

Emotion

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Proposing a Model Whereby Negative Valence Bias Increases the Risk for More Severe Dysphoric Posttraumatic Stress Disorder and Depression Symptomology

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Experiencing trauma increases risk for posttraumatic stress disorder (PTSD) and depression, and individuals who experience psychopathology after a traumatic event often experience symptoms from both disorders. Because a tendency to view events in a more negative light and a propensity toward threat appraisals are risk factors for both PTSD and depression, negative valence bias—a tendency to appraise emotional ambiguity as having a more negative (less positive) meaning—may be a transdiagnostic risk factor. In other words, we expect individuals with a negative valence bias experience greater PTSD and depression symptoms. We measured valence bias and self-reported PTSD and depression symptoms in a sample of college students in 2021 ($n = 287$; 72.5% reported experiencing trauma). Although valence bias was not associated with PTSD symptoms as a whole, we found in our exploratory model that more negative bias was associated with greater dysphoria-related PTSD symptoms and greater depression symptoms (indirect effect $p = .03$). Thus, we propose a model whereby a more negative valence bias contributes to increased susceptibility for maladaptive stress responses, which may be associated with greater likelihood of symptoms of dysphoria-related PTSD and depression. These findings suggest that valence bias represents a transdiagnostic affective risk factor, warranting future research examining the impacts of bias-altering interventions (e.g., mindfulness-based treatments) as a means for managing symptoms in individuals with heightened dysphoria-related PTSD and/or depression symptoms. Additionally, in post hoc analyses it emerged that Latinx participants displayed a more negative valence bias, indicating the need for more research in diverse samples.

Keywords: posttraumatic stress disorder, depression, valence bias, ambiguity

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Over half of the U.S. population will experience a traumatic event during their lifetime (Ozer et al., 2003). Trauma exposure increases one's risk of developing posttraumatic stress disorder (PTSD; Bernat et al., 1998; Vrana & Lauterbach, 1994) as well as depressive disorders (Roley et al., 2015). Although most people exposed to trauma do not develop psychopathology (Kessler et al., 1995), those who do often experience comorbid PTSD and depression (Chiu et al., 2011; Spinhoven et al., 2014), such that comorbidity is the

rule rather than the exception (Flory & Yehuda, 2015). Individuals who experience PTSD symptoms subsequently have more severe depression symptoms (Subica et al., 2012) with some work suggesting a causal pathway from PTSD to depression (Breslau, 2009; Stander et al., 2014). Existing literature also points to the role of affective mechanisms, such as elevated dysphoria, higher levels of negative affect, lower levels of positive affect, and deficits in emotion regulation abilities as factors that may be sources of co-occurring PTSD

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Claudia Clinchard served as lead for investigation, methodology, project administration, visualization, and writing—review and editing and contributed equally to conceptualization, data curation, and software. Nicholas R. Harp served in a supporting role for conceptualization, data curation, methodology, and writing—review and editing. Tierney Lorenz served in a supporting role for conceptualization, methodology, and writing—review and editing. Maital Neta served in a supporting role for conceptualization, writing—original draft, and writing—review and editing. Claudia Clinchard and Nicholas R. Harp contributed equally to formal analysis and writing—original draft. Nicholas R. Harp and Maital Neta contributed equally to supervision.

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and depression (Post et al., 2011, 2021). Thus, there is a critical need for models accounting for pre-trauma affective risk factors and how such factors contribute to the development of comorbid psychopathology.

Notably, both PTSD (Elwood et al., 2009) and depression (Beck, 1967) are characterized by a pervasive tendency to view events in a more negative light, as well as the tendency to avoid experiences or events that bring up undesired internal experiences (Flory & Yehuda, 2015; Moulds et al., 2007). For instance, attention to and recall biases for negative information have been proposed to foster symptoms such as negative cognitions related to the self and the world in those with PTSD (Imbriano et al., 2022). Such a tendency might fuel maladaptive posttraumatic stress responses that ultimately give rise to posttrauma depression. If so, clinical interventions for PTSD and depression may benefit from targeting the underlying affective risk factors linking both disorders. To test this model, we used a sample of young people with and without a history of trauma to examine the extent to which individual differences in affective processing (i.e., valence bias) may putatively lead to increased PTSD and depression symptoms.

Individual differences in affective processing styles can be assessed via responses to emotional ambiguity. By definition, emotionally ambiguous cues have equally valid positive and negative meanings. Thus, in the absence of a clarifying context, individuals vary in their tendency to interpret these cues as positive or negative (i.e., valence bias; Neta et al., 2009). Supporting its usefulness as a prognostic risk factor, valence bias is a trait-like difference that is stable over time (Harp et al., 2022) and generalizes across responses to emotionally ambiguous faces, scenes, and words (Harp et al., 2021; Neta et al., 2013). In the short-term, valence bias guides one's view of the world (e.g., approach-avoidance behaviors; Harp et al., 2021; Krieglmeier et al., 2010), and over the longer-term, valence bias is associated with well-being in physical (Neta et al., 2019), psychological (Park et al., 2016; Petro et al., 2021), and social domains (Neta & Brock, 2021).

