i.e., BPD symptoms). However, we continued recruiting to allow for a larger sample on which to explore within-treatment effects. Thus, the remaining participants were assigned to immediate treatment, which was analogous to the BPD Compass condition (BPD Compass-Naturalistic; *n* = 49; 49%). Those assigned to the BPD Compass conditions completed 18 treatment sessions within a 7-month treatment window immediately following their baseline assessment. Each patient completed brief questionnaires before and after each session. Following their final session, patients completed a posttreatment diagnostic assessment and another battery of self-report questionnaires. Participants in the WLC completed brief self-report questionnaires every 4 weeks during their 18-week waiting period to monitor for worsening symptoms and to ensure they still met study inclusion criteria (i.e., refraining from concurrent behavioral treatment and refraining from medication changes). Procedures were in place to refer participants for immediate care in the case of significant clinical deterioration, though no patients required these contingencies. Once the 18-week waiting period elapsed, patients in the WLC completed a second post-waitlist diagnostic assessment and battery of self-report questionnaires. They were then offered BPD Compass.

Of those assigned to WLC, 72.0% (*n* = 18) completed the post-waitlist assessment. Of those randomized to the BPD Compass condition, 58% (*n* = 15) completed all 18 sessions, although fewer (*n* = 12; 46%) attended their posttreatment assessment (one case was withdrawn due to therapist nonadherence). Across all conditions (i.e., WLC, randomized to immediate BPD Compass, nonrandomized participants), 93 patients initiated BPD Compass and 63 (68%) people completed the treatment protocol. Complete posttreatment data is available for 56 patients. Participants were more likely to drop out of the BPD Compass-Randomized condition relative to the WLC, OR = 2.21, 95% CI:.66: 7.39. Presence or severity of BPD or any of the comorbid conditions assessed did not predict drop out, with the exception of premenstrual dysphoric disorder; those with more severe symptoms of this disorder were more likely to drop out ($\beta = .04$, $p < .05$).

TREATMENT

BPD Compass is an 18-session cognitive-behavioral intervention designed to target the three BPD-relevant AMPD personality dimensions (i.e., negative affectivity, antagonism, and disinhibition). The first session is devoted to psychoeducation regarding BPD, as well as providing patients with an overview of the treatment. The

Table 1
Baseline Demographic Characteristics

Characteristic	Total ($N = 97$)	BPD Compass-R ($n = 25$[b])	WLC ($n = 25$)	BPD Compass-NR ($n = 49$)
Age (Mean, *SD*)	28.14 (8.80)	26.96 (9.24)	29.91 (9.69)	27.92 (8.07)
Gender[a]				
Female	73 (73.7)	21 (84)	19 (82.6)	33 (67.3)
Male	16 (16.2)	2 (8)	3 (13.0)	11 (22.4)
Genderqueer/Non-binary	10 (10.1)	2 (8)	3 (13.0)	5 (10.2)
Transgender	6 (6.1)	2 (8)	2 (8.7)	2 (4.1)
Other	1 (1.0)	1 (4)	0 (0.0)	0 (0.0)
Racial/Ethnic Background[a]				
White	88 (88.9)	23 (92)	22 (95.7)	43 (87.8)
African-American	8 (8.1)	3 (12)	1 (4.3)	4 (8.2)
Indigenous/Native American	4 (4.0)	2 (8)	0 (0.0)	2 (4.1)
Latinx	10 (10.1)	3 (12)	1 (4.3)	6 (12.2)
Other	3 (3.1)	0 (0.0)	1 (4.3)	2 (4.1)
Sexual Orientation[a]				
Heterosexual/Straight	51 (51.5)	12 (48)	14 (60.9)	25 (51)
Bisexual	33 (33.3)	8 (32)	11 (47.8)	14 (28.6)
Asexual	6 (6.1)	3 (12)	1 (4.3)	2 (4.1)
Queer	13 (13.1)	2 (8)	4 (17.4)	7 (14.3)
Gay/Lesbian	12 (12.1)	3 (12)	3 (13)	6 (12.2)
Some post-secondary education	88 (90.9)	23 (92)	21 (91.3)	44 (42.9)
Married	15 (15.2)	0 (0)	5 (21.7)	10 (20.4)
Current Psychotropic Medication	66 (66.7)	18 (72)	16 (64)	32 (65.3)

Note: BPD Compass-R = Randomized to immediate treatment; BPD Compass-NR = Assigned (not randomized) to BPD Compass. Data are presented as number (percentage) of patients unless otherwise indicated.
[a] Values may not sum to total in each column because participants could select multiple racial/ethnic backgrounds.
[b] One participant was not included in analyses due to therapist non-compliance.

next two sessions focus on identifying patients' values and assessing the extent to which they are currently living in accordance with them. Four sessions are then spent teaching skills to improve cognitive flexibility around emotional situations, relationships, and beliefs regarding their ability to resist impulsive urges. Six sessions are then dedicated to behavior change, in which patients are asked to identify unhelpful patterns of behavior and instead practice behaviors that are better aligned with their values. Next, four sessions focus on cultivating mindfulness, wherein patients learn skills to respond non-judgmentally to thoughts, sensations, interpersonal conflicts, and impulsive urges and remain in the moment. The final session is devoted to relapse prevention.

