The interplay of stress and electrocortical reactivity to reward in the prospective prediction of depression symptoms during COVID-19

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ABSTRACT

Rates of depression have increased during the novel coronavirus disease 2019 (COVID-19) pandemic, potentially due to associated stress exposure. However, it remains unclear which individuals are most susceptible. Electrocortical markers of reward processing, such as the reward positivity (RewP), are implicated in depression risk and may provide insights into who is most vulnerable to stress during the COVID-19 pandemic. The current study examined whether pre-pandemic neural correlates of reward reactivity (i.e., RewP) moderated the impact of social and financial stress on changes in youth and mother depression symptoms pre-to-post pandemic onset. Youth ($n = 45$) and mothers ($n = 45$) in the current sample were recruited prior to the COVID-19 pandemic as part of a larger study. RewP was assessed pre-pandemic, and depression symptoms were assessed pre- and post-pandemic onset for both youth and mothers. Additionally, social and financial chronic stress severity was assessed post-pandemic onset using a modified version of the UCLA Life Stress Interview. Financial stress was associated with prospective increases in depression for youth exhibiting blunted RewP at baseline. Similarly, family stress was associated with prospective increases in depression symptoms for mothers exhibiting blunted RewP at baseline. Findings suggest reduced reward responsiveness at the neural level may predispose both youth and mothers to future depression symptoms when exposed to higher levels of stress in the context of a pandemic.

1. Introduction

Though the novel coronavirus disease 2019 (COVID-19) pandemic is ongoing, and its long-term effects on mental health is uncertain, there is increasing evidence that the pandemic has resulted in an unprecedented mental health crisis (Pfefferbaum and North, 2020). Rates of depression, anxiety, and post-traumatic stress have significantly increased during the current pandemic among children, adolescents, and adults across the globe (de Miranda et al., 2020; Racine et al., 2020; Xiong et al., 2020). Nonetheless, epidemiological studies suggest that not all individuals exposed to stress will develop psychopathology or an enduring stress response, highlighting factors that may exacerbate or mitigate risk trajectories (Galatzer-Levy et al., 2018; Pfefferbaum and North, 2020). Identifying factors that increase risk for depression within the context of the current COVID-19 pandemic is vital to mitigate the pandemic's impact on mental health through targeted intervention efforts.

Unprecedented shutdowns, school closures, and public health mandates to mitigate COVID-19 spread (e.g., social distancing, stay-at-home orders) have undoubtedly contributed to increased social and financial strain for families. These elevated levels of stress likely augment risk for prospective increases in depressive symptoms for youth and their parents. Indeed, stress exposure is hypothesized to play a significant role in the etiology of depression (Grant et al., 2003; Hammen, 2005), and elevated levels of stress within both social (Hammen, 2003; Vrshek-Schallhorn et al., 2015) and financial (Mistry et al., 2009; Swift et al., 2020) domains have been shown to prospectively predict depression in both youth and adult samples. Yet, there are significant individual differences in susceptibility to the deleterious effects of stress exposure, underscoring the need to identify variables that moderate the impact of stress exposure on risk for depression.

Vulnerability-stress models of psychopathology suggest that stress in isolation may not increase risk for psychopathology, but rather, the interplay between stress exposure and preexisting vulnerabilities increases risk for psychopathology (Hankin and Abela, 2005). Individual

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differences in reward reactivity have been linked to the emergence of depression in youth (for review, see Kujawa and Burkhouse, 2017) and are consistently observed in depressed individuals (Admon and Pizzagalli, 2015; Proudfit et al., 2015). Utilizing event-related potentials (ERPs), derived from electroencephalogram (EEG), to capture individual differences in reward processing, researchers have identified a promising biomarker for depression. Specifically, the reward positivity (RewP), an ERP component occurring 250–350 ms following reward feedback, is a reliable index of reward responsiveness across development (Kujawa et al., 2020; Proudfit, 2015). Across several studies, an attenuated RewP is observed among youth and adults with major depressive disorder (Belden et al., 2016; Klawohn et al., 2020; Liu et al., 2014), prospectively predicts first onset of depression (Bress et al., 2013; Nelson et al., 2016), and appears to be relatively specific to vulnerability for depression, rather than anxiety (Burkhouse et al., 2017; Nelson et al., 2016).