Interpretation bias, which refers to the tendency to interpret ambiguous or neutral information as negative or threatening (Beck et al., 2005) has also been studied in individuals with PTSD (Bomyea et al., 2017) and depression (Everaert et al., 2017, 2018). There are important similarities between valence bias and interpretation bias: for example, both constructs capture individual differences in the evaluation of emotional ambiguity that are associated with internalizing symptomology (Everaert et al., 2018; Neta & Brock, 2021; Petro et al., 2021). However, valence bias leverages relatively simple socioemotional signals such as an image of a surprised facial expression, which is ambiguous in that it could be interpreted as positive (winning a prize) or a negative (losing something important) that are developmentally appropriate and highly sensitive to individual differences in bias toward both positivity and/or negativity (J. E. Pierce et al., 2023; Puccetti et al., 2023), and not specific to a trauma-related experience. In contrast, other measures of interpretation bias leverage more complex stimuli (rearranging words to construct a meaningful sentence) or social scenarios that are more challenging for children and, in many paradigms, can only be interpreted as negative/threatening or neutral, but not dual-valence (i.e., there is not an equally valid positive alternative interpretation). Further, these paradigms are often based on trauma exposure status, where the negative/threatening interpretation is related to the trauma (e.g., an image of a man and a woman kissing on a couch may be positive to a nontrauma-exposed individual but may trigger the trauma of someone that survived sexual violence; Elwood et al., 2007).

Previous work has linked a more negative (less positive) valence bias to increased internalizing symptoms. For instance, in a study of individuals without known trauma histories, greater elevations in cortisol (a marker of stress reactivity) were associated with more negative responses to surprised faces following a stressor (Brown et al., 2017). Likewise, heightened emotional arousal to threat of an electric shock was associated with a more negative valence bias (Neta et al., 2017). Further, there is evidence that a more negative valence bias is associated with greater symptoms of depression (Neta & Brock, 2021; Petro et al., 2021) and anxiety (Park et al., 2016).

Moreover, a negative valence bias could contribute to a maladaptive response following a stressful and traumatic event—including symptoms that contribute to PTSD. PTSD is characterized by an elevated subjective sense of threat (Dunmore et al., 2001), such as viewing the world (and people in it) as more threatening. This may be more adaptive in the short term as it could serve to protect an individual from experiencing future harm (Pollak & Kistler, 2002). However, attributing ambiguous—that is, potentially threatening or rewarding—social information to predominantly negative dimensions may increase behavioral avoidance of all but the most overtly positive cues. Over time, such avoidance would powerfully reinforce further threat perceptions (Espejo et al., 2017), contributing to heightened traumatic stress, but also leading to fewer opportunities for social reward—which itself is a significant maintaining factor in depression (Lancaster et al., 2016). Additionally, diminished access to social rewards may account for part of the association between PTSD and depression. It has been shown that anhedonia, a crucial diagnostic feature of PTSD, can result from deficits in reward functioning (Nawijn et al., 2015). While PTSD was found to be associated with reduced reward anticipation, the approach, arousal, and valence ratings of positive images were not associated with PTSD (Nawijn et al., 2015). Thus, both processes of increases in negative interpretations for ambiguous stimuli and diminished reward functioning are likely at play in the overlap between PTSD and depression, consistent with the tripartite model of anxiety and depression (Clark & Watson, 1991).

Here, we used an undergraduate sample to test our proposed model in which a negative valence bias increases the risk for more severe PTSD and depression symptoms. Recruiting a college sample may be more representative of a trauma-exposed population than other adult community members. Indeed, the prevalence of lifetime PTSD ranges from 5.6% (Frans et al., 2005) to 7.8% (Kessler et al., 1995) in the general population, but has been found to be higher, at approximately 9% (Read et al., 2011), in college students. We expected that PTSD symptoms may account for the association between negative valence bias and depression symptoms. We hypothesized that measures of valence bias, PTSD symptoms, and depression symptoms would all be positively correlated. Then, we tested whether a more negative valence bias was associated with higher levels of PTSD symptoms, and subsequently associated with more severe depression symptoms. Given extant literature showing high comorbidity between PTSD and mood disorders (e.g., depression; Chiu et al., 2011; Spinhoven et al., 2014), and literature indicating stronger support for a directionality from PTSD to depression (Flory & Yehuda, 2015; Stander et al., 2014), a more negative valence bias may be a source of these comorbid psychopathologies. That is, a more negative valence bias may first explain a more maladaptive response to trauma, by way of higher PTSD symptoms, which may then explain the development and/or maintenance of depression symptoms.

Method

Transparency and Openness

The current study was not preregistered. Deidentified data is available on the Open Science Framework (<https://osf.io/kt3m6>). The list of stimuli is included in Table S1 in the online supplemental materials. Data were analyzed with R, primarily using the lavaan package (Rosseel, 2012) and the psych package (Revelle, 2023).