Patients received individual treatment sessions lasting 45–60 minutes weekly. Sessions were conducted via a HIPAA-compliant telehealth service (Zoom). Treatment was delivered by nine study therapists who were primarily advanced clinical psychology graduate students with a background in cognitive-behavior therapy, in addition to the treatment developers/licensed clinical psychologists (SSZ; MWS). Student therapists received didactic training in BPD Compass prior to delivering the treatment and took part in weekly supervision meetings with the treatment developers. Treatment adherence was assessed for each student therapist by the treatment developers, who reviewed all sessions of their first case using a BPD Compass-specific fidelity checklist. In addition, all sessions were video-recorded, and 20% were randomly selected for review by treatment developers and rated on a 5-point scale. Average treatment fidelity was high (97.32%, $SD = 11.42$), and average competence, intended to measure therapeutic skill, was adequate to good ($M = 3.61$, $SD = .99$).

MEASURES

Doctoral students trained to reliability and masked to treatment conditions administered clinician-rated diagnostic and severity assessments. Self-report questionnaires were administered using Research Electronic Data Capture (REDCap; Harris et al., 2019) and accessed via links sent by study assessors and therapists.

Diagnostic Measures

Participants completed a diagnostic assessment to confirm eligibility prior to randomization. The

BPD module of the SCID-II (First et al., 2015) was administered first to confirm participants met *DSM-5* criteria for BPD (American Psychiatric Association, 2013). The SCID-II is a semistructured diagnostic interview used to assess the presence of personality disorders. The BPD module has demonstrated excellent inter-rater reliability in a mixed sample of outpatients, inpatients, and healthy controls ($\kappa = .91$; Lobbestael et al., 2011).

Modules of the Diagnostic Interview for Anxiety, Mood, and Obsessive-Compulsive and Related Neuropsychiatric Disorders (DIAMOND; Tolin et al., 2018), a semistructured diagnostic interview for *DSM-5* disorders were used to assess exclusion criteria and comorbid *DSM-5* diagnoses. Assessors assigned a clinical severity rating (CSR) based on the degree of distress/functional impairment related to each diagnosis. CSRs were rated on a seven-point scale (1–7), wherein a CSR ≥ 3 suggests clinically significant distress/impairment. Diagnostic assessments were also audio-recorded, and 20% of tapes were rated by an assessor masked to initial ratings and treatment condition. Assessors demonstrated excellent reliability determining study eligibility (Krippendorff's $\alpha = 1.00$), making categorical determinations of the presence of diagnoses (Krippendorff's $\alpha = 1.00$), and rating CSRs, Krippendorff's $\alpha = .89$, 95% CI [.68, .99].[2]

Symptom Measures

BPD Symptom Severity. The self-report version of the Zanarini Rating Scale for BPD (ZAN-BPD-SR; Zanarini et al., 2015) is a 9-item continuous measure intended to measure BPD symptom severity within the previous week. Respondents rate the degree to which each *DSM-5* criteria for BPD affected them on a five-point scale with unique anchors for each item ranging from 0 (no symptoms) to 4 (severe symptoms). Items are summed to create a total score. ZAN-BPD-SR items demonstrated good internal consistency across at pre- and posttreatment assessments, McDonald's ωs: .83 and .89, respectively.

Depression Symptoms. The Overall Depression Severity and Impairment Scale (ODSIS; Bentley et al., 2014) is a 5-item self-report measure designed to assess depression symptoms over the past week. Items are rated on a 4-point Likert-type scale with unique anchors for each item and summed to create a total score. Scores ≥ 8 indicate clinically significant depression symptoms. ODSIS items demonstrated excellent internal consistency at pre- and posttreatment assessments, ωs: .91 and .96, respectively.

Anxiety Symptoms. The Overall Anxiety Severity and Impairment Scale (OASIS; Norman et al., 2006) is a 5-item self-report questionnaire designed to measure anxiety symptoms over the past week. Items are rated on a 4-point Likert-type scale with unique anchors for each item and summed to create a total score. Scores ≥ 8 indicate clinically significant anxiety symptoms. OASIS items demonstrated good-to-excellent internal consistency at pre- and posttreatment assessments, ωs: .84 and .94, respectively.