Importantly, there is preliminary evidence that this biomarker exacerbates the impact of stress on depression symptoms. For example, greater stress exposure is associated with increased depressive symptoms, specifically for adolescents who exhibit a blunted RewP to social (Pegg et al., 2019) or monetary (Burani et al., 2019; Goldstein et al., 2020) reward. However, it is unclear whether RewP also moderates the impact of stress on prospective increases in depression symptoms in adults. Furthermore, although these previous studies have examined interactions between RewP and the occurrence of acute stressful life events or lifetime stress exposure, it is unknown if RewP also moderates the impact of chronic stress on prospective increases in depression symptoms. This is especially important to examine within the context of the COVID-19 pandemic, which has contributed to enduring, heightened levels of stress within several domains of functioning for many families.

Therefore, the current study aimed to replicate and extend these prior findings by examining RewP as a moderator of the impact of chronic stress severity across multiple domains of functioning on prospective increases in youth's and mothers' symptoms of depression during the COVID-19 pandemic. As part of a pre-existing study, a sample of 9–16 year-old youth and their mothers completed a reward processing task while ERPs (i.e., RewP) were recorded prior to COVID-19. In addition, youth and mothers completed self-report measures of depression symptoms. Following the onset of the COVID-19 pandemic, participants were invited to participate in an additional follow-up assessment involving depression questionnaires and an interviewer-based assessment of chronic life stress. We predicted that youth and mothers exposed to elevated levels of social (i.e., peer, mother-child, family) and financial chronic strain within the context of the current pandemic would show prospective increases in depression symptoms pre-to-post pandemic onset, and that these effects would be moderated by RewP. Specifically, given the relation between blunted RewP and depression risk (for reviews, see Kujawa et al., 2020; Proudfit, 2015), we expected that greater levels of stress would predict COVID-19-related increases in depressive symptoms among youth and mothers exhibiting a more attenuated RewP at baseline.

2. Method

2.1. Participants

Participants included 45 biological mother-child dyads initially recruited based on maternal history of major depressive disorder (MDD; present versus absent) prior to the COVID-19 pandemic as part of two larger studies on the intergenerational transmission of depression. The first study ($n = 15$) examined neural predictors of intergenerational depression transmission and the second study ($n = 30$) evaluated the impact of a preventative intervention. To be included in the MDD group, mothers were required to have a history of MDD. Exclusionary criteria across both studies for mothers and children were neurological disorders, traumatic brain injury, active suicidal ideation, lifetime history of

bipolar disorder, schizophrenia, or psychosis, or current alcohol and/or substance use disorder in the past 6 months. Mothers without a history of MDD were required to be lifetime free of any psychiatric disorder. Additional exclusionary criteria for youth included a lifetime history of MDD in the first study, and current MDD in the second study. Youth average age at the baseline assessment was 12.42 ($SD = 2.31$; Range = 9–16), and 82.2% were female. In terms of youth racial identity, 53.3% identified as White, 20.0% as Black, 11.1% as Asian, and 15.6% as multiracial or another race. Additionally, 24.4% of youth were Hispanic/Latinx. Mother average age at the baseline assessment was 42.09 ($SD = 6.44$; Range = 30–56). In terms of mother racial identity, 53.3% identified as White, 24.4% as Black, 11.1% as Asian, and 11.2% as multiracial or another race. Additionally, 25.0% of mothers identified as Hispanic/Latinx.

2.2. Clinical measures

Maternal current and lifetime history of DSM-5 psychiatric disorders were assessed at baseline using the Structured Clinical Interview for DSM–5 (First et al., 2015). Twenty mothers (44.4%) met diagnostic criteria for at least a single MDD episode.

Youth were administered the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) at both baseline and COVID-19 follow-up assessments to assess their depression symptoms. The CES-D demonstrated acceptable internal consistency across both assessments (αs = .78, .88).

Mothers were administered the Beck Depression Inventory-II (BDI-II; Beck et al., 1996) at both baseline and COVID-19 follow-up assessments to assess their depression symptoms. The BDI-II demonstrated good internal consistency across both assessments (αs = .88, 91).

2.3. Chronic stress

Youth and mother chronic stress exposure during the COVID-19 pandemic was assessed using a modified version of the UCLA Life Stress Interview (LSI). The LSI is semi-structured contextual threat interview that assesses chronic stress severity across a variety of domains. Specifically, the LSI probes for objective contextual information regarding functioning within specific domains, and interviewers assign a chronic stress severity rating on a scale of 1 ("superior functioning") to 5 ("severe stress"). To assess social chronic stress, youth were administered the peer, mother-child, and other-family (i.e., other household family members) sections of the UCLA LSI for Children (Adrian and Hammen, 1993) and mothers were administered the close friendships, mother-child, and other-family (i.e., family of origin, nuclear family excluding target child) sections of the UCLA LSI for Adults (Hammen et al., 1987). To reduce participant burden for mothers, the other-family domain was modified to include functioning in their relationship with a spouse or partner rather than assessing this domain separately. Additionally, mothers were administered the financial section of the LSI to assess familial financial chronic stress. Importantly, the LSI was modified for the current study to focus on overall levels of functioning during the pandemic, rather than across a prolonged window (e.g., 6 months). Twenty two percent of interviews ($n = 10$) were coded by two independent raters to assess interrater reliability for stress severity scores, yielding excellent intraclass correlations for all domains (ICCs: 0.85–0.97).

