NEW RESEARCH

Longitudinal Associations Between Reward Responsiveness and Depression Across Adolescence

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Objective: Lower neural response to reward predicts subsequent depression during adolescence. Both pubertal development and biological sex have important effects on reward system development and depression during this period. However, relations among these variables across the transition from childhood to adolescence are not well characterized.

Method: Depressive symptoms, pubertal status, and the reward positivity (RewP) event-related potential component, a neural indicator of reward responsivity, were assessed in 609 community-recruited youth at 9, 12, and 15 years of age. Structural equation modeling was used to examine concurrent and prospective relations within and between depression and reward responsiveness as well as the influence of pubertal status and biological sex on these variables across assessments.

Results: Stability paths for depression, the RewP, and pubertal status were significant across assessments. Compared with male participants, female participants reported more advanced pubertal status at all assessments, a smaller RewP at age 9, and higher levels of depression at age 15. More advanced pubertal status was associated with a larger RewP at age 15. Most importantly, there were bidirectional prospective effects between the RewP and depression from ages 12 to 15; a lower RewP at age 12 predicted increases in depression at age 15, whereas increased depression at age 12 predicted a lower RewP at age 15.

Conclusion: These findings indicate that there are bidirectional prospective effects between reward responsiveness and depression that emerge between ages 12 and 15. This may be a crucial time for studying bidirectional reward responsiveness–depression associations across time.

Key words: depression; longitudinal; reward positivity; reward responsiveness; RewP

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Depression rates increase dramatically in early adolescence and continue to increase throughout adolescence.[1] Adolescence is also characterized by increases in reward-seeking behaviors that correspond to developmental changes in the mesocorticolimbic pathway, a network connecting dopamine-rich regions of the brain that process reward-related information.[2,3] Studies examining reward responsiveness during adolescence suggest that, compared with children and adults, adolescents demonstrate greater reward responsiveness.[2,3] It is hypothesized that atypical development of the reward system during adolescence may partially explain the increase in depression during this period, possibly through its role in reinforcement learning and in emotions central to depression, such as anhedonia.[4,5]

Event-Related Potentials as a Method for Studying the Reward System

Event-related potentials (ERPs) provide a temporally precise measure of neural responses to stimuli. The reward positivity (RewP) is an ERP component that assesses reward responsiveness. RewP is a positive deflection in the ERP signal occurring approximately 250 to 350 ms after reward-related feedback, such as receiving monetary gains and losses, and the neural response is larger in response to gains.[6] The RewP is frequently scored as the neural response to gains minus losses and can be measured reliably across childhood and adolescence.[7,8]

Reward Processing and Depression During Adolescence

Reward processing dysfunction has emerged as one of the most promising biological markers of depression.[9-11] A lower RewP is concurrently and prospectively associated with depression during adolescence.[9,12-18] A recent meta-analysis concluded that, when combining cross-sectional and prospective studies, there is a significant and moderate-sized effect of the RewP on depression among youth younger than 18 years of age.[9] While these studies have established a lower RewP as an important risk factor for the development of depression, no studies have

examined the RewP–depression association across time. This is critical; while the RewP has typically been examined as a symptom correlate or vulnerability for depression, other models, such as a "scarring effect"[19] of depression on the RewP, are plausible.[20] Examination of the bidirectional associations between the RewP and depression across time allows for a more comprehensive understanding of the directionality and age-specific relations between constructs across development.

