

Neurophysiological Responses to Interpersonal Emotional Images Prospectively Predict the Impact of COVID-19 Pandemic-Related Stress on Internalizing Symptoms

Lindsay Dickey, Michael West, Samantha Pegg, Haley Green, and Autumn Kujawa

ABSTRACT

BACKGROUND: Exposure to stressful events related to the COVID-19 coronavirus pandemic has been associated with increases in the prevalence of depression and anxiety, raising questions about vulnerabilities that make some individuals more susceptible to internalizing symptoms following stress exposure.

METHODS: This prospective study examined the effects of neurophysiological reactivity to positive and threatening interpersonal stimuli, indexed by the late positive potential (LPP) event-related potential, in conjunction with exposure to interpersonal pandemic-related stressors in the prediction of internalizing symptom changes from before to during the pandemic. Emerging adults ($n = 75$) initially completed measures of internalizing symptoms and an interpersonal emotional images task while an electroencephalogram was recorded pre-pandemic and were recontacted during the COVID-19 pandemic in May 2020 to complete measures of exposure to pandemic-related stressful events and current internalizing symptoms.

RESULTS: Results indicated that emerging adults experienced numerous stressful events associated with the pandemic, as well as overall increases in symptoms of depression and traumatic intrusions during the pandemic. Furthermore, significant interactions between LPP reactivity to positive and threatening interpersonal stimuli and interpersonal stress exposure emerged in the prediction of internalizing symptoms, controlling for baseline symptoms. Under high exposure to interpersonal stressors, reduced positive LPPs predicted increases in depressive symptoms while enhanced threatening LPPs predicted increases in traumatic intrusions.

CONCLUSIONS: These findings highlight the mental health impacts of the COVID-19 pandemic on emerging adults, and the role of individual differences in neurophysiological reactivity to emotional stimuli in vulnerability for depression and traumatic intrusions following stress exposure.

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Depressive and anxiety disorders are highly comorbid, prevalent psychopathologies (1,2). Robust evidence demonstrates that stressful events, particularly interpersonal events, prospectively predict depression and anxiety (3–7). However, not all individuals who experience stressful events develop depression or anxiety. Understanding factors underlying risk and resilience following exposure to stressful life events is imperative for identifying those at greatest risk and processes to target through intervention.

In addition to constituting an international physical health crisis, the COVID-19 coronavirus pandemic has had profound mental health impacts, with prevalence estimates of clinical levels of depression and anxiety symptoms during the pandemic far exceeding prior epidemiological research (22.8%–45.1% for depression and 20.0%–37.1% for anxiety) (8–10). Given the existing stress literature, pandemic-related

stressors potentially contributing to the elevation in internalizing symptoms include interpersonal conflicts (11), social isolation and loneliness (12), job loss (13), general life disruption (14), and the uncontrollable nature of these experiences (15).

Emerging adulthood is a time of increasing independence and salience of peer and romantic relationships (16) and a high-risk period for the emergence of psychopathology, particularly mood and anxiety disorders (17,18). Our prior research indicates that emerging adults experienced abrupt disruptions in education, occupations, and relationships because of university closures, economic recession, and social distancing mandates associated with the pandemic, which correlated with depression and anxiety (9). Of these, interpersonal pandemic-related stressors may confer particular risk for internalizing symptoms to this population. Longitudinal data on

the pandemic are needed to disentangle preexisting vulnerability factors making some individuals more susceptible to symptom changes following stress exposure. Considering that experiences of stress confound with individuals' dependent behaviors and cognitions (7,19), the unanticipated, ubiquitous impact of the COVID-19 pandemic provides a unique opportunity to study risk processes that predict individual differences in responses to stressors.

The late positive potential (LPP) is a neurophysiological measure of emotional reactivity that could be applied to understand stress vulnerability. The LPP is an event-related potential (ERP) characterized by a sustained positive deflection beginning approximately 300 ms after stimulus onset (20,21). The LPP reflects the elaborative processing of motivationally salient stimuli and is consistently enhanced in response to emotional compared with neutral stimuli (22). Combined electroencephalography (EEG)-functional magnetic resonance imaging studies show that scalp-recorded LPP correlates with activation within a broad network of cortical and subcortical regions, including the amygdala, nucleus accumbens, medial and ventrolateral prefrontal cortices, and visual cortices (23–26). The LPP demonstrates sensitivity to individual differences in emotion processing and is reliably elicited across development (27–29).

Previous research shows reduced LPPs to positive stimuli in individuals with and at risk for depression (30–33), but elevated LPPs, generally to negative or threatening stimuli, in individuals with and at risk for anxiety (33–38). The literature on LPPs to negative stimuli in depression is more equivocal, with some evidence of reductions (31) and other studies revealing enhancements (39,40). These discrepancies may be attributable to differences in the orientation of the stimuli (e.g., self-relevant stimuli vs. other-oriented stimuli) and/or comorbid anxiety symptoms. However, one study found enhanced LPPs to emotional faces when controlling for comorbid anxiety (41), supporting heterogeneous patterns of reactivity within major depressive disorder, potentially because of specific symptom combinations, such as the presence or absence of anhedonia. These discrepancies may also relate to developmental differences in reactivity, given the evidence for overall reductions in LPPs with age (29). Reduced LPPs in depression fit with emotion context insensitivity theories (42), possibly reflecting the inability to sustain activation in motivational systems (30,43), particularly for positive stimuli (44). Conversely, elevated LPPs associated with anxiety are consistent with the broader literature on heightened attention for threatening stimuli (45,46). This highlights the potential sensitivity of the LPP in distinguishing heterogeneous patterns of emotion processing within depression and between highly comorbid psychopathologies.

