Neural Response to Rewards, Stress and Sleep Interact to Prospectively Predict Depressive Symptoms in Adolescent Girls

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Neural Response to Rewards, Stress and Sleep Interact to Prospectively Predict Depressive Symptoms in Adolescent Girls

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Blunted reward processing both characterizes major depressive disorder and predicts increases in depressive symptoms. However, little is known about the interaction between blunted reward processing and other risk factors in relation to increases in depressive symptoms. Stressful life events and sleep problems are prominent risk factors that contribute to the etiopathogenesis of depression and have been linked to reward dysfunction; these factors may interact with reward dysfunction to predict increased depressive symptoms. In a large sample of 8- to 14-year-old adolescent girls, the current study examined how blunted reward processing, stressful life events, and sleep problems at baseline interacted to predict increases in depressive symptoms 1 year later. Reward processing was indexed by the reward positivity (RewP), an event-related potential elicited during a simple monetary reward paradigm (i.e., Doors task). Two-way interactions confirmed that a blunted RewP predicted increased depressive symptoms at (a) high levels of stress but not average or low levels of stress, and (b) high and average levels of sleep problems but not low levels of sleep problems. Finally, a 3-way interaction confirmed that a blunted RewP predicted increased depressive symptoms at high levels of stress and sleep problems but not average or low levels of stress and sleep problems. Thus, adolescents characterized by low reward response (i.e., blunted RewP) were at an increased risk of developing depressive symptoms if they experienced increased stressful life events or sleep problems; moreover, risk was greatest among adolescents characterized by all 3.

Depression is increasingly linked to reward system dysfunction (Whitton, Treadway, & Pizzagalli, 2015). Neuroimaging research examining deficits in reward processing in depression has focused mainly on the mesolimbic pathway and on the striatum in particular (Nestler & Carlezon, 2006). Adults and adolescents with major depressive disorder (MDD) show reduced ventral striatum activation to rewards compared to their healthy peers (Forbes et al., 2009; Pizzagalli et al., 2009). Blunted striatal response to rewards is also evident in healthy young adults and adolescents who report elevated depressive symptoms (Forbes et al., 2010; McCabe, 2015).
Woffindale, Harmer, & Cowen, 2012), as well as in adolescents who are at increased risk for depression based on maternal history of depression (Gotlib et al., 2010). In fact, blunted ventral striatum activation prospectively predicts increases in depressive symptoms in adolescence (Keren et al., 2018; Stringaris et al., 2015). Collectively, these data suggest that reduced activation in reward-related circuitry is part of the etiopathogenesis of depression.

In addition to functional magnetic resonance imaging (fMRI) studies, electroencephalography (EEG) research has focused on an event-related potential (ERP) referred to as the reward positivity (RewP). The RewP is evident 250 ms to 350 ms at frontocentral electrode sites following the presentation of rewards compared to nonrewards. Although a relative negativity was originally interpreted in the ERP following nonrewards (i.e., the feedback negativity), more recent research supports the possibility that the difference between gains and losses is driven by variability in the response to rewards (Holroyd, Pakzad-Vaezi, & Krigolson, 2008). Using principal component analysis, Foti, Weinberg, Dien, and Hajcak (2011) found that the differentiation between reward and loss ERPs was driven by a reward-related positivity that was absent or reduced on nonreward trials. In support, an increased RewP is related to heightened ventral striatal activation to reward measured using fMRI (Carlson, Foti, Mujica-Stringaris et al., 2015) and is related to person-ality traits linked to depression, such as low positive emotionality (Kujawa et al., 2015; Speed et al., 2018). In addition, a blunted RewP is evident among depressed children and adults (Belden et al., 2016; Brush, Ehmann, Hajcak, Selby, & Alderman, 2018; Foti & Hajcak, 2009; Liu et al., 2014) and is observable prior to the onset of depression and can predict increases in depressive symptoms and first-onset MDD (Bress, Foti, Kotov, Klein, & Hajcak, 2013; Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). Recent evidence also indicates that the RewP may interact with other risk factors in predicting the development of depression. For instance, one investigation found that a maternal history of depression predicted increased levels of depressive symptoms, but only in the context of a blunted RewP (Kujawa, Hajcak, & Klein, 2019). Overall, reward system function indexed by the RewP appears robustly related to risk for developmental increases in depressive symptoms and disorders (Proudfit, 2015).