The RewP Across Development

Despite the RewP being a developmentally appropriate measure of reward responsiveness, research examining developmental changes in the RewP is limited, and most existing work has employed cross-sectional designs. Findings examining the RewP during adolescence are mixed; while some cross-sectional studies have found decreases[21-23] or nonlinear changes[24] in the magnitude of the RewP across children, adolescents, and adults, others found no differences in RewP magnitude across groups.[25] Only 3 studies have examined reward responsiveness during adolescence longitudinally using ERPs. One study of adolescents found that the ERP response to gains, but not to losses, increased from baseline to the 2-year follow-up for younger, but not older, adolescents.[26] A second study of children and adolescents found that across a 2-year follow-up, there were significant increases in neural responses to gains and losses, but that there were no significant changes in the RewP.[13] Finally, a third study, which employed a subset of participants from the current investigation, examined a wider window of development and found that RewP magnitude did not significantly change across 3 assessments that included late childhood, early adolescence, and middle adolescence.[7]

Current Study

There is a surprising dearth of longitudinal studies examining the prospective associations between reward responsiveness and depression and their age-specific timing in childhood and adolescence. Relatedly, while several studies have demonstrated that a lower RewP predicts increases in depression, none of these studies assessed associations between the RewP and depression at multiple time points, limiting conclusions about the direction, developmental timing, and types of effects that can be examined.

Two additional factors that have been implicated in the development of both depression and the reward system are biological sex and pubertal status. Sex differences in the prevalence of depression, reward responsiveness, and the neural development of dopamine-rich reward regions emerge in early adolescence around the onset of puberty.[27-30] However, few previous studies have considered the effects of sex and puberty on the association between reward responsivity and depression, and none have assessed puberty at multiple waves to examine the effects of pubertal development.

We used structural equation modeling (SEM) to study the codevelopment of reward responsiveness and depression across childhood and adolescence, while accounting for the influences of biological sex and pubertal development in a community sample of youth assessed at ages 9, 12, and 15. Specifically, we aimed to establish at what age or ages a lower RewP predicts subsequent depression, adjusting for prior depression, pubertal development, and biological sex, and whether depression symptoms predict subsequent RewP, adjusting for prior RewP, biological sex, and pubertal development.

METHOD

Participants

This study draws on the Stony Brook Temperament Study, a study of 609 youth and at least one coparticipating biological parent.[31] There were 559 families recruited at age 3, and 50 children were added at age 6 to increase diversity. The sample has been reevaluated at 3-year intervals. Families were screened to ensure that the parent spoke English and that children did not have significant developmental or medical conditions. Written consent from a primary caregiver was obtained, and families were compensated for participation in the study. All procedures were approved by the Stony Brook University Institutional Review Board.

Demographic characteristics of the sample are reported in Table 1. The current study primarily used data from youth participants and a coparticipating parent at the most recently completed waves once ERP reward tasks were added: age 9 (mean [SD] = 9.17 [0.37]), 12 (mean [SD] = 12.67 [0.42]), and 15 (mean [SD] = 15.16 [0.40]). Of the 609 families in the larger study, 531 provided data during at least one of the relevant assessments. The age 9 wave included 481 youth and 487 parents; the age 12 wave included 470 youth and 467 parents; the age 15 wave included 448 youth and 454 parents.

Measures

Depression. In-person interviews were conducted using the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS)[32] separately with youth and a coparticipating parent (nearly all mothers) at each assessment to assess depression symptoms experienced by youth in the past month. Interviewers made summary ratings integrating both informants' reports for each of 10

TABLE 1 Demographic Characteristics

Variable	n	(%)
Child race		
White	542	(89.0)
Black	49	(8.0)
Asian	14	(2.3)
Other[a]	4	(0.7)
Child ethnicity (Hispanic)		
Hispanic	75	(12.3)
Non-Hispanic	534	(87.7)
Parent graduated college[b]		
Mother (age 9)	277	(56.3)
Father (age 9)	218	(44.3)

[a]*Multiracial, Native American or Alaskan Native, Native Hawaiian or Other Pacific Islander.*
[b]*Number (%) of the 492 families who provided data on that variable at age 9.*

depression symptoms on a 3-point Likert-type scale (absent = 1, subthreshold = 2, threshold = 3). These scores were summed to assess past-month depression severity. The K-SADS was administered by extensively trained research staff closely supervised by a clinical psychologist and a child and adolescent psychiatrist. Interrater reliabilities were substantial[33] at age 9 (intraclass correlation coefficient = 0.83) and ages 12 and 15 (α = .97).

