RESEARCH ARTICLE

ANXIETY AND DEPRESSION ASSOCIATION OF AMERICA WILEY

Neural markers of emotion regulation difficulties moderate effects of COVID-19 stressors on adolescent depression

Resh S. Gupta[1,2] | Lindsay Dickey[3] | Autumn Kujawa[3]

[1]Department of Psychiatry, University of California San Diego, La Jolla, California, USA

[2]Sam and Rose Stein Institute for Research on Aging, University of California San Diego, La Jolla, California, USA

[3]Department of Psychology and Human Development, Vanderbilt University, Nashville, Tennessee, USA

Correspondence

Autumn Kujawa, Department of Psychology and Human Development, Vanderbilt University, Peabody College #552, 230 Appleton Pl, Nashville, TN 37203-5721, USA.
Email: autumn.kujawa@vanderbilt.edu

Abstract

Background: Stressful events, such as those imposed by the COVID-19 pandemic, are associated with depression risk, raising questions about processes that make some people more susceptible to the effects of stress on mental health than others. Emotion regulation may be a key process, but methods for objectively measuring emotion regulation abilities in youth are limited. We leveraged event-related potential (ERP) measures and a longitudinal study of adolescents oversampled for depression and depression risk to examine emotion regulation difficulties as prospective predictors of depressive symptoms in response to pandemic-related stress.

Methods: Before the pandemic, adolescents with ($n = 28$) and without ($n = 34$) clinical depression ($N = 62$ total) completed an explicit emotion regulation task while ERP data were recorded and measures of depressive symptoms. Adolescents were re-contacted during the pandemic to report on COVID-19 related stressful events and depressive symptoms ($n = 48$).

Results: Adolescents who had never experienced a depressive episode showed an increase in depressive symptoms during the pandemic, but adolescents who were clinically depressed before the pandemic did not exhibit significant changes in symptoms. Neural markers of emotion regulation abilities interacted with pandemic-related stressful events to predict depressive symptoms during the pandemic, such that stressors predicted increases in depressive symptoms only for adolescents with greater difficulty modulating responses to negative images before the pandemic.

Conclusions: Results provide insight into adolescent mental health during the COVID-19 pandemic and highlight the role of emotion regulatory brain function in risk and resilience for depression.

KEYWORDS

COVID-19 pandemic, depression, emotion regulation, event-related potentials, stress

1 | INTRODUCTION

Depression is a prevalent and impairing disorder often emerging in adolescence (Kessler et al., 2005). Stressful events are known to precede the onset of depression (Hammen, 2005), but many people experience stressful events without developing depression, raising questions about factors that shape susceptibility to stress.

The ability to adaptively respond to and regulate negative emotions, particularly in the context of stressful events, is critical for mental health, and maladaptive emotion regulation has been shown to play a key role in depression (Joormann & Stanton, 2016). Further, there is some evidence that difficulty with emotion regulation moderates effects of stress on depression (Troy et al., 2010).

Emotion regulation is often measured at the self-report level, but questionnaires require insight into emotions and regulation strategies and query general tendencies, regardless of type of emotion or context (Gross & John, 2003). Neural measures offer real time, objective measures of emotion regulation that can assess individuals' ability to regulate dysphoric emotions, including sadness, anhedonia, and distress, which are core features of depression (Kovacs & Yaroslavsky, 2014). Meta-analyses indicate that emotion regulation activates cognitive control circuits, including dorsomedial, dorsolateral, and ventrolateral prefrontal cortex, anterior cingulate cortex, and temporal cortex, and downregulates activation of the amygdala and parahippocampal gyrus (Buhle et al., 2014; Frank et al., 2014).

Event-related potentials (ERPs) derived from the electroencephalogram (EEG) are particularly well-suited neural measures for examining the temporal dynamics of emotion regulation. In particular, the late positive potential (LPP) is a sustained positivity in the ERP waveform that begins around 300 ms after stimulus onset, is enhanced for salient stimuli compared to neutral stimuli, and extends over centroparietal and frontal electrode sites in emotion regulation tasks (Moran et al., 2013; Moser et al., 2014). The LPP is modulated by the meaning of the stimulus (MacNamara et al., 2009) and efforts to regulate emotions through techniques like cognitive reappraisal (Hajcak & Nieuwenhuis, 2006; Krompinger et al., 2008). Cognitive reappraisal involves changing interpretations of a negative stimulus to more neutral or pleasant thoughts (Gross, 1998) and is associated with positive outcomes, including lower depression and reduced stress reactivity (Carlson et al., 2012; Troy et al., 2010). The LPP elicited to emotional stimuli has been shown to be decreased when participants are instructed to use regulation strategies like cognitive reappraisal, although the timing and topographical distributions of these patterns vary across studies (e.g., Fitzgerald et al., 2016; Shushakova et al., 2018).

In addition to insights into typical emotion regulatory processes, the LPP has also shown utility in elucidating patterns of emotionality in depression. Several studies have indicated that the LPP is blunted to emotional stimuli in those with elevated depressive symptoms or diagnoses (Proudfit et al., 2015). Additionally, alterations in the LPP have been observed in youth at risk for depression before the development of symptoms (Kujawa et al., 2012; Nelson et al., 2015), but little work has examined associations between depression and the LPP in explicit emotion regulation tasks. The use of emotion regulation tasks with objective measures like the LPP may be particularly useful for translating neuroscience to intervention because they allow for a direct examination of the effects of specific skills training on a neural response.

