Supplementary Materials for Villanueva et al. (2022)

Supplementary Analyses Part 1: Controlling for BD Mood Symptoms

Consistent with previous studies on bipolar risk and emotion functioning, we ran parallel analyses controlling for clinical symptoms, to help determine whether any observed associations between emotion differentiation and bipolar risk (HPS-20) were independent of transient elevation in current symptomatology (e.g., Ford, Mauss & Gruber, 2015; Gruber et al., 2018; Gruber & Johnson, 2009). Specifically, we re-ran our main analyses for ED-General, as well as ED-Positive and ED-Negative, controlling for current mood symptom severity in Block 3, with emotion differentiation scores in Block 4 (either ED-General, or ED-Positive and ED-Negative). This analysis approach is consistent with previous research controlling for symptom severity as a potential confound in studies of BD trait risk using the HPS.

Study 1 Supplementary Results: Controlling for BD Mood Symptoms

We note that for the following parallel analyses described below, of the 113 participants included in the Study 1 main analyses (after excluding 23 participants with zero variance ICCs), 24 additional participants were excluded who did not have usable symptom measures, which fell into one of three non-mutually exclusive categories: no symptoms collected in the same semester as the ESM study (n = 19), incomplete or inconsistent responding on symptom measures (n = 0), and failed any attention check items (n = 5). This resulted in a final subsample of 89 participants from Study 1 that were included in supplementary analyses controlling for symptom measures.

First, we examined whether the ED-General score was a predictor of trait BD risk when controlling for current symptoms (see **Table S1**). Specifically, results indicated that demographic variables in Block 1 were not predictors of BD risk scores using the HPS-20 (Model 1: $F_{(2, 86)}$ =

0.63, $p = .533$; $R^2 = .02$). When current PA and NA intensity were added in Block 2, the overall model was still not significant (Model 2: $F_{(4, 84)} = 0.46$, $p = .766$; $R^2 = .02$, $\Delta R^2 = .01$). When symptom measures (DSM5-Depression, DSM5-Mania, and ASRM) were added in Block 3, the overall model was significant (Model 3: $F_{(7, 81)} = 2.43$, $p = .026$; $R^2 = .17$, $\Delta R^2 = .15$); notably, mania symptoms as measured by the DSM5-Mania score were associated with increased BD risk, but the other mania symptom measure (ASRM) was not. When ED-General was added in Block 4, the overall model remained significant (Model 4: $F_{(8, 80)} = 2.11$, $p = .044$; $R^2 = .18$, $\Delta R^2 = .00$). However, as seen in **Table S1**, examination of individual beta weights from Model 4 indicated that ED-General was not associated with BD risk (HPS-20: $\beta = 0.03$, $p = .785$), which is not consistent with our hypotheses. Taken together, this suggests that the Study 1 main results do not change from those reported in the main text; that is, ED-General did not predict trait BD risk.

We next examined whether the ED-Positive and ED-Negative valence scores were predictors of trait BD risk when controlling for current symptoms. As seen in **Table S2**, results indicated that demographic variables in Block 1 were not predictors of BD risk scores using the HPS-20 (Model 1: $F_{(2, 86)} = 0.63$, $p = .533$; $R^2 = .02$). When current positive and negative affect intensity were added in Block 2, the overall model was not significant (Model 2: $F_{(4, 84)} = 0.46$, $p = .766$; $R^2 = .02$, $\Delta R^2 = .01$). When symptom measures (DSM5-Mania, ASRM, DSM5-Depression, and BDI-SF) were added in Block 3, the overall model became significant (Model 3: $F_{(7, 81)} = 2.43$, $p = .026$; $R^2 = .17$, $\Delta R^2 = .15$); notably, mania symptoms as measured by the DSM5-Mania score were associated with increased BD risk, but the other mania symptom measure (ASRM) was not. When ED-Positive and ED-Negative were added in Block 4, the overall model was no longer significant (Model 4: $F_{(9, 79)} = 1.93$, $p = .059$; $R^2 = .18$, $\Delta R^2 = .01$).

Examination of individual beta weights from Model 4 indicated that neither ED-Positive (HPS-20: β = -0.07, p = .597) or ED-Negative (HPS-20: β = 0.09, p = .455) were associated with BD risk, which is not consistent with our hypotheses. Taken together, this suggests that the Study 1 main results did not change from those reported in the main analyses when mood symptoms were controlled for; that is, ED-Positive and ED-Negative did not predict trait BD risk.

Study 2 Supplementary Results: Controlling for BD Mood Symptoms

We note that for these parallel analyses described below, of the 122 participants included in the Study 2 main analyses (after excluding 14 participants with zero variance ICCs), 8 additional participants were excluded who did not have usable symptom measures, which fell into one of three non-mutually exclusive categories: no symptoms collected in the same semester as the ESM study (n = 5), incomplete or inconsistent responding on symptom measures (n = 3), and failed any attention check items (n = 5). This resulted in a final subsample of 114 participants from Study 2 that were included in supplementary analyses controlling for symptom measures.

First, we examined whether the ED-General score was a predictor of trait BD risk when controlling for current symptoms (see **Table S1**). Specifically, results indicated that demographic variables in Block 1 were not predictors of BD risk scores using the HPS-20 (Model 1: $F_{(2, 112)}$ = 0.38, p = .68; R^2 = .01). When current PA and NA intensity were added in Block 2, the overall model became significant (Model 2: $F_{(4, 110)}$ = 4.38, p = .003; R^2 = .022, ΔR^2 = .13); notably, both PA and NA intensity were associated with increased BD risk scores. When symptom measures (DSM5-Mania, ASRM, DSM5-Depression, and BDI-SF) were added in Block 3, the overall model remained significant (Model 3: $F_{(8, 106)}$ = 3.71, p = .001; R^2 = .22, ΔR^2 = .08); notably, mania symptoms as measured by the DSM5-Mania score were associated with increased BD

risk, but the other mania symptom measure (ASRM) was not. Consistent with our hypotheses, when ED-General was added in Block 4, the overall model remained significant (Model 4: $F_{(89, 105)} = 4.03$, $p < .001$; $R^2 = .26$, $\Delta R^2 = .04$). Examination of individual beta weights indicated that ED-General was associated with decreased trait BD risk ($\beta = -0.20$, $p = .023$); in other words, lower emotion differentiation was associated with increased trait BD risk. Furthermore, we note that after controlling for symptoms in Block 3, ΔF from Model 3 to Model 4 was significant ($p = .023$), suggesting incremental validity to adding ED-General to the model to predict trait BD risk. This suggests that the Study 2 main results did not change when controlling for current mood symptoms.