Importantly, much of the literature on the LPP and internalizing psychopathologies has relied on cross-sectional designs, but individual differences in emotional reactivity assessed by the LPP may actually reflect an underlying vulnerability for the later emergence of symptoms following stress exposure. Supporting this possibility, we previously showed that an enhanced LPP to unpleasant stimuli and a reduced LPP to pleasant stimuli prospectively predicted increases in psychiatric symptoms in children exposed to greater stress related to a natural disaster (47). This theory is

further supported by a larger literature examining ERPs, brain activation, and pupillometry in emotional contexts as moderators of symptom change following stress exposure (14,48–50). Specifically, other ERP research demonstrates an interactive effect between reward positivity, an ERP marker of positive valence systems function, and exposure to stressful life events in the prediction of depression (48). There is also evidence that amygdala reactivity to threatening stimuli in conjunction with exposure to acute stressful events may be a candidate neural index of stress vulnerability (14,49). Finally, pupillometry research shows that reduced pupil dilation in response to affective stimuli predicted depressive symptoms following exposure to natural disaster-related stress (50).

The majority of the ERP literature on emotion uses broad categories of unpleasant and pleasant stimuli (51) or emotional faces presented out of context. Considering the centrality of social processes to internalizing psychopathologies (52), LPP reactivity to interpersonal stimuli specifically could be highly relevant for understanding vulnerabilities for internalizing symptoms. Research shows that LPP is sensitive to stimulus attributes (53–55), with social aspects of stimuli demonstrating particular salience (55,56). We recently validated a novel set of stimuli to elicit neurophysiological reactivity to interpersonal emotional images (L. Dickey, M.Ed., unpublished data, June 2020), which may be particularly relevant for understanding symptom changes during the pandemic, given the interpersonal impacts of the pandemic and social distancing. Hyper-reactivity to threat and hyporeactivity to positive interpersonal stimuli may result in tendencies to disengage and withdraw socially (e.g., avoidance of potential threat and low approach motivation toward positive interactions). These tendencies may then be exacerbated by stress exposure, such as stress related to the ongoing pandemic where social isolation decreases the availability of positive social interactions, thus contributing to the onset and maintenance of depression.

This study is among the first to examine prospective predictors of responses to COVID-19 stressful events. Extending findings from Kujawa *et al.* (47), we examined LPP reactivity to positive and threatening interpersonal emotional images as predictors of internalizing symptom changes during a global pandemic. In an effort to understand the impact of the COVID-19 pandemic on mental health, we first developed a measure of pandemic-related stressful events in a separate sample of emerging adults (9). In this study, LPP reactivity to interpersonal images and baseline internalizing symptoms were assessed pre-pandemic. In May 2020, follow-up assessments of exposure to pandemic-related events and internalizing symptoms were completed to examine stress exposure and changes in depressive and anxiety symptoms during the pandemic. Given the potency of interpersonal stressors as predictors of internalizing disorders, the pervasive social disruption associated with the COVID-19 pandemic, and the focus on neurophysiological reactivity to interpersonal emotional images, we examined both total pandemic-related stressors and interpersonal stressors, specifically, as predictors of internalizing symptom change as main effects and interactive effects with the LPP to positive and threatening interpersonal stimuli. We expected significant increases in symptoms of depression and anxiety across time and main effects of both types of stress on symptoms. Furthermore, we

predicted that under high exposure to stress, particularly interpersonal stress, reduced LPP reactivity to emotional stimuli would predict changes in depressive symptoms, whereas heightened LPP reactivity to threatening stimuli would predict anxiety symptom changes.

METHODS AND MATERIALS

Participants

Participants ($N = 130$) were undergraduate students originally recruited as part of a study on emotional and social functioning in emerging adults. At T1, participants completed a series of self-report questionnaires followed by counterbalanced computer-based tasks while EEG was continuously recorded (57). During May 11–13, 2020, all participants were contacted with the option to complete additional self-report questionnaires by May 21, 2020 (T2). The analyzed sample ($n = 75$) with T1 and T2 data had a mean age (SD) of 19.25 (1.16) years at T1 and were 76.0% female, 10.67% Hispanic/Latino, 54.67% White/Caucasian, 29.33% Asian, 9.33% Black/African American, and 6.67% multiracial. The average time between assessments was 313.14 (102.26) days. Participants identifying as female ($\chi^2_{1,113} = 6.78, p = .01$), Black/African American, ($\chi^2_{1,113} = 5.47, p = .02$), and with higher baseline panic symptoms ($t_{111} = 2.54, p = .02$) were more likely to complete the follow-up assessment. The retained sample did not differ from the baseline sample on LPPs or baseline symptoms of depression, social anxiety, or traumatic intrusions ($ps > .08$). The Institutional Review Board at Vanderbilt University approved this study, and informed consent was obtained from all participants.