The COVID-19 pandemic has been a time of widespread stress and increases in depression prevalence in adolescents (Racine et al., 2021). There is a need to better understand the specific types of stressful events youth have experienced, how these events contribute to depression risk, and individual differences that make some youth particularly vulnerable to stress effects on mental health. Research on widespread stressors impacting large groups of people may advance more general understanding of processes that make some people more susceptible to the effects of stress on mental health than others. Longitudinal research objectively measuring pre-existing individual differences and testing prospective predictors of later responses to stress provides insights into vulnerability-stress models of psychopathology and intervention targets.

The current study leveraged a study of adolescents oversampled for clinical depression and depression risk to test neural markers of emotion regulation ability as a predictor of depressive symptoms during the COVID-19 pandemic. Participants completed an emotion regulation task and depressive symptom measure pre-pandemic. We developed a COVID-19 stress measure for adolescents (adapted from Kujawa et al., 2020), and participants were re-assessed early in the pandemic to measure stressful events and symptoms. Goals were to examine endorsement of COVID-19 related stressful events in adolescents, examine changes in symptoms of depression from pre- to during the pandemic in adolescents with and without clinical depression pre-pandemic, and test neural measures of emotion regulation as prospective predictors of depression during the pandemic as main effects and interacting with stress exposure. We hypothesized that depression would increase overall from pre- to during the pandemic, and that greater difficulties modulating the LPP would be associated with a greater increase in depressive symptoms. Further, we hypothesized that neural markers of emotion regulation would moderate the effects of COVID-19-related stress on symptoms, such that stronger effects of stress on depressive symptoms would be observed for adolescents with difficulty modulating emotional responses pre-pandemic.

2 | METHODS

2.1 | Participants

Three groups of adolescents aged 14–17 were enrolled pre-pandemic as part of a larger study: adolescents with current depression, adolescents with no history of depression but at high risk based on maternal history of depression, and adolescents with no history of depression at relatively low risk due to no maternal history of depression. Sixty-two participants (46 females) completed assessments before the pandemic; at baseline, 28 participants had clinical depression diagnoses and 34 had no lifetime history of depression (15 high risk based on maternal depression and 19 relatively low risk). Mean age was 15.10 years (SD = 1.07) at baseline; 3.23% Hispanic/Latinx, 72.58% White/Caucasian, 11.29% Black/African American, 3.23% Asian, 1.61% American Indian/Alaska Native, 1.61% Native Hawaiian/Pacific Islander, and 8.06% identified as another race. One participant did not report race/ethnicity. Of these 62 participants, 3 did not complete the EEG and 6 were excluded for noisy EEG data. One participant had noisy EEG data at frontal, but not centroparietal, sites and was thus excluded from analyses using frontal electrodes. Fifty-two participants were included in the within-subjects EEG analyses, and 48 participants completed the COVID-19 follow-up assessment in April-May 2020 and were included in the frequencies of COVID-19 stressful events (40 participants who completed the follow-up assessment had usable frontal EEG data).

2.2 | Procedures

The Vanderbilt University Institutional Review Board approved this study. Informed consent was obtained from parents and assent from minor participants. Following consent/assent, participants and biological mothers were interviewed using the mood disorders module of the Structured Clinical Interview for DSM-5 Disorders (SCID; First et al., 2016) to determine diagnoses for mothers and the DSM-5 version of the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children (KSADS; Kaufman et al., 2016) for adolescent diagnoses (see Supporting Information). Next, participants completed questionnaires assessing baseline depressive symptoms and an EEG assessment including the emotion regulation task in a counterbalanced order with other tasks. Results of other tasks will be presented separately (e.g., Pegg et al., 2020). In April 2020, participants were contacted by email to complete the same depressive symptom measure and a questionnaire assessing exposure to stressful events due to the pandemic. Participants were compensated financially for completing each assessment. The mean time between baseline and follow-up assessments was 244.78 days (SD = 116.76).

2.3 | Measures

2.3.1 | Emotion regulation task

EEG was recorded continuously while participants completed the emotion regulation task adapted from Moser et al. (2014). The task included 25 negative images selected to elicit dysphoric emotions (e.g., crying or mourning people), consistent with prior research (Kudinova et al., 2016), and 25 neutral images in a pseudorandom order. Images were acquired from the International Affective Picture System (IAPS; Lang et al., 2008; details in Supporting Information). Negative images were presented twice, once with instructions to look at the images and respond naturally, and once with instructions to decrease emotional responses to the images. Before starting the task, participants were provided with examples of how to modulate their emotional responses to the images on decrease trials through strategies like cognitive reappraisal and completed a practice block of four trials. On each trial, the instructions "look neutral," "look negative," or "decrease negative" appeared for 2 s, followed by a fixation cross for 500 ms and an image for 6 s. A fixation cross was again presented for 2.5 s, and participants were then asked to rate the intensity of their reaction to the stimulus using a scale of 0 (none) to 7 (very strong; see Supporting Information).

