Reduced reward responsiveness moderates the effect of maternal depression on depressive symptoms in offspring: evidence across levels of analysis

Autumn Kujawa,[1] Greg Hajcak,[2] and Daniel N. Klein[3]

[1]Department of Psychology and Human Development, Vanderbilt University, Nashville, TN; [2]Departments of Psychology and Biomedical Science, Florida State University, Tallahassee, FL; [3]Department of Psychology, Stony Brook University, Stony Brook, NY, USA

Background: Reduced reward responsiveness (RR) may contribute to depression vulnerability. At the neurophysiological level, RR is reliably and validly assessed using the reward positivity (RewP) event-related potential component. We previously identified a blunted RewP in 9-year-old children at high risk for depression due to maternal depression, but the role of RR in pathways from parental history to the development of depressive symptoms has not been examined. **Methods:** At age 9, never-depressed children (N = 369) completed a task in which RewP was measured in response to monetary reward and loss feedback. Parental history of depression was assessed using semistructured interviews, and children reported on their depressive symptoms. At age 12, youth depressive symptoms were reassessed, along with a self-report measure of RR. We tested RR as a moderator of the effects of parental depression on depressive symptoms at age 12, using both neurophysiological and self-report measures and controlling for age 9 symptoms. **Results:** Main effects of RR and interactions with maternal depression were significant. Maternal depression predicted greater depressive symptoms in youth with blunted and average RewP but was not a significant predictor in youth with an enhanced RewP. A similar pattern was observed for self-reported RR. The two measures of RR were not correlated with each other and accounted for unique variance in symptoms. Interactions between RR and paternal depression were not significant. **Conclusions:** Reduced RR, as measured by neurophysiology and self-report, moderates the effects of maternal depression on depressive symptoms in offspring. Assessment of RR along with risk factors such as parental depression may aid in identifying children at greatest risk and enhancing RR could be a potential target for prevention. Results highlight the utility of multimethod approaches for advancing understanding of depression risk. **Keywords:** reward; event-related potentials; depression; adolescence; vulnerability; resilience; maternal depression.

Introduction

The Research Domain Criteria (RDoC) initiative emphasizes integrating multiple levels of analysis of constructs relevant to the development of psychopathology. Depression is characterized by alterations in the reward responsiveness (RR) constructs which include both subjective experiences of pleasure and neural activation to reward attainment (National Institute of Mental Health, 2018), and may be components of approach motivation systems (e.g., Gray, 1994). For example, youth and adults with depression exhibit blunted activation to rewards in the ventral striatum (Forbes & Dahl, 2012; Pizzagalli, 2014; Stringaris et al., 2015). Behaviorally, depression is characterized by reduced tendencies to adjust behavior or expend effort for rewards (Pizzagalli, Jahn, & O'Shea, 2005; Treadway, Bossaller, Shelton, & Zald, 2012). Critically, rather than a state marker of the disorder, alterations in RR have been observed in youth at risk for depression prior to the onset of the disorder, suggesting a possible vulnerability contributing to the development of depression (Kujawa & Burkhouse, 2017; Nelson, Perlman,

Klein, Kotov, & Hajcak, 2016; Stringaris et al., 2015).

Alterations in RR can be reliably and validly assessed using neurophysiological measures, such as the reward positivity (RewP) event-related potential (ERP). We have previously referred to this component as the feedback negativity (FN), consistent with conceptualizations of it reflecting a negative deflection in the ERP wave in response to errors and losses (e.g., Kujawa, Proudfit, & Klein, 2014). However, recent research indicates that this component actually reflects a relative positivity in the ERP wave to rewards approximately 300 ms after feedback over frontocentral sites and more accurately termed RewP (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Kujawa et al., 2018). RewP is consistently observed across development and demonstrates acceptable reliability (Bress, Meyer, & Proudfit, 2015; Kujawa et al., 2018). The magnitude of RewP has been correlated with positive emotionality in children (Kujawa, Proudfit, Kessel et al., 2015), behavioral, and self-report measures of reward sensitivity in older adolescents and young adults (Bress & Hajcak, 2013), and activation of ventral striatum (Becker, Nitsch, Miltner, & Straube, 2014; Luking, Nelson, Infantolino, Sauder, & Hajcak, 2017). When labeled as

FN, this component is often scored as the loss minus gain difference score; however, it can also be calculated as the reward minus loss difference such that more positive values reflect greater differentiation between conditions.