Participants

Participants were university students recruited from the University of Nebraska-Lincoln subject pool and received course credit for participation. Study advertisements described all measures, including assessment of traumatic life events. The data were collected in 2021. All procedures were approved by the institutional review board of the university, and participants provided informed consent, in accordance with the Declaration of Helsinki. We expected a small association between depression symptoms and valence bias and a small association between PTSD and valence bias, given prior findings with depression and anxiety measures (Neta & Brock, 2021; Park et al., 2016). We expected a association relationship between PTSD and depression, given prior work (Morris et al., 2012). According to post hoc simulation-based estimates, a percentile bootstrapped path analysis with one small and one small-to-moderate effect would require approximately 412 participants (Fritz & MacKinnon, 2007). We recruited a total of 385 participants, but 98 were removed for poor data quality (described below). Of the remaining 287 participants, 84.3% ($n = 242$) identified as White, 2.8% ($n = 8$) as Black, 7.0% ($n = 20$) as Asian, 1.0% ($n = 3$) as American Indian/Alaskan Native, 4.2% ($n = 12$) as multiracial, and 0.07% ($n = 2$) were unreported. Ages ranged from 17 to 36 years ($M_{\text{age}} = 20.03$, $SD = 1.84$). The majority of participants were female (female = 222; male = 64; one participant did not report their sex); gender identity was not assessed separately from sex. Participants self-reported their ethnicity. In terms of ethnicity, 9.8% ($n = 28$) of participants identified as Latinx, 88.5% ($n = 254$) as not Latinx, and 1.7% ($n = 5$) did not report on ethnicity. Information on socioeconomic status was not collected.

Measures

The PTSD Checklist for *DSM-5* (PCL-5; Weathers et al., 2013) and the Beck Depression Inventory-II (BDI-II; Beck et al., 1996) were used to measure PTSD and depression symptoms, respectively. Both measures are extensively validated and show good reliability in determining symptoms of these disorders (Faro & Pereira, 2020; Ghazali & Chen, 2018; Seligowski & Orcutt, 2016; Wang & Gorenstein, 2013). In the present sample, our observed reliability was similarly good ($\alpha = .95$ for both measures). Importantly, both measures also detect subdiagnostic (but still clinically relevant) psychopathology in community populations (Brancu et al., 2016; Faro & Pereira, 2020). The PCL-5 cutoff score indicating probable PTSD is suggested to be between 31 and 33 (Bovin et al., 2016), and commonly accepted cutoffs for BDI-II designate mild (14–19), moderate (20–28), and severe (29–63) categories of depression (Beck et al., 1996). Additionally, the self-reported Life Events Checklist for *DSM-5* (LEC-5; Weathers et al., 2013) scores were used as a qualitative measure of the type of trauma (e.g., interpersonal violence and combat) and to better characterize the trauma experience of this sample (see Table 1).

Table 1

Breakdown of Trauma Types, as Measured by the LEC-5, for Participants Who Met the Cutoff Score of 31 on the PCL-5

Trauma type	Number of participants (%)
Intimate partner/sexual violence	15 (5.2)
Accident	14 (4.9)
Trauma of loved ones	7 (2.4)
Other traumas	6 (2.1)
Unexpected death of a loved one	4 (1.4)
Physical violence	3 (1)
Did not report	18 (6.3)
Any trauma (total)	67 (23.3)

Note. LEC-5 = Life Events Checklist for *DSM-5*; PCL-5 = PTSD checklist for *DSM-5*; PTSD = posttraumatic stress disorder.

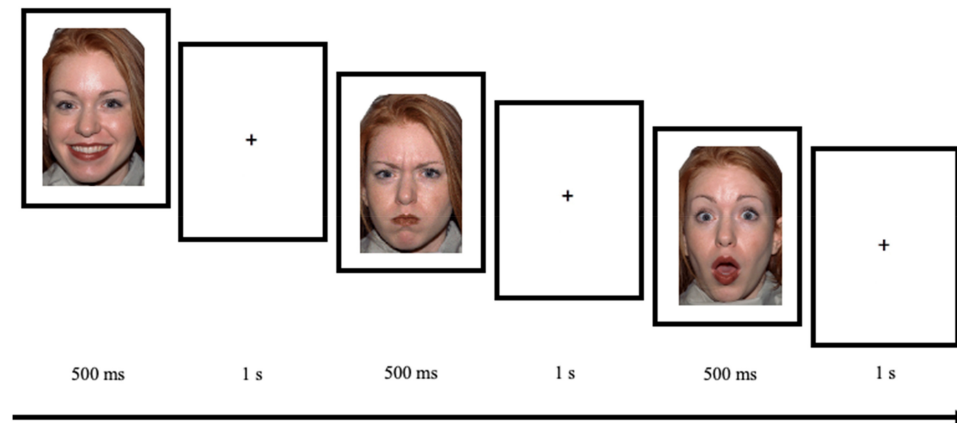
Procedure

We used a validated two-alternative forced choice categorization task for assessing valence bias (Harp et al., 2021), delivered online via PsychoPy (J. W. Pierce et al., 2019) and hosted on Pavlovia (Pavlovia, n.d.). Participants completed the study on their own devices in the location of their choice (e.g., in their homes). See Figure 1 for the task layout. In the task, participants viewed six task blocks—two from each stimulus category (faces, scenes, and words). All stimuli were taken from prior work demonstrating ambiguity for each stimulus category (see Table S1 in the online supplemental materials for the list of stimuli). We included three categories of stimuli—rather than relying on only one—to produce a robust and generalizable measure of valence bias (Harp et al., 2021). Analyses were collapsed across the stimulus categories (i.e., faces, scenes, and words) as there is evidence that the same individuals who categorize ambiguous faces as more positive respond similarly to ambiguous scenes (Neta et al., 2013) and words (Harp et al., 2021).