2.3.2 | EEG data collection and processing

EEG data were recorded using a 32-channel BrainProducts acti-CHamp System and BrainVision Recorder software with a 1000 Hz sampling rate and impedances below 30 kΩ. Facial electrodes were attached approximately 1 cm above and below one eye and 1 cm on the outer corner of eac eyes to measure electrooculogram. EEG data were processed offline using BrainVision Analyzer software, filtered from 0.01 to 30 Hz, and re-referenced to the linked mastoids. Data were segmented from −200 ms before to 6000 ms after stimulus onset. Data were corrected for eye movements (Gratton et al., 1983), and artifacts were removed using semi-automated procedures, with the following criteria: maximal allowed voltage step: 40 μV/ms; maximal allowed difference of values in intervals: 150 μV (interval length: 500 ms); minimal allowed amplitude: −200 μV; maximal allowed amplitude: 200 μV; and lowest allowed activity in intervals: 0.5 μV (interval length: 100 ms). Additional artifacts were identified using visual inspection and removed.

Average ERPs were computed for each condition and baseline corrected to 200 ms preceding stimulus onset. We focused on "look negative" and "decrease negative" trials because we were interested in neural responses during explicit emotion regulation. The LPP is sustained across time and tends to change from more centroparietal to frontal sites at later stages of processing. To quantify emotion regulation effects on the LPP, we first conducted repeated-measures ANOVAs across two regions (frontal electrode sites: F3, F4, and Fz; centroparietal electrode sites: CP1, CP2, P3, P4, and Pz) and three time windows (400–1000, 1000–3500, and 3500–6000 ms; similar to Moser et al., 2014) to determine the timing and scalp distribution of emotion regulation effects in the overall sample (see Supporting Information). The LPP was reduced overall on decrease relative to look trials 3500–6000 ms after stimulus onset over frontal sites, and this scoring was used to examine predictors of symptom change. To isolate variability in the ERP wave attributed to emotion regulation, unstandardized residual scores were calculated predicting LPP decrease negative amplitudes from LPP look negative amplitudes (Meyer et al., 2017; referred to as LPP decrease residuals). More negative LPP decrease residuals indicate a reduction in neural responses when regulating emotions.

Split-half reliability of the LPP was generally acceptable to good across windows (frontal: .66–.81; centroparietal: .60–.82), although the late frontal LPP for the look negative condition was more borderline at .51. To investigate this, we examined split-half reliability at the frontal electrodes separately in this window and it was acceptable to good for Fz and F4 (.62–.74) but poor for F3 look negative (.34) which appeared to be due to the impacts of ocular artifacts on this channel. We repeated analyses with a pooling of Fz/F4 as a more reliable measure of LPP and results were generally consistent with the original results (see Supporting Information).

2.3.3 | COVID-19 stressful events

Participants completed an adolescent version of the Pandemic Stress Questionnaire (PSQ; adapted from Kujawa et al., 2020; full measure in Supporting Information), a 22-item self-report measure of exposure to events due to the COVID-19 pandemic. Participants responded "yes/no" to indicate whether they had experienced each

event, followed by a perceived severity rating from 1 to 5 for endorsed events. Only the total number of endorsed events was analyzed in the present study. We previously presented data on test–retest reliability of the PSQ and correlations with established measures (Kujawa et al., 2020).

2.3.4 | Depressive symptoms

Depressive symptoms were measured using the Mood and Feelings Questionnaire (MFQ; Angold et al., 1995) administered at baseline and then again in the follow-up questionnaires. The self-report 33-item MFQ assesses depressive symptoms in the past 2 weeks using a 3-point Likert scale. The MFQ had good internal consistency at baseline ($n = 61$, $\alpha = .95$) and follow-up ($n = 48$, $\alpha = .95$).

2.4 | Data analysis

First, we examined frequencies of COVID-19 stressful events ($n = 48$). Next, given the possibility that distinct patterns of symptom change may be observed for those who were symptomatic at baseline relative to those who were not, a linear mixed-effects model with a random intercept for subject was conducted in Matlab R2021a to determine the effects of group (clinically depressed vs. never depressed) and time (baseline vs. follow-up) on depressive symptoms ($n = 62$). Subsequent paired-samples t-tests were performed to examine symptom changes in each group (clinically depressed $n = 28$, never depressed $n = 34$), with restricted maximum likelihood to

estimate missing data using lme4 in R (Bates et al., 2015). Finally, we examined LPP decrease residuals and the interaction with COVID-19 stressful events as predictors of depressive symptoms at follow-up in multiple regression analyses using lavaan in R (Rosseel, 2012) with full information maximum likelihood to handle missing data (Enders, 2013; $n = 62$). Time from baseline to follow-up, baseline depressive symptoms, age, and gender were included as covariates.

3 | RESULTS

3.1 | Frequencies of COVID-19-related stressful events

Frequencies of endorsed events are shown in Figure 1. Participants reported an average of 4.5 total PSQ events ($SD = 2.58$; range = 1–12), with canceling or postponing important events, canceling travel, inability to be with close family and friends, and conflicts and arguments with family members due to the pandemic particularly common.