We next examined whether the ED-Positive and ED-Negative valence scores were predictors of trait BD risk when controlling for current symptoms. As seen in **Table S2**, results indicated that demographic variables were not predictors of BD risk scores using the HPS-20 (Model 1: $F_{(2, 110)} = 0.37$, $p = .695$; $R^2 = .01$). When current PA and NA intensity were added in Block 2, the overall model was significant (Model 2: $F_{(4, 106)} = 4.43$, $p = .002$; $R^2 = .14$, $\Delta R^2 = .14$); notably, both PA and NA intensity were associated with increased BD risk scores. When symptom measures (DSM5-Mania, ASRM, DSM5-Depression, and BDI-SF) were added in Block 3, the overall model remained significant (Model 3: $F_{(8, 102)} = 3.77$, $p < .001$; $R^2 = .23$, $\Delta R^2 = .09$); notably, mania symptoms as measured by the DSM5-Mania score were associated with increased BD risk, but the other mania symptom measure (ASRM) was not. When ED-Positive and ED-Negative were added in Block 4, the overall model was also significant (Model 4: $F_{(10, 100)} = 3.66$, $p < .001$; $R^2 = .27$, $\Delta R^2 = .04$). Examination of individual beta weights from Model 4 indicated that neither ED-Positive (HPS-20: $\beta = -0.15$, $p = .109$) nor ED-Negative (HPS-20: $\beta =$ -

0.09, p = .387) alone predicted BD risk scores. Taken together, this suggests that the Study 2 main analyses did not change when controlling for current mood symptoms.

Supplementary Analyses Part 2: Exploring Interactions of ED-Positive and ED-Negative

Past research has been mixed on links between ED and psychopathology, depending on whether differentiation of negative emotions, positive emotions, or both, were measured. However, given established emotion-related disturbances of both positive and negative valence emotions in bipolar prone and diagnosed populations, we conducted exploratory analyses to test for potential unique interplay between differentiation of positive and negative emotions in the context of bipolar disorder risk. These exploratory analyses described below paralleled the approach of main analyses in Study 1 and Study 2, with the addition of the interaction term of ED-Positive x ED-Negative in the final step of regression analyses.

Study 1 Supplementary Results: ED-Positive x ED-Negative Predicting Trait Bipolar Risk

To examine the relationship between interactions of positive ED and negative ED on bipolar disorder risk, we used a hierarchical linear regression analysis. Specifically, we re-ran our main regression analyses with trait bipolar disorder risk (HPS-20) as the outcome, and the interaction term of ED-Positive and ED-Negative as the predictor. Block 1 included age and gender (Male=0, Female=1), Block 2 included PA and NA intensity, and Block 3 included ED-Positive and ED-Negative. The interaction term of ED-Positive x ED-Negative was in Block 4. No significant outliers were revealed with Cook's distance, multicollinearity diagnostics suggested satisfactory tolerance statistics, and missing cases were deleted listwise. This allowed us to examine the potential influence of interactions between positive and negative ED subscale scores on bipolar disorder risk while accounting for common confounds.

As seen in **Table S3**, the demographic variables in Block 1 were not predictors of bipolar disorder risk scores using the HPS-20 (Model 1: $F_{(2, 110)}$=0.87, p=.421; R^2=.02). When current PA and NA intensity were added in Block 2, the overall model was still not significant (Model 2: $F_{(4, 108)}$=1.06, p=.378; R^2=.04, ΔR^2=.02). Inconsistent with our hypotheses, the overall model for Block 3 was not significant (Model 3: $F_{(6, 106)}$=0.78, p=.591; R^2=.04, ΔR^2=.00). When the ED-Positive x ED-Negative interaction term was entered in Block 4, the overall model was also not significant (Model 4: $F_{(7, 105)}$=0.77, p=.611; R^2=.05, ΔR^2=.01). These results remained consistent when controlling for current mood symptoms. Taken together, this suggests that ED-Positive and ED-Negative did not predict trait bipolar disorder risk.

Study 2 Supplementary Results: ED-Positive x ED-Negative Predicting Trait Bipolar Risk

Study 2 utilized a similar regression analysis approach to Study 1. Two separate regressions were run where trait bipolar disorder risk (HPS-20) was the outcome, first for global ED (ED-General) and separately for the positive and negative ED indices (ED-Positive, ED-Negative). Block 1 included relevant confounds of age and gender (Male=0, Female=1), Block 2 included mean PA and NA intensity, and Block 3 included ED-Positive and ED-Negative. We included the additional interaction term of ED-Positive x ED-Negative in Block 4. No significant outliers were revealed with Cook's distance, multicollinearity diagnostics suggested satisfactory tolerance statistics, and missing cases were deleted listwise.

As seen in **Table S3**, the demographic variables in Block 1 were not associated with bipolar disorder risk scores using the HPS-20 (Model 1: $F_{(2, 118)}$=0.34, p=.716; R^2=.01). When current PA and NA intensity were added in Block 2, the overall model was significant (Model 2: $F_{(4, 116)}$=4.27, p=.003; R^2=.13, ΔR^2=.12); notably, both PA and NA intensity were associated with increased bipolar risk scores. When ED-Positive and ED-Negative were added to Block 3, the

overall model was significant (Model 3: $F_{(6,114)}$=3.68, p=.002; R^2=.16, ΔR^2=.03). However, examination of the individual beta weights indicated that neither ED-Positive (β=-0.02, p=.812) nor ED-Negative (β=-0.14, p=.161) alone predicted bipolar disorder risk scores and the ΔF from Model 2 to Model 3 was also not significant (p=.103). However, when the ED-Positive x ED-Negative interaction term was entered in Block 4, the overall model was significant (Model 4: $F_{(7, 113)}$=3.82, p=.001; R^2=.19, ΔR^2=.03) and examination of individual beta weights indicated that the interaction term significantly predicted increased bipolar disorder risk scores (β=0.19, p=.046). Furthermore, ΔF from Model 3 to Model 4 was significant (p=.046), further suggesting incremental validity to adding the ED-Positive x ED-Negative interaction term to the model to predict trait bipolar disorder risk. Simple slope analyses showed that ED-Negative was a significant negative predictor (β=-0.15, p<.02) of trait bipolar risk as measured by the HPS-20 only when ED-Positive was also low (-1 SD). ED-Negative did not predict bipolar disorder risk scores at mean or high (+1 SD) levels of ED-Positive (ps > .18). As seen in **Figure S1**, lower scores on ED-Positive and ED-Negative combined, but not separately, predicted increased bipolar disorder risk scores (HPS-20). We note that this pattern of findings remained consistent when controlling for current mood symptoms.

Contrary to Study 1, but in partial support of our hypotheses, Study 2 results suggested that ED, as measured by the co-occurrence of low ED-Positive and low ED-Negative, was associated with increased trait bipolar risk (HPS-20). See **Figure S2** for a summary and comparison of Study 1 and Study 2 results.

Supplementary Analyses Part 3: Associations with Emotion Dynamic Constructs

Additional supplementary analyses examined whether relevant emotion dynamic constructs were associated with bipolar risk using the same experience-sampling assessment approach. Specifically, we examined whether relevant emotion dynamic constructs—including emotion variability, emotion covariation, emodiversity, emotion inertia, and emotion regularity—were associated with increased bipolar risk. We also examined whether emotion differentiation was a unique predictor of bipolar risk, above and beyond any secondary emotion dynamic constructs that were also associated with bipolar risk. This enabled us to examine whether emotion differentiation was uniquely associated with bipolar risk versus other relevant emotion dynamic constructs that were also associated with bipolar risk across an approximately two-week sampling period. Although we had no specific a priori predictions regarding the relationship between BD risk and these secondary constructs, we anticipated that in cases where emotion differentiation predicted BD risk it would do so after accounting for any of the secondary emotion dynamic measures that were also associated with BD risk. In other words, we predicted that emotion differentiation should have incremental predictive value for BD risk when compared to other related dynamic emotion constructs.