RewP is consistently observed to be blunted in youth and adults with symptoms of depression (e.g., Belden et al., 2016; Foti, Carlson, Sauder, & Proudfit, 2014). Moreover, there is growing evidence that reduced RR, as measured by RewP, may play a role in the development of depression. For example, a blunted RewP to monetary rewards has been observed in children at risk for depression and prospectively predicts the onset of depression in adolescent girls (Bress, Foti, Kotov, Klein, & Hajcak, 2013; Kujawa et al., 2014; Nelson et al., 2016). Reductions in RewP may reflect tendencies to disengage from positive events or reduced motivation to adjust behavior to seek out rewards, which may predispose to depression risk in combination with other vulnerabilities and stress.

Parental depression is a well-established risk factor for depression in children, with offspring of depressed parents exhibiting up to three times the risk of depression as offspring of nondepressed parents (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Weissman et al., 2006). Although both maternal and paternal depression are associated with risk in offspring, there has been greater focus on maternal depression, partly due to higher prevalence of depression in women compared to men, as well as evidence that the effects of maternal depression on internalizing symptoms in children may be somewhat stronger than the effects of paternal depression (Connell & Goodman, 2002).

Vulnerabilities for depression should be more apparent among children of depressed parents compared to nondepressed parents. Consistent with this, there is substantial evidence that children of depressed mothers show abnormalities in RR. For example, reduced striatal activation during reward anticipation and feedback has been observed in youth with maternal histories of depression compared to youth with no maternal depression (Olino et al., 2014; Sharp et al., 2014). In a large community sample of children, we previously found that never-depressed offspring of mothers with a history of depression without comorbid anxiety exhibited a blunted RewP compared to offspring of nondepressed mothers (Kujawa et al., 2014).

These findings suggest that reduced RR likely contributes to depression vulnerability. Yet, understanding of the role of RR in linking parental depression to the emergence of symptoms in offspring is needed in order to identify children at greatest risk. Although parental depression is associated with substantially increased risk of depression in offspring, many offspring of depressed parents will not develop depression. Thus, one possibility is that reduced RR may moderate the association between parental depression and symptoms in offspring, such that the greatest risk exists for children with multiple risk factors (e.g., reduced RR and parental depression). A second possibility is that RR may be a mechanism of intergenerational transmission of depression, such that offspring of depressed parents are likely to exhibit reduced RR, which in turn contributes to the development of depression. Prospective examination of the role of RR in pathways to depression is needed to test these possibilities.

Although there is evidence that risk for depression in youth is characterized by reduced RR across levels of analysis (for a review, Kujawa & Burkhouse, 2017), most studies examine a single measure of RR at a time. As such, the utility of multiple measures of RR remains unclear. Additional research is needed to evaluate the relative variance accounted for by distinct measures, as well as associations between measures, in order to develop methods to maximize accuracy in predicting outcomes and minimize burden and expense.

This study extends our previous cross-sectional studies of RewP in children of depressed parents (Kujawa, Proudfit, Laptook, & Klein, 2015; Kujawa et al., 2014) by examining RR as a prospective predictor of change in depressive symptoms. Never-depressed children ($N = 369$) completed an EEG reward task and measures of depressive symptoms at age 9, and both biological parents were interviewed regarding their histories of depression. Youth were followed up at age 12, completed a self-report of RR and depressive symptoms, and diagnoses were reassessed. The primary goal was to examine whether RR moderates or mediates the effects of parental depression on changes in offspring depressive symptoms from late childhood to early adolescence. The second goal was to test whether neurophysiological and self-report indicators of RR demonstrate similar patterns and whether each measure accounts for unique variance in depressive symptoms.

Method
Participants

Participants were part of a community sample of children initially recruited when they were 3 or 6 years old. Children with no significant medical conditions or developmental disabilities and living with at least one biological, English-speaking parent were eligible to participate. The sample was evaluated again at age 9, at which time the reward task was administered. We previously reported on cross-sectional associations between maternal depression and RewP in 407 children with EEG data at age 9 (Kujawa et al., 2014). To examine RR as a vulnerability that precedes the onset of depression, children who met criteria for a depressive episode at age 9 ($n = 4$) were excluded from analyses. Given evidence of distinct alterations in RR in bipolar disorder (Nusslock & Alloy, 2017), children of parents with bipolar disorders were also excluded.