3.2 | Change in depressive symptoms during the pandemic

Descriptive statistics are presented in Table 1. Baseline depressive symptoms did not differ from follow-up depressive symptoms in the overall sample, $t(50.24) = 0.21$, $p = .84$. Linear mixed-effects analyses indicated that the interaction between group (clinically depressed vs.
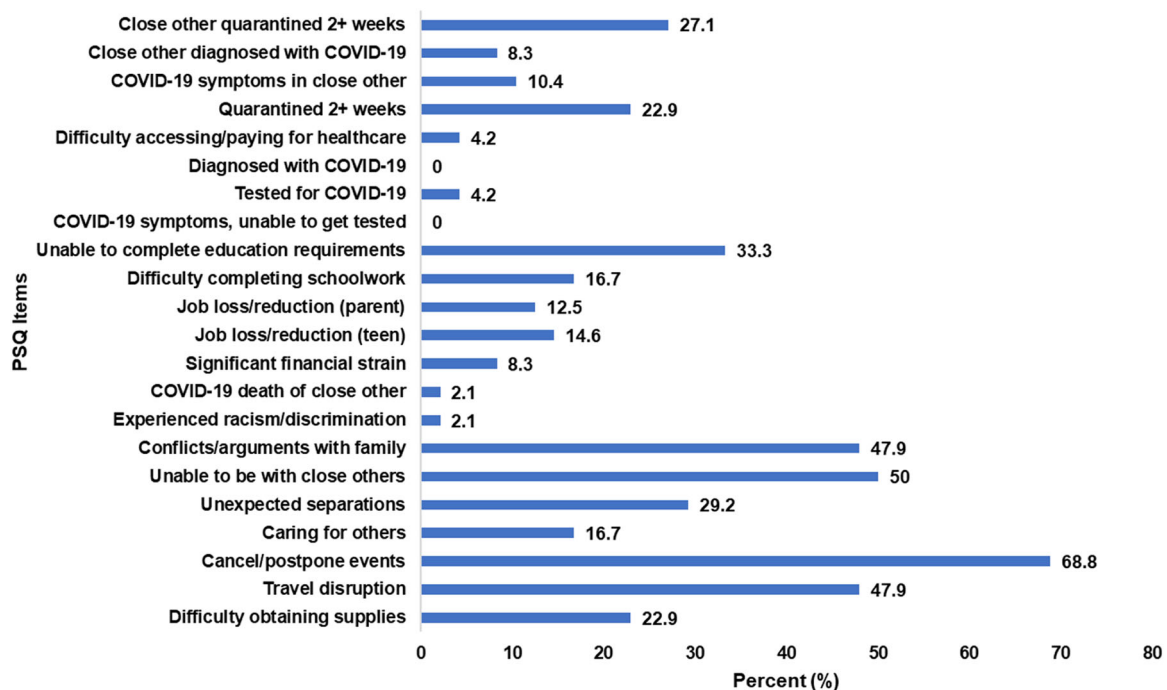
FIGURE 1 Frequency of exposure to events assessed by the Pandemic Stress Questionnaire (PSQ) in April and May 2020.

TABLE 1 Descriptive statistics and bivariate correlations among primary study variables.

	M (SD)	1	2	3	4
1. LPP decrease negative	4.50 (8.89)	–			
2. LPP look negative	6.59 (7.65)	.70***	–		
3. Depressive symptoms (baseline)	18.20 (14.57)	.03	.19	–	
4. Depressive symptoms (follow-up)	18.69 (14.48)	.27	.31	.68***	–
5. COVID-19 stressful events	4.50 (2.58)	.17	−.01	.32*	.45**

Abbreviation: LPP, late positive potential.

*p < .05.; **p < .01.; ***p < .001.

never depressed) and time on depressive symptoms was significant (b = 8.34, SE = 3.11, t(105) = 2.68, p < .01). In the depressed group, depressive symptoms during the pandemic (M = 24.15, SD = 16.34) did not significantly differ from baseline (M = 27.86, SD = 14.15, t (21.13) = −1.58, p = .13, d = .15). In the never depressed group, depressive symptoms during the pandemic (M = 14.79, SD = 11.80) increased from baseline (M = 10.00, SD = 8.83, t(30.02) = 2.27, p = .03, d = .27).

3.3 | Emotion regulation, stress, and depressive symptom change

ERPs depicting emotion regulation effects in the overall sample are shown in Figure 2, and bivariate correlations are presented in Table 1. Results from multiple regression analyses examining predictors of depressive symptoms during the pandemic are presented in Table 2. The interaction between LPP decrease residuals and COVID-19 stressful events was significant and interpreted with the web utility developed by Preacher et al. (2006). The effect of stressful events on depressive symptoms was significant for LPP decrease residuals one standard deviation above the mean (b = 2.92, SE = 1.04, t = 2.80, p < .01), but not for LPP decrease residuals at the mean (b = 1.15, SE = .64, t = 1.80, p = .08) or one standard deviation below the mean (b = −.62, SE = .84, t = −.74, p = .46). Specifically, the effect of COVID-19 stress on depressive symptoms was significant for LPP decrease residuals greater than .58 based on procedures in Johnson and Neyman (1936) (Figure 3). A scatterplot of the association is presented in the Supporting Information.

The regression interaction model was tested separately in the clinically depressed and never depressed subsets of the sample to determine whether a similar pattern was observed in each of these groups. The interaction was significant in both the clinically depressed (b = .42, SE = .20, p = .04) and never depressed groups (b = .28, SE = .12, p = .02). The regression model for the never depressed group was also tested with maternal depression history (low risk vs. high risk) as a covariate and the interaction remained significant (b = .28, SE = .12, p = .02).