Eating Disorder Symptoms. The Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a self-report questionnaire consisting of 28 items designed to assess the frequency and severity of behaviors and cognitions related to eating disorders in the last month. Twenty-three EDE-Q items are rated on a six-point Likert-type scale from 0 (*No days/ None of the times/Not at all*) to 6 (*Every day/Every time/Markedly*) and averaged to create a total score. Scores ≥ 2.80 indicate clinically significant eating disorder symptoms (Velkoff et al., 2023). EDE-Q items demonstrated excellent internal consistency at pre- and posttreatment assessments, ωs: .93 and .93, respectively.

Posttraumatic Stress Disorder (PTSD) Symptoms. The PTSD Checklist for *DSM-5* (PCL-5; Blevins et al., 2015) is a 20-item self-report questionnaire intended to assess PTSD symptoms over the last month. Respondents rate the extent to which each *DSM-5* symptom of PTSD has applied to them on a 5-point scale ranging from 0 (*Not at all*) to 4 (*Extremely*). Scores ≥ 33 are indicative of a probable PTSD diagnosis (Blevins et al., 2015). PCL-5 items demonstrated excellent internal consistency at pre- and posttreatment assessments, ωs: .96 and .98, respectively.

ANALYTIC PLAN

Our first aim was to characterize patterns of diagnostic comorbidity at baseline. We first examined the average number of comorbid *DSM-5* diagnoses and compared the average number of comorbid diagnoses in each condition using a chi-squared test in SPSS Version 29 (IBM Corp. 2023). We then examined the frequency of each comorbid *DSM-5* diagnosis at baseline and compared the proportion of each diagnosis among conditions using a series of chi-squared tests. We examined the mean baseline CSRs of each comorbid condition and compared the mean baseline CSRs among conditions using a series of one-way ANOVAs.

[2] Krippendorff's $\alpha s \geq .80$ are considered to indicate reliable variables, and αs between .67 and .80 indicate tentative reliability (Krippendorff, 2004).

Finally, we examined the mean baseline scores on all self-reported symptom measures (i.e., ZAN-BPD-SR, ODSIS, OASIS, EDE-Q, PCL-5) and compared the mean baseline scores of each measure among conditions using a series of one-way ANOVAs.

Our second aim was to examine whether comorbid conditions improve as a function of treatment with BPD Compass. We first compared BPD Compass-Randomized to the WLC. Specifically, we examined the frequency and percentage of each *DSM-5* diagnosis at posttreatment for each condition (small cell sizes precluded statistical comparisons) and compared average CSRs at posttreatment between conditions using independent samples *t*-tests. Based on a sensitivity analysis using G*Power (version 3.1; Faul et al., 2007) assuming $\alpha = .05$, the smallest between-condition difference in CSRs at posttreatment we had 80% power to detect varied from $g = 1.05$ for BPD (BPD Compass-R: $n = 13$; WLC: $n = 18$) to $g = 6.51$ for both substance use disorder (SUD) and binge eating disorder (BED; BPD Compass-R: $n = 3$; WLC: $n = 1$). The smallest between-condition differences in self-reported symptom measures we were powered to detect ranged from $g = 1.17$ for BPD, depression, and anxiety symptoms to $g = 1.26$ for eating disorder symptoms.

Finally, we collapsed across all participants to maximize our power to test for pre- to posttreatment changes in the frequency, percentage, and CSRs of comorbid diagnoses and severity of self-report measures. We calculated within-condition effect sizes using Hedges's *g* with standard deviation of the difference to examine the magnitude of change in each indicator from pre- to posttreatment. Assuming $\alpha = .05$, the smallest differences in CSRs we had 80% power to detect ranged from $g = .37$ for BPD ($n = 57$) to $g = 1.68$ for bulimia nervosa (BN; $n = 5$) and the smallest differences in self-report scores we were powered to detect ranged from .51 for BPD, anxiety, depressive symptoms to .55 for PTSD symptoms.

Results

BASELINE PATTERNS OF COMORBIDITY

In addition to their BPD diagnosis, participants met criteria for an average of 3.26 ($SD = 2.03$, range: 0–8) comorbid diagnoses at baseline.[3] The modal number of comorbid diagnoses was two ($n = 20$; 20.2%), followed by three ($n = 18$; 18.2%). Of note, only five people (5.1%) did *not* meet criteria for an additional diagnosis beyond BPD. There were no significant differences in the number of comorbid diagnoses at baseline as a function of treatment condition (BPD Compass-Randomized, BPD Compass-Naturalistic, WLC), $\chi^2(16, 96) = 20.50$, $p = .20$. The most common comorbid diagnoses were social anxiety disorder (SAD; $n = 54$; 54.5%), generalized anxiety disorder (GAD; $n = 45$; 45.5%), PTSD ($n = 29$; 29.29%), and obsessive compulsive disorder (OCD; $n = 27$; 27.3%). Similarly, there were no significant differences in the number of people endorsing each diagnosis as a function of condition, $\chi^2 < 4.23$, ps $> .12$, with the exception of OCD, $\chi^2(2, 99) = 6.87$, $p < .05$; significantly more patients met criteria for OCD in the BPD Compass-Naturalistic condition ($n = 19$; 38.80%) than in the BPD Compass-Randomized ($n = 3$; 12%; $p < .01$) and WLC ($n = 5$; 20%; $p < .01$) conditions.