2.4. Doors task

Youth and mothers completed the Doors task (Proudfit, 2015) to assess RewP. During the task, participants were shown two identical adjacent doors and instructed to guess which door had a prize behind it by using the left and right mouse buttons. After the selection was made, a fixation cross appeared on the screen for 1000 ms, followed by either a green arrow pointing up, indicating that the participant won $0.50, or a

red arrow pointing down, indicating that the participant lost $0.25. After the arrow, a fixation mark was presented for 1500 ms, followed by a screen reading "click for the next round." Once the participant responded, a new trial began. Participants completed 20 gain and 20 loss trials, presented in random order.

2.5. EEG data collection and processing

Continuous EEG data was recorded throughout the Doors tasks with the ActiveTwo BioSemi system (BioSemi, Amsterdam, The Netherlands). An elastic cap with thirty-four standard electrode sites was used (including Fz and Iz), based on the 10/20 system, along with one electrode on each mastoid. Four facial electrodes were placed on participants for electrooculogram (EOG) recordings generated from eye blinks and eye movements (two electrodes were placed 1 cm above and below the right eye to measure vertical eye blinks and movements, and two electrodes were placed 1 cm beyond the outer edge of each eye to measure horizontal eye blinks and movements). The data were digitized at 24-bit resolution with a cutoff of 1024 Hz.

Offline data analyses were performed with BrainVision Analyzer 2 software (Brain Products, Gilching, Germany). EEG data were re-referenced offline to the average of the mastoids and band-passed filtered with low-pass of 0.1 Hz and high-pass of 30 Hz. Data were segmented beginning 200 ms prior to reward feedback onset and ending 800 ms after reward feedback. For each trial, baseline correction was performed using the 200 ms before reward feedback presentation. Eye blink and ocular corrections followed the method of Gratton et al. (1983). Standard artifact analysis procedures were used, identifying a voltage step of more than 50 μV between sample points, a voltage difference of 300 μV within a segment, and a maximum voltage difference of less than 0.50 μV within 100-ms intervals. In addition, trials were inspected visually, and data from individual channels with remaining artifacts were removed on a trial-to trial basis. Consistent with prior research that has localized the RewP to fronto-central electrodes in both adults and youth using principal components analysis (e.g., Ethridge et al., 2017; Liu et al., 2014) and consistently scored the RewP at these sites (Bress et al., 2012; Burkhouse et al., 2017; Goldstein et al., 2020), the RewP was calculated as the average activity 250–350 ms following reward feedback pooled at Fz, FCz, and Cz, where the difference between gain and loss trials was maximal (see Fig. 1 for scalp maps). Paired-samples t-tests confirmed that ERPs for gain trials was significantly larger than ERPs for loss trials across each of these three electrodes for both mothers and youth (all $ps < .001$). Average ERPs were calculated separately for gain and loss trials (Fig. 1). Consistent with previous research (e.g., Belden et al., 2016; Pegg et al., 2019), residual RewP was calculated by regressing ERPs to gain trials onto ERPs for loss trials, saving the unstandardized residual. More positive values for the residual RewP indicate greater reward reactivity.

2.6. Procedures

Participants were recruited from the community prior to the pandemic using flyers, mass emails, and webpage advertisements. At the initial assessment, informed consent and assent were obtained from mothers and youth, respectively. Mothers were administered psychiatric diagnostic interviews to confirm study eligibility. Both mothers and youth completed self-report measures of depression symptoms (i.e., BDI-II for mothers and the CES-D for youth) as well as a reward processing task while continuous EEG was collected.

Following the onset of the COVID-19 pandemic, participants were invited to participate in a follow-up assessment between June and September of 2020. On average, participants completed the COVID-19 assessment 19.31 months after their baseline assessment ($SD = 9.30$; Range = 4.44–33.67). During this follow-up, youth and mothers completed the same self-report measures of depression symptoms administered at baseline. Additionally, youth and mothers completed an

interviewer-based assessment of chronic stress. Dyads were compensated for their participation in the study, and all study procedures were approved by the University of Illinois at Chicago Institutional Review Board and were carried out in accordance with the Declaration of Helsinki. Participants were included in the current study if they completed both baseline and COVID-19 assessments and at least one dyadic member had useable EEG data for the reward task.