Measures

Interpersonal Emotional Interrupt Task. EEG was continuously recorded while participants completed an interpersonal version of an emotional interrupt paradigm, which reliably elicits the LPP (29,58). Stimuli were selected for relevance to the social experiences of emerging adults, including 15 threatening interpersonal images (e.g., bullying by peers, arguing with parents or friends), 15 pleasant interpersonal images (e.g., friends laughing, happy couples), and 15 nonsocial neutral images (e.g., nature and city scenes). Stimuli were obtained through stock image sites and the Open Affective Standardized Image Set (59). Trials consisted of a fixation cross (+) presented for 800 ms, an image presented for 1000 ms, a single target arrow (< or >) for 150 ms, and the same image presented for an additional 400 ms. To ensure attention throughout the task and to measure emotional interference on behavioral performance, participants were instructed to click the right or left mouse button to indicate the target arrow direction on each trial. Only correct trials with responses within 150–2150 ms were included in the analysis. Intertrial intervals varied randomly from 1500 ms to 2000 ms. Participants completed six practice trials followed by two blocks of the task for a total of 90 trials. Behavioral data are presented in the Supplement.

Pandemic-Related Stress. Participants completed the college student version of the Pandemic Stress Questionnaire

(PSQ) (9), a 24-item measure of perceived exposure and subjective severity of events due to the COVID-19 pandemic (full measure in the Supplement). Participants responded “yes/no” to indicate experiencing each event, followed by a perceived severity rating from 1 to 5 for endorsed events. In addition to PSQ total event scores, we previously tested face valid subscale scores, including an interpersonal subscale that includes five items assessing exposure to interpersonal conflicts, unexpected separations, inability to be with close others, loss of a close other because of COVID-19, and experiences of racism and discrimination (9). Only the total and interpersonal PSQ scales were analyzed in this study.

Internalizing Symptoms. Internalizing symptoms were measured using the 64-item Inventory of Depression and Anxiety Symptoms (60). Participants rated the extent to which each item was experienced in the previous 2 weeks from 1 to 5. The Inventory of Depression and Anxiety Symptoms consists of two broad scales, general depression and dysphoria, and 10 symptom-specific scales. We first examined symptom changes from before to during the pandemic in general depression, social anxiety, panic, and traumatic intrusions. To minimize the number of tests conducted, primary analyses of LPPs and interpersonal stress focused on symptom scales that showed an overall increase during the pandemic. Items assessing suicidal ideation (items 7, 9, 14, 15, 41, and 43) were not assessed at follow-up and were excluded from the calculation of the general depression scale. Internal consistencies for the analyzed scales at the initial and follow-up assessments were $\alpha = 0.92$ – 0.93 for general depression, $\alpha = 0.67$ – 0.80 for traumatic intrusions, $\alpha = 0.86$ – 0.75 for social anxiety, and $\alpha = 0.88$ – 0.86 for panic.

EEG Data Collection and Processing

EEG data were continuously recorded using a 64-channel actiCHamp system (Brain Products, Munich, Germany). Cz served as the online reference, and data were collected at a sampling rate of 1000 Hz. Electrooculogram was recorded by facial electrodes placed 1 cm vertically and horizontally around the eyes and referenced to an electrode on the back of the neck, per the Brain Products bipolar-to-auxiliary adapter design. Offline processing was completed using BrainVision Analyzer software (Brain Products, Munich, Germany). Data were re-referenced to the linked mastoid recordings (TP9/TP10) and bandpass filtered from 0.01 to 30 Hz. Trials were segmented from -200 ms to 1000 ms after image onset. Ocular correction and semiautomated procedures identifying voltage steps greater than $50 \mu\text{V}/\text{ms}$ between sampling points, voltage differences greater than $175 \mu\text{V}$ within a trial, and lowest allowed activity of $0.50 \mu\text{V}$ within 100-ms intervals were applied. Remaining artifacts were removed through visual inspection. Faulty recordings at single electrodes were resolved through interpolation. Included participants had a minimum of 12 artifact-free trials per condition to obtain a stable LPP (61). Segments were averaged within each condition and baseline corrected to -200 ms. LPP was scored from 400 to 1000 ms (47,55,62,63) at a pooling of occipitoparietal sites (POz, PO3, PO4) (34,64–67), consistent with cross-sectional

research on this sample (L. Dickey, M.Ed., unpublished data, June 2020), and the region of overlap in the maximal distributions for both emotional conditions compared with neutral (Figure 1). Exploratory analyses testing a broader occipitoparietal pooling are presented in the Supplement. Split-half reliability for the LPP was acceptable to good (Spearman-Brown coefficients: positive = 0.83, threatening = 0.78, neutral = 0.68). Ten participants were missing EEG data: 1 did not complete the task, 1 had a recording issue, 5 had poor data quality, and 3 had fewer than 12 correct artifact-free trials.

Data Analysis

Paired-samples *t* tests were conducted to analyze internalizing symptom changes from before to during the pandemic. Restricted maximum likelihood was used to estimate missing data using the lme4 package in R (66). Frequencies of endorsed events and event sums were calculated to characterize exposure to pandemic-related stressors.

Consistent with ERP recommendations, unstandardized residual scores were computed to evaluate the LPP in each emotional condition, partialling out variance associated with the neutral condition (67). Multiple linear regressions were conducted to test the effects of LPPs to positive and threatening stimuli and pandemic-related stressful events and their interaction in the prediction of internalizing symptom changes. Interaction terms were calculated by taking the product of LPP residual scores and mean-centered interpersonal stressful events. To isolate change in symptoms from T1 to T2, T1 symptoms were included as covariates. Furthermore, to differentiate effects for depression versus anxiety, T2 anxiety was included as a covariate when examining predictors of depression, and T2 depression was included as a covariate when examining predictors of anxiety. Full-information maximum likelihood was used to estimate missing data using the lavaan package in R (68). Significant interactions were

probed by examining simple slopes at the mean and 1 SD above and below the mean LPP, region of significance using the Johnson-Neyman technique (69), and confidence bands for the simple slopes through a web-based utility (70). To account for type I errors from multiple comparisons, regression results are also presented, with false discovery rate (FDR) corrections applied (71).