Among people who met criteria for each diagnosis, the average CSR ranged from 3.30 (for SUD) to 4.91 (for persistent depressive disorder). The average CSR of each comorbid diagnosis except SUD was >4, suggesting moderately distressing and impairing symptoms. There were no significant differences in baseline CSRs for any assessed diagnoses between as a function of condition Fs < 2.66, ps $> .05$. See Supplemental Table 1.

Finally, to complement our clinician-rated data, we examined self-reported symptom severity. On average, our sample endorsed moderately severe BPD symptoms ($M = 17.10$, $SD = 7.33$), along with anxiety ($M = 10.52$, $SD = 3.94$), depression ($M = 10.20$, $SD = 4.71$), and PTSD ($M = 35.50$, $SD = 21.60$) severity scores that were above established clinical thresholds (Bentley et al., 2014; Blevins et al., 2015; Norman et al., 2006). Eating disorder severity ($M = 2.51$, $SD = 1.50$) was slightly lower than the clinical threshold (Velkoff et al., 2023). No significant differences were observed on any self-report measure as a function of condition at baseline, Fs < 2.01, ps $> .14$. See Supplemental Table 1.

IMPROVEMENT IN COMORBID DIAGNOSES ACROSS TREATMENT

BPD Compass-Randomized Compared to Waitlist Control

Across all conditions, fewer participants met criteria for each diagnosis at their second assessment (i.e., posttreatment or post-waitlist; Table 2). The percentage of patients in the BPD Compass-Randomized condition who met criteria for each

[3] Pretreatment for patients who started treatment immediately (BPD Compass-Randomized, BPD Compass-Non-randomized) and prewaitlist for those in the Waitlist/Delayed Treatment condition.

Table 2
Diagnostic Characteristics at Pretreatment/Waitlist and Posttreatment/Waitlist

	BPD-Compass Randomized			Waitlist Control			All BPD Compass		
	Pre-Treatment		Post-Treatment	Pre-Waitlist		Post-Waitlist	Pre-Treatment[a]		Post-Treatment
	n (%)	CSR	CSR	# (%)	CSR	CSR	# (%)	CSR	CSR
BPD	25 (100)	16.04 (6.46)	6.62 (6.04)	25 (100%)	15.00 (6.10)	14.11 (7.50)	92 (100)	14.61 (6.63)	7.44 (6.27)
BP-II	3 (12%)	5.33 (2.08)	3.00 (0.00)	5 (20%)	3.60 (1.34)	2.60 (1.53)	11 (11.1%)	4.55 (1.44)	2.60 (1.51)
OCD	3 (12%)	4.33 (1.53)	3.00 (2.82)	5 (20%)	4.20 (0.87)	3.33 (1.52)	27 (27.3%)	4.22 (1.05)	2.63 (1.80)
SAD	12 (48%)	4.75 (1.14)	2.60 (1.95)	13 (52%)	4.62 (1.12)	4.00 (1.31)	48 (58.5%)	4.54 (1.06)	2.77 (1.54)
GAD	11 (44%)	4.64 (1.29)	2.75 (1.50)	12 (48%)	4.83 (1.34)	3.00 (1.85)	37 (45.1%)	4.51 (1.10)	2.13 (1.69)
PD	6 (24%)	4.17 (1.33)	2.50 (2.12)	7 (28%)	4.00 (1.53)	4.25 (2.10)	16 (20.5%)	4.38 (1.36)	2.50 (1.77)
Ag	0 (0%)	–	–	4 (16%)	4.75 (1.71)	2.67 (2.08)	8 (10.4%)	4.75 (1.30)	2.60 (1.34)
MDD	7 (28%)	3.86 (1.46)	2.00 (1.41)	8 (32%)	4.38 (1.19)	2.80 (1.80)	18 (23.1%)	4.50 (1.34)	1.43 (0.78)
PDD	6 (24%)	4.67 (1.63)	2.20 (0.84)	10 (40%)	5.00 (1.25)	4.67 (1.51)	23 (28.7%)	4.67 (1.63)	2.20 (0.84)
PMDD	3 (12%)	3.67 (1.16)	–	6 (24%)	3.83 (0.98)	3.40 (1.52)	17 (24.1%)	3.82 (0.95)	3.57 (1.27)
PTSD	5 (20%)	4.55 (1.15)	3.14 (1.16)	10 (40%)	4.70 (0.82)	4.00 (1.55)	18.5 (28.7%)	4.57 (1.19)	2.75 (1.60)
BN	1 (4%)	6.00 –	–	2 (8%)	5.00 (0.00)	4.00 (0.00)	6 (7.9%)	4.83 (0.98)	2.60 (1.52)
BED	5 (20%)	4.60 (1.14)	3.00 (2.00)	3 (12%)	4.33 (0.58)	5.00 –	13 (17.3%)	4.38 (.96)	2.00 (1.53)
SUD	7 (28%)	3.14 (0.38)	2.67 (0.56)	2 (8%)	3.00 (0.00)	3.00 –	20 (20.2%)	3.30 (0.80)	2.82 (0.41)
		Severity	Severity		Severity	Severity		Severity	Severity
ZAN-BPD-SR	–	17.88 (7.96)	4.00 (3.04)	–	17.39 (6.82)	14.26 (7.29)	–	16.44 (7.52)	6.33 (5.70)
ODSIS	–	11.44 (4.47)	4.33 (4.85)	–	10.78 (4.82)	10.63 (5.84)	–	10.14 (4.92)	5.20 (4.40)
OASIS	–	11.12 (3.80)	4.67 (4.85)	–	10.83 (4.10)	10.36 (6.00)	–	10.41 (4.40)	5.86 (4.17)
EDE-Q	–	2.61 (1.59)	1.64 (1.26)	–	2.40 (1.37)	2.56 (1.51)	–	2.55 (1.89)	1.89 (1.31)
PCL-5	–	35.74 (23.10)	15.22 (21.80)	–	43.05 (21.65)	36.82 (26.51)	–	33.95 (22.20)	16.22 (19.53)