4 | DISCUSSION

The current study characterized adolescent experiences of COVID-19-related stressful events early in the pandemic, examined changes in symptoms of depression from pre- to during the pandemic in adolescents with and without clinical depression pre-pandemic, and tested a neural measure of emotion regulation ability as a prospective predictor of follow-up depressive symptoms as a main effect and interacting with stress exposure. Youth endorsed experiencing multiple stressful events, and we observed distinct patterns of symptom change for adolescents with clinical depression pre-pandemic compared to those with no prior history of depression. Stress was associated with increases in depressive symptoms only for adolescents with high LPP decrease residuals at baseline, reflecting difficulty modulating responses using emotion regulation strategies like reappraisal. This is consistent with vulnerability-stress models in that emotion regulation difficulties may be an underlying vulnerability that is activated in the context of stress and leads to increased depression risk.

Youth reported multiple COVID-19 stressful events in spring 2020, including canceling important events and inability to be with loved ones and family members due to the pandemic, which were also associated with depressive symptoms. Patterns of hange in symptoms during the pandemic depended on depression status pre-pandemic. For those who had not experienced depression pre-pandemic, depressive symptoms increased, consistent with prior research (Racine et al., 2021). However, adolescents who were depressed pre-pandemic did not exhibit an increase in symptoms. Some depressed adolescents may have been in remission when re-assessed, leading to less of an overall increase in depression. Stable symptoms in the clinically depressed group may also reflect failure to remit or recurrence of depression due in part to the pandemic. In addition, depressed adolescents may have been experiencing greater academic and interpersonal stress pre-pandemic (Field et al., 2001), and restrictions may have temporarily mitigated some of these stressors. Consistent with the possibility of the pandemic buffering against interpersonal strain, we previously observed a reduction in social anxiety in college students during the pandemic (Dickey et al., 2021).

Consistent with hypotheses, pandemic-related stress predicted increases in depressive symptoms only for adolescents with relatively high LPP decrease residuals at baseline, reflecting more difficulty modulating emotional responses using strategies like reappraisal. To our knowledge, this is the first study to examine objective ERP markers of emotion regulation abilities as a prospective predictor of depressive symptoms, highlighting the potential utility of these methods. Prior research has shown that LPP alterations when viewing emotional images are associated with depression (Proudfit et al., 2015), and the present findings of emotion regulation-related LPP alterations further support and corroborate the LPP as a potential target for intervention and prevention. Somewhat surprisingly, we did not observe an overall correlation between the LPP and depressive symptoms, but this is consistent with prior evidence that
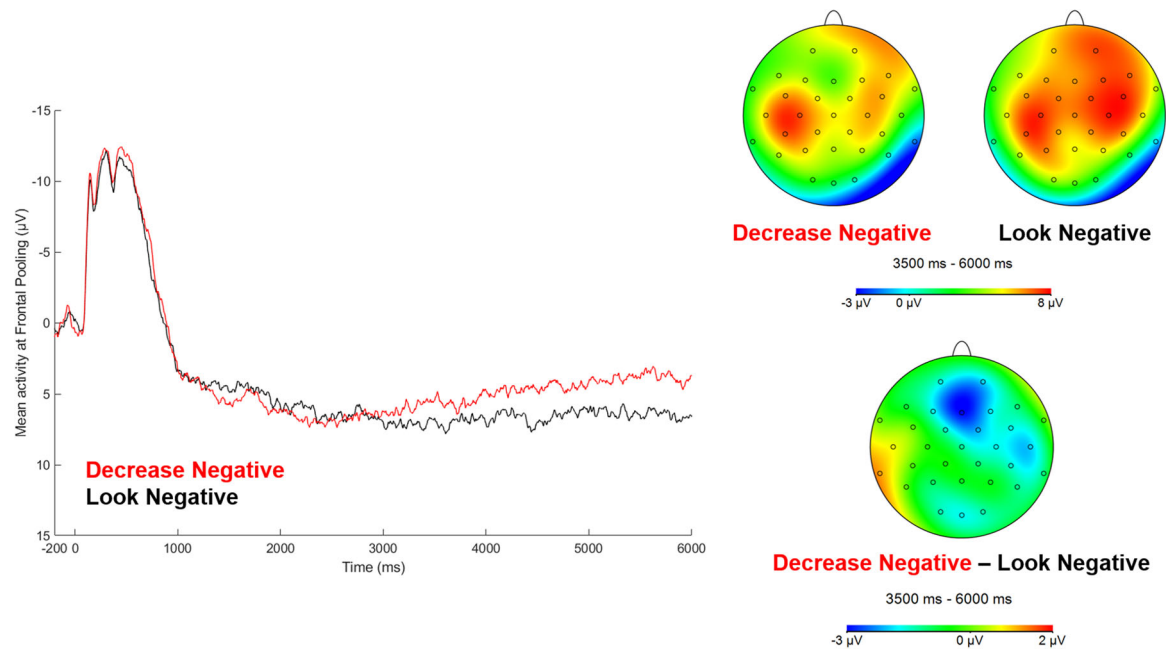
FIGURE 2 Decrease negative and look negative ERP waveforms pooled at frontal electrodes Fz, F3, and F4 (left) and scalp distributions for decrease negative, look negative, and decrease negative minus look negative conditions 3500–6000 ms poststimulus in the overall sample (right). ERP, event-related potential.