Of the 407 participants who met criteria for our initial study, 369 completed self-report measures of depressive symptoms and RR at the age 12 assessment and were included in the current prospective study. The sample was 43.9% female, 11.7% Hispanic, 91.1% White, 6.2% Black, and 2.7% Asian. Mean age was 9.17 years (*SD* = .37) at the EEG assessment and 12.65 (*SD* = .43) at the follow-up assessment.

Procedure

Procedures were approved by the Stony Brook University Institutional Review Board. Parents provided informed consent and participants provided assent. When children were 3 or 6 years old, both biological parents were asked to complete a semistructured interview to assess their histories of depression. At approximately age 9, children returned to the laboratory for the EEG assessment (Kujawa et al., 2014), and parents completed an interview assessing psychopathology since the initial evaluation. At approximately age 12, adolescents completed self-report measures of RR and depressive symptoms.

Measures

Parental depression. When children entered the study at either age 3 or 6, biological mothers and fathers were interviewed using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2002) supervised by a licensed psychologist. An advanced doctoral student and a masters-level clinician conducted interviews by telephone, which yields comparable results to in-person interviews (Sobin, Weissman, Goldstein, & Adams, 1993). Interrater reliability (kappa) was 0.93 for depressive disorders (*n* = 30). Parents were interviewed again at the age 9 assessment to assess psychopathology in the years since the initial assessment, with diagnoses combined across assessments to obtain lifetime diagnoses. At age 9, advanced doctoral students and a masters-level clinician administered the SCID. Interrater reliability (kappa) was 0.91 for depressive disorders (*n* = 45).

A small proportion of biological fathers were unavailable for the interview and family history information was obtained from the other parent (Andreasen, Endicott, Spitzer, & Winokur, 1977). At the age 9 assessment, diagnoses for 16 fathers (4.3%) were obtained through family history interviews. More details on interviews and parental diagnoses are presented in Kujawa et al. (2014).

Offspring depression. Youth completed the 27-item self-report Children's Depression Inventory (CDI; Kovacs, 1992) at both assessments. The CDI is a widely used measure of depressive symptom severity in children and adolescents with good psychometric properties. Internal consistency of the CDI in this sample was acceptable (Cronbach's α = .74 at age 9 and .83 at age 12).

At both assessments, one parent and the child were interviewed using the DSM-IV version of the Schedule of Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime (K-SADS; Axelson, Birmaher, Zelazny, Kaufman, & Gill, 2009) to assess depressive disorders. Advanced doctoral students in clinical psychology and a masters-level clinician administered the K-SADS, which were supervised in a group format by an experienced child psychiatrist and licensed clinical psychologist.

Self-reported reward responsiveness. At age 12, participants completed a self-report RR scale derived from the behavioral inhibition system/behavioral activation system (BIS/BAS) scales (Carver & White, 1994) with the addition of items to assess RR (see Van den Berg, Franken, & Muris, 2010). The RR scale consists of eight items rated on a 4-point scale (e.g., 'When I see an opportunity for something I like, I get excited right away'). The RR scale has been shown to have acceptable internal consistency, test–retest reliability, and convergent and discriminant validity (Van den Berg et al., 2010). The BIS/BAS scales demonstrate acceptable reliability and validity in adolescents (Yu, Branje, Keijsers, & Meeus, 2011), and internal consistency was good for the RR scale in our sample (Cronbach's α = .80).

Neurophysiological measure of reward responsiveness. The reward task consisted of 60 trials in three blocks. For each trial, participants were presented with two doors and instructed to select one by button press. Next, a fixation (+) appeared for 1,000 ms, and feedback was presented for 2,000 ms. Participants were told that they could either win $0.50 or lose $0.25 on each trial. A win was indicated by a green '↑,' and a loss by a red '↓.' Finally, a fixation appeared for 1,500 ms, followed by the message 'Click for the next round', which remained on the screen until the participant responded and the next trial began (task presented in Figure S1). The task included 30 win and 30 loss trials in a random order. Participants were informed they could win up to $5 and received $5 cash at the end of the task.