2.7. Analytic plan

Analyses were conducted in SPSS (Version 27). Due to the presence of missing data (baseline CES-D: 2.2%, child-reported mother-child stress: 2.2%, child-reported other-family stress: 2.2%, child RewP: 4.4%, mother RewP: 6.7%), we conducted Little's missing completely at random (MCAR) test to evaluate whether data were missing at random. Results for Little's MCAR test was not significant $\chi2(85) = 89.89$, p = .34, thereby justifying the use of imputation methods (i.e., estimation maximization) for the estimation of missing values (cf. Schafer and Graham, 2002).[2] A power analysis conducted in G*Power 3.1.9.7 indicated that the current sample was powered at 80% at a threshold of $p < .05$ to detect moderate to large (r = .39) interaction effects.

Regression analyses were conducted to examine the main and interactive effects of chronic stress and RewP on prospective increases in depression symptoms in both youth and their mothers. In these models, follow-up depression symptoms were the dependent variable, the main effects of baseline symptoms, maternal MDD history (presence versus absence), chronic stress, and RewP were entered as independent variables in the first step of the regression, and the stress × RewP interaction was entered in the second step. Analyses were conducted separately for youth and mothers and were repeated for each domain of chronic stress. All continuous predictors were mean-centered for all analyses. For significant findings in youth, we individually controlled for youth age, sex, racial/ethnic identity (non-Hispanic White: yes versus no), and time since baseline assessment in a series of tests of robustness. For significant findings in mothers, we examined if results were maintained individually controlling for mother age, racial/ethnic identity (non-Hispanic White: yes versus no), and time since baseline assessment as tests of robustness. Additionally, as some dyads received a group intervention through participation in one of the studies ($n = 6$), we also examined if results held controlling for intervention status (received intervention: yes versus no). Finally, given hypotheses that financial stress may contribute to distress during the pandemic through increases in family stress (Prime et al., 2020), we also examined whether significant results for financial stress were maintained controlling for mother-child and family stress.

3. Results

3.1. Preliminary analyses

Descriptive statistics and correlations among study variables for mothers and youth are presented in Table 1. None of the correlations between residual RewP and depression symptoms or chronic stress were significant. Regarding relations between symptoms and stress, youth baseline depression symptoms were significantly correlated with child-reported other-family stress, and mother follow-up depression symptoms were associated with greater mother-reported other-family stress. Of note, youth and mothers' report of chronic stress within their relationship (i.e., mother-child stress) was significantly correlated.

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[2] Analyses conducted with non-estimated data yielded identical results to those presented. Details of analyses are available upon reasonable request from the corresponding author.
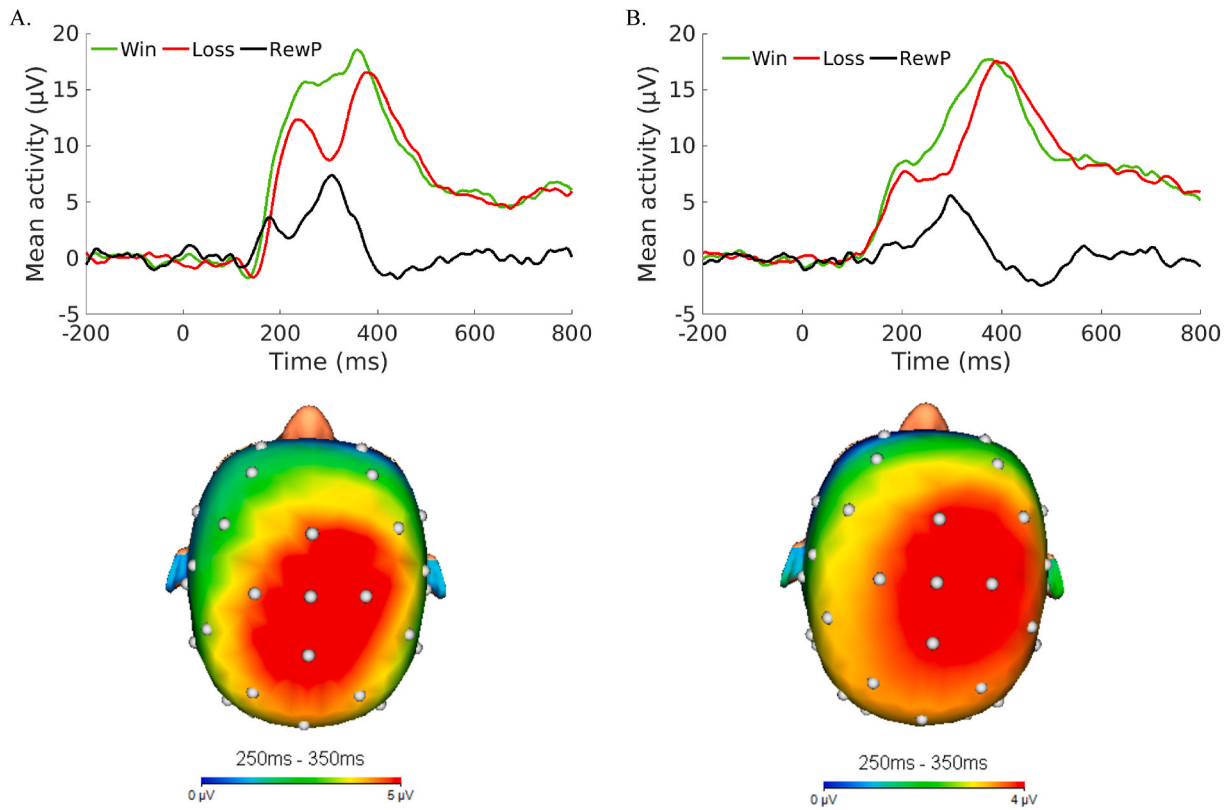
Fig. 1. Waveforms (top) and scalp maps (bottom) depicting the reward positivity (RewP) in response to gain and loss trials 250–350 ms following feedback presentation for (A) youth and (B) mothers.