TABLE 2 Multiple regression analysis testing the main and interactive effect of LPP decrease residuals and COVID-19 stressful events predicting depressive symptoms during the COVID-19 pandemic.

	b (SE)	β	Partial R²
Step 1			
Age	−1.13 (1.40)	−.09	.003
Gender	2.76 (3.82)	.09	.008
Time from baseline to follow-up	0.02 (0.01)	.18^	.087
Depressive symptoms (baseline)	0.60 (0.10)	.61***	.436
LPP decrease residuals	0.26 (0.26)	.12	.003
COVID-19 stressful events	0.81 (0.66)	.15	.017
Step 2			
LPP decrease residuals × stress	0.28 (0.11)	.61*	.061

Abbreviation: LPP, late positive potential.

^$p < .10.$

*$p < .05.$; ***$p < .001.$

the LPP may reflect a vulnerability for later psychopathology in combination with other risk factors, like acute stress (Kujawa et al., 2016).

Surprisingly, significant emotion regulation effects on the LPP were only apparent in the 3500–6000 ms window over frontal sites. Although the LPP is typically maximal over centroparietal sites in emotion reactivity tasks with shorter stimulus durations, emotion regulation-related LPP modulation has also been observed over frontal sites in prior studies (Moser et al., 2014; Shushakova et al., 2018). Although the scalp distribution of ERPs does not directly correspond with activation of specific brain regions, some research in adults suggests that the LPP over frontal sites is enhanced overall when attempting to reappraise emotional stimuli, potentially due to increased cognitive control (Moser et al., 2014). The current results indicate that in adolescents, more adaptive emotion regulation is characterized by relative reductions in the LPP over frontal sites at later stages of processing, potentially reflecting reductions in the perceived salience of or attentional allocation to the stimulus.

In terms of limitations, a relatively small sample completed all assessments, but the sample is unique in terms of the proportion with clinical depression pre-pandemic and we accounted for missing data in analyses. Some of the LPP variables exhibited relatively low split-half reliability, potentially due in part to the young sample, high rates of depression, and/or variability in responses to images, and more trials may be needed in future research on emotion regulation tasks across development. A strength is that this is one of the first longitudinal studies of responses to COVID-19-related stress using neural measures and one of the first to examine ERP markers of emotion regulation abilities as prospective predictors of depressive symptoms. Although replication in larger longitudinal samples is needed, the results of the current study offer unique insights into the role of emotion regulation difficulties in vulnerability-stress pathways to psychopathology and tools for objectively assessing emotion regulation.
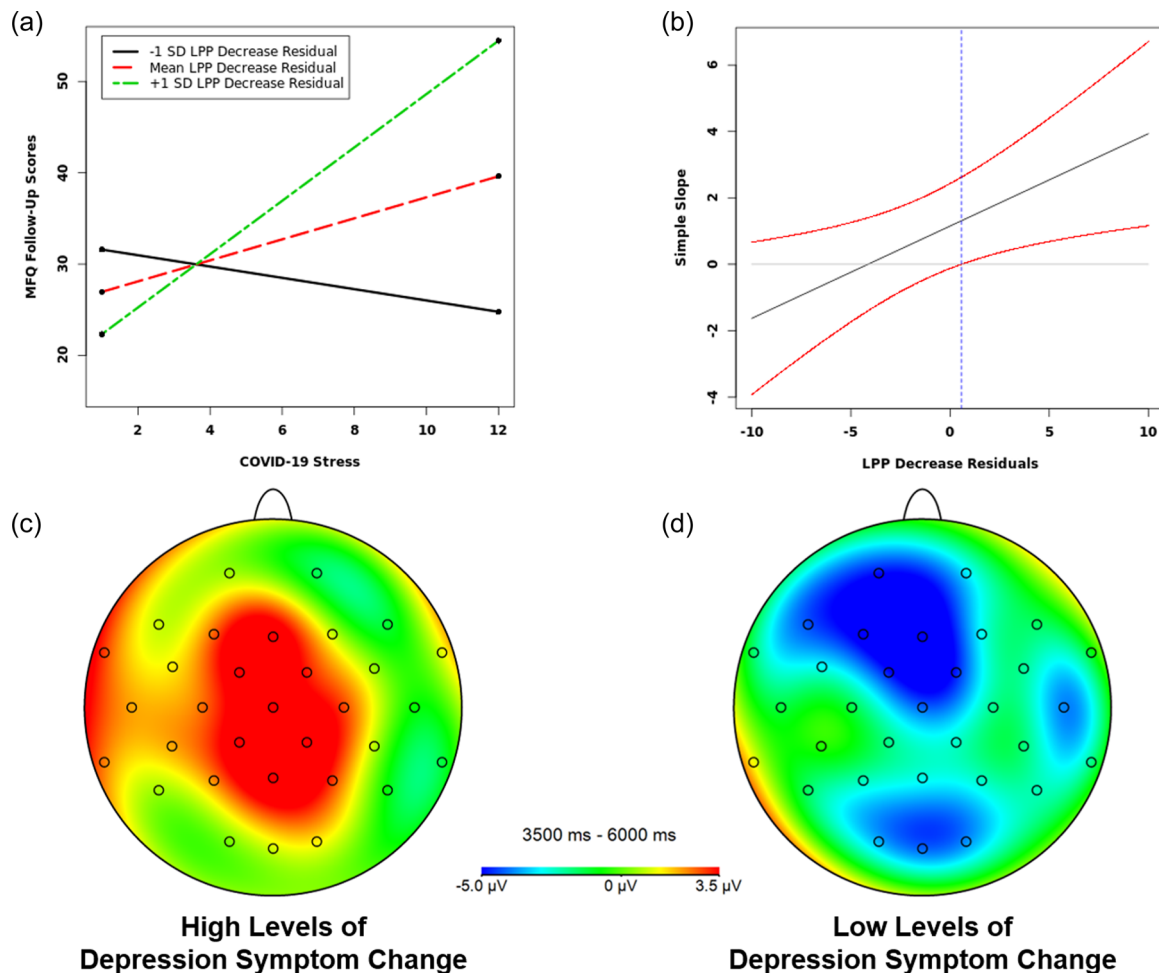
FIGURE 3 (a) Simple slopes plot for the interaction effect between COVID-19 stress at −1SD, mean, and +1SD LPP decrease residuals in the prediction of follow-up depressive symptoms. (b) Region of significance and confidence bands for the simple slope of the association between COVID-19 stress and follow-up depressive symptoms as a function of LPP decrease residuals. (c, d) Scalp distributions depicting the responses to decrease negative minus look negative trials for adolescents above the mean for COVID-19 stress exposure who showed relatively high (c) and low (d) levels of depression symptom changes. *Note*: Median splits of symptom changes were used for illustrative purposes, but analyses used continuous measures.