EEG was recorded using a 34-channel Biosemi system (10/20 system; 32 channel cap plus Iz and FCz). Electrodes were placed on the mastoids, and eye movements were recorded from facial electrodes above and below and on the outer corners of the eye. Data were digitized with a sampling rate of 1,024 Hz. Offline analysis was performed using Brain Vision Analyzer. Data were referenced to average mastoid, filtered with cutoffs of 0.01 and 30 Hz, segmented for each trial 500 ms before feedback and continuing for 1,000 ms after onset. Data were corrected for eye blinks (Gratton, Coles, & Donchin, 1983). Artifact rejection was completed using semiautomated procedures: a voltage step of more than 50 μV between sample points, a voltage difference of 300 μV within a trial, and a voltage of <0.50 μV within 100-ms intervals. Visual inspection was used to remove additional artifacts. ERPs were averaged across gain and loss trials and baseline corrected to the window 500 ms prior to stimulus onset. Participants had at least 19 trials per condition at FCz and Cz after artifact rejection. RewP was scored as the mean amplitude 275–375 ms following feedback at FCz/Cz (Figure 1). To isolate variance in the ERP wave attributed to processing of gain versus loss feedback, the RewP difference score was calculated as the mean amplitude on gain minus loss trials, with more positive values indicating greater RR. In our earlier studies with this sample (Kujawa, Proudfit, Kessel et al., 2015; Kujawa et al., 2014), we included Fz in the pooling, but our recent work indicates RewP is maximal over more central sites at this age (Kujawa et al., 2018; see Figure 1). In earlier publications, we calculated the difference score as the loss minus gain difference, but we have reversed the scoring here so that more *positive* values reflect greater differentiation between conditions, consistent with the name change from FN to RewP. Analyses of RewP at Fz, FCz, and Cz and in response to loss and gain separately are presented in Supporting Information (SI).

Results
Descriptive statistics and bivariate correlations

Descriptive statistics and bivariate correlations are presented in Table 1. At the age 12 assessment, two participants had developed major depression or persistent depressive disorder, and 38.5% of mothers and 18.2% of fathers had a lifetime history of a depressive disorder. Child depressive symptoms were relatively low on average but varied across participants (range = 22 at age 9 and 28 at age 12). Boys
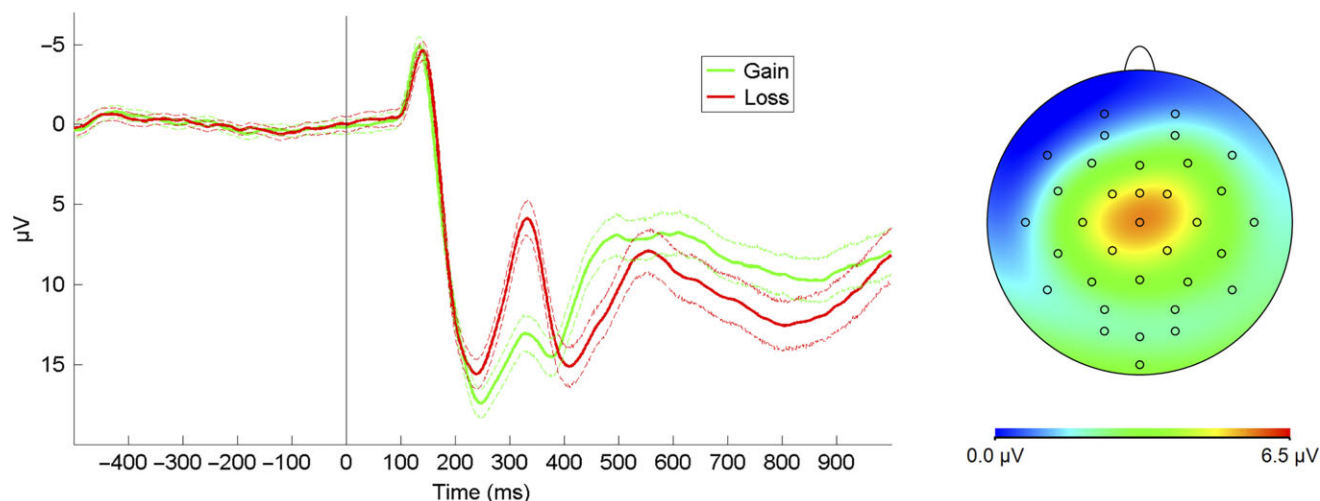
Figure 1 ERPs (with 95% confidence intervals) to gains and losses at FCz/Cz and scalp distribution depicting the gain minus loss difference 275–375 ms after feedback in the overall sample [Colour figure can be viewed at wileyonlinelibrary.com]

Table 1 Descriptive statistics and bivariate correlations between study variables