In addition to blunted neural reward measures, stressful life events are associated with increases in depressive symptoms and prospectively predict the onset of depression in adolescence (Abela & Skitch, 2007; Allgood-Merten, Lewinsohn, & Hops, 1990; Monroe, Rohde, Seeley, & Lewinsohn, 1999). Other studies have similarly found that stressful life events predict increases in depressive symptoms, even after controlling for baseline depressive symptoms (Auerbach, Bigdal-Peyton, Eberhart, Webb, & Ho, 2011)—results that may be particularly true for females (Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin, Mermelstein, & Roesch, 2007; Rudolph & Hammen, 1999). Collectively, these studies suggest that both a blunted neural response to reward and stressful life events are prominent risk factors for the development of depressive symptoms.

Increasingly, theory and research suggest that stress may interact with reward-related neural systems to impact depression, such that those at the greatest risk for depression may be individuals characterized by reduced reward activation and increased stressful life events (Pizzagalli, 2014). To our knowledge, only one study has examined how neural measures of reward processing and stressful life events relate to depressive symptoms within an adolescent sample. Specifically, Luking, Nelson, Infantolino, Sauder, and Hajcak (2018) found that ventral striatum activity interacted with negative life events to explain increased depressive symptoms cross-sectionally (Luking et al., 2018). One aim of the current study is to investigate how reduced reward processing, as measured by the RewP, and stressful life events interact to predict longitudinal increases in depressive symptoms in an adolescent sample.

Similar to stressful life events, sleep disturbances have depressogenic effects (Roberts & Duong, 2013, 2014). Nearly three fourths of adolescents with MDD or elevated depressive symptoms suffer from sleep disturbances (Liu et al., 2007). One study found that sleep disturbance during childhood predicted adolescent-onset depression (Ong, Wickramaratne, Tang, & Weissman, 2006); other studies have similarly found that sleep problems predicted increased depressive symptoms (Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009; Roberts & Duong, 2014). Indeed, a recent meta-analysis confirmed a directional relationship between sleep problems and depressive symptoms such that sleep problems precede the onset of MDD and increases in depressive symptoms in adolescents (Lovato & Gradisar, 2014).

In addition to directly contributing to the onset of MDD and increased depressive symptoms, sleep problems have also been linked to reduced reward processing in adolescence: Poor sleep quality has been related to reduced ventral striatum activation to reward feedback (Hasler et al., 2012; Holm et al., 2009). Collectively, these studies suggest that sleep disturbances are related to reduced reward processing and significantly enhance the risk for developing depression. A second goal of the current study is to examine whether increased sleep problems moderate the relationship between blunted reward processing and prospective increases in depressive symptoms. Moreover, the current study examines whether the experience of stress and sleep disturbances are unique or overlapping
moderators of the association between the RewP and subsequent increases in depressive symptoms.

The current study seeks to examine whether a blunted RewP, stressful life events, and sleep problems interact to predict increases in depressive symptoms during late childhood and early adolescence. We conceptualize neural response to reward (i.e., the RewP) as a diathesis, whereas both stressful life events and sleep problems act as stressors. In support of this view, there is evidence that a blunted RewP precedes the onset of MDD (Nelson et al., 2016). Moreover, RewP appears to aggregate in families, as it is reduced in individuals with a maternal history of depression (Kujawa, Proudfit, & Klein, 2014). In addition, an individual’s level of positive affect is associated with their sibling’s RewP, such that decreased positive affect in probands is linked with decreased RewP in siblings—even after controlling for the sibling’s own level of positive affect (Weinberg, Liu, Hajcak, & Shankman, 2015).

Finally, there is evidence from a twin study that reward system measures apart from the RewP may be heritable (Macoveanu, Miskowiak, Kessing, Vinberg, & Siebner, 2016). However, we note that sleep problems may be conceptualized as a diathesis because they may precede the onset of depression.