5 | CONCLUSION

The present study indicates distinct patterns of depression symptom change for adolescents with pre-pandemic clinical depression versus those without. Additionally, neural markers of emotion regulation interacted with stressful events such that pandemic-related stress predicted increases in depression only for adolescents with greater difficulty modulating emotional responses pre-pandemic. These results provide insight into adolescent mental health during the COVID-19 pandemic, with general implications for the role of emotion regulatory brain function in risk and resilience.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data are available by email request to the corresponding author.

ORCID

Resh S. Gupta https://orcid.org/0000-0003-2337-5473
Autumn Kujawa http://orcid.org/0000-0002-0560-4589

REFERENCES

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). Development of a short questionnaire for use in

epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*, 5, 237–249.

Bates, D., Mächler, M., Bolker, B. M., & Walker, S. C. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, 67(1), https://doi.org/10.18637/jss.v067.i01

Buhle, J. T., Silvers, J. A., Wager, T. D., Lopez, R., Onyemekwu, C., Kober, H., Weber, J., & Ochsner, K. N. (2014). Cognitive reappraisal of emotion: A meta-analysis of human neuroimaging studies. *Cerebral Cortex*, 24(11), 2981–2990.

Carlson, J. M., Dikecligil, G. N., Greenberg, T., & Mujica-Parodi, L. R. (2012). Trait reappraisal is associated with resilience to acute psychological stress. *Journal of Research in Personality*, 46(5), 609–613.

Dickey, L., West, M., Pegg, S., Green, H., & Kujawa, A. (2021). Neurophysiological responses to interpersonal emotional images prospectively predict the impact of COVID-19 pandemic-related stress on internalizing symptoms. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 6(9), 887–897. https://doi.org/10.1016/J.BPSC.2021.03.004

Enders, C. K. (2013). Dealing with missing data in developmental research. *Child Development Perspectives*, 7(1), 27–31. https://doi.org/10.1111/CDEP.12008

Field, T., Miguel, D., & Sanders, C. (2001). Adolescent depression and risk factors. *Adolescence*, 36(143), 491–498.

First, M. B., Williams, J. B., Karg, R. S., & Spitzer, R. L. (2016). *Structured clinical interview for DSM-5 disorders, clinician version (SCID-5-CV)*. American Psychiatric Association.

Fitzgerald, J. M., MacNamara, A., DiGangi, J. A., Kennedy, A. E., Rabinak, C. A., Patwell, R., & Phan, K. L. (2016). An electrocortical investigation of voluntary emotion regulation in combat-related posttraumatic stress disorder. *Psychiatry Research: Neuroimaging*, 249, 113–121. https://doi.org/10.1016/j.pscychresns.2015.12.001

Frank, D. W., Dewitt, M., Hudgens-Haney, M., Schaeffer, D. J., Ball, B. H., Schwarz, N. F., & Sabatinelli, D. (2014). Emotion regulation: Quantitative meta-analysis of functional activation and deactivation. *Neuroscience & Biobehavioral Reviews*, 45, 202–211.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, 55(4), 468–484.

Gross, J. J. (1998). Antecedent-and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74(1), 224–237.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85(2), 348–362. https://doi.org/10.1037/0022-3514.85.2.348

Hajcak, G., & Nieuwenhuis, S. (2006). Reappraisal modulates the electrocortical response to unpleasant pictures. *Cognitive, Affective and Behavioral Neuroscience*, 6(4), 291–297. https://doi.org/10.3758/CABN.6.4.291

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology*, 1(1), 293–319. https://doi.org/10.1146/annurev.clinpsy.1.102803.143938

Johnson, P. O., & Neyman, J. (1936). Tests of certain linear hypotheses and their application to some educational problems. *Statistical Research Memoirs*, 1, 57–93.