Secondary measures consisted of the following five dynamic emotion constructs: (1) emotion variability, (2) emotion covariation, (3) emodiversity, (4) emotion inertia, and (5) emotion regularity. These comparison measures were selected to represent a diverse range of complex emotional measures while eliminating redundant emotion dynamic constructs that have been previously examined in populations at risk for or diagnosed with mood disorders. Individual items are described below and descriptives can be found in **Table S4**.

Emotion variability (or lability) is defined as the intra-individual variability of positive emotions and negative emotions, which was operationalized as the standard deviation of position

and negative emotions across the experience-sampling period, computed separately for *positive emotion variability* and *negative emotion variability* (e.g., Grühn et al., 2013). Greater emotion variability has been associated with bipolar disorder both during mood episodes and periods of euthymia (e.g., Henry et al., 2008). Emotion variability has also been correlated with elevated bipolar symptoms and bipolar risk (e.g., Angst et al., 2003; Sperry & Kwapil, 2019; 2020), as well as poorer clinical prognosis, including earlier age of onset of BD and significantly higher risk for comorbidities (Henry et al., 2008).

Emotion covariation (or dialecticism) was defined as the simultaneous occurrence of pleasant and unpleasant emotion states, which was operationalized as the Pearson correlation between positive and negative emotions across the experience-sampling period (e.g., Grühn et al., 2013). A correlation near zero indicates that positive emotions and negative emotions are experienced independently.

Emodiversity indicates the richness (number of unique emotional states) and evenness (extent to which specific emotions are experienced in the same proportion) of a person's emotional experiences (Quoidbach et al., 2014, 2018). Both greater positive and negative emodiversity have been demonstrated to be beneficial for mental and physical health over and above mean levels of positive and negative emotion in initial studies. However, some recent research has suggested that only greater positive emodiversity is associated with better mental and physical health outcomes; more diverse negative emotions (i.e., negative emodiversity), on the other hand, was either associated with more depressive and anxiety symptoms and more health symptoms (Urban-Wojcik et al., 2020), or was unrelated to indicators of physical health, such as biomarkers of inflammation (Ong et al., 2018). In the present study, as in past research, operationalization of emodiversity was based on an adaptation of the Shannon biodiversity

index, a measure of the number and evenness of species in a biological ecosystem (Magurran, 2004; Shannon, 1948). Emodiversity was computed once per ESM survey, and then averaged across the experience-sampling period for all types of emotions (i.e., *global emodiversity*), and, where appropriate, it can also be examined in two subscales separately for positively-valenced emotions (i.e., *positive emodiversity*) and negatively-valenced emotions (i.e., *negative emodiversity*). Across all emodiversity indices, higher values represented more diverse emotional experiences.

Emotion inertia reflects the degree to which emotions are resistant to change, and may indicate that a person's emotions are non-responsive to external cues (i.e., environmental context) or internal cues (i.e., emotion regulation efforts; Kuppens et al., 2010). Emotion inertia was computed as the autocorrelation among consecutive time points across the experience-sampling period, separately for positively-valenced emotions (i.e., *positive emotion inertia*) and negatively-valenced emotions (i.e., *negative emotion inertia*).

Lastly, *emotion regularity* was defined as the degree to which a person maintains and returns to a set of stable emotional states over time (D'Mello & Gruber, 2021). For example, using a 7-pt Likert scale to measure positive and negative emotions, a person who rates positive emotions as '2' and negative emotions as '5' on three separate occasions across a sampling period of 25 timepoints, would have 3 recurrent points in which they returned to this "stable" emotional state. Emotional regularity was computed using recurrence quantification analysis (RQA), which measures recurrent patterns (i.e., repeated emotion ratings) over time, by comparing time series data (in this study, experience-sampling data), to itself, at various time lags (Webber & Zbilut, 2005).

Study 1 Supplementary Results: Associations Between BD Risk and Symptoms with Extant Emotion Dynamic Constructs

We first conducted bivariate correlations among our primary variables with the secondary emotion dynamics measures (i.e., positive emotion variability, negative emotion variability, emotion covariation, global emodiversity-- and where appropriate separately for positive and negative emodiversity--, positive emotion inertia, negative emotion inertia, and emotion regularity). As seen in **Table S5,** consistent with previous literature (e.g., Gruber et al., 2013b; Sperry & Kwapil, 2020), BD risk was associated with increased positive and negative emotion variability. BD risk was not associated with any of the other emotion dynamic measures. Current mania symptom severity was associated with increased positive emodiversity and current depression symptom difficulties were associated with increased positive and negative variability, increased global and negative emodiversity, decreased positive emodiversity, and decreased emotion regularity. Notably, all three measures of emotion differentiation (ED-Global, ED-Positive, ED-Negative) were associated with decreased positive and negative emotion variability and with increased emotion covariation. ED-Positive and ED-Global were also associated with decreased positive and global emodiversity. In addition, ED-Positive was associated with decreased positive and negative emotion inertia; ED-Negative was associated with increased emotion regularity, and decreased negative and global emodiversity; and ED-Global was associated with decreased negative emotion inertia. No other correlations were significant.

Study 1 Supplementary Results: BD Risk with Extant Emotion Dynamic Constructs

A secondary aim of the current investigation explored whether BD risk was associated with our extant emotion dynamic constructs (i.e., positive emotion variability, negative emotion variability, emotion covariation, global emodiversity--and where appropriate separately for

positive and negative emodiversity subscales--, positive emotional inertia, negative emotion inertia, and emotion regularity). Examination of skewness and kurtosis indices revealed that emotion regularity was positively skewed and leptokurtic (skewness statistic = 4.70, kurtosis statistic = 26.06) and positive emodiversity was negatively skewed and leptokurtic (skewness statistic = -3.05, kurtosis statistic = 13.32). We attempted to normalize them using a log transformation [LG10+1] for use in final analyses. We display non-transformed values in **Table S4** to facilitate interpretation.

We followed a similar analytic approach used in main analyses to explore whether our secondary emotion constructs were associated with trait BD risk. Specifically, Block 1 included relevant confounds of age and gender (Male = 0, Female = 1), Block 2 included mean PA and NA intensity, and Block 3 included our secondary emotion dynamic constructs as concurrent predictors of trait BD risk. No significant outliers were revealed with Cook's distance, multicollinearity diagnostics suggested satisfactory tolerance statistics, and missing cases were deleted listwise. Similar to the main results for Study 1, results indicated that demographic variables in Block 1 were not predictors of HPS-20 scores (Model 1: $F_{(2, 113)} = 0.87$, $p = .423$; $R^2 = .01$). When current PA and NA intensity were added in Block 2, the overall model was also not significant (Model 2: $F_{(4, 131)} = 0.91$, $p = .462$; $R^2 = .03$, $\Delta R^2 = .01$). The model for Block 3, which included our secondary emotion dynamic constructs, was not significant (Model 3: $F_{(11, 122)} = 1.11$, $p = .358$; $R^2 = .09$, $\Delta R^2 = .06$). As seen in **Table S6**, individual beta weights indicated that none of the secondary emotion constructs significantly predicted BD risk. Taken together, this suggests that none of our secondary emotion constructs were associated with trait BD risk. Therefore, no subsequent regression analyses were performed.