The Children's Depression Inventory (CDI) was also administered to assess depressive symptoms during the past 2 weeks in youth via self-report and mother report.[34] It includes 27 items for youth and 17 items for mothers, each rated on a 3-point Likert-type scale (0-2) with higher scores reflecting greater severity. The CDI was administered to youth and mothers at each assessment. Although in a small proportion of cases, fathers completed K-SADS interviews, the mother-reported CDI was used for all participants, as other analyses indicated that mother-reported CDI is superior to father-reported CDI as an indicator of youth depression in the current sample. Internal consistency was moderate to substantial[33] for youth (α = .74-.83) and mothers (α = .79-.81) across assessments.

Pubertal Development. The Pubertal Development Scale (PDS)[35] is a widely used self-report measure of pubertal development. Youth report on growth, body hair, and skin changes. Male youth were asked about voice changes and growth of facial hair, while female youth were asked about breast development and age at menarche. Items were rated on a 4-point Likert-type scale (ranging from "not yet started" to "seems complete") except for menarche, which was rated as yes/no. The scale had slight-to-fair reliability[33] at age 9 (male: α = .50; female: α = .38), moderate reliability at age 12 (male: α = .72; female: α = .72), and fair-to-moderate reliability at age 15 (male: α = .76; female: α = .51).

Reward Processing. The doors reward task has been used extensively in prior studies to elicit the RewP.[12,36,37] It consisted of 3 blocks of 20 trials. Trials began with 2 identical doors on the screen. Participants were told that they could win $0.50 or lose $0.25 on each trial and were asked to select the right or left door by clicking a mouse. Rewards trials are twice as large in magnitude because losses are subjectively about twice as valuable as gains,[38] while ensuring that participants accrue money during the task. The doors were presented on the screen until the participant selected one. After the selection, a fixation cross (+) was presented for 1,000 ms, then feedback was presented for 2,000 ms. A green arrow (↑) represented a gain, whereas a red arrow (↓) indicated a loss. The randomly determined feedback was followed by a fixation cross presented for 1,500 ms, followed by the message "Click for next round"; this message remained on the screen until the participant responded to begin the next trial. All participants received 30 gain and 30 loss trials.

Consistent with prior investigations,[17,18,39] feedback-locked ERPs were scored as the mean amplitude from 250 to 350 ms after feedback at the FCz electrode site. Scores were averaged separately for gain and loss trials. The RewP was quantified as the difference between gain and loss trials (gains minus losses), which is necessary for isolating the RewP component.[6] Split-half reliability based on the Spearman-Brown prophecy formula was moderate to substantial[33] for neural response to gain (age 9 = 0.64; age 12 = 0.83; age 15 = 0.85) and loss (age 9 = 0.73; age 12 = 0.75; age 15 = 0.82) scores. When using the Furr and Bacharach formula to calculate reliability for the RewP difference score, results show that it is slight to fair[33] (age 9 = 0.42; age 12 = 0.36; age 15 = 0.40). These results are consistent with results reported in prior work on the reliability of the RewP.[8]

Electroencephalography Data Acquisition and Processing. The doors reward task was administered on a computer via Presentation Version 17.2 (Neurobehavioral Systems, Inc., Albany, California). The same electroencephalography (EEG) recording and processing parameters used in previous studies were implemented in the current study.[7,37] Continuous EEG was recoded with a 34-electrode elastic cap (32 channels with FCz and Iz added) with sites placed according to the 10/20 system using a BioSemi system (BioSemi B.V., Amsterdam, Netherlands). Electro-oculography was recorded using 4