Variable	Mean(SD)/%	1	2	3	4	5	6	7	8
1. Sex (female)	43.9%	–							
2. Age 9 depressive symptoms	4.89 (4.15)	−.12*	–						
3. Age 12 depressive symptoms	4.57 (5.00)	.08	.29***	–					
4. Maternal depression	38.5%	.02	.06	.14**	–				
5. Paternal depression	18.2%	−.01	.09	.03	.12*	–			
6. RewP Gain	14.06 (10.10)	−.11*	.02	−.14**	.03	.02	–		
7. RewP Loss	8.62 (9.58)	.03	.02	−.04	.08	−.03	.69***	–	
8. RewP Difference	5.44 (7.78)	−.18**	.00	−.13*	−.05	.06	.45***	−.34***	–
9. Self-reported RR	26.88 (3.21)	.06	−.13*	−.29***	−.02	−.02	.01	.01	.00

RewP, Reward positivity; RR, Reward responsiveness.
*p < .05; **p < .01; ***p < .001

showed a larger RewP and reported greater depressive symptoms at age 9. Bivariate correlations with sex were not significant at age 12. Maternal depression correlated with greater depressive symptoms in offspring at age 12 but not age 9. Paternal depression was not significantly correlated with offspring symptoms. Depressive symptoms at age 12 were modestly negatively correlated with both measures of RR, but depressive symptoms at age 9 were only correlated with self-reported RR. The two RR measures were not correlated with each other. Parental depression was not significantly correlated with either RR measure in offspring,[1] indicating that RR did not mediate the effects of parental depression on offspring symptoms. Therefore, regression analyses focused on RR as a moderator.

Reward responsiveness as a moderator of parental depression effects

A multiple regression analysis was computed using PROCESS (Hayes, 2013) and heteroscedasticity-consistent standard errors (HC3; Hayes & Cai, 2007) to examine RewP and self-reported RR as moderators of

the effects of parental depression on depressive symptoms at age 12, adjusting for age 9 symptoms. Main effects of sex, age 9 depressive symptoms, maternal depression, paternal depression, RewP, and self-reported RR were entered into Step 1. Interactions between parental depression and each measure of RR entered into Step 2. Continuous measures were centered before calculating interaction terms.

Results from the multiple regression analysis are presented in Table 2. The overall model was significant, R^2 = .21, $F(10, 358)$ = 7.45, p < .001. Sex (female), maternal depression, RewP, and self-reported RR were significant and unique predictors of age 12 depressive symptoms. The interactions between maternal depression and both measures of RR uniquely predicted depressive symptoms (Figures 2 and 3). Among children with low (−1 SD below the mean) and mean RewP, maternal depression predicted elevated depressive symptoms (simple slope = 2.07; SE = .71; t = 2.90; p = .004; and simple slope = 1.17; SE = .49; t = 2.41; p = .02, respectively). For children with high (+1 SD) RewP, maternal depression did not predict offspring depressive symptoms (p = .66). Similarly, among children with low (−1 SD) and mean self-reported RR, maternal

Table 2 Regression analysis testing the main and interaction effects of parental history of depression and neurophysiological and self-report measures of RR on child depressive symptoms at age 12

Predictor	Unstandardized b (SE)
1. Main effects	$R^2 = .19$***
Age 9 depressive symptoms	.31 (.07)***
Sex	.98 (.49)*
Maternal depression	1.20 (.49)*
Paternal depression	−.04 (.65)
RewP	−.07 (.03)*
Self-reported RR	−.41 (.09)***
2. Interaction effects	Change $R^2 = .02$*
Maternal depression X RewP	−.12 (.06)*
Maternal depression X Self-reported RR	−.38 (.17)*
Paternal depression X RewP	.03 (.07)
Paternal depression X Self-reported RR	−.17 (.21)

RewP, Reward positivity; RR, Reward responsiveness.
*$p < .05$; ***$p < .001$

depression predicted elevated depressive symptoms (simple slope = 2.38; SE = .78; t = 3.05; $p < .01$; and simple slope = 1.17; SE = .49; t = 2.40; p = .02, respectively). For children with high (+1 SD) self-reported RR, maternal depression did not predict depressive symptoms (p = .94).[2] Paternal depression was not a significant predictor of offspring depressive

symptoms. Effects of RewP appeared driven more by RewP to rewards than losses (see SI).