RESULTS

Frequencies of PSQ Events

Frequencies of endorsed PSQ items are presented in Table 1. Participants endorsed an average of 7.39 total PSQ events (SD = 3.38; range, 1–17) and 2.24 interpersonal events (SD = 0.98; range, 0–4). Commonly experienced stressors included unexpected separations and moves, inability to be with close others, and cancellation of important events and travel, while the least frequently endorsed stressors included the death of a close other, visa problems, and COVID-19 diagnosis.

Symptoms of Depression and Anxiety

Descriptive statistics and bivariate correlations between primary study variables are presented in Table 2. Paired-samples *t* tests revealed significant increases in symptoms of depression ($t_{74} = 4.06, p < .001$) and traumatic intrusions ($t_{74} = 4.41, p < .001$), a decrease in social anxiety ($t_{74} = -3.04, p < .01$), and no change in panic symptoms ($p = .45$). At T1, 12.0% of the sample were above the balanced clinical cutoff for major depressive disorder and 6.7% were above the balanced clinical cutoff for posttraumatic stress disorder. At T2, this proportion increased to 32.0% for depression and 26.7% for posttraumatic stress disorder (72). Subsequent primary analyses of LPP and stress focused on changes in depression and traumatic intrusions, given that both increased from T1 to T2. Exploratory analyses of social anxiety symptom changes are presented in the Supplement.

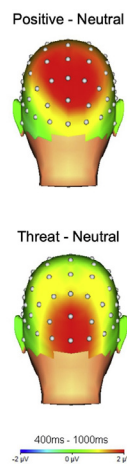
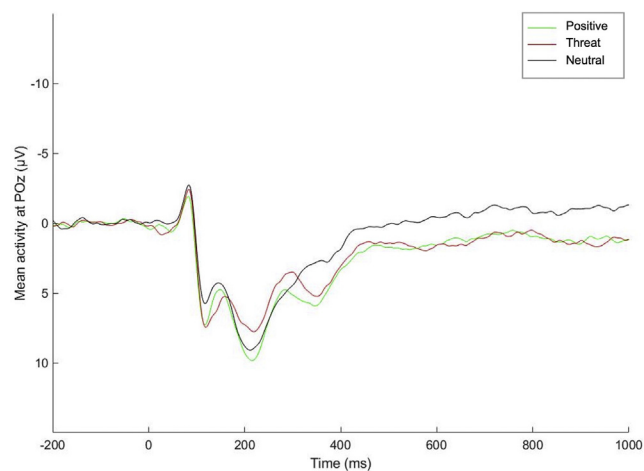


Figure 1. Grand average event-related potential waveform for the late positive potential across POz, PO3, and PO4. Scalp distributions reflect the response to the interpersonal emotional condition minus the response to the neutral condition.

Table 1. Frequency of Exposure to Events Assessed by the Pandemic Stress Questionnaire

Subscale/Item	%
I had difficulty obtaining basic supplies because of the coronavirus pandemic (e.g., food, medicine, toilet paper).	27.6
I had to move unexpectedly because of the coronavirus pandemic.	67.1
I had problems with my visa or the Student and Exchange Visitor Information System because of the coronavirus pandemic (e.g., unable to renew).	2.6
I had to cancel travel or experienced a major disruption in travel plans because of the coronavirus pandemic.	50.0
I had to cancel or postpone important events because of the coronavirus pandemic (e.g., events for a club, sporting events, major celebrations).	75.0
I had to take on additional responsibilities caring for others (e.g., siblings, other family members) due to the coronavirus pandemic.	39.5
I was unexpectedly separated from family, friends, or others close to me because of the coronavirus pandemic.	89.5
I was unable to be with close family, friends, or partners because of the coronavirus pandemic.	64.5
I had conflicts or arguments with my partner or family members due to coronavirus (e.g., conflicts about living arrangements, shared work space, schedule expectations).	59.2
I experienced racism or discrimination due to the coronavirus pandemic.	5.3
Someone close to me died from COVID-19.	3.9
I experienced significant financial strain due to the pandemic (e.g., due to travel, purchasing supplies, paying for housing).	28.9
I temporarily or permanently lost a job or had my work hours greatly reduced due to the coronavirus pandemic.	35.5
My parent(s) temporarily or permanently lost a job or had their work hours greatly reduced because of the coronavirus pandemic.	34.2
I was unable to complete important requirements for my education or professional goals due to the coronavirus pandemic (e.g., coursework, taking the SAT or GRE, thesis).	28.9
I had problems with online courses and/or remote work (e.g., slow connection, no computer or internet access, major differences in time zone).	48.7
I had symptoms of COVID-19 (e.g., cough, fever, trouble breathing) but was unable to get tested.	10.5
I was tested for COVID-19.	10.5
I was diagnosed with COVID-19.	1.3
I had difficulty accessing or paying for physical or mental health care and/or difficulties with health insurance due to the coronavirus pandemic.	23.7
I was quarantined for 2 weeks or longer due to possible exposure to COVID-19 or due to international travel.	31.6
Someone close to me had symptoms of COVID-19 (e.g., cough, fever, trouble breathing) but was unable to get tested.	14.5
Someone close to me was diagnosed with COVID-19.	18.4
Someone close to me was quarantined for 2 weeks or longer due to possible exposure to COVID-19 or due to international travel.	35.5

GRE, Graduate Record Examinations; SAT, Scholastic Assessment Test.