Participants were instructed to categorize the stimulus on each trial as either positive or negative by pressing either the “A” or “L” key on their keyboard (response keys counterbalanced across participants). Both the face and scene blocks included 12 ambiguous images and 12 clear images (six positive, six negative), for a total of 48 faces and scenes across blocks. The faces were taken from the NimStim (Tottenham et al., 2009) and Karolinksa Directed Emotional Faces (Lundqvist et al., 1998), and reflected either clearly positive (happy) or negative (angry) affect, or ambiguous affect (surprise). Scenes were taken from the International Affective Picture System (Lang et al., 2008), and were validated to reflect either clearly positive (e.g., puppies) or negative contexts (e.g., a bad car accident) or ambiguous emotional contexts that are appraised as positive by some individuals and as negative by others (e.g., a tearful scene that could be a reunion or a parting ways; Neta et al., 2013). Each word block consisted of 11 ambiguous and 11 clear (either five or six positive, either five or six negative) words, for a total of 44 words (Harp et al., 2021). Words were presented in capital letters in plain black font on a white background. Thus, participants completed 140 total trials.

As data quality checks, we (a) trimmed trials in which the reaction times were faster than 250 milliseconds or in which the reaction times were slower than three times the standard deviation for that individual’s reaction time, and (b) used responses to the clear valence images (e.g., happy and angry). Specifically, if participants failed to categorize the clearly valenced stimuli above 60% accuracy in any single stimulus category (the cutoff used in extensive prior

Figure 1
Valence Bias Task



Note. Blocks of faces, scenes, and words—each comprising 50% ambiguous images (surprise) and 50% clear/unambiguous (25% positive and 25% negative; happy and angry)—were presented to participants for 500 ms with 1 s fixation crosses in between each image. Participants categorized every image as either positive or negative. The facial expressions shown here in this figure come from the NimStim data set (Actor 01F) (Tottenham et al., 2009). See the online article for the color version of this figure.

work; Harp et al., 2021; Neta et al., 2013, 2017), then the values for that stimulus category were treated as missing and the average of the two remaining categories were used to calculate valence bias; participants with two or more stimulus categories missing were removed prior to analysis, as in previous work (Harp et al., 2021). Of the 385 participants recruited, 98 were eliminated due to inaccurate categorizations in two or more categories. Of the 287 remaining participants, 61 were missing data in one category (due to inaccurate categorizations), and thus their valence bias scores were calculated from the two remaining stimulus categories. We note, due to the method of online data collection, a fair number of participants were removed for poor-quality data following manipulation checks, but this is consistent with other reports stating an expected data loss of 10%–50% (Curran, 2016). We found no significant differences between participants with no missing categories or one missing category for all study variables (see Section S1 in the online supplemental materials). After completing the task, participants completed the PCL-5, LEC-5, and BDI-II.

Data Analysis

Data analysis was completed in R (Version 3.3.3; R Core Team, 2021). First, we calculated valence bias for each participant as the percentage of negative categorizations of ambiguous faces, words, and scenes out of the total number of trials for that condition (excluding omissions; Neta et al., 2009). For example, if a participant categorized 12 out of 24 ambiguous faces as negative, then that individual's valence bias would be 50% for faces.

The distribution of all key variables departed significantly from normality (PCL-5: $W = 0.91, p < .001$; BDI-II: $W = 0.91, p < .001$; valence bias: $W = 0.99, p = .04$). As such, bivariate associations are reported using Spearman's rho. Demographic characteristics, including age, sex, ethnicity, and racial identity were screened for associations with the independent and dependent variables and included as controls if significantly related. Path analysis and

exploratory factor analysis models were conducted using the lavaan package (Rosseel, 2012). To account for the relatively small amount of missing data (covariance coverage: 0.98–1.00), we used maximum likelihood estimation which is preferable to traditional missing data treatments (e.g., listwise deletion; Enders, 2010).

For the key test of our hypothesis, we examined the degree to which the effect of valence bias on depression occurs through the path of PTSD symptoms. Specifically, we conducted a regression analysis to examine if valence bias was associated with depression symptoms (i.e., direct path). Then, we used a regression analysis to examine if valence bias was associated with PTSD symptoms. To test our mediation hypothesis, we used maximum likelihood and a percentile bootstrap estimation approach with 10,000 samples (Hayes, 2009) to examine the indirect effect from valence bias to depression by way of PTSD symptoms. Some have noted that such analyses can be useful in determining indirect pathways where there are clear theoretic grounds and interpretation is non-causal (Shrout, 2011). We conceptualized valence bias as a stable trait likely present prior to trauma experience (e.g., Harp et al., 2022), and reviews have noted that the preponderance of research supports stronger directionality from PTSD to depression than from depression to PTSD (e.g., Flory & Yehuda, 2015; Stander et al., 2014). Thus, we ordered the chain of our path analysis from valence bias to PTSD symptoms to depression symptoms.