Discussion

In this prospective study, we examined reward responsiveness as a moderator of the effects of parental depression on depressive symptoms in young adolescents using both neurophysiological and self-report measures. Both measures of RR uniquely moderated the effect of maternal depression on depressive symptoms in offspring. Among children with an average or relatively blunted RewP to monetary reward versus loss in late childhood, maternal history of depression predicted greater depressive symptoms in early adolescence. Among children with an enhanced RewP, maternal depression was not a significant predictor of depressive symptoms in offspring. Similar but unique associations were observed with self-reported measures of RR assessed concurrently with depressive symptoms. RR did not appear to mediate the effects of maternal depression on symptoms in offspring. This longitudinal study provides insight into the role of altered RR in developmental pathways to depression, highlights interactions between multiple risk factors, and supports examination of RR across levels of analysis.
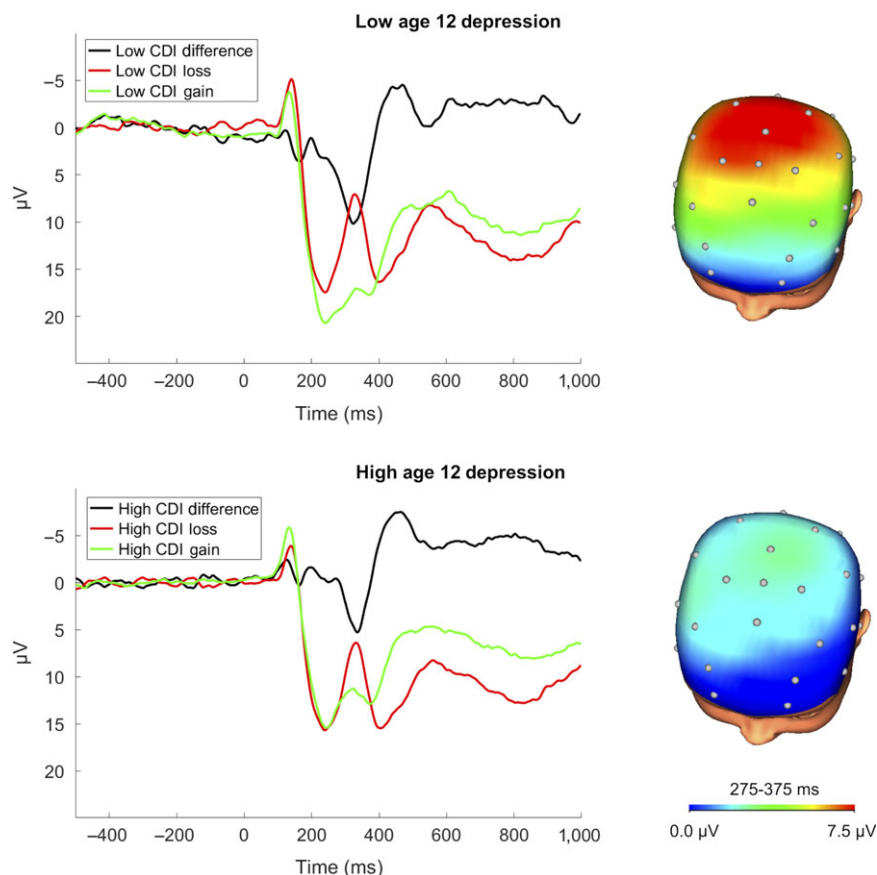