LPP Predicting Internalizing Symptom Changes

Multiple regression results testing the main and interactive effects of LPP to positive and threatening images and interpersonal and total pandemic-related stressors in the prediction of depression and traumatic intrusion symptom

changes are presented in Tables 3–6. Interpersonal events predicted change in depressive symptoms (β s = 0.21–0.25, z s = 2.21–2.86, p s = .004–.03, FDR-corrected p s = .01–.04) but not traumatic intrusions (β s = 0.09–0.16, z s = 0.92–1.57, p s = .12–.36). Total events predicted changes in

Table 2. Descriptive Statistics and Bivariate Correlations Among Primary Study Variables

Variable	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11
1 Total PSQ Events	7.39 (3.38)	–	–	–	–	–	–	–	–	–	–	–
2 Interpersonal PSQ Events	2.24 (0.98)	0.57 ^a	–	–	–	–	–	–	–	–	–	–
3 T1 General Depression	40.64 (12.39)	0.18	0.08	–	–	–	–	–	–	–	–	–
4 T1 Traumatic Intrusions	6.29 (2.50)	–0.17	0.00	0.47 ^a	–	–	–	–	–	–	–	–
5 T1 Social Anxiety	10.36 (4.50)	–0.06	0.06	0.54 ^a	0.36 ^a	–	–	–	–	–	–	–
6 T1 Panic	11.83 (4.68)	0.16	0.12	0.54 ^a	0.46 ^a	0.50 ^a	–	–	–	–	–	–
7 T2 General Depression	47.53 (13.94)	0.50 ^a	0.39 ^a	0.38 ^a	0.15	0.30 ^a	0.27 ^b	–	–	–	–	–
8 T2 Traumatic Intrusions	8.09 (3.46)	0.38 ^a	0.31 ^a	0.13	0.33 ^a	0.02	0.31 ^a	0.53 ^a	–	–	–	–
9 T2 Social Anxiety	8.63 (3.63)	0.18	0.14	0.18	0.18	0.28 ^b	0.19	0.44 ^a	0.40 ^a	–	–	–
10 T2 Panic	12.17 (4.94)	0.33 ^a	0.31 ^a	0.31 ^a	0.26 ^b	0.24 ^b	0.66 ^a	0.56 ^a	0.50 ^a	0.45 ^a	–	–
11 Positive LPP Residuals	0.00 (3.28)	0.05	0.14	0.05	0.03	0.12	0.22 ^c	0.06	0.16	0.22 ^c	0.33 ^a	–
12 Threat LPP Residuals	0.00 (2.88)	–0.02	0.20	–0.02	0.02	0.06	0.04	0.07	0.14	0.23 ^c	0.08	0.57 ^a

LPP, late positive potential; PSQ, Pandemic Stress Questionnaire; T, time.

^a $p < .01$.

^b $p < .05$.

^c $p < .10$.

Table 3. Multiple Regression Analyses Testing the Main and Interactive Effect of Pandemic-Related Interpersonal Stressful Events and LPP to Emotional Interpersonal Stimuli in the Prediction of Depressive Symptom Changes From Before to During the Pandemic

Positive LPP			Threatening LPP		
Variable	b (SE)	β	Variable	b (SE)	β
T1 General Depression	0.37 (0.10)	0.32 ^a	T1 General Depression	0.36 (0.10)	0.31 ^a
T2 Traumatic Intrusions	1.85 (0.37)	0.45 ^a	T2 Traumatic Intrusions	1.85 (0.38)	0.45 ^a
Positive LPP Residuals	-0.04 (0.39)	-0.01	Threatening LPP Residuals	-0.01 (0.41)	-0.00
Interpersonal PSQ Events	3.65 (1.28)	0.25 ^b	Interpersonal PSQ Events	2.92 (1.32)	0.21 ^c
Int. Events \times Pos LPP Res.	-0.82 (0.40)	-0.19 ^c	Int. Events \times Threat LPP Res.	-0.66 (0.42)	-0.15
Total Model $R^2 = 0.49$			Total Model $R^2 = 0.48$		

Int. Events \times Pos LPP Res., interaction between interpersonal stressful events and LPP residuals to positive images; Int. Events \times Threat LPP Res., interaction between interpersonal stressful events and LPP residuals to threatening images; LPP, late positive potential; PSQ, Pandemic Stress Questionnaire.

^a $p < .001$.

^b $p < .01$.

^c $p < .05$.

both (β s = 0.25–0.31, z s = 2.26–3.38, p s = .001–.02, FDR-corrected p s = .008–.04). The main effects of LPPs were not significant (p s = .24–.91). There were no significant interactions between LPPs and total events (p s > .08); however, the interaction between interpersonal stressful events and positive LPPs predicted depressive symptom change ($\beta = -0.19$, $z = -2.05$, $p = .04$, FDR-corrected $p = .16$), and the interaction between interpersonal events and threatening LPPs predicted change in traumatic intrusion symptoms ($\beta = 0.25$, $z = 2.39$, $p = .02$, FDR-corrected $p = .14$). These interactions remained significant at $p < .05$ when controlling for age, gender, race, and time between assessments.