Results

Characteristics of the Sample

Out of the 287 participants, 208 (72.5%) reported having experienced at least one traumatic event in their lifetime. Sixty-seven participants (23.3%) met the cutoff of 31 on the PCL-5 (Bovin et al., 2016); however, the average symptom scores were below the clinical cutoff ($M = 18.88; SD = 16.14$) as is typical for a university sample. While the average depression scores were similarly moderate ($M = 13.31, SD = 11.66$), when considering established cutoffs for

BDI-II (Beck et al., 1996), 39 met the criteria for mild depression, 37 met the criteria for moderate depression, and 34 met the criteria for severe depression.

Bivariate Relationships

Group means for the categorization of positive, negative, and ambiguous stimuli from the valence bias task are provided in Table S2 in the online supplemental materials. Correlations among concurrent measures were all in the expected direction (Table 2). Valence bias was correlated with PTSD symptoms, $r(285) = .11$, $p = .05$, and depression symptoms, $r(285) = .19$, $p = .001$, such that a more negative valence bias was related to more severe symptoms. Further, we replicated earlier work showing that PTSD symptoms and depression symptoms were positively correlated, $r(285) = .72$, $p < .001$ (Chiu et al., 2011), such that higher levels of PTSD symptoms were associated with higher depression symptoms. Female participants showed a more negative valence bias than males, $t(284) = 3.52$, $p < .001$; one did not report their sex and thus was not included in the analysis, as in previous work (Neta et al., 2019). Female participants also reported greater levels of both PTSD, $t(283) = 3.34$, $p < .001$; one did not answer the PCL questionnaire and thus was not included in the analysis, and depression symptoms, $t(279) = 3.75$, $p < .001$; five did not answer the BDI questionnaire and thus were not included in the analysis. Ethnicity was related to valence bias, $t(280) = 2.21$, $p = .03$; five had missing ethnicity data and thus were not included in the analysis, PTSD symptoms, $t(279) = 2.20$, $p = .03$, and depression symptoms, $t(275) = -2.40$, $p = .02$, such that Latinx individuals had a more negative valence bias and greater symptoms of PTSD and depression than non-Latinx individuals.

Path Analysis Results

A series of regression analyses were used to test the predicted effect of PTSD symptoms on the relationship between valence bias and depression symptoms. The model was just identified. Results indicated that valence bias ($b = 0.18$, $SE = 2.70$, $t = 1.27$, $p < .001$) and PTSD symptoms ($b = 3.50$, $SE = 0.76$, $t = 4.63$, $p < .001$) were both significantly associated with depression symptoms in separate regression models. Further, valence bias remained significantly associated with depression symptoms ($b = 0.09$, $SE = 0.04$, $z = 2.34$, $p = .02$) even when including PTSD symptoms in the model. Notably, this effect was observed after controlling for sex differences in PTSD symptoms ($b = -6.04$, $SE = 2.07$, $z = -2.91$, $p = .004$)

and depression symptoms ($b = -1.90$, $SE = 0.93$, $z = -2.03$, $p = .04$), as well as after controlling for ethnicity differences in PTSD symptoms ($b = 6.53$, $SE = 2.98$, $z = 2.19$, $p = .03$) and depression symptoms ($b = 1.70$, $SE = 1.75$, $z = 0.97$, $p = .33$). In the full model (see Figure 2), PTSD symptoms were significantly associated with depression symptoms ($b = 0.49$, $SE = 0.03$, $z = 14.55$, $p < .001$). Bootstrap estimation indicated the indirect coefficient was not significant ($b = 0.05$, $SE = 0.03$, $z = 1.74$, $p = .08$).

Exploratory Analyses

We examined the existing four-factor *DSM-5* symptom clusters and did not find evidence of these clusters as mediators (see Section S2 in the online supplemental materials). As a follow-up analysis, we used a data-driven approach to conduct an exploratory factor analysis to identify different dimensions of PTSD symptoms within our sample (see Section S3 in the online supplemental materials). We tested the path analysis model described above using each of the factors identified in our exploratory factor analysis. The indirect effects were not significant for Factor 1 (intrusions and avoidance; $b = 3.13$, $SE = 2.55$, $z = 1.23$, $p = .22$) or Factor 3 (anxious arousal; $b = 1.23$, $SE = 2.36$, $z = 0.52$, $p = .60$). In contrast, the path analysis model for Factor 2 (dysphoria symptom cluster) revealed a significant indirect effect ($b = 0.08$, $SE = 0.04$, $z = 2.13$, $p = .03$) indicative of a more negative valence bias being associated with increased dysphoria-related PTSD and depression symptoms. When including Factor 2 in the path model (see Figure 3), the direct path between valence bias and depression was no longer statistically significant ($b = 0.06$, $SE = 0.03$, $z = 1.89$, $p = .06$). Additionally, we tested our models with lifetime exposure to traumatic events as a covariate, and the results were unchanged (see Section S4 in the online supplemental materials).