Figure 2 Age 9 ERPs at FCz/Cz and scalp distributions depicting the gain minus loss difference for children with a maternal history of depression with low and high depressive symptoms at follow up. Note: A median split of depressive symptoms at age 12 (residuals adjusting for symptoms at age 9) was used to group participants for illustrative purposes [Colour figure can be viewed at wileyonlinelibrary.com]
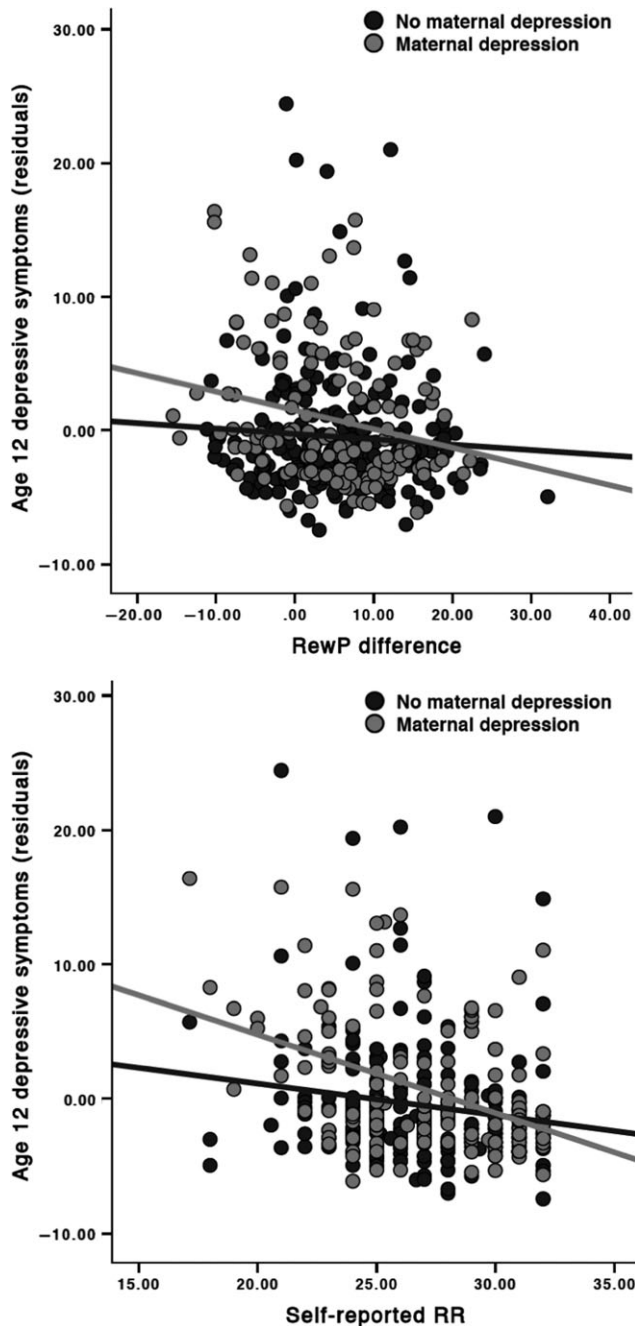
Figure 3 Scatter plots depicting the associations between neurophysiological and self-report measures of reward responsiveness (RR) on depressive symptoms at age 12 (residuals adjusting for symptoms at age 9) for youth with and without maternal histories of depression

Risk for depression has previously been associated with abnormalities in reward processing, and at least some of these measures predict the emergence of depressive symptoms and disorders across development (e.g., Nelson et al., 2016; Stringaris et al., 2015). The goal of our study was to clarify the role of RR in pathways from established risk factors such as parental depression to the development of symptoms. Our findings support reduced RR as a moderator, in that the combination of maternal depression and relatively blunted RR was associated with greater depressive symptoms. As such, RR may help to explain

multifinality among offspring of depressed mothers. Despite clear links between parental depression and risk for depression in offspring (Weissman et al., 2006), this effect was not apparent among children who showed enhanced RR. That is, the combination of risk factors was associated with greater depressive symptoms, but enhanced RR appeared protective against the effects of maternal depression. Thus, greater reactivity to and engagement with rewards and positive events may play a role in reducing likelihood of developing depression among children at risk. It is important to note, however, that clinical depression in this sample was still very rare, and further work is needed to evaluate whether similar effects are apparent later in development when rates of depression diagnoses increase.

The effects of parental depression on depressive symptoms in offspring were specific to maternal rather than paternal depression. This is consistent with evidence of weaker effects of paternal compared to maternal depression on internalizing symptoms in children (Connell & Goodman, 2002). In addition, we previously observed specificity for the effects of maternal internalizing disorders on RewP in offspring (Kujawa et al., 2014), which may be partly attributed to some mothers being more involved in early parenting than fathers. Indeed, we previously observed links between paternal depression and blunted RewP in children in combination with less positive parenting by mothers, suggesting that maternal parenting style may buffer effects of paternal depression (Kujawa, Proudfit, Laptook, et al., 2015). It should also be noted that rates of depression were lower in fathers, which could limit power to detect effects. Lastly, examination of the effects of paternal depression on offspring symptoms later in life is needed, given evidence that the effects of paternal depression may be specific to more moderate and severe cases of depression in offspring (Klein et al., 2005).

Given the lack of associations between parental depression and RR, our findings did not support RR as a mechanism of the effects of parental depression on symptoms in offspring. Further work is needed to explore these associations, however, as heterogeneity in parent and child symptoms may have limited our ability to detect mediation. That is, RR may be a mechanism of the development of more specific symptom dimensions (e.g., anhedonia). Questions remain regarding how or why alterations in RR develop. We previously found RewP to be relatively stable across development and associated with positive emotionality assessed in early childhood (Kujawa et al., 2018; Kujawa, Proudfit, Kessel et al., 2015), suggesting that individual differences in RR may be early emerging. As such, research is needed to evaluate the utility of RR as an intervention target, including whether it is modifiable through parenting or child-focused interventions.