We focused on late childhood and early adolescence, as this developmental period is characterized by increases in depressive symptoms. A large sample of adolescent girls completed measures of reward processing, depressive symptoms, stressful life events, and sleep problems at baseline. One year later, they completed the same measure of depressive symptoms. First, we examine whether baseline RewP interacts with exposure to stressful life events to predict increases in depressive symptoms 1 year following the baseline visit. We also test whether baseline RewP similarly interacts with sleep problems at baseline to predict a prospective increase in depression. We hypothesize that a blunted RewP will predict increased depressive symptoms, but only in the context of increased stress or sleep disturbances. Finally, we conducted exploratory analysis to investigate the outcome of a three-way interaction between RewP, stressful life events, and sleep problems to determine their combined effect on prospective increases in depression.

METHODS

Participants

Participants were part of a community sample of 317 adolescent girls between 8 and 14 years of age ($M = 12.4, SD = 1.8$) from Long Island, New York, and were predominantly Caucasian (86%), with the remaining participants identifying as 5% African American, 5% Hispanic, and 4% Other. Participants were recruited from the community through a commercial mailing list, word of mouth, and use of study-related flyers. Most parents reported having obtained a bachelor’s degree (31.3%) or a master’s degree (27.9%). Depressive symptoms were assessed 1 year later in 252 participants. Of the 252 participants with data available at both assessments, nine participants were excluded due to poor quality EEG data. In total, data from 243 participants were available for analysis. Of the 243 participants, 187 participants (baseline age: $M = 12.6, SD = 1.7$) completed the Adolescent Life Events Questionnaire (ALEQ) at the initial assessment, as the stress questionnaire was introduced after data collection had begun; 238 participants (baseline age: $M = 12.4, SD = 1.8$) completed the Pittsburgh Sleep Quality Index (PSQI) at the initial assessment. For the post hoc moderation analysis testing the three-way interaction between the reward positivity, stressful life events, and sleep problems, the sample included 183 participants (baseline age: $M = 12.6, SD = 1.7$). Participants without follow-up data reported significantly more stressful life events at baseline ($t = 2.02, p = .045$). However, participants with and without follow-up data did not differ on baseline measures of RewP ($t = 1.34, p = .18$), age ($t = .270, p = .79$), depressive symptoms ($t = 1.44, p = .15$), or PSQI total score ($t = 1.96, p = .15$). Along the same lines, participants with ($n = 187$) and without ($n = 56$) the ALEQ at baseline did not differ on the RewP ($t = 0.73, p = .47$), depressive symptoms ($t = 1.31, p = .19$), or PSQI total score ($t = 0.62, p = .53$). All participants and their parents provided informed assent and consent, respectively, as approved by the Institutional Review Board at Stony Brook University.

Measures

**Adolescent Life Events Questionnaire**

The ALEQ measures a range of stressful life events including school and family problems, and friendship and romantic relationship difficulties, and has good psychometric properties (Hankin & Abramson, 2002). Participants completed the ALEQ at the baseline visit. Participants rated the frequency with which each of 57 stressful life events had occurred in the past 3 months. A 5-point rating scale from 0 (never) to 4 (always) was used to rate each event. The total score was calculated by summing the frequency scores of each of the events.

**Children’s Depression Inventory**

The Children’s Depression Inventory (CDI) is a measure of depressive symptoms in children (Kovacs, 1992) and has good internal consistency and test–retest reliability (Masip, Amador-Campos, Gómez-Benito, & Del Barrio Gándara, 2010). Participants completed the CDI at both baseline and 1-year follow-up visits. This measure consists of 27 items rated on a 3-point scale (0–2). The internal consistency of the CDI total score at baseline ($\alpha = .89$) as well as at follow-up ($\alpha = .89$) was good.
The Pittsburgh Sleep Quality Index (PSQI) was administered at the baseline visit to assess sleep disturbances in the past month. It has good psychometric properties (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). To be more age appropriate, the current study slightly modified the PSQI by removing the item that asks whether the respondent has a roommate. Overall, the measure consists of 18 items rated on a 4-point scale (0–3). The internal consistency of the total PSQI sleep score in our sample was moderate (α = .67). Although the PSQI is commonly used in adults, some research studies have used the PSQI in children and adolescents and report similar internal consistency as the current study (de la Vega et al., 2015; Lund, Reider, Whiting, & Prichard, 2010; Siu, Chan, Wong, & Wong, 2012).