Discussion

We examined the associations between negative valence bias, PTSD symptoms, and depression symptoms. Depression symptoms were positively associated with both valence bias and PTSD symptoms, though the association between valence bias and overall PTSD symptoms was not statistically significant. That said, we found in our exploratory data analysis that the dysphoria-related subset of PTSD symptoms was associated with a more negative bias. Having a more negative valence bias may not be a risk factor for PTSD symptoms as a whole, but rather a risk factor for the development of the comorbidity of PTSD and depression. For example, it has been

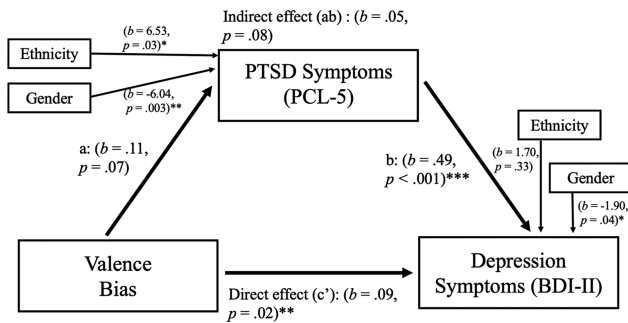
Table 2
Descriptive Statistics and Correlations

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. Age	20.03	1.84	—					
2. Sex (female = 1, male = 0)	0.77	0.43	-.02	—				
3. Valence bias	53.60	14.30	.07	.20***	—			
4. PTSD symptoms	18.88	16.14	.07	.19**	.13 [†]	—		
5. Dysphoria-related PTSD symptoms	7.32	6.24	.05	-.15*	.16**	.90***	—	
6. Depression symptoms	13.31	11.66	.11	.23***	.19**	.72***	.77***	—
7. Ethnicity (Hispanic or Latino/a = 1, not = 0)	0.11	0.31	-.05	.00	.15*	.16*	.16*	.15*

Note. Thirty-nine participants met the criteria for mild depression (score of 14–19), 37 met criteria for moderate depression (20–28), and 34 met criteria for severe depression (29–63). PTSD = posttraumatic stress disorder.

[†] $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2
Path Model Examining the Indirect Effect of Valence Bias Through the Sum of All Items on the PCL-5 to Depression Symptoms

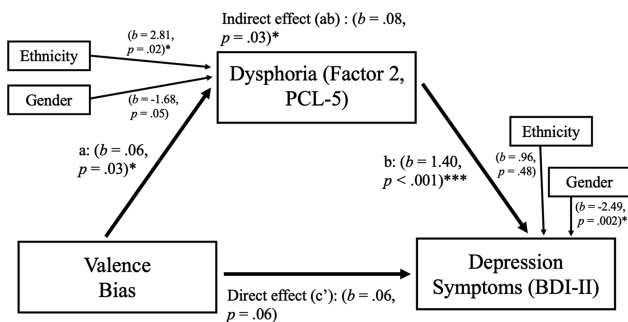


Note. Path c' represents the direct effect of valence bias to depression symptoms. Path ab represents the indirect effect of valence bias through PTSD symptoms to depression symptoms. PCL-5 = PTSD Checklist for DSM-5; PTSD = posttraumatic stress disorder; BDI-II = Beck Depression Inventory-II.

found that negative interpretation biases, which is a construct highly related to valence bias, play a causal role in the development of anxiety and depression (Yoon et al., 2020). Further, because most of our participants had only been exposed to one trauma during their life, having a more negative valence bias may be a risk factor primarily for comorbid PTSD and depression prior to multiple trauma exposures.

Although we did not find evidence that overall PTSD symptoms explained the link between valence bias and depression symptoms, dysphoria-related PTSD symptoms did explain this link. That is, a more negative valence bias was associated with greater levels of dysphoria-related PTSD symptoms, which explained the association between a more negative bias and more severe depression symptoms. This model is consistent with evidence that PTSD symptoms may contribute to risk for depression (Breslau, 2009; Stander et al., 2014). Notably, we found this pattern in a sample spanning subclinical and clinical levels of symptoms on both measures. We discuss the clinical implications of these findings and situate them in a

Figure 3
Path Model Examining the Indirect Effect of Valence Bias Through the Dysphoria-Related Items on the PCL-5 to Depression Symptoms



Note. Path c' represents the direct effect of valence bias to depression symptoms. Path ab represents the indirect effect of valence bias through Factor 2 (dysphoria-related) of PTSD symptoms to depression symptoms. PCL-5 = PTSD Checklist for DSM-5; PTSD = posttraumatic stress disorder; BDI-II = Beck Depression Inventory-II.

broader theoretical framework—the initial negativity hypothesis—that may explain why a more negative valence bias serves as a transdiagnostic affective risk factor that could contribute to the development of comorbid PTSD and depression.

Clinical Implications

Our findings and proposed model have several clinical implications. While a large majority of our sample experienced a traumatic event (72.5%), only 23.3% of our sample reported clinically significant levels of PTSD symptoms at the time of the study. Although most of the sample did not report clinically severe PTSD symptoms, there is much to gain from studying individuals with subclinical symptoms. Indeed, those with subclinical symptoms respond better to treatment (Korte et al., 2016), and subclinical levels of PTSD may reflect preclinical symptoms that accumulate over multiple trauma exposures into clinically severe PTSD. Understanding how valence bias is associated with dysphoria-related PTSD symptoms, including at subclinical levels, may thus facilitate the identification of individuals who could benefit from early intervention. That is, valence bias might represent an affective risk factor and possible treatment target.