Study 2 Supplementary Results: Associations Between BD Risk and Symptoms with Extant Emotion Dynamic Constructs

We included the same secondary measures as in Study 1 in order to examine whether and how emotion differentiation relates to BD risk, in comparison to other relevant emotion dynamics constructs (i.e., positive emotion variability, negative emotion variability, emotion covariation, global emodiversity -- and where appropriate separately for positive and negative emodiversity-- positive emotional inertia, negative emotion inertia, and emotion regularity).

We first examined bivariate correlations among our primary variables with the secondary emotion dynamics measures (i.e., positive emotion variability, negative emotion variability, emotion covariation, global emodiversity, positive emodiversity, negative emodiversity, positive emotional inertia, negative emotion inertia, and emotion regularity). Similar patterns of correlations emerged as in Study 1. Specifically, as seen in **Table S7**, results indicated that BD risk was associated with increased positive and negative emotion variability, as in Study 1, as well as increased positive, negative, and global emodiversity—no other secondary dynamic emotion construct was correlated with trait bipolar risk as measured by the HPS-20. Difficulty with manic symptoms was significantly correlated with increased positive and negative emotion variability and increased positive and global emodiversity. Depressive symptom severity and difficulties correlated with increased negative emotion variability, decreased emotion covariation, increased negative and global emodiversity, and decreased emotion regularity. Similar to Study 1, ED-Positive was associated with decreased positive and negative emotion variability, increased emotion covariation, and decreased positive emotional inertia. ED-Negative was also associated with decreased positive and negative emotion variability, increased emotion covariation, decreased negative and global emodiversity, and decreased negative

emotion inertia. As in Study 1, ED-General was associated with decreased positive and negative emotion variability, increased emotion covariation, and decreased negative and global emodiversity; unlike in Study 1, ED-General was associated with decreased positive emotional inertia, but unrelated to negative emotional inertia. PA intensity was associated with decreased emotion covariation, increased positive and global emodiversity, and decreased negative emodiversity, and increased emotion regularity. NA intensity was associated with increased negative emotion variability, increased negative and global emodiversity, and decreased emotion regularity. No other correlations were significant.

Study 2 Supplementary Results: Associations Between BD Risk and Extant Emotion Dynamic Constructs

As in Study 1, a secondary aim of Study 2 explored whether BD risk was associated with our extant emotion dynamic constructs (i.e., positive emotion variability, negative emotion variability, emotion covariation, global emodiversity--and where appropriate separate examination of positive and negative emodiversity subscales, positive emotional inertia, negative emotion inertia, and emotion regularity). Examination of skewness and kurtosis indices revealed that emotion regularity was positively skewed and leptokurtic (skewness statistic = 5.48, kurtosis statistic = 33.02) and so attempts to transform this variable were used with a (LG(10)+1) transformation in our final analyses. We display non-transformed values in **Table S4** to facilitate interpretation.

We followed a similar analytic approach used in the main study analyses to explore whether BD risk was associated with our secondary emotion constructs. Specifically, Block 1 included relevant confounds of age and gender (Male = 0, Female = 1), Block 2 included mean PA and NA intensity, and Block 3 included our secondary emotion dynamic constructs as

concurrent predictors of trait BD risk. No significant outliers were revealed with Cook's distance, multicollinearity diagnostics suggested satisfactory tolerance statistics, and missing cases were deleted listwise. Similar to the main results for Study 2, results indicated that demographic variables in Block 1 were not predictors of HPS-20 scores (Model 1: $F_{(2, 132)} = 0.21$, $p = .808$; $R^2 = .00$). When current PA and NA intensity were added in Block 2, the overall model became significant (Model 2: $F_{(4, 130)} = 5.53$, $p < .001$; $R^2 = .15$, $\Delta R^2 = .14$). The model for Block 3, which included our secondary emotion dynamic constructs, remained significant (Model 3: $F_{(11, 121)} = 3.17$, $p = .001$; $R^2 = .22$, $\Delta R^2 = .08$). However, as seen in **Table S8**, individual beta weights indicated that only positive emotion variability significantly predicted BD risk ($\beta = 0.23$, $p = .039$). Of note, the significant ΔF for Model 3 was also not significant ($p = .114$).

Study 2 Supplementary Results: Controlling for Emotion Dynamic Constructs

In order to ascertain whether BD risk was associated with emotion differentiation in Study 2 above and beyond other extant emotion dynamic constructs that were also associated with BD risk (i.e., positive emotion variability), we re-ran our regression analyses including positive emotion variability in Block 3, following PA and NA in Block 2, prior to our measures of emotion differentiation. Specifically, ED-General or ED-Positive and ED-Negative were included in Block 3, respectively.

With respect to ED-General, when we re-ran main analyses for Study 2, the overall models for Block 3 (Model 3: $F_{(5, 115)} = 5.95$, $p < .001$; $R^2 = .21$, $\Delta R^2 = .08$) and Block 4 (Model 4: $F_{(6, 114)} = 4.92$, $p < .001$; $R^2 = .21$, $\Delta R^2 = .00$) were significant. However, as seen in **Table S8**, examination of individual beta weights indicated that ED-General ($\beta = -.02$, $p = .875$) no longer predicted trait BD risk after accounting for positive variability ($\beta = .28$, $p = .014$). Similarly, when we examined whether the ED-Positive and ED-Negative valence scores (Block 4) were

predictors of trait BD risk after controlling for positive variability, *p*-value of the Δ*F* from Block 3 to Block 4 was not significant ($p = .343$). Taken together, these results suggest that emotion differentiation no longer predicted trait BD risk after accounting for positive emotion variability.

These results are consistent with a growing body of work suggesting that increased positive emotion variability is a predictor of clinically-relevant outcomes in at-risk bipolar samples (e.g., Angst et al., 2003; Henry et al., 2008; Sperry & Kwapil, 2019, 2020) as well as negative clinical outcomes more generally (Gruber et al., 2013b). One possibility is that emotion differentiation is a significant predictor of BD risk, but perhaps emotion differentiation and variability are capturing conceptually overlapping constructs. This interpretation could be due to similarities in how emotion variability and emotion differentiation are commonly measured (e.g., averaging multiple emotion ratings across experience-sampling timepoints). Disconnections between the conceptualization and measurement of emotion differentiation may have contributed to the non-significant incremental predictive validity of emotion differentiation in the current study (Thompson et al., 2021). Emotion differentiation research tends to define people low in emotion differentiation as identifying emotions in global positive and negative terms, such as "good" or "bad", pleasant or unpleasant, as opposed to people high in emotion differentiation who use more precise terms like angry, frustrated, calm. In practice, however, emotion differentiation is commonly measured using intraclass correlations (ICCs) that effectively average across multiple emotion ratings across timepoints, and do not require participants to generate and label their own emotion experiences, which is a central feature of the conceptualization of emotion differentiation. Thompson and colleagues (2021) also highlighted that this disconnect between conceptualization and measurement of emotion differentiation muddles researchers' ability to parse apart multiple emotion dynamics constructs. However,

other studies have found that emotion differentiation is significantly associated with clinically-relevant mood symptoms even after controlling for emotion intensity and emotion variability (e.g., Boden et al., 2013; Demiralp et al., 2012). Future studies that can address these issues to better align the conceptualization and measurement of emotion differentiation (e.g., inclusion of vague terms to describe emotions as “bad” or “unpleasant”, open-ended response formats) will be critical to advancing the existing research to elucidate the role of emotion differentiation in the context of bipolar disorder. Given consistent associations between positive affect, positive variability, and positive emotion differentiation, it will be important to include emotion variability as a confounding variable in future studies on emotion differentiation in the context of bipolar disorder, in line with current conventions that control for mean emotional intensity.