Table 1
Descriptive statistics and correlations among study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. T1 CES-D	–												
2. T2 CES-D	.17	–											
3. YR – Peer Stress	.25	.11	–										
4. YR – Mother-Child Stress	.28	.15	.33*	–									
5. YR – Other-Family Stress	.42**	.21	.35*	.44**	–								
6. Youth Residual RewP	.15	.10	-.19	-.05	.09	–							
7. T1 BDI-II	.18	.03	.06	-.19	.08	.06	–						
8. T2 BDI-II	-.08	.24	.15	.18	-.01	-.08	.61***	–					
9. MR – Friendship Stress	.24	-.17	.10	.13	.34*	.16	.05	.002	–				
10. MR – Mother-Child Stress	.34*	.17	.03	.44**	.29	-.01	.19	.24	.28	–			
11. MR – Other-Family Stress	-.03	.004	.21	.08	.27	-.25	.20	.33*	.03	.16	–		
12. MR – Financial Stress	-.03	-.01	.007	.07	.08	-.09	.03	.07	-.11	.02	.10	–	
13. Mother Residual RewP	-.05	-.07	-.13	.06	-.07	.07	-.24	-.09	.003	.20	-.22	-.06	–
Mean (SD)	9.92 (6.42)	12.93 (8.91)	2.20 (0.71)	2.03 (0.52)	2.25 (0.60)	0.00 (6.93)	7.60 (6.89)	9.24 (7.85)	2.30 (0.97)	2.03 (0.53)	2.50 (0.60)	2.66 (0.70)	0.00 (4.35)

Note. CES-D = Center for Epidemiological Studies Depression Scale. BDI-II=Beck Depression Inventory-II. YR=Youth Report. MR = Mother Report. RewP = reward positivity.
*p < .05; **p < .01; ***p < .001.

3.2. Impact of stress × RewP interactions on youth depressive symptoms

First, we examined the impact of youth chronic stress, RewP, and their interaction on prospective increases in youth's depressive symptoms. Results revealed a significant RewP × Financial Stress interaction, $\beta = -0.61$, $t(39) = -4.03$, $p < .001$, $r_{effect\ size} = .54$. Following the guidelines of Aiken and West (1991), we examined simple slopes for financial stress at high (+1 SD) and low (−1 SD) levels of residual RewP (Fig. 2). Financial stress positively associated with depressive symptoms for youth exhibiting blunted residual RewP (−1 SD), $\beta = 0.54$, $t(39) = 2.85$, $p = .007$, $r_{effect\ size} = .42$, and negatively associated with depressive symptoms for youth exhibiting increased residual RewP (+1 SD), $\beta =$
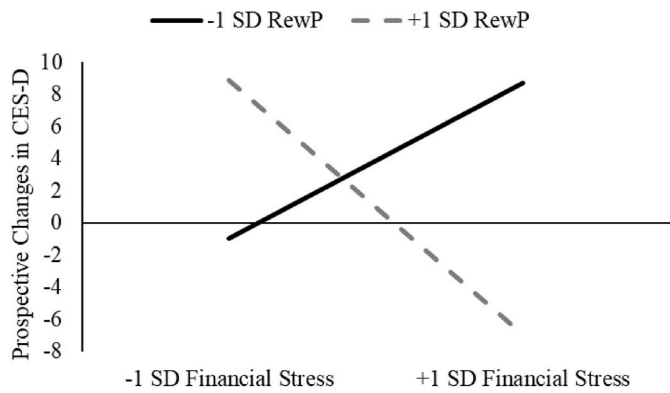
Fig. 2. Interaction between financial stress and youth residual RewP predicting prospective increases in youth depression symptoms. CES-D = Center for Epidemiological Studies Depression Scale. RewP = residual reward positivity.