Most studies of RR in depression and risk rely on a single measure of RR, and the integration of self-report

and neurophysiological measures is a strength of this study. Importantly, both measures of RR accounted for unique variance in the emergence of depressive symptoms, indicating that multi-method approaches may be particularly fruitful for understanding developmental pathways to depression and identifying children at risk. Self-report and neurophysiological measures of RR were not correlated with one another in this study, which differs from previous evidence of a correlation between self-reported RR and RewP in older adolescents and young adults (Bress & Hajcak, 2013). This may be attributed to the variability of young adolescents' insight into their behaviors and/or different patterns of developmental changes in reward systems at each level. These measures assess distinct aspects of reward processing, with RewP reflecting an immediate neural response to monetary reward feedback and self-report reflecting broad patterns of reward-seeking behaviors across domains. Between middle childhood and young adulthood, developmental changes occur both in cognitive processes that may influence youths' perspectives on their own RR and in the neural circuitry underlying RR. RewP in particular appears to be more stable across adolescence than from childhood to adolescence (Kujawa et al., 2018), suggesting the possibility that associations with self-report measures of RR may not emerge until later in adolescence. Additional longitudinal work is needed to test this possibility.

A few limitations should be noted. We were unable to examine self-reported RR as a prospective predictor of change in depressive symptoms because it was only administered at age 12. Our ERP task does not allow us to disentangle responses to reward versus performance feedback, and future research is needed to compare reactivity to specific types of positive feedback in depression risk. Effects of RR measures on depressive symptoms were modest in size, consistent with prior work examining neural measures as vulnerabilities for depression in large samples (for a review, Kujawa & Burkhouse, 2017). Although this raises questions about the clinical significance of these results, we observed unique effects of multiple measures of RR, prior depressive symptoms, sex, and maternal depression in predicting symptoms in early adolescence, supporting the utility of combining measures and risk factors to improve prediction of outcomes. Cross-sectional associations between RewP and age 9 depressive symptoms were not significant, possibly due to relatively less variance in symptoms at age 9 compared to age 12 or because of developmental changes in associations between symptoms and RR. Rates of depression in this sample were still very low, raising questions about generalizability to clinical disorders. Subthreshold symptoms should be strong predictors of the later development of depressive disorders (Keenan et al., 2008; Shankman et al., 2009), and follow-up data with this sample will allow us to continue to explore these pathways across adolescence.

Conclusions

Using a longitudinal sample and multiple measures of RR, we found that children's reward responses moderate the effects of maternal depression on the development of depressive symptoms. These results provide insight into the role of altered reward processing in the development of depression, support the utility of both neurophysiological and self-report measures, and highlight potentially fruitful avenues for future research.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Further information.
Table S1. Descriptive statistics and bivariate correlations between RewP scoring approaches.
Figure S1. ERP monetary reward task (Doors).

Acknowledgements

Correspondence

Autumn Kujawa, Department of Psychology and Human Development, Vanderbilt University, 230 Appleton Place, Nashville, TN 37203-5721, USA; Email: autumn.kujawa@vanderbilt.edu

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Key points

- Previous research has identified blunted reward responsiveness (RR) in offspring of depressed parents, but the role of RR in pathways from familial risk to emergence of depressive symptoms has yet to be examined.
- Using neurophysiological and self-report measures, we found that reduced RR moderated the effects of maternal depression on changes in offspring symptoms from late childhood to early adolescence.
- Among youth with low and average RR, maternal depression predicted increases in symptoms, but effects of maternal depression were not significant among children with high RR.
- Offspring of depressed mothers who show relatively blunted reward responses may be at greatest risk for depression. Enhancing RR could be a potential target for prevention.

Notes

1. As in Kujawa et al., 2014, the interaction between maternal depression and maternal anxiety was a significant predictor of RewP in offspring, $F(1, 365) = 4.59$, $p = .03$, such that a blunted RewP was apparent among offspring of mothers with a history of depression without comorbid anxiety. The main effect of maternal depression significantly predicted change in offspring depressive symptoms from age 9 to age 12, $F(1, 364) = 4.61$, $p = .03$, but the effects of maternal anxiety and interaction between maternal depression and anxiety were not significant for offspring depressive symptoms (ps > .13).

2. To test the possibility that sex moderates the effects of RR or interactions with maternal depression, we also tested the model with the addition of the sex by RewP, sex by self-reported RR, and three-way interactions between sex, RR variables, and maternal depression. None of these interactions were significant (ps > .24).

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