Table S1

Regression Analyses using ED-General Scores to Predict BD Risk (HPS-20 Scores) Controlling for BD-Relevant Symptoms Across Study 1 and Study 2

	HPS-20 Study 1 (n = 89)			**HPS-20** Study 2 (n = 110)		
Predictor	R^2	ΔR^2	β	R^2	ΔR^2	β
Block 1	.02	.02		.01	.01	
Age			.14			.06
Gender			.12			-.12
Block 2	.02	.01		**.14****	**.13****	
PA			.03			**.23***
NA			.09			.05
Block 3	**.17***	**.15****		**.22****	**.08***	
DSM5-Depression			-.01			.06
DSM5-Mania			**.37****			**.21***
ASRM			.09			.12
BDI-SF			--			.21
Block 4	**.18****	.00		**.26****	**.04***	
ED-General			.03			**-.20***

Note: Regression analyses were conducted using a subset of eligible participants with usable ED scores and completed symptom measures (i.e., additional participants were excluded who did not have usable emotion differentiation indices due to having ICCs with zero variance (Study 1: n = 23; Study 2: n = 14) and who did not have usable symptom measures completed within the same semester of the ESM data collection period (Study 1: n = 24; Study 2: n = 8). BD Risk was assessed with HPS-20 = 20-item Hypomanic Personality Scale; Gender (Male = 0, Female = 1); PA = Positive Affect; NA = Negative Affect; ED = Emotion Differentiation; ED-General = General Emotion Differentiation (i.e., ED-Positive + ED-Negative); β = Standardized beta coefficients (beta values are from Model 4); *p < .05, **p < .01.

Table S2

Regression Analyses using ED-Positive and ED-Negative to Predict BD Risk (HPS-20 Scores) Controlling for BD-Relevant Symptoms Across Study 1 and Study 2

	HPS-20 Study 1 (n = 89)			**HPS-20** Study 2 (n = 110)		
Predictor	R^2	ΔR^2	β	R^2	ΔR^2	β
Block 1	.02	.02		.01	.01	
Age			.11			.05
Gender			.10			-.12
Block 2	.02	.01		**.14****	**.14****	
PA			.04			**.24***
NA			.12			.06
Block 3	**.17***	**.15****		**.23****	**.09***	
DSM5-Mania			**.35****			**.23***
ASRM			.11			.10
DSM5-Depression			-.03			.06
BDI-SF			--			.22
Block 4	.18	.01		**.27****	.04	
ED-Positive			-.07			-.15
ED-Negative			.09			-.09

Note: Regression analyses were conducted using a subset of eligible participants with usable ED scores and completed symptom measures (i.e., additional participants were excluded who did not have usable emotion differentiation indices due to having ICCs with zero variance (Study 1: n =

23; Study 2: n = 14) and who did not have usable symptom measures completed within the same semester of the ESM data collection period (Study 1: n = 24; Study 2: n = 8). BD Risk was assessed with HPS-20 = 20-item Hypomanic Personality Scale; Gender (Male = 0, Female = 1); PA = Positive Affect; NA = Negative Affect; ED = Emotion Differentiation; ED-Positive=Positive Emotion Differentiation; ED-Negative=Negative Emotion Differentiation; β = Standardized beta coefficients (beta values are from Model 4); *p < .05, **p < .01.

Table S3

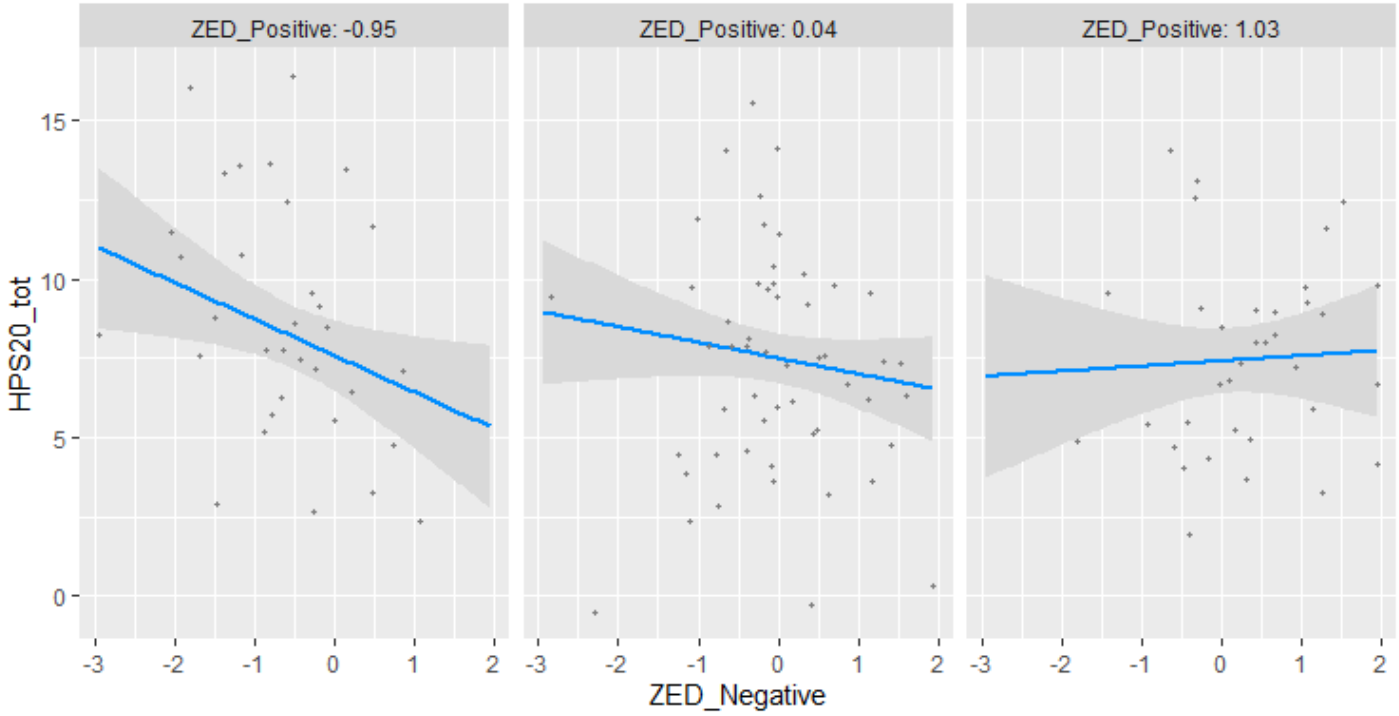
Regression Analyses Using ED-Positive x ED-Negative Interaction to Predict HPS-20 Scores