-0.89, t (39) $= -3.45$, $p < .001$, $r_{effect\ size} = .48$. Next, we conducted a series of analyses to test finding robustness. The significant simple slopes for financial stress at both low and high levels of residual RewP were maintained controlling for youth age, sex, racial/ethnic identity, intervention group, time since baseline assessment, mother-child stress, and family stress (all $ps \leq .015$). No other main effects of stress, RewP, or their interaction were significantly associated with prospective changes in youth depression symptoms (all $ps \geq .32$; see Supplementary Table 1).

3.3. Impact of stress × RewP interactions on mother depressive symptoms

Mirroring analyses for youth, we examined the impact of mother chronic stress, RewP, and their interaction on prospective increases in mothers' depressive symptoms. There was a significant interaction between mother-reported other-family stress and residual RewP, $\beta = -0.27$, t (39) $= -2.33$, $p = .03$, $r_{effect\ size} = .35$, predicting prospective increases in mothers' depression symptoms. Simple slopes for residual RewP (Fig. 3) indicated that other-family stress positively associated with depression symptoms for mothers exhibiting blunted residual RewP (-1 SD), $\beta = 0.45$, t (39) $= 2.94$, $p = .006$, $r_{effect\ size} = .43$, but not for mothers exhibiting increased residual RewP ($+1$ SD), $\beta = -0.08$, t (39) $= -0.44$, $p = .67$, $r_{effect\ size} = .07$. Tests of robustness indicated that the positive relation between other-family stress and prospective increases in depression symptoms for mothers exhibiting blunted residual RewP was maintained controlling for the influence of mother age, racial/ethnic identify, intervention status, and time since baseline assessment (all $ps \leq .009$). No other main effects of mother stress, RewP, or their interaction were significantly associated with prospective changes in mothers' depression symptoms (all $ps \geq .08$; see Supplementary
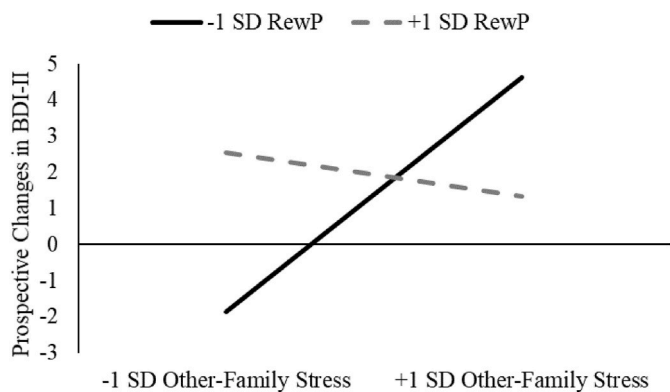

Fig. 3. Interaction between mother-reported other-family stress and residual RewP predicting prospective increases in mother depression symptoms. BDI-II = Beck Depression Inventory-II. RewP = residual reward positivity.

Table 2).

4. Discussion

The current study sought to examine the impact of chronic stress severity on prospective increases in youth's and mothers' depression symptoms within the context of the COVID-19 pandemic. We examined whether a pre-pandemic electrocortical marker of reward responsiveness interacted with chronic stress exposure to predict future depression symptom risk among youth and their mothers. We found no evidence that either chronic stress or pre-pandemic RewP individually predicted increases in depression symptoms for youth or mothers. However, consistent with hypotheses, blunted RewP moderated the impact of stress on prospective increases in depression among both youth and their mothers. Importantly, these findings were maintained when adjusting for demographic variables (i.e., age, sex, racial/ethnic identity).