Doors Task

The Doors task is a simple monetary reward task in which gains and losses are equiprobable on each trial (Proudflit, 2015). Using the left and right mouse buttons, participants were instructed to select between two identical doors displayed on a computer screen. After making a selection, a fixation cross was presented for 1,500 ms, followed by feedback stimuli (either a green arrow pointing upward signified +$0.50) or a red arrow pointing downward signified –$0.25) on that trial. The feedback stimuli remained on the screen for 2,000 ms. Text appeared on the screen that instructed participants to “Click for next round,” followed by a fixation cross presented for 1,000 ms. The task contained 30 gain trials and 30 loss trials that were presented in pseudorandom order, using Presentation, version 17.2 (Neurobehavioral Systems, Albany, CA). Participants were told that they could receive between $0 and $15 dollars at the end of the task based on their cumulative earnings; all participants received $8 for completing the task.

Psychophysiological Data Recording and Processing

Continuous EEG was recorded while participants completed the doors task. EEG signal was recorded using the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands) with an elastic cap containing 34-electrode sites placed according to the 10/20 system (i.e., 32 channels plus FCz and Iz). Additional electrodes were placed above and below the left eye, and near the outer canthi of the left and right eyes to monitor vertical electrooculographic activity and horizontal electrooculographic activity. Two electrodes were placed on the left and right mastoids. The EEG signal was preamplified at the electrode to improve signal-to-noise ratio, and data were digitized at a 24-bit resolution with a sampling rate of 1024 Hz using a low-pass fifth-order sinc filter with a half-power cutoff of 204 Hz. Active electrodes were measured online with reference to a common mode sense active electrode constructing a monopolar channel.

EEG data were analyzed using BrainVision Analyzer, version 2.1 (Brain Products, Gilching, Germany). The raw EEG data were re-referenced offline to the average of the left and right mastoids and band-pass filtered from 0.1 to 30 Hz. Eyeblink and ocular-movement corrections were performed using established standards described by Gratton, Coles, and Donchin (1983). Feedback-locked epochs were extracted with a duration of 1,000 ms, starting 200 ms before feedback presentation. The 200-ms segment prior to feedback onset served as the baseline. Epochs containing a voltage greater than 50 μV between consecutive sample points, a voltage difference of 300 μV within a segment, or a maximum voltage difference of less than 0.5 μV within 100-ms intervals were automatically rejected. The reward positivity was scored by using the mean activity in a 100-ms window around the peak of the gain minus loss difference waveform within a 200- to 400-ms time frame at electrode site FCz for each subject.

Data Analysis

Analyses were conducted with the statistical software package SPSS, Version 23 (SPSS Inc, Armonk, NY). Bivariate correlations were assessed using Pearson’s r correlations between the baseline RewP, ALEQ total stress score, PSQI total sleep score, CDI at baseline and follow-up, and the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). Moderation analyses were conducted using the PROCESS macro (Version 3.1; Hayes, 2017) Model 1 for the first two moderation models and Model 3 to test the three-way interaction (i.e., double moderation model). The a priori analyses included two separate moderation models. The first moderation model included the reward positivity as the independent variable, CDI total score at follow-up as the dependent variable, CDI baseline total score as a covariate, and ALEQ total score as the moderator. The second moderation model included the same independent and dependent variables and covariate as the first model; however, PSQI total sleep score was used as the moderator. A follow-up exploratory analysis was conducted using both ALEQ total stress and PSQI sleep scores as moderators to examine whether a three-way interaction effect existed between the reward positivity, stress, and sleep problems in predicting changes in depressive symptoms. All moderation models used a bootstrapping approach with 5,000 bootstrapped samples, and significance was determined using a 95% confidence interval.