[a] The pretreatment assessment was used for BPD Compass-R and BPD Compass-NR, whereas the post-waitlist assessment was used for the delayed treatment condition.

diagnosis at posttreatment (4–8%) was numerically lower than the percentage of patients with each diagnosis post-waitlist (4–48%); however, the degree of dropout makes it difficult to draw meaningful conclusions about the superiority of either condition (see Figure 2). Among those who received BPD Compass (i.e., BPD Compass-Rando mized/Naturalistic, Delayed treatment), average posttreatment CSRs were below the clinical threshold for all diagnoses except premenstrual dysphoric disorder. By contrast, the average CSRs for those assigned to the WLC remained above the clinical threshold for all disorders except bipolar II, agoraphobia, and major depressive disorder (MDD).

Clinician-rated BPD symptoms in the past week, $g = -1.05$, $p = .01$, as well as CSRs for persistent depressive disorder, $g = 1.27$, $p = .03$, and SAD, $g = 1.80$, $p = .01$, were significantly lower at the end of the BPD Compass-Randomized condition than at the end of the WLC. A large, yet nonsignificant, difference between conditions was observed for SAD, $g = .82$, $p = .20$, whereas moderate (nonsignificant) effects favoring BPD Compass-Randomized were observed for MDD, $g = -.40$, $p = .60$, PTSD, $g = -.79$, $p = .31$, and BED, $g = -.56$, $p = .47$. The differences between conditions for bipolar II, $g = -.22$, $p = .40$, generalized anxiety disorder $g = -.13$, $p = .82$ and SUD, $g = -.33$, $p = .66$, were nonsignificant and small in magnitude. Similarly, BPD Compass-Randomized patients demonstrated significantly lower self-reported BPD, depressive, anxiety, and PTSD symptoms at posttreatment than WLC patients did at the end of the WLC that were large in magnitude, $gs > -.83$, $ps < .03$. Although not statistically significant, BPD Compass-Randomized patients reported less severe eating disorder symptoms relative to WLC at posttreatment and this difference was medium-sized, $g = -.62$, $p = .13$.

All Participants

Collapsing across all participants, pre- to posttreatment change in CSRs was significant and large in magnitude for BPD, bipolar II, OCD, SAD, GAD, agoraphobia, MDD, persistent depressive disorder, PTSD, and BED ($gs > .81$, $ps < .02$; Table 3). Nonsignificant, medium-sized improvements were observed for panic disorder and BN severity, $gs > .70$, $ps < .12$; along with small-to-medium-sized, nonsignificant improvements in SUD severity, $g = .43$, $p = .17$.