The RewP indexes initial response to reward (Kujawa et al., 2020; Proudfit, 2015), and prior studies show that attenuated RewP is implicated in depression risk across development (Klawohn et al., 2020; Kujawa and Burkhouse, 2017; Liu et al., 2014). Consistent with prior research showing that blunted reward response interacts with episodic or lifetime stress to predict depression symptoms in adolescents (Burani et al., 2019; Goldstein et al., 2020; Pegg et al., 2019), the current study extends these findings and demonstrates that individuals with preexisting neural vulnerabilities (i.e., blunted reward reactivity) are also at greatest risk for increases in depression symptoms within the context of elevated chronic stress. Furthermore, the current study extends these prior findings to show that blunted RewP interacts with chronic stress in predicting psychopathology during the COVID-19 pandemic and in adult samples. Future research is needed to evaluate the precise mechanisms through which this biological vulnerability exacerbates the impact of stress on depression risk. However, it may be that individuals exhibiting blunted RewP are less likely to seek out or enjoy reward within the context of high stress, which increases risk for depressive symptoms.

Findings highlight financial stress as a predictor of depression symptoms within the context of the current pandemic for youth exhibiting blunted reward reactivity. Financial strain may be a potent stressor that increases depression risk for youth during the pandemic given significant economic repercussions and widespread job loss (de Miranda et al., 2020). Indeed, the impact of financial stress during the COVID-19 pandemic on youth may be exacerbated by school closures due to limited access to technology (e.g., laptops, internet access), thereby affecting virtual learning, and reduced access to school-sponsored supports (e.g., free school lunches, recreational activities, school-based mental health care) (Hoffman and Miller, 2020). It has also been hypothesized that financial strain may influence youth by increasing parenting stress, placing a strain on familial relations (Prime et al., 2020). However, current findings were, at least partially, independent of familial stress, suggesting that financial strain may have a direct impact on depression risk for youth who exhibit preexisting neural vulnerabilities.

Current findings indicated that higher financial strain was also associated with decreases in depression symptoms for youth exhibiting adaptive patterns of reward responsiveness (i.e., increased RewP). Although unexpected, this finding is consistent with emerging literature suggesting some individuals demonstrate psychological improvement following stress exposure, potentially due to increases in prosocial behavior and received social support (for review, see Mancini, 2019). Therefore, it may be that youth exhibiting adaptive reward responsiveness solicit social support from others in the context of financial chronic stress exposure. Alternatively, the positive side-effects of increased financial strain, such as increased time with a parent due to decreased hours, may be especially impactful for youth displaying increased RewP. Future studies including larger samples and comprehensive assessments of family social support are needed to more

explicitly test this hypothesis.

For mothers exhibiting blunted reward reactivity, familial chronic stress prospectively predicted increased depression symptoms. Interpersonal stress within the family, which has been consistently recognized as a risk factor for psychological distress (Hammen and Brennan, 2002; Whisman and Baucom, 2012), has been exacerbated by the COVID-19 pandemic. Indeed, stress within the family unit (e.g., conflict with partners, parenting stress) may be particularly impactful during the pandemic due to COVID-19-related experiences such as isolation and social distancing recommendations (Prime et al., 2020). Shelter in place orders have confined families to their homes for prolonged periods without previous interruptions such as work, school, and extracurriculars. In addition, social distancing guidelines and travel restrictions may have enhanced the saliency of familial stress for mothers by restricting access to in-person social support through family members outside of their household (e.g., parents and siblings). Taken together, familial chronic stress enhanced by the unique circumstances of the COVID-19 pandemic may be especially overwhelming for mothers with preexisting deficits in reward reactivity.

Of note, pre-pandemic blunted RewP interacted with different domains of chronic stress to predict prospective increases in depression symptoms for both youth and mothers. Therefore, it may be that financial and familial stress differentially impact youth and mothers during the COVID-19 pandemic. For example, familial stress may have emerged as a significant predictor of maternal depression symptoms given the inclusion of functioning in relationships with a spouse or partner and with family of origin to the maternal familial stress domain. Given the link between relationship discord and depressive symptoms (Whisman and Baucom, 2012), partner or spouse chronic stress may have driven relations with prospective increases in depression symptoms, which might explain differences in the role of familial stress for youth and mothers. It may also be that financial and interpersonal chronic stress interact to predict risk. For example, in youth, the impact of interpersonal stress on depression risk has been shown to diminish in the context of low socioeconomic status (Vrshek-Schallhorn et al., 2015), whereas in adults, financial stress may be more salient within the context of low social support (Åslund et al., 2014). Conclusions regarding which specific forms of chronic stress interact with aberrant reward reactivity to predict pandemic-related changes in depression symptoms should remain tentative, pending replication in adequately powered samples.

Unexpectedly, although pre-to-post pandemic onset scores for the BDI-II were significantly correlated for mothers, no significant correlation emerged between CES-D scores for youth. Youth in our sample were between 9 and 16 years old at the baseline assessment, and on average participants completed the follow-up assessment over a year and a half later. As risk for depression increases during the transition to adolescence (Hankin et al., 1998), it is possible that youth do not exhibit high long-term stability of depression symptoms across this window of escalating risk. This is consistent with prior research showing that depression symptoms increase during early adolescence before stabilizing or decreasing during later adolescence, particularly for girls (Ge et al., 1994; Holsen et al., 2000), and that the stability of adolescents' CES-D scores decreases as the time between assessments increases (Garrison et al., 1990).