RESULTS

Table 1 displays descriptive statistics and bivariate correlations among the baseline RewP, ALEQ stress score, PSQI sleep score, Pubertal Development Scale, and CDI depressive symptoms, as well as CDI at follow-up. Figure 1 displays the waveform and scalp topography of the RewP at baseline.
The first moderation model involved the RewP and stressful life events. This model yielded no main effects of RewP or stress on depressive symptoms; however, there was a significant interaction between the RewP and stress (see Table 2). The simple slopes analyses (see Figure 2) revealed that a more blunted RewP predicted increased depressive symptoms among adolescents who had higher levels of stress (+1 SD; b = −0.26, t = −2.73, p < .01) compared to adolescents who experienced average levels (b = −0.11, t = −1.63, p = .11) or low levels of stress (−1 SD; b = 0.04, t = 0.43, p = .67).1

The second moderation model involved the RewP and sleep disturbance. This model yielded a significant main effect of the RewP and sleep on depressive symptoms, as well as a significant interaction between the RewP and total sleep score (see Table 3). The simple slopes analyses (see Figure 3) revealed that a more blunted RewP predicted increased depressive symptoms when sleep problems were at a high level (+1 SD; b = −0.38, t = −3.7, p < .005) and average level (b = −0.14, t = −2.31, p < .05) but not a low level (−1 SD; b = 0.10, t = 1.22, p = .23).

The third—exploratory—moderation model examined whether stress and sleep score together moderated the relationship between the RewP and depressive symptoms. This model yielded no main effect of the RewP, stress, and sleep on depressive symptoms. In addition, there was no two-way interaction between the RewP and stress or the RewP and sleep; however, there was a significant two-way interaction between stress and sleep. Finally, there was a significant three-way interaction between the RewP, stress, and sleep score (see Table 4). The simple slopes analyses (see Figure 4) revealed that the interaction between the RewP, ALEQ stress score, and sleep problems significantly predicted depressive symptoms at a high level of stressful life events (+1 SD) and a high level of sleep problems only (+1 SD; b = −0.44, t = −3.74, p < .005).2

In contrast, the interaction between the RewP, stress, and sleep problems was not significant at high stress (+1 SD) and low sleep (−1 SD; b = 0.15, t = 0.93, p = .35), low stress (−1 SD) and high sleep (+1 SD; b = 0.09, t = 0.39, p = .70), or low stress (−1 SD) and low sleep (+1 SD; b = −0.07, t = −0.56, p = .57).

### DISCUSSION

The goal of the current study was to examine potential moderators of the relationship between blunted neural reward system activation and subsequent increases in depressive symptoms in a community sample of adolescent girls during late childhood and early adolescence. We found that a blunted RewP predicted increased depressive symptoms in individuals exposed to high levels of stress; however, a blunted RewP did not predict increased depressive symptoms in individuals exposed to average or low stress levels. In addition, a blunted RewP predicted increased depressive symptoms among individuals with elevated and average sleep problems but not in individuals with low sleep problems. Finally, we observed a significant three-way interaction between the RewP, stressful life events, and sleep problems, such that participants at baseline who were characterized by a blunted RewP, more stressful life events, and sleep problems had the greatest subsequent increase in depressive symptoms.

Given the potential utility of leveraging reward system activation to predict risk for depression, it is important to

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1 The ALEQ was also completed by participants at the follow-up assessment. When ALEQ total score at follow-up was used as the moderator, the model yielded no main effect of the RewP or stress on depressive symptoms. In addition, the interaction between the RewP and stress did not significantly predict changes in depressive symptoms (b = −0.004, SE = .003, p = .10), though the effect was at a trend level and in the same direction as the moderation model using baseline ALEQ.

2 When pubertal status was included as a covariate in all of the moderation models tested, the interaction between the RewP and stressful life events trended toward significance (b = −0.0053, SE = .0028, p = .06), whereas the interaction between the RewP and sleep problems (b = −0.08, SE = .02, p < .001) and the three-way interaction between the RewP, stressful life events, and sleep problems (b = −0.003, SE = .001, p < .05) continued to be significant.
consider moderators that may affect this relationship. The current study suggests that exposure to stressful life events and sleep problems may be particularly potent moderators of the relationship between reduced reward system activation and subsequent increases in depression during adolescence. Previous research has consistently found that increased sleep problems and stressful life events have depressogenic effects; however, the current study is the first to indicate that these factors interact to impact the relationship between reduced reward processing and prospective increases in depressive symptoms. The results of the current study are in line with Luking et al. (2017), who showed that stressful life events interacted with fMRI reward processing components to explain concurrent depressive symptoms. The current study extends those results by examining prospective increases in depressive symptoms and integrating the effects of sleep problems on the relationship between a blunted RewP and increases in depressive symptoms.