Simple slopes revealed that the effect of interpersonal events in the prediction of depressive symptom change was significant for positive LPP amplitudes at the mean ($b = 3.65$, $SE = 1.28$, $p = .006$) and 1 SD below the mean ($b = 6.35$, $SE = 1.95$, $p = .002$) but not LPPs 1 SD above the mean ($p = .58$). The Johnson-Neyman region of significance indicated that the effect of interpersonal stress on depression was significant for LPP amplitudes below 1.25 (observed amplitude range = -7.56 to 7.79) (Figure 2). The effect of interpersonal events predicting change in traumatic intrusions was

significant for threatening LPPs 1 SD above the mean ($b = 1.34$, $SE = 0.51$, $p = .01$) but not for LPP amplitudes at the mean or 1 SD below the mean (p s = .12–.58). The Johnson-Neyman region of significance indicated that this effect was significant for LPP amplitudes above 0.75 (observed range = -6.98 to -6.39) (Figure 2). Given the evidence that the LPP is composed of several distinct positivities (29,73), exploratory analyses examining P300/early LPP (300–400 ms) and late LPP (900–1000 ms) are presented in the Supplement, along with exploratory analyses of early visual processing components (i.e., P1, N1, N2).

DISCUSSION

This study is among the first longitudinal studies to characterize the impact of the COVID-19 pandemic on emerging adults by assessing the frequencies of exposure to pandemic-related events and changes in internalizing symptoms. We additionally examined the predictive utility of neurophysiological reactivity to interpersonal emotional images in conjunction with pandemic-related stressful events on internalizing symptom change during the pandemic. We found overall increases in symptoms of depression and

Table 4. Multiple Regression Analyses Testing the Main and Interactive Effect of Pandemic-Related Interpersonal Stressful Events and LPP to Emotional Interpersonal Stimuli in the Prediction of Traumatic Intrusion Symptom Changes From Before to During the Pandemic

Positive LPP			Threatening LPP		
Variable	b (SE)	β	Variable	b (SE)	β
T1 Traumatic Intrusions	0.31 (0.13)	0.23 ^a	T1 Traumatic Intrusions	0.33 (0.13)	0.25 ^b
T2 General Depression	0.11 (0.03)	0.45 ^c	T2 General Depression	0.11 (0.02)	0.44 ^c
Positive LPP Residuals	0.07 (0.10)	0.06	Threatening LPP Residuals	0.02 (0.11)	0.02
Interpersonal PSQ Events	0.32 (0.35)	0.09	Interpersonal PSQ Events	0.55 (0.35)	0.16
Int. Events \times Pos LPP Res.	0.19 (0.11)	0.17 ^d	Int. Events \times Threat LPP Res.	0.25 (0.10)	0.23 ^a
Total Model $R^2 = 0.39$			Total Model $R^2 = 0.38$		

Int. Events \times Pos LPP Res., interaction between interpersonal stressful events and LPP residuals to positive images; Int. Events \times Threat LPP Res., interaction between interpersonal stressful events and LPP residuals to threatening images; LPP, late positive potential; PSQ, Pandemic Stress Questionnaire; T, time.

^a $p < .05$.

^b $p < .01$.

^c $p < .001$.

^d $p < .10$.

Table 5. Multiple Regression Analyses Testing the Main and Interactive Effect of Total Pandemic-Related Stressful Events and LPP to Emotional Interpersonal Stimuli in the Prediction of Depressive Symptom Changes From Before to During the Pandemic

Variable	Positive LPP		Variable	Threatening LPP	
	b (SE)	β		b (SE)	β
T1 General Depression	0.32 (0.10)	0.29 ^a	T1 General Depression	0.33 (0.10)	0.29 ^a
T2 Traumatic Intrusions	1.56 (0.38)	0.39 ^b	T2 Traumatic Intrusions	1.54 (0.37)	0.38 ^b
Positive LPP Residuals	-0.09 (0.37)	-0.02	Threatening LPP Residuals	0.16 (0.38)	0.04
Total PSQ Events	1.30 (0.38)	0.31 ^a	Total PSQ Events	1.26 (0.38)	0.30 ^a
Tot. Events \times Pos LPP Res.	-0.09 (0.13)	-0.06	Tot. Events \times Threat LPP Res.	-0.18 (0.10)	-0.15 ^c
Total Model $R^2 = 0.46$			Total Model $R^2 = 0.49$		

Tot. Events \times Pos LPP Res., interaction between total stressful events and LPP residuals to positive images; Tot. Events \times Threat LPP Res., interaction between total stressful events and LPP residuals to threatening images; LPP, late positive potential; PSQ, Pandemic Stress Questionnaire, T, time.

^a $p < .01$.
^b $p < .001$.
^c $p < .10$.

traumatic intrusions and decreases in social anxiety symptoms during the pandemic, but no change in panic symptoms. Reactivity to emotional interpersonal stimuli before the pandemic, measured by the LPP, moderated the impact of interpersonal events, specifically, on internalizing symptom changes, such that hyporeactivity to positive stimuli predicted increased depressive symptoms and hyperreactivity to threatening stimuli predicted increased traumatic intrusion symptoms in combination with greater interpersonal stress exposure.