Joormann, J., & Stanton, C. H. (2016). Examining emotion regulation in depression: A review and future directions. *Behaviour Research and Therapy*, 86, 35–49. https://doi.org/10.1016/J.BRAT.2016.07.007

Kaufman, J., Birmaher, B., Axelson, D., Perepletchikova, F., Brent, D., & Ryan, N. (2016). *The schedule for affective disorders and schizophrenia—Present and lifetime version for DSM-5*.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(June), 593–602. https://doi.org/10.1001/archpsyc.62.6.593

Kovacs, M., & Yaroslavsky, I. (2014). Practitioner review: Dysphoria and its regulation in child and adolescent depression. *Journal of Child Psychology and Psychiatry*, 55(7), 741–757. https://doi.org/10.1111/JCPP.12172

Krompinger, J. W., Moser, J. S., & Simons, R. F. (2008). Modulations of the electrophysiological response to pleasant stimuli by cognitive reappraisal. *Emotion*, 8(1), 132–137. https://doi.org/10.1037/1528-3542.8.1.132

Kudinova, A. Y., Owens, M., Burkhouse, K. L., Barretto, K. M., Bonanno, G. A., & Gibb, B. E. (2016). Differences in emotion modulation using cognitive reappraisal in individuals with and without suicidal ideation: An ERP study. *Cognition and Emotion*, 30(5), 999–1007. https://doi.org/10.1080/02699931.2015.1036841

Kujawa, A., Green, H., Compas, B. E., Dickey, L., & Pegg, S. (2020). Exposure to COVID-19 pandemic stress: Associations with depression and anxiety in emerging adults in the United States. *Depression and Anxiety*, 37(12), 1280–1288. https://doi.org/10.1002/da.23109

Kujawa, A., Hajcak, G., Danzig, A. P., Black, S. R., Bromet, E. J., Carlson, G. A., & Klein, D. N. (2016). Neural reactivity to emotional stimuli prospectively predicts the impact of a natural disaster on psychiatric symptoms in children. *Biological Psychiatry*, 80(5), 381–389. https://doi.org/10.1016/j.biopsych.2015.09.008

Kujawa, A., Hajcak, G., Torpey, D., Kim, J., & Klein, D. N. (2012). Electrocortical reactivity to emotional faces in young children and associations with maternal and paternal depression. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 53(2), 207–215. https://doi.org/10.1111/J.1469-7610.2011.02461.X

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instructional manual*. University of Florida.

MacNamara, A., Foti, D., & Hajcak, G. (2009). Tell me about it: Neural activity elicited by emotional pictures and preceding descriptions. *Emotion*, 9(4), 531–543. https://doi.org/10.1037/A0016251

Meyer, A., Lerner, M. D., De Los Reyes, A., Laird, R. D., & Hajcak, G. (2017). Considering ERP difference scores as individual difference measures: Issues with subtraction and alternative approaches. *Psychophysiology*, 54(1), 114–122. https://doi.org/10.1111/psyp.12664

Moran, T. P., Jendrusina, A. A., & Moser, J. S. (2013). The psychometric properties of the late positive potential during emotion processing and regulation. *Brain Research*, 1516, 66–75. https://doi.org/10.1016/J.BRAINRES.2013.04.018

Moser, J. S., Hartwig, R., Moran, T. P., Jendrusina, A. A., & Kross, E. (2014). Neural markers of positive reappraisal and their associations with trait reappraisal and worry. *Journal of Abnormal Psychology*, 123(1), https://doi.org/10.1037/a0035817

Nelson, B. D., Perlman, G., Hajcak, G., Klein, D. N., & Kotov, R. (2015). Familial risk for distress and fear disorders and emotional reactivity in adolescence: An event-related potential investigation. *Psychological Medicine*, 45(12), 2545–2556. https://doi.org/10.1017/S0033291715000471

Pegg, S., Dickey, L., Green, H., & Kujawa, A. (2020). Differentiating clinically depressed adolescents with and without active suicidality: An examination of neurophysiological and self-report measures of reward responsiveness. *Depression and Anxiety*, 37(9), 876–884. https://doi.org/10.1002/da.23012

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics*, 31(4), 437–448.

Proudfit, G. H., Bress, J. N., Foti, D., Kujawa, A., & Klein, D. N. (2015). Depression and event-related potentials: Emotional disengagement and reward insensitivity. *Current Opinion in Psychology*, 4, 110–113. https://doi.org/10.1016/J.COPSYC.2014.12.018

Racine, N., McArthur, B. A., Cooke, J. E., Eirich, R., Zhu, J., & Madigan, S. (2021). Global prevalence of depressive and anxiety symptoms in

children and adolescents during COVID-19: a meta-analysis. *JAMA Pediatrics*, *175*(11), 1142–1150. https://doi.org/10.1001/JAMAPEDIATRICS.2021.2482

Rosseel, Y. (2012). lavaan: An R package for structural equation modeling and more Version 0.5-12 (BETA). *Journal of Statistical Software*, *48*(2), 1–36. http://cran.r-project.org

Shushakova, A., Ohrmann, P., & Pedersen, A. (2018). Exploring deficient emotion regulation in adult ADHD: Electrophysiological evidence. *European Archives of Psychiatry and Clinical Neuroscience*, *268*(4), 359–371. https://doi.org/10.1007/s00406-017-0826-6

Troy, A. S., Wilhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the silver lining: Cognitive reappraisal ability moderates the relationship between stress and depressive symptoms. *Emotion*, *10*(6), 783–795. https://doi.org/10.1037/A0020262

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.