Relatedly, baseline concurrent associations between RewP and depressive symptoms were not significant among mothers and youth. Although blunted RewP is consistently associated with diagnoses of depression and prospective MDD risk (Belden et al., 2016; Bress et al., 2013; Klawohn et al., 2020; Liu et al., 2014; Nelson et al., 2016), relations between the RewP and concurrent symptom severity have been inconsistent. While some studies demonstrate a significant association between RewP and concurrent depression symptoms (e.g., Bress et al., 2012; Burkhouse et al., 2017), many studies do not report significant correlations between RewP and symptom severity measures (e.g., Bress et al., 2013; Burani et al., 2019; Goldstein et al., 2020; Klawohn et al.,

2020; Liu et al., 2014; Pegg et al., 2019). Thus, these prior findings, in combination with the current study, may suggest that rather than being a correlate of psychiatric symptoms, individual differences in the RewP reflect a vulnerability contributing to future depression, especially in combination with stress exposure.

The current study had multiple strengths including pre-pandemic assessments of symptoms and electrocortical reward reactivity in both youth and mothers, enabling longitudinal examination of increases in depression symptoms. Additionally, the study utilized an objective contextual threat interview, which is the "gold-standard" of stress exposure assessment (Harkness and Monroe, 2016). Most profoundly, the current study replicated interactions between stress and reward reactivity in the prospective prediction of depression symptoms in youth using a related but separate adult sample. However, there were also some limitations which warrant attention. First, the current study was underpowered to detect small to moderate effects, which may have contributed to Type II error. Furthermore, as smaller sample sizes can contribute to over-estimation of effect sizes (Button et al., 2013), there is also the possibility of Type I error. This said, current findings regarding interactions between RewP and stress in the prospective prediction of depression symptoms are not only consistent with prior findings (Burani et al., 2019; Goldstein et al., 2020; Pegg et al., 2019) but were also replicated in two related but separate samples in the current study. Therefore, although results point towards blunted RewP as a vulnerability that may increase risk for depression within the context of elevated levels of chronic stress, strong conclusions regarding the specificity of effects to particular domains of stress should not be drawn prior to replication in larger samples. Relatedly, the small sample sizes precluded examinations of potential moderators such as age, maternal depression history, or sex (for youth). Thus, future, larger samples are needed to replicate current findings and test potential moderators of interest.

In addition to the sample size, there were methodological limitations to the current study that should be addressed in future work. Specifically, although findings were replicated in both youth and mothers, different clinical measures were administered to assess symptoms of depression in the two samples. While previous research suggests that the CES-D and BDI-II are highly correlated (for review, see Wang and Gorenstein, 2013) and psychometrically valid assessments of depression (Radloff, 1977; Steer et al., 2001), it may be that the two measures assess different symptoms profiles. Future studies should replicate associations between the RewP, chronic stress, and prospective increases in depression utilizing identical clinical measures across mothers and youth. Additionally, the current study did not assess other forms of stress exposure (e.g., acute stressful life events), which may allow for more nuanced examinations of the objective negative impact of pandemic-related events (e.g., school closures, job loss). Finally, as chronic stress was not assessed pre-pandemic, it is unclear whether chronic stress patterns reflected changes in stress due to the pandemic or were a continuation of pre-pandemic chronic stress.

In summary, these novel findings support a dual-risk model in which chronic stress interacts with deficits in the neural reward system to indicate risk for depressive symptomology in the context of the COVID-19 pandemic. Importantly, the interaction between chronic stress and neural vulnerabilities in the prospective prediction of depression was replicated in both youth and mothers. Preliminary findings suggest that preventative efforts may consider targeting reward processing in families who experience high levels of chronic stress, given associations with prospective changes in depression symptoms.

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CRediT authorship contribution statement

Cope Feurer: Conceptualization, Investigation, Methodology, Formal analysis, Writing – original draft. **Maria Granros:** Conceptualization, Investigation, Writing – original draft. **Alison E. Calentino:** Investigation, Project administration, Writing – review & editing. **Jennifer H. Suor:** Investigation, Writing – review & editing. **Khushboo Patel:** Investigation, Writing – review & editing. **Katie L. Burkhouse:** Conceptualization, Methodology, Supervision, Funding acquisition, Writing – review & editing.

Declaration of competing interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2021.05.034.

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