	HPS-20 Study 1 (*n*=113)			**HPS-20** Study 2 (*n*=121)		
Predictor	R^2	ΔR^2	β	R^2	ΔR^2	β
Block 1	.02	.02		.01	.01	
Age			.06			-.00
Gender			.10			-.03
Block 2	.04	.02		**.13****	**.12****	
PA			.08			**.24****
NA			.17			**.23***
Block 3	.04	.00		**.16****	.03	
ED-Positive			-.05			-.02
ED-Negative			.05			-.14
Block 4	.05	.01		**.19****	**.03***	
ED-Positive x ED-Negative			.09			**.19***

Note: Regression analyses were conducted using a subset of eligible participants who had usable ED scores (see Footnote 5). HPS-20=20-item Hypomanic Personality Scale; Gender (Male=0, Female=1); PA=Positive Affect Mean Intensity; NA=Negative Affect Mean Intensity; ED=Emotion Differentiation; ED-Positive=Positive Emotion Differentiation; ED-Negative=Negative Emotion Differentiation; ED-Positive x ED-Negative=Interaction of Positive Emotion Differentiation and Negative Emotion Differentiation; β =Standardized beta coefficients from Model 4; *p<.05, **p<.01

Figure S1

Interaction of ED-Positive and ED-Negative to Predict Trait Bipolar Disorder Risk (HPS-20 Scores) in Study 2



Note: Trait bipolar disorder risk (HPS-20 scores) as a function of negative emotion differentiation (ED-Negative), moderated by positive emotion differentiation (ED-Positive). As in main analyses, we controlled for demographics (age, gender) and emotion intensity (NA, PA). Shaded regions delineate the 95% confidence bands for the simple slopes at -1 standard deviation (left), mean (center), and +1 standard deviation (right) levels of positive emotion differentiation.

Figure S2

Summary of results indicating whether emotion differentiation was associated with bipolar disorder risk dimensions across Study 1 and Study 2

Bipolar Disorder Risk Dimension	**Study 1** (pre-COVID)	**Study 2** (during COVID)
Trait - Lifetime Risk (e.g., HPS-20)	No	Yes ED-General
State - Mania Symptoms (e.g., ASRM, DSM5-Mania)	Yes ED-Positive	No
State - Depression Symptoms (e.g., BDI-SF, DSM5-Depression)	No	No

Table S4

Descriptives for Secondary Experience-Sampling Variables Across Study 1 and Study 2

	Study 1 (*N* = 136)	Study 1 Range	Study 2 (*N* = 136)	Study 2 Range	Statistic
Secondary Variables					
Positive Variability	1.01 (0.30)	0.35-1.86	0.85 (0.33)	0.11-2.03	*F* = 17.50**
Negative Variability	0.64 (0.31)	0.09-1.75	0.56 (0.29)	0.07-1.73	*F* = 4.54*
Emotion Covariation	-0.48 (0.25)	-0.85-0.52	-0.40 (0.32)	-0.89-0.88	*F* = 5.06*
Emodiversity-Global	1.49 (0.25)	1.04-2.12	1.75 (0.34)	0.34-2.37	*F* = 49.19**
Emodiversity-Positive	1.00 (0.13)	0.17-1.10	1.35 (0.31)	0.02-1.61	*F* = 146.67**
Emodiversity-Negative	0.67 (0.47)	0.00-1.73	0.71 (0.50)	0.00-1.88	*F* = 0.47
Emotion Inertia-Positive	0.17 (0.19)	-0.30-0.67	0.14 (0.20)	-0.42-0.76	*F* = 2.04
Emotion Inertia-Negative	0.20 (0.19)	-0.16-0.71	0.15 (0.18)	-0.45-0.67	*F* = 5.77*
Emotion Regularity	3.51 (4.89)	0.34-38.44	4.81 (11.61)	0.00-86.68	*F* = 1.46

Note: Experience-sampling measure descriptives are reported for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, and completed at least 50% of ESM survey prompts. Values display average values with standard deviations in parentheses. *$p < .05$ comparing Study 1 and Study 2; **$p < .01$ comparing Study 1 and Study 2

Table S5

Bivariate Correlations Between Primary and Secondary Study Measures in Study 1

	HPS-20	DSM5-Mania	ASRM	DSM5-Depression	ED-General	ED-Positive	ED-Negative	PA	NA
Positive Variability	**0.21***	0.09	-0.12	**0.30****	**-0.57****	**-0.63****	**-0.32****	**-0.17***	**0.24****
Negative Variability	**0.17***	-0.04	0.03	**0.26****	**-0.62****	**-0.20***	**-0.77****	**-0.19***	**0.71****
Emotion Covariation	-0.11	0.08	0.01	-0.06	**0.35****	**0.36****	**0.22***	-0.15	0.01
Emodiversity-Global	0.06	0.06	0.03	**-0.30****	**-0.28****	-0.01	**-0.41****	**-0.20***	**0.87****
Emodiversity-Positive	-0.06	-0.06	**0.25****	**-0.37****	0.12	0.05	0.13	**0.70****	**-0.27****
Emodiversity-Negative	0.07	0.08	-0.01	**-0.33****	**-0.30****	-0.03	**-0.43****	**-0.33****	**0.90****
Emotion Inertia-Positive	0.10	0.01	0.11	-0.67	-0.16	**-0.20***	-0.06	-0.09	-0.01
Emotion Inertia-Negative	0.15	0.06	0.04	0.05	**-0.20***	**-0.17***	-0.16	**-0.17***	0.13
Emotion Regularity	-0.05	-0.01	0.07	**-0.25****	0.14	0.03	**0.19***	**0.47****	**-0.37****

Note: Bivariate correlations are reported for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, and completed at least 50% of ESM survey prompts. HPS-20 = 20-item Hypomanic Personality Scale; ASRM = Altman Self Rating Mania; BDI-SF = Beck Depression Inventory – Short Form; ED = Emotion Differentiation; ED-General = General Emotion Differentiation (i.e., ED-Positive + ED-Negative); ED-Positive = Positive Emotion Differentiation; ED-Negative = Negative Emotion Differentiation; PA = Positive Affect; NA = Negative Affect. **p* <.05; ***p*<.01.

Table S6

Regression Analyses using Secondary Emotion Variables to Predict BD Risk (HPS-20 Scores)

	HPS-20 Study 1 (n = 133)			**HPS-20** Study 2 (n = 135)		
Predictor	R^2	ΔR^2	β	R^2	ΔR^2	β
Block 1	.01	.01		.00	.00	
Age			.06			-.01
Gender			.05			-.05
Block 2	.03	.01		**.15****	**.14****	
PA			.18			.21
NA			.13			.10
Block 3	.10	.06		**.22****	.08	
Positive Variability			.13			**.23***
Negative Variability			.02			.06
Emotion Covariation			.01			.04
Emodiversity-Global			-.14			.17
Emotion Inertia-Positive			.04			-.00
Emotion Inertia-Negative			.06			.07
Emotion Regularity			-.19			-.00

Note: Regression analyses were conducted for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, and completed at least 50% of ESM survey prompts. Prior to analyses, emotion regularity was transformed using a (LG(10)+1) transformation. BD Risk was assessed with HPS-20 = 20-item Hypomanic Personality Scale; Gender (Male = 0, Female = 1); PA = Positive Affect Mean Intensity; NA = Negative Affect Mean Intensity; β = Standardized beta coefficients (beta values are from Model 3); *$p < .05$, **$p < .01$.