Collapsing across all participants, BPD Compass was associated with large, significant improvements in self-reported symptoms of BPD, depression, and anxiety, and $gs > .81$, $ps < .01$, and medium-sized, significant improvements in

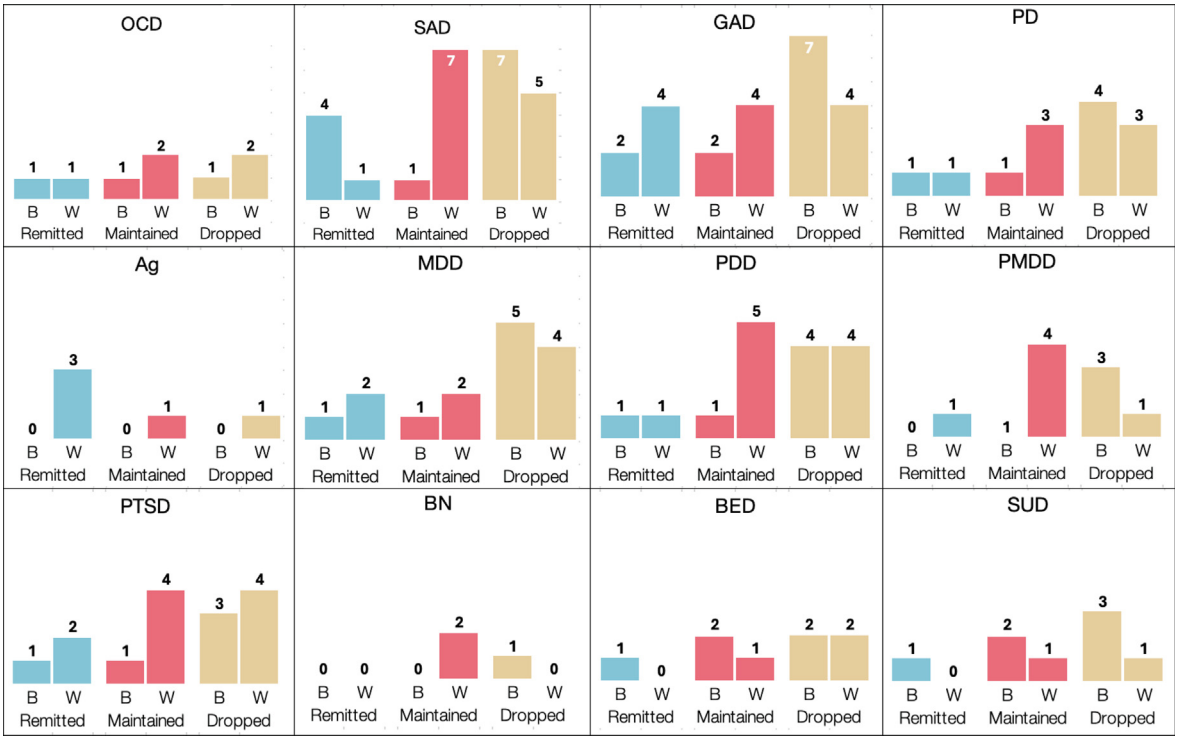

FIGURE 2 BPD Compass trial outcomes as a function of treatment condition (BPD Compass-R, Waitlist Control)

Table 3
Within and Between Condition Effect Sizes Examining the Magnitude of Change in Clinical Severity by Diagnosis

	Within Condition Effects						Between Condition effects			
	Pre- to Post-Treatment All BPD Compass		Pre- to Post-Treatment for BPD Compass-R		Pre- to Post-Waitlist		BPD-R vs Waitlist at Pre-treatment		BPD-R vs Waitlist at Post-treatment	
	Hedges' *g*	CI	Hedges' *g*	CI	Hedges' *g*	CI	Hedges' *g*	CI	Hedges' *g*	CI
BPD	**.92**	.60 : 1.21	**1.13**	.44 : 1.81	–.02	–46 : .43	.16	–.39 : .71	**–1.05**	–1.79 : -.30
BP2	**.86**	.13 : 1.50	.40	–.57 : 1.26	.80	–.11 : 1.65	.03	–.52 : .57	.22	–1.17 : 1.60
OCD	**.84**	.23 : 1.36	1.20	–.37 : 2.82	.32	–.41 : 1.00	–.19	–.73 : .36	–.12	–1.46 : 1.20
SAD	**1.10**	.64 : 1.52	**1.19**	.39 : 2.28	.42	–.24 : 1.06	–20	–.57 : .51	–.82	–1.91 : .28
GAD	**1.23**	.69 : 1.76	.71	–.34 : 1.68	**.91**	.09 : 1.70	–.70	–.62 : .47	–.13	–1.24 : 1.06
PD	.70	–.06 : 1.34	.27	–.62: 1.10	–.10	–.80 : .63	–.25	–.81 : .31	–.67	–2.06 : .80
Ag	**1.24**	.06 : 2.10	–	–	.60	–.27 : 1.40	–.38	–.93 : .16	–	–
MDD	**1.94**	.70 : 2.93	.80	–.45 : 2.00	.37	–.39 :1.10	–.12	–.67 : .42	–.40	–1.77 : 1.02
PDD	**1.50**	.75 : 2.14	**1.27**	.08 : 2.41	.00	–.67 : .67	–.35	–.90 : .20	**–1.80**	–1.13 : –41
PMDD	.16	–.50 : .80	–	–	.07	–.64 : .77	–.30	–.87 : .23	–	–
PTSD	**1.23**	.45: 1.90	1.99	–.26 : 4.56	.50	–.26 : 1.22	–.42	–1.00 : .10	–.79	–2.21 : .70
BN	.81	–.16 : 1.53	–	–	–	–	.04	–.51 : .59	–	–
BED	**1.83**	.54: 2.81	.92	–.35 : 2.01	–	–	.24	–.31 : .80	–.56	–1.90 : .87
SUD	.43	–.17 : .98	.46	–.58 : 1.42	–	–	**.58**	.19 : 1.40	–.33	–1.60 : 1.02
BPD-SR	**1.30**	.92 : 1.70	**1.31**	.47 : 2.16	.29	–.15 : .73			**–1.50**	–2.45 : –.71
ODSIS	**.82**	.49 : 1.12	**.98**	.18 : 1.74	–.04	–.48 : .40	.14	–.42 : .70	**–1.10**	–1.92 : –.27
OASIS	**1.04**	.70 : 1.38	**1.04**	.22 : 1.82	.08	–.36 : .62	.07	–.48 : .63	**–.98**	–1.78 : –.15
EDE	**.59**	.28 : .91	.32	–.40 : 1.03	.72	–.27 : .70	.16	–.42 : .72	–.62	–1.4 :.22
PCL-5	**.76**	.44 : 1.07	**.80**	.04 : 1.51	.38	–.10 : .87	–.32	–.90 : .27	**–.84**	–1.64 : –.11