Interventions that target a more negative valence bias may be most beneficial if they are promoted as a protective factor against adversity (i.e., prior to repeated traumatic experiences). As such, addressing valence bias may add to existing interventions to promote emotional flexibility and adaptability, which have in turn proven critical for the success of resilience training programs for individuals in high-risk occupations such as military personnel (Brassington & Lomas, 2021; Horn et al., 2016). Methods of intervention that alter valence bias could include mindfulness practice (Harp et al., 2022) and cuing cognitive reappraisal use (Neta et al., 2023). Such efforts may be useful in conjunction with traditional interventions for PTSD and depression (e.g., cognitive behavioral therapy). Moreover, it is possible that these known interventions for comorbid PTSD and depression may act, at least in part, by altering valence bias. For example, the positive effects of mindfulness-based treatments in reducing symptoms of PTSD (Hopwood & Schutte, 2017) and depressive symptomatology (McCarney et al., 2012) may be attributable to the impacts of mindfulness training on reducing negative valence bias (Harp et al., 2022). Considering that a more negative valence bias may make one more prone to experiencing a maladaptive stress response, these interventions focusing on altering valence bias may be most beneficial if they are promoted as a protective factor against adversity. However, we note that the bias-altering interventions require randomized clinical trials. Such work could better examine the durability of the intervention effects as well as causal associations of the interventions on valence bias and symptoms.

Additionally, given that valence bias and overall PTSD symptoms were not correlated, one important caveat of such an approach focused on altering valence bias is that it might alleviate only dysphoria-related symptoms, rather than the full range of PTSD symptoms. Dysphoria symptoms include difficulties with sleep, irritability, and concentration, but also include alterations in one's worldview, with adverse impacts on social connections. Given our findings that a more negative bias was associated only with worsened dysphoria-related symptoms of PTSD, it may be the case that only this cluster of PTSD symptoms is alleviated by interventions that target valence bias. One possibility for this pattern of results is that the dysphoria subset of symptoms identified here may be

bridge symptoms (i.e., symptom clusters that span different disorders) between PTSD and depression. For instance, previous research suggests that dysphoria symptoms play a prominent role in bridging these two disorders (Afzali et al., 2017), possibly linking PTSD symptoms to later depression. As such, altering an individual's valence bias may reduce the likelihood of dysphoria symptoms post-trauma, eradicating this bridge to posttraumatic depression symptoms. Identifying such bridge symptoms is crucial, as addressing the symptoms can prevent contagion of one mental disorder to another. In other words, minimizing dysphoric-related PTSD symptoms may prevent the likelihood of individuals developing comorbid disorders (e.g., depression; Afzali et al., 2017; Jones et al., 2021).

One other notable clinical implication relates to the role of ethnicity in our model. Other research has indicated that Latinx individuals in the United States are at greater risk for developing PTSD (Pole et al., 2005) and depression (Skaer et al., 2000), likely due to minority stress and exposure to systemic discrimination (Sibrava, et al., 2019). In addition to replicating this finding, we also showed an extension of this pattern in that, on average, the Latinx participants had a more negative valence bias than the non-Latinx participants. This is both a novel contribution and a notable strength of the present research. Specifically, much previous work assessing valence bias relied on White participants to control for the possibility of "out-group" effects while viewing facial stimuli consisting solely of White faces; however, in our more diverse sample, we found significant effects of ethnicity on valence bias. This finding suggests one of two causes: (a) an "out-group" effect in which facial expressions may be more arousing and threatening to people viewing the faces when they are from a racial or ethnic group that differs from the facial stimuli (Fani et al., 2012; see also Basyouni et al., 2022 for out-group effects on valence bias), or (b) enhanced threat perception within marginalized groups. Given the inclusion of nonface stimuli (scenes, words), we believe the latter interpretation is more likely. The more negative valence bias in Latinx versus non-Latinx identifying individuals could also be a result of greater trauma exposure; future work is needed in this area. It is possible that Latinx individuals are exposed to forms of trauma that are not captured by traditional trauma measures (including the one used in the current study) that may be further associated with a more negative valence bias. For example, immigration trauma and discrimination stress are associated with symptoms of PTSD and depression but not assessed with the current measure (de Arellano et al., 2018).

A Mechanistic Account of Valence Bias

The working model for understanding individual variability in valence bias is the initial negativity hypothesis, positing that the initial interpretation of ambiguous cues tends to be negative (Neta & Whalen, 2010), and that positive interpretations require regulatory processing to help override this initial response (Petro et al., 2018). Given that more frequent use of emotion regulation strategies, like cognitive reappraisal, buffers stress-related increases in negativity (Raio et al., 2021), improving emotion regulation skills may help disrupt the relationships among valence bias, PTSD symptoms, and depression symptoms. For example, valence bias became more positive throughout an 8-week Mindfulness-Based Stress Reduction program (Harp et al., 2022), suggesting that mindfulness and acceptance-based emotion regulation strategies are likely helpful for overcoming the initial negativity (Kober et al., 2019). Indeed,

difficulties in emotion regulation play a role in both depression (Joormann & Stanton, 2016) and PTSD symptom severity, even when controlling for negative affect (Tull et al., 2007), further underscoring this possibility. In other words, it could be that a more negative valence bias reflects difficulties in emotion regulation that give rise to PTSD and depression.