Table S7

Bivariate Correlations Between Primary and Secondary Study Measures in Study 2

	HPS-20	DSM5-Mania	ASRM	DSM5-Depression	BDI-SF	ED-General	ED-Positive	ED-Negative	PA	NA
Positive Variability	**0.28****	**0.22****	0.06	0.05	0.03	**-0.62****	**-0.70****	**-0.32****	0.08	0.08
Negative Variability	**0.30****	**0.27****	0.12	**0.28****	**0.38****	**-0.72****	**-0.36****	**-0.79****	-0.02	**0.59****
Emotion Covariation	-0.10	0.10	0.07	**-0.21***	-0.13	**0.28****	**0.30****	**0.18***	**-0.24****	-0.021
Emodiversity-Global	**0.32****	**0.24****	0.04	**0.19***	**0.32****	-0.06	-0.06	**-0.33****	**0.35****	**0.63****
Emodiversity-Positive	**0.25****	0.14	0.15	-0.12	-0.09	-0.06	-0.06	-0.12	**0.75****	0.06
Emodiversity-Negative	**0.21***	**0.24****	-0.09	**0.39****	**0.53****	-0.06	-0.06	**-0.36****	**-0.19***	**0.86****
Emotion Inertia-Positive	0.08	0.00	-0.03	-0.02	-0.02	**-0.25****	**-0.25****	-0.03	-0.01	0.01
Emotion Inertia-Negative	0.10	-0.02	-0.07	0.09	0.16	-0.04	-0.44	-0.18	0.01	0.08
Emotion Regularity	0.01	-0.17	0.15	**-0.26****	**-0.25****	-0.01	-0.01	0.06	**0.41****	**-0.28****

Note: Bivariate correlations are reported for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, and completed at least 50% of ESM survey prompts. HPS-20 = 20-item Hypomanic Personality Scale; ASRM = Altman Self Rating Mania; BDI-SF = Beck Depression Inventory – Short Form; ED = Emotion Differentiation; ED-General = General Emotion Differentiation (i.e., ED-Positive + ED-Negative); ED-Positive = Positive Emotion Differentiation; ED-Negative = Negative Emotion Differentiation; PA = Positive Affect; NA = Negative Affect. *p <.05; **p<.01.

Table S8

Regression Analyses using ED Scores to Predict BD Risk (HPS-20 Scores) Controlling for Positive Emotion Variability in Study 2

	HPS-20 Study 2 (n = 121)				**HPS-20** Study 2 (n = 121)		
Predictor	R^2	ΔR^2	β	Predictor	R^2	ΔR^2	β
Block 1	.01	.01		**Block 1**	.01	.01	
Age			-0.02	Age			-0.03
Gender			-0.07	Gender			-0.08
Block 2	.13	**.12****		**Block 2**	**.13****	**.12****	
PA			**.23****	PA			**0.23***
NA			**.25****	NA			**0.22***
Block 3	.21	**.08****		**Block 3**	**.21****	**.08****	
Positive Variability			**.27***	Positive Variability			**0.35****
Block 4	.21	.00		**Block 4**	**.22****	.02	
ED-General			-.02	ED-Positive			.14
				ED-Negative			-0.11

Note: Regression analyses were conducted for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, and completed at least 50% of ESM survey prompts. BD Risk was assessed with HPS-20 = 20-item Hypomanic Personality Scale; Gender (Male = 0,

Female = 1); PA = Positive Affect Mean Intensity; NA = Negative Affect Mean Intensity; ED = Emotion Differentiation; ED-Positive = Positive Emotion Differentiation; ED-Negative = Negative Emotion Differentiation; β = Standardized beta coefficients (beta values are from Model 4); *$p < .05$, **$p < .01$.

Table S9

Descriptives for Primary Experience-Sampling Variables for Participants with Usable ED Scores Across Study 1 and Study 2

	Study 1 (*n*=113)	Study 1 Range	Study 2 (*n*=122)	Study 2 Range	Statistic
Primary Variables					
ED-General	0.11 (0.55)	-1.09 - 1.82	0.10 (0.63)	-1.74 - 1.47	*F*=0.02
ED-Positive	-0.04 (0.31)	-0.73 - 0.82	-0.07 (0.37)	-1.60 - 0.74	*F*=0.52
ED-Negative	0.15 (0.36)	-0.57 - 1.00	0.17 (0.40)	-0.99 - 1.00	*F*=0.19
Mean PA	3.33 (1.02)	0.35 - 5.72	2.79 (1.30)	0.02 - 5.92	*F*=12.68**
Mean NA	1.18 (0.78)	0.06 - 3.65	1.01 (0.74)	0.05 - 4.49	*F*=2.90

Note: Experience-sampling measure descriptives are reported for eligible participants who met the initial study inclusion criteria, had HPS-20 scores, completed at least 50% of ESM survey prompts, and had usable ED scores (i.e., additional participants were excluded who did not have reliable emotion differentiation indices due to having ICCs with zero variance; Study 1: *n*=23; Study 2: *n*=14; see Footnote 5). Values display average values with standard deviations in parentheses. Range is the minimum and maximum value in Study 1 and Study 2. ED=Emotion Differentiation; ED-General=General Emotion Differentiation (i.e., ED-Positive + ED-Negative); ED-Positive=Positive Emotion Differentiation; ED-Negative=Negative Emotion

Differentiation; PA=Positive Affect; NA=Negative Affect. *$p < .05$ comparing Study 1 and Study 2; **$p < .01$ comparing Study 1 and Study 2.

Table S10

List of Additional Survey Measures Administered in the Broader Study Protocol for Study 1

Measure	Scale Citation (if applicable)
Participant Characteristics	
Study ID number	n/a
Demographic questions	n/a
Social media use questionnaire	n/a
Health Information Questionnaire	n/a
Counseling and Treatment Questionnaire	Sachs et al. (2003)
Current medication use (past month)	n/a
Affective Decision-Making and Behavior	
CARE	Fromme, Kratz, & Rivet (1997)
Sexual Risk Behavior Index	Hendershot, Magnan & Bryan (2010)
SUPPS-P	Cyders, Littlefield, Coffey, & Karyadi (2014)
Monetary Choice Questionnaire	Kirby, Petry, & Bickel (1999)
Behavioral Activation System-Reward Responsiveness	Carver & White (1994)
Emotion and Decision-Making Beliefs	Gatchpazian & Ford (in prep)
Emotion and Well-Being	
Modified Differential Emotions Scale (mDES)	Cohn et al. (2009)
Subjective Happiness Scale (SHS)	Lyubomirsky & Kepper (1999)
Satisfaction with Life Scale (SWLS)	Diener, Emmons, Larsen, & Griffin (1985)
Extreme Valuing of Happiness Scale (VHQ)	Mauss et al. (2011)
Fear of Happiness (FOH)	Joshanloo (2013)
Emotion and Decision-Making Beliefs	Gatchpazian & Ford (in prep)
Emotion Control Beliefs (Items 5-16)	Mauss, Butler, Roberts, & Chu (2010)
Perth Alexithymia Scale (PAQ)	Preece et al. (2018)
Domain-Specific Impulsivity in Children	Tsukayama, Duckworth, & Kim (2013)
Brief Resilience Scale	Smith et al. (2008)
Positive Emotion Persistence (PEP)	Gruber et al. (in prep)