Insofar as the moderator and independent variable are interchangeable statistically, the current results could also be interpreted as evidence that a large neural response to reward is a protective factor—such that stressful life events and sleep disturbance did not predict increases in depression among

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>Lower CI</th>
<th>Upper CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RewP</td>
<td>0.09</td>
<td>0.11</td>
<td>−.13</td>
<td>.30</td>
</tr>
<tr>
<td>ALEQ Total**</td>
<td>0.10</td>
<td>0.03</td>
<td>.04</td>
<td>.16</td>
</tr>
<tr>
<td>RewP × ALEQ*</td>
<td>−0.006</td>
<td>0.003</td>
<td>−0.012</td>
<td>−0.0007</td>
</tr>
</tbody>
</table>

Note: N = 187. ALEQ = Adolescent Life Events Questionnaire; RewP = reward positivity; CI = confidence interval.

* p < .05. ** p < .01.
participants characterized by a large and robust RewP. Regardless, variation in reward sensitivity could serve as a preexisting neurobehavioral trait such that a blunted RewP characterizes at-risk individuals, and increased response to rewarding stimuli may reflect resilience against the development of depression in the face of stressors and poor sleep. Although increased reward processing may serve as a resilience factor for depression, it may relate to other negative outcomes. For instance, among vulnerable individuals, potentiated responses to rewards could lead to excessive approach-related affect characteristic of hypomania and mania symptoms (Alloy & Nusslock, 2019; Boland et al., 2016). A possible mechanism explaining our findings is that stressful life events and sleep problems might both inhibit dopaminergic activity of the reward circuit, and thereby have negative consequences on reward circuit activation over time (Pizzagalli, 2014). In this way, stressful life events and sleep problems might be harbingers of later reductions of the RewP. Along these lines, it is important to note that the RewP was measured concurrently with sleep problems and stressful life events. Future studies should examine the sequential or bidirectional relationships between these variables to determine the timescale on which stressful life events and sleep disturbance influence each other and their potential impact on reward-related neural activity. In the current study, we are unable to draw inferences about casual pathways among reward processing, sleep problems and stressful events. Future studies are needed to understand whether these results generalize to different populations such as males, older adolescents, and adults. Given that female adolescents are at increased risk for depression, it is conceivable that associations between reward processing, stress, sleep, and subsequent development of depression are stronger in girls compared to boys. Second, it will be important for future

### TABLE 3
Main Effects of Sleep Problems ($M = 4.7$, $SD = 2.9$), the RewP ($M = 5.8$, $SD = 6.0$), and Their Interaction Predicting Depressive Symptoms at Follow-Up While Covarying for Depressive Symptoms at Baseline

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>Lower CI</th>
<th>Upper CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reward Positivity*</td>
<td>0.24</td>
<td>.11</td>
<td>.02</td>
<td>.46</td>
</tr>
<tr>
<td>PSQI Total**</td>
<td>0.83</td>
<td>.20</td>
<td>.44</td>
<td>1.2</td>
</tr>
<tr>
<td>RewP × PSQI**</td>
<td>-0.08</td>
<td>.02</td>
<td>-.13</td>
<td>-.04</td>
</tr>
</tbody>
</table>

Note: $N = 238$. RewP = reward positivity; CI = confidence interval; PSQI = Pittsburgh Sleep Quality Index.

* $p < .05$. ** $p < .01$. 