Note: Bolded Hedges's g values represent effects that are significant at the .05 level. We were unable to calculate within BPD Compass-R effects for Ag, PMDD, and BN as there were no individuals with these conditions at one or both timepoints. We were unable to calculate waitlist effects for BN because the standard error of the different was 0, nor for BED and SUD because there was only one person with each of these conditions who completed the posttreatment assessment. We were unable to calculate between condition effects (BPD Compass-R vs. WLC) at posttreatment for Ag, PMDD, and BN because there was no one who endorsed these conditions in the BPD Compass-R condition.

self-reported PTSD and eating disorder symptoms, *gs* > .58, *p* = .01, pre- to posttreatment. By contrast, pre- to post-waitlist change in each symptom measure was non-significant, *g*s < .39 , *p*s > .12.

Discussion

In this secondary analysis of Sauer-Zavala et al. (2023a), we assessed the extent to which BPD Compass functions as a transdiagnostic intervention in outpatient settings, in line with its design. Considering the high prevalence of coexisting mental disorders among people with BPD (Leichsenring et al., 2024), it was unsurprising that the vast majority of our participants met criteria for additional *DSM-5* disorders, with SAD, GAD, OCD, and PTSD emerging as the most prevalent comorbid diagnoses. Further, the symptoms associated with these comorbid disorders were, on average, consistently reported as moderately distressing and impairing.

As anticipated, fewer participants in the BPD Compass-Randomized condition met criteria for any comorbid disorder at posttreatment compared to those in the WLC at the post-waitlist timepoint. Participants in BPD Compass-Randomized demonstrated significantly lower CSRs in both BPD and persistent depressive disorder compared to those in the WLC. Additionally, although not statistically significant, medium-to-large sized effects were observed for improvements in SAD, GAD, MDD, PTSD, and BED, all favoring the BPD Compass condition. The statistical significance observed in specific conditions like BPD and persistent depressive disorder suggests that BPD Compass may be particularly effective with these disorders, whereas the non-significant but medium-to-large sized effects in other comorbidities imply broader yet impactful effects. Although promising, this pattern of results should be interpreted with caution until they are replicated in larger study, as our small sample size and dropout rate may have biased our outcomes.

Furthermore, posttreatment CSRs for all participants receiving BPD Compass were consistently below the clinical cutoff for all assessed conditions except premenstrual dysphoric disorder. In contrast, participants on the waitlist consistently maintained CSRs above the clinical threshold for all disorders except bipolar II, agoraphobia, and MDD. Again, these findings suggest that BPD Compass may efficaciously reduce both the percentage of patients with comorbid disorders and the severity of such conditions, though the notable dropout rate in posttreatment and post-waitlist assessments limits our confidence in these results. BPD Compass includes a range of therapeutic strategies (e.g., values, cognitive flexibility, behavior change) that are applicable to different psychiatric conditions. This versatility may result in a more transdiagnostic and potent approach to mental health, addressing not only the primary symptoms but also related comorbidities. Importantly, our results provide a signal that BPD Compass is efficacious not only in reducing BPD symptoms but also in addressing comorbid disorders, warranting further study. Specifically, all participants who received BPD Compass exhibited significant and large reductions in clinician-rated symptoms across a spectrum of disorders, including BPD, bipolar II, OCD, SAD, GAD, agoraphobia, MDD, persistent depressive disorder, PTSD, and BED. Further, these results extended to self-reported symptoms of depression, anxiety, and PTSD. Conversely, these outcomes were not replicated in the WLC.