A more positive valence bias, which is associated with increased well-being and resilience when facing possible threats (Brown et al., 2017; Neta et al., 2017; Raio et al., 2021), may be indicative of seeing stressors as opportunities for growth. Although the mechanism of reappraisal, which putatively supports a more positive valence bias, may be maladaptive in situations of uncontrollable stress like acute trauma (Troy et al., 2013), there may be other adaptive strategies to improve posttraumatic outcomes. Certainly, experiencing a traumatic event—while clearly not inherently good—could set the stage for subsequent growth and development (Thomas, 2015; Westphal & Bonanno, 2007). Children who have been previously institutionalized show a more positive valence bias that is likely associated with increased resilience (Vanteghem et al., 2017), and attentional bias toward resilience-related affective stimuli is associated with greater posttraumatic growth (Gonzales-Mendez et al., 2020). It is possible that a more positive valence bias buffers against comorbid PTSD and depression by lending to a mindset of seeing high-stress events as opportunities for growth (Jamieson et al., 2013).

Constraints on Generality

Due to the restricted age range, we are unable to determine if these effects generalize to other age groups. It would be useful to examine different age groups (e.g., children and older adults), as age may play a role in how individuals react to stressful situations and the degree to which they can regulate their emotions. For example, it has been found that older age is associated with a shift from an initially negative to an initially positive response to emotional ambiguity (Petro et al., 2021). Additionally, the sample had predominantly female participants and so our model may not generalize across sexes or genders. Future work should consider recruiting a more representative sample in terms of age and sex and should replicate and further explore the impacts of minority stress or marginalization on valence bias.

Limitations and Future Directions

Of course, the work is not without its limitations. Because these analyses may have been underpowered, according to simulation-based estimates (Fritz & MacKinnon, 2007), the results should be interpreted with caution and treated as preliminary. Nonetheless, the analyses remain valuable because they provide an estimate of the effect sizes relevant for planning future work. One caveat is that screening questionnaires—though convenient—are not as precise as clinical diagnoses or diagnostic interviews. That said, the questionnaires do reflect a range of symptoms associated with these disorders, both above and below clinical thresholds, which allows researchers to explore the full range of symptom severity. Future work could consider using trauma-specific stimuli rather than the more general emotional stimuli used here and could aim to replicate and extend these findings in a broader population with particular focus on Latinx samples, thus establishing effect sizes and potential moderators.

Additionally, 23.3% of our sample had met the criteria for PTSD according to the PCL-5. While this is a higher rate than most general community samples, future work should recruit a larger, more

diverse sample, with a wider array of experienced traumatic events and greater levels of PTSD symptoms to ensure sufficient power and further examine generalizability of the present findings. Further, the proposed model should be extended to studying individuals who shared similar experiences with trauma (e.g., veterans).

Further, these data were collected at a single time point, limiting the ability to track these changes over time. While there is a strong theoretical background indicating the directionality of PTSD beginning before depression (Breslau, 2009), it is also possible that there is a parallel (rather than sequential) process. That is, the findings presented here have directional paths that are consistent with our hypothesized causal model, but because they are derived from a cross-sectional data set, they cannot establish causality per se. Thus, to replicate these findings and establish causality, several avenues for future work should be considered. For example, longitudinal studies can capture pre-trauma valence bias as predictor of posttraumatic depression responses and would help establish directionality. For example, Beevers et al. (2011) used this kind of design to establish that affective biases measured in soldiers prior to deployment predicted development of depression symptoms following war-zone exposure. In addition, valence bias may be manipulated in a well-controlled randomized clinical trial for depression in people who have experienced trauma. Establishing PTSD symptoms as a treatment mediator—that is, a causal mechanism by which changes in valence bias influence changes in depression symptoms—would require repeated measures of both process and outcome variables as well as establishing a dose–response association (Cuijpers et al., 2019).

Conclusion

We found evidence for our proposed model whereby a negative valence bias was associated with greater PTSD symptoms and higher levels of depression symptoms, but only for a dysphoric subset of PTSD symptoms. How individuals respond to ambiguous cues gives a more textured representation of how individuals process their surroundings and is linked to differences in stress responses and mood symptomatology. A negative valence bias in PTSD and depression may reflect difficulties in emotion regulation that help lay the foundation for the maintenance of PTSD and depression symptoms. While enhanced stress reactivity is associated with more negative interpretations of ambiguity, emotion regulation skills have been found to shift interpretations in the positive direction. Altogether, these results suggest that a maladaptive response to a stressor may arise from a more negative valence bias, which may contribute to greater symptoms of depression. However, this proposed model needs further testing to establish directionality and causality.

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