Psychological Adjustment	
DSM-5 Cross Cutting Measure	American Psychiatric Association (2013)
Patient Safety Screener-3	Boudreaux et al. (2015)
Schedule for Nonadaptive and Adaptive	Clark et al. (1993)
Personality-Self-Harm	Algorta et al. (2013)
PROMIS	Yu et al. (2012)
Alcohol Frequency Questionnaire (AQF)	Cocco & Carey (1998)
RAPI Alcohol Problems Questionnaire	White & Labouvie (1989)
Perceived Stress Scale (PSS)	Cohen et al. (1983)
Drug Abuse Screening Test (DAST-10)	Cocco & Carey (1998); Skinner (1982)
Cannabis Use Problems Identification Test	Bashford, Flett, & Copeland (2010)
Healthy Living Questionnaire 2011	Ware et al. (2001)
Prodromal Questionnaire for Psychosis	Loewy et al. (2011)
TriPM	Patrick et al. (2009)
GAD-7 (Generalized Anxiety Disorder)	Spitzer et al., (2006)
Social Functioning and Connection	
Perceived Social Support and Conflict-friends	Schuster, Kessler, & Aseltine (1990); Whalen & Lachman (2000)
Social Identity Scale at CU Boulder	Leach et al. (2008)
Belonging Uncertainty Scale	Walton & Cohen (2007)
Social Network-Quantity	Parkinson, Kleinbaum, & Wheatley (2018)
Social Network-Quality	Morelli et al. (2017)
Academic Adjustment	
National Survey of Student Engagement	Kuh et al. (2001)
Academic Self-Efficacy	Gaumer Erickson et al. (2016)
Miscellaneous	
Brief Social Desirability Scale	Haghighat (2007)
Catch Items	n/a

Note: n/a = Not officially published scale.

Table S11

List of Additional Survey Measures Administered in the Broader Study Protocol for Study 2

Measure	Scale Citation (if applicable)
Participant Characteristics	
Study ID number	n/a
Demographic questions	n/a
Social media use questionnaire	n/a
Health Information Questionnaire	n/a
Counseling and Treatment Questionnaire	Sachs et al. (2003)
Current medication use (past month)	n/a
Affective Decision-Making and Behavior	
CARE	Fromme, Kratz, & Rivet (1997)
Sexual Risk Behavior Index	Hendershot, Magnan & Bryan (2010)
SUPPS-P	Cyders, Littlefield, Coffey, & Karyadi (2014)
Monetary Choice Questionnaire	Kirby, Petry, & Bickel (1999)
Behavioral Activation System-Reward Responsiveness	Carver & White (1994)
Emotion and Decision-Making Beliefs	Gatchpazian & Ford (in prep)
Emotion and Well-Being	
Modified Differential Emotions Scale (mDES)	Cohn et al. (2009)
Subjective Happiness Scale (SHS)	Lyubomirsky & Kepper (1999)
Satisfaction with Life Scale (SWLS)	Diener, Emmons, Larsen, & Griffin (1985)
Extreme Valuing of Happiness Scale (VHQ)	Mauss et al. (2011)
Fear of Happiness (FOH)	Joshanloo (2013)
Emotion Regulation Questionnaire (ERQ)	Gross & John (2003)
Emotion and Decision-Making Beliefs	Gatchpazian & Ford (in prep)
Emotion Control Beliefs (Items 1-4)	Tamir, John, Srivastava, & Gross (2007)
Emotion Control Beliefs (Items 5-16)	Mauss, Butler, Roberts, & Chu (2010)
Domain-Specific Impulsivity in Children	Tsukayama, Duckworth, & Kim (2013)
Brief Resilience Scale	Smith et al. (2008)
Brief COPE	Carver (1997)
Emotion Regulation COVID-19 Items	n/a
Positive Emotion Persistence (PEP)	Gruber et al. (in prep)

Psychological Adjustment	
DSM-5 Cross Cutting Measure	American Psychiatric Association (2013)
Patient Safety Screener-3	Boudreaux et al. (2015)
Schedule for Nonadaptive and Adaptive Personality-Self-Harm	Clark et al. (1993)
Family Index of Risk for Mood	Algorta et al. (2013)
PROMIS	Yu et al. (2012)
Alcohol Frequency Questionnaire (AQF)	Rehm (1998)
RAPI Alcohol Problems Questionnaire	White & Labouvie (1989)
Perceived Stress Scale (PSS)	Cohen et al. (1983)
Drug Abuse Screening Test (DAST-10)	Cocco & Carey (1998); Skinner (1982)
Cannabis Use Problems Identification Test	Bashford, Flett, & Copeland (2010)
Healthy Living Questionnaire 2011	Ware et al. (2001)
Prodromal Questionnaire for Psychosis	Loewy et al. (2011)
GAD-7 (Generalized Anxiety Disorder)	Spitzer et al., (2006)
Social Functioning and Connection	
Perceived Social Support and Conflict-friends	Schuster, Kessler, & Aseltine (1990); Whalen & Lachman (2000)
Social Identity Scale at CU Boulder	Leach et al. (2008)
Belonging Uncertainty Scale	Walton & Cohen (2007)
Social Network-Quantity	Parkinson, Kleinbaum, & Wheatley (2018)
Social Network-Quality	Morelli et al. (2017)
UCLA Loneliness Scale	Russell et al. (1978)
Inclusion of Self in Other Scale	Aron et al. (1991)
Academic Adjustment	
National Survey of Student Engagement	Kuh et al. (2001)
Academic Self-Efficacy	Gaumer Erickson et al. (2016)
Miscellaneous	
Brief Social Desirability Scale	Haghighat (2007)
Catch Items	n/a
Creativity Items	
Free Association Task	Gray et al. (2019)

COVID-19 Items	
COVID-19 Health Exposure	n/a
COVID-19 Health Behavioral Changes	n/a
COVID-19 Perceived Control	n/a
COVID-19 Perceived Threat	n/a
COVID-19 Health Mindset	n/a
COVID-19 Health Impact	n/a
COVID-19 Health Anxiety	n/a
Belief in Conspiracy Theories	n/a
Political Orientation	n/a
ESM Measures (2 week daily sampling)	
Daily Prompt – DRM Emotions	Kahneman et al. (2004)
Daily Prompt – Emotion Regulation Strategies	Gruber, Kogan, Mennin, & Murray (2013)
Daily Prompt – Mind wandering	Killingsworth & Gilbert (2010)
Daily Prompt – Context	Gruber, Kogan, Mennin, & Murray (2013)
Daily Prompt – Context (COVID-19)	Gruber, Kogan, Mennin, & Murray (2013)
End of Day – DRM Emotions	Kahneman et al. (2004)
End of Day – Emotion Regulation	n/a
End of Day – Mind Wandering	Killingsworth & Gilbert (2010)
End of Day – Context	Gruber, Kogan, Mennin, & Murray (2013)
End of Day – Daily Events Scale	Gruber, Kogan, Mennin, & Murray (2013)
End of Day – Daily Satisfaction	Gruber, Kogan, Mennin, & Murray (2013)
End of Day – SF-36	Gruber, Kogan, Mennin, & Murray (2013)
End of Day – Sleep	n/a

Note: n/a = Not officially published scale.

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