Together, these findings suggest that it may be possible to achieve simultaneous improvements in symptoms across various conditions by specifically targeting personality traits —negative affectivity, antagonism, and disinhibition—that confer risk for multiple disorders. Although there is data to suggest that BPD Compass is associated with large reductions in neuroticism, moderate reductions in some facets of antagonism (i.e., mistrust, manipulativeness), and small statistically signficant reductions in disihibition (Sauer-Zavala et al., 2023b), future research with a larger sample is needed confirm the mediating effects of personality trait change on symptom improvement. This positions BPD Compass as a promising transdiagnostic treatment, offering an avenue for addressing a broad range of psychiatric symptoms in a parsimonious and effective manner (e.g., Hood et al., 2024).

Transdiagnostic treatments undeniably offer compelling advantages. Protocols designed for specific disorders fail to effectively address the presence of multiple conditions. Moreover, training in single-disorder protocols places a considerable burden on clinicians in terms of training expenses (McHugh & Barlow, 2010). Clinicians who implement BPD Compass instead may only need proficiency in this treatment to effectively address BPD symptoms and co-occurring conditions commonly encountered in routine outpatient practice (Sauer-Zavala & Southward, 2023).

A transdiagnostic intervention based on personality traits can also offer the advantage of personalization to individual patient needs. By creating dimensional personality profiles for each patient, specific personality-based modules can be selected based on the mechanisms that contribute to their

particular symptoms (Samuel & Widiger, 2006; Sauer-Zavala, Southward, Hood, et al., 2022). For example, if a patient has elevated negative affectivity and disinhibition and these personality dimensions are also contributing to their comorbid disorders, treatment modules targeting these specific traits can be chosen, whereas modules focusing on remaining traits may not be necessary. Future research examining whether personalizing treatment based on personality trait elevations leads to more robust and efficient improvements is needed, similar to other studies that compare personalized and standard delivery (e.g., Weisz et al., 2012).

LIMITATIONS

These findings should be considered in the context of the study's limitations. Despite the high proportions of participants from sexual and ethnic or racial minority statuses, most of our participants primarily identified as White and female with at least some postsecondary education, limiting the generalizability of results beyond these characteristics. Although we included patients with substance use disorders and bipolar disorder I in our sample, we did exclude people with these conditions when symptoms were severe (SUD) or uncontrolled (mania in the past 12 months), along with those with a history of psychotic features, for whom alternative care (i.e., medication) is the standard. Thus, our findings may not accurately capture patterns of BPD comorbidity at the higher end of the severity spectrum.

Considering the nature of this study as a secondary data analysis, it is important to note that it was not designed with the statistical power necessary to detect small between-condition effects of interest, or moderating effects of demographics or pretreatment processes of interest. This is because we employed a sequential analysis design in which we stopped randomizing after we were powered to detect a large effect on the parent study's primary outcome of interest (i.e., BPD symptoms). We continued recruiting for the naturistic phase of the study in order to have a larger sample to conduct within-treatment analyses. Although we acknowledge that we would be able to draw stronger causal conclusions on the efficacy of BPD Compass for comorbid conditions if we had a larger randomized sample, we feel our sequential analysis design balances service to the community with scientific inquiry. It is also possible that patients who were randomized to receive BPD Compass immediately, those we received this intervention after a waiting period, and those who were in the naturalistic condition may have had different expectancies about the effect of treatment, biasing results. As a result of our small sample, we focused on interpreting effect sizes rather than statistical significance; nevertheless, examining patterns of comorbidity across treatment and between conditions required multiple comparisons and statistically significant findings should be interpreted with caution due to the possibility of false positives.

Finally, the dropout rate in this study was notable. This poses a challenge in evaluating whether BPD Compass would maintain its effectiveness for patients who did not complete the treatment. The extent to which the intervention could yield improvements for those who did not finish the course of treatment warrants further investigation. Understanding the efficacy of BPD Compass across the entire participant spectrum, including those who discontinued treatment, is important for a comprehensive assessment of its impact.

Despite these limitations, we demonstrated an important preliminary signal that BPD Compass can serve as a transdiagnostic intervention. While keeping our small sample size and considerable dropout in mind, BPD Compass not only significantly reduced symptoms associated with BPD but also demonstrated efficacy in addressing a broad spectrum of disorders, including bipolar II, OCD, SAD, GAD, agoraphobia, MDD, persistent depressive disorder, PTSD, and BED. These findings suggest that BPD Compass may present a more parsimonious and potent approach for simultaneously treating BPD and comorbid conditions while reducing the training burden on clinicians, warranting further study.

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