### TABLE 4
Main Effects of the RewP ($M = 6.0$, $SD = 5.9$), Stress ($M = 31.1$, $SD = 23.8$), and Sleep Problems ($M = 4.7$, $SD = 2.9$) at Baseline as well as Their Interactions on Depressive Symptoms at Follow-Up While Covarying for Depressive Symptoms at Baseline

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>Lower CI</th>
<th>Upper CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reward Positivity</td>
<td>-0.20</td>
<td>.23</td>
<td>-.65</td>
<td>.27</td>
</tr>
<tr>
<td>ALEQ Total Score</td>
<td>-0.04</td>
<td>.06</td>
<td>-.15</td>
<td>.07</td>
</tr>
<tr>
<td>RewP × ALEQ</td>
<td>0.01</td>
<td>.006</td>
<td>-.003</td>
<td>.02</td>
</tr>
<tr>
<td>PSQI Total Sleep</td>
<td>-0.28</td>
<td>.40</td>
<td>-.11</td>
<td>.51</td>
</tr>
<tr>
<td>RewP × PSQI</td>
<td>0.05</td>
<td>.06</td>
<td>-.07</td>
<td>.16</td>
</tr>
<tr>
<td>ALEQ × PSQI*</td>
<td>0.02</td>
<td>.009</td>
<td>.006</td>
<td>.04</td>
</tr>
<tr>
<td>RewP × PSQI × ALEQ*</td>
<td>-0.003</td>
<td>.001</td>
<td>-.005</td>
<td>-.0004</td>
</tr>
</tbody>
</table>

Note: $N = 183$. RewP = reward positivity; CDI = Children’s Depression Inventory; CI = confidence interval; ALEQ = Adolescent Life Events Questionnaire; PSQI = Pittsburgh Sleep Quality Index.

* $p < .05$. 

FIGURE 3 Predicted Children’s Depression Inventory (CDI) depression scores at follow-up from the three-way interaction between baseline reward positivity (RewP) and Pittsburgh Sleep Quality Index (PSQI) sleep. Note: CDI scores at baseline were included as a covariate.

FIGURE 4 Predicted Children’s Depression Inventory (CDI) depression scores at follow-up from the three-way interaction between baseline reward positivity (RewP), Adolescent Life Events Questionnaire (ALEQ) stress score, and Pittsburgh Sleep Quality Index (PSQI) sleep score. Note: CDI scores at baseline were included as a covariate.
studies to determine whether the effects of stress, sleep problems, and reduced reward processing predict depression diagnoses, as opposed to symptoms, which were generally in the milder range. Third, depressive symptoms were assessed more than 1 year following the exposure to stressful life events. Research suggests that the effects of stressful life events are weaker after 6 months (Brown & Harris, 2012; Kendler, Karkowski, & Prescott, 1998)—thus, the extended time between the stressful events and the assessment of depressive symptoms may underestimate the magnitude of the association. Fourth, the current study used a between-subjects approach to analyze the effects of reward processing, stressful life events, and sleep problems on change in depressive symptoms. Future studies might use a within-subjects analytic approach by obtaining repeated measures of reward processing, stressful life events, sleep problems, and depression; this would provide information about the circumstances in which youth are likely to experience increases in depression at the individual level.

Fifth, the current study examined only how negative events affect the relationship between reward responding and depression. The vantage sensitivity model provides a theoretical framework to investigate how positive experiences can mitigate risk factors for developing depression (Pluess & Belsky, 2013). Future studies are needed to examine whether positive events might interact with reward processing to impact depressive symptoms. Finally, self-report measures of stressful life events and sleep problems have important limitations. Self-reports of life events may not accurately distinguish between stress exposure and stress response, and most questionnaire measures do not consider the context in which life events occur (Harkness & Monroe, 2016). For example, one of the items in the ALEQ asks participants how often they failed a test or class project but does not account for the percentage of the final class grade or whether the class was important to the individual or not. Self-report measures of stressful life events can also be biased by depressogenic personality traits that may affect subjective reporting of stressful events (Liu, 2013). Similarly, the PSQI is not an objective measure of sleeping habits and is not commonly used in children and adolescents. It would behoove future researchers to use more objective measures of sleep, such as actigraphs.

In conclusion, the results of the current study provide novel and valuable insights into the pathogenesis of depression during adolescence and could be most useful for developing targeted intervention and prevention efforts that focus on increasing reward sensitivity (i.e., potentiating RewP), decreasing sleep problems, and/or aiding in stress management. Indeed, due to the heterogeneity of depression, the most effective treatments may be those that concurrently target multiple factors, include sleep problems, external stressors, and reward responsiveness.

DISCLOSURE STATEMENT

No potential conflict of interest was reported by the authors.

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NEURAL RESPONSE TO REWARDS, STRESS, AND SLEEP