Emerging adults endorsed many pandemic-related events with high frequency in May 2020, including being unexpectedly separated from close others, unexpected moves, the inability to be with close others, and cancellation of important events and travel. Compared with our prior research validating PSQ in an online community sample of young adults (9), differences emerged from this sample. General disruptions and financial items, such as difficulty obtaining supplies, financial strain, and job loss, were endorsed at relatively higher rates in the community sample, while interpersonal items, including unexpected separation and conflicts/arguments, were endorsed more frequently in this sample. Although additional research is needed across the life span, these comparisons suggest that some types of interpersonal stressors due to COVID-19 may be more common in younger populations, but financial, work-

related, and health-related stressors may be more common in older adults.

Interpersonal stressors are especially robust predictors of depression and anxiety (3–5,7), and the high rates of interpersonal events due to COVID-19 informed our hypothesis that individual differences in emotional reactivity in the context of interpersonal scenarios, indexed by the LPP, may predict responses to interpersonal stressors, specifically. Consistent with the robust associations between stress and psychopathology (3–5,7), we observed increases in depressive symptoms and trauma-related anxiety symptoms but decreases in social anxiety symptoms. Considering social distancing mandates and limited in-person interactions, it is possible that reduced exposures to socioevaluative situations alleviated symptoms of social anxiety.

In examining neurophysiological predictors of symptom change, our results showed that under high exposure to stressful interpersonal events, reduced neural reactivity to positive interpersonal images conferred risk for depressive symptoms, while enhanced reactivity to threatening images predicted increased traumatic intrusions. These findings suggest that LPPs to emotional interpersonal stimuli may reflect individual differences in vulnerability to stress or general susceptibility to the environment and further distinguish risk for depression from trauma-related anxiety. Individuals who have

Table 6. Multiple Regression Analyses Testing the Main and Interactive Effect of Pandemic-Related Total Stressful Events and LPP to Emotional Interpersonal Stimuli in the Prediction of Traumatic Intrusion Symptom Changes From Before to During the Pandemic

Variable	Positive LPP		Variable	Threatening LPP	
	b (SE)	β		b (SE)	β
T1 Traumatic Intrusions	0.43 (0.13)	0.31 ^a	T1 Traumatic Intrusions	0.45 (0.13)	0.32 ^a
T2 General Depression	0.09 (0.03)	0.35 ^a	T2 General Depression	0.09 (0.03)	0.35 ^a
Positive LPP Residuals	0.12 (0.10)	0.11	Threatening LPP Residuals	0.12 (0.10)	0.10
Total PSQ Events	0.25 (0.11)	0.25 ^b	Total PSQ Events	0.27 (0.11)	0.26 ^b
Tot. Events \times Pos LPP Res.	0.02 (0.03)	0.05	Tot. Events \times Threat LPP Res.	0.03 (0.03)	0.10
Total Model $R^2 = 0.42$			Total Model $R^2 = 0.38$		

Int. Events \times Pos LPP Res., interaction between total stressful events and LPP residuals to positive images; Int. Events \times Threat LPP Res., interaction between total stressful events and LPP residuals to threatening images; LPP, late positive potential; PSQ, Pandemic Stress Questionnaire.

^a $p < .01$.
^b $p < .05$.

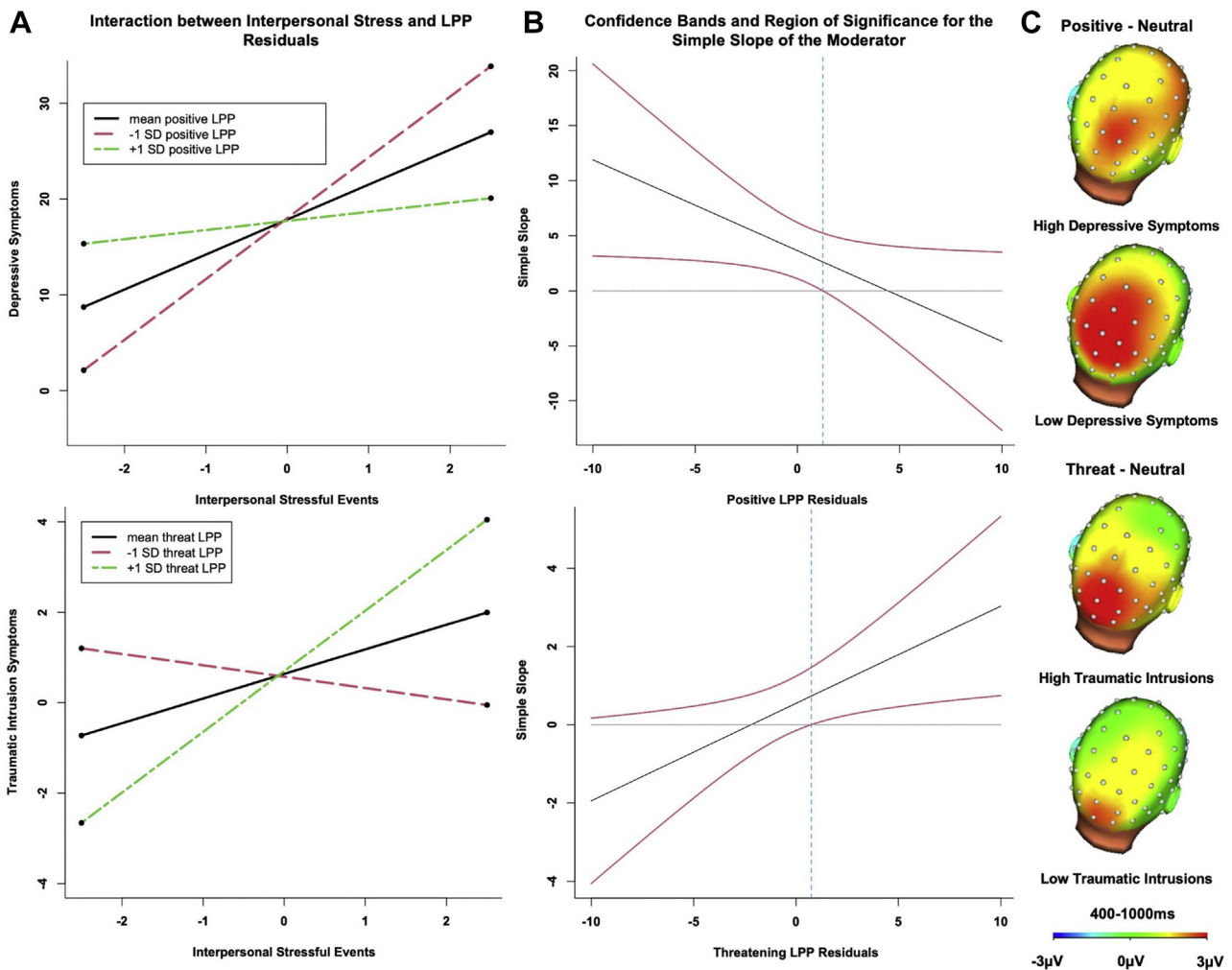


Figure 2. (A) Plots of the simple slopes for the interaction effect between pandemic-related interpersonal events at low, average, and high levels of late positive potential (LPP) reactivity to positive stimuli (top) and threatening stimuli (bottom) in the prediction of depressive symptoms (top) and traumatic intrusion symptoms (bottom). (B) Plot of the confidence bands and region of significance for the simple slopes. (C) Scalp distributions depicting the LPP for participants above the mean for interpersonal stress exposure with high and low levels of symptom changes. The median splits for interpersonal stress and high and low residualized internalizing symptom changes were used for illustrative purposes only; analyses examined stress and symptoms as continuous variables.

difficulty maintaining attention toward positive interpersonal events may be at risk for depression in an environment in which social rewards are further limited, which is consistent with evidence of stronger effects for reduced reactivity for positive compared with negative stimuli in depression (44,74). It is also possible that individuals with reduced LPPs to interpersonal stimuli pre-pandemic tend to generate dependent interpersonal stressors during the pandemic, thus increasing risk for depressive symptoms (5,6,19).

Interestingly, a distinct pattern of results emerged for traumatic intrusions, such that enhanced LPPs to interpersonal stimuli predicted increased traumatic intrusions in combination with interpersonal events. This is consistent with prior evidence of reductions in emotional reactivity in depression but elevated reactivity in anxiety (33,37,75). These patterns were particularly apparent for LPPs to threatening stimuli, which is consistent with previous LPP and neuroimaging research on responses to stress in youth (47,49) and the broader literature

on threat hypervigilance in anxiety (33,37,38). It is important to note that LPP interaction effects were specific to interpersonal rather than total events, which could be explained by the selective use of interpersonal images in our task, with LPPs to interpersonal scenarios eliciting vulnerability primarily in the context of interpersonal stress.

Although we aimed to minimize the number of tests conducted to examine pathways to internalizing psychopathologies, our LPP results did not survive corrections for multiple comparisons, and replication is needed in a larger sample. Though the results showed small to medium effect sizes, there is increasing recognition that small effects are typical in research on complex psychological processes, such as the development of psychopathology (76). The lack of nonsocial emotional image conditions to directly test the specificity of the effects to interpersonal stimuli is a limitation of this study. However, previous ERP research directly comparing LPPs to social and nonsocial neutral images shows enhanced LPPs to

neutral images containing people (56), supporting the potential of this specificity. Other limitations include the susceptibility of PSQ to subjective interpretations of events compared with stress interview measures (7). However, by asking participants to first report the presence or absence of events before rating severity, this subjectivity is mitigated. Furthermore, although Inventory of Depression and Anxiety Symptoms permits a dimensional approach to assessing risk for internalizing psychopathologies, and we did observe high rates for symptoms above established clinical cutoffs, it is unclear whether our results would generalize to clinical samples based on diagnostic categories. Finally, we focused on ERP components indexing individual differences in attentional processing of emotional images, particularly the LPP, and consideration of the role of ERPs indexing conflict monitoring and other cognitive processes is needed in future work.

To our knowledge, this prospective study is among the first to evaluate neurophysiological predictors of the impact of COVID-19-related stress on internalizing symptoms in emerging adults. Our results support a stress sensitivity model, where the LPP may reflect vulnerabilities for internalizing symptoms when exposure to interpersonal stressors is high. Other research using psychophysiological measures, such as pupillometry (77), to assess alterations in emotion further support the possible clinical applications of these methods for identifying those at greatest risk during times of crisis. Notably, our findings also demonstrate the sensitivity of neurophysiological measures of emotion for distinguishing between risk for depression and trauma-related anxiety, with the potential for informing more personalized prevention efforts.

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ARTICLE INFORMATION

From the Department of Psychology and Human Development (LD, SP, AK), Vanderbilt University, Nashville, Tennessee; Department of Psychiatry (MW), Pennsylvania State College of Medicine, Hershey, Pennsylvania; and Department of Psychology (HG), Western University, London, Ontario, Canada.

Address correspondence to Autumn Kujawa, Ph.D., at autumn.kujawa@vanderbilt.edu.

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