additional facial electrodes: 1 each placed approximately 1 cm outside the left and right eyes and 2 placed approximately 1 cm above and below the right eye. Sintered Ag/AgCl electrodes were used. The BioSemi ActiveTwo system was used to record EEG and electro-oculography, and the signal was digitized with a sampling rate of 1,024 Hz using a low-pass fifth-order sinc filter with a half-power cutoff of 204.8 Hz. A common mode sense active electrode producing a monopolar (nondifferential) channel was used as a recording reference for the EEG electrodes. The electro-oculography electrodes produced 2 bipolar channels measuring horizontal and vertical eye movement. BrainVision Analyzer Version 2.1 (Brain Products GmbH, Gilching, Germany) was used to analyze EEG data. An average of the left and right mastoids, band-pass filtered (0.1-30 Hz) and corrected for eye movement artifacts according to the technique outlined by Gratton *et al.*,[40] was used as an offline reference. Feedback-locked epochs with durations of 1,000 ms were extracted beginning 200 ms before feedback, with the 200-ms interval before feedback used for baseline correction. Epochs containing a maximum voltage difference of less than 0.5 mV within 100-ms intervals, a voltage greater than 50 mV between sample points, or a voltage difference of 300 mV within a segment were automatically rejected. Additional artifacts were identified and removed via visual inspection. Topographic interpolation was used to compute a weighted average for an electrode when there were issues with an EEG channel for a specific subject (ie, those with less than 15 segments of data after artifact rejection), although all participants had a minimum of 15 segments per condition at FCz after artifact rejection, and therefore none of the analyzed data were interpolated.

Data Analytic Strategy

Single-factor confirmatory factor analysis was used to create latent depression variables at ages 9, 12, and 15 using youth CDI, mother CDI, and K-SADS depression scores as indicators to reduce the measurement error. Youth CDI was used as the first indicator, and its unstandardized factor loading was fixed to 1.00 for model identification because it loaded most strongly on the depression factor at 2 waves. Residuals between common indicators across waves were correlated to account for method and reporter variance.

SEM was used because it allows for simultaneous evaluation of the associations between all variables. Stability paths within all repeatedly measured variables were estimated by regressing the scores from later assessments onto the score from the previous wave. Covariances between the RewP and latent depression variables were estimated at each assessment. The influence of the RewP on subsequent depression, and of depression on the subsequent RewP, was estimated using cross-lagged regression paths between the RewP and latent depression scores; the score from the later assessment was regressed on the score of the opposing variable from the previous assessment. To account for influences of biological sex and pubertal status on depression and reward responsiveness, latent depression scores and the RewP at each assessment were regressed on biological sex and the concurrent PDS score. Covariances were estimated between biological sex and PDS at each assessment to account for sex differences in pubertal development. Exploratory analyses were also conducted by replicating and slightly modifying this model: once replacing the RewP with the neural response to gain at each age and once replacing the RewP with the neural response to loss at each age.

Finally, to examine whether there were differences in model parameters across time (ie, do the means of variables differ at ages 9, 12, or 15; does the strength of the associations between variables change across time), a series of follow-up model comparisons and Wald χ^2 tests[41] were conducted on the model examining the RewP. To best manage the latent depression variables, comparisons investigating whether depression levels changed across waves were conducted by constraining the depression variables to equality, progressively freeing these parameters, and comparing χ^2 scores; all other comparisons were conducted using Wald χ^2 tests.

Analyses were conducted in Mplus 8[42] using a maximum likelihood estimator, which is suitable for continuous data. Full information maximum likelihood estimation was used to account for missing data.[43] This allows for all individuals providing data on at least one variable to be included in model estimation. Therefore, all 609 participants are included in our models based on having available data on biological sex. However, the pattern of findings is identical when analyzing the subset of 531 participants who provided data at the age 9, 12, and/or 15 assessments. Similarly, the pattern of findings was identical to those presented when controlling for mother and father education status as a covariate.

RESULTS

Descriptive Statistics

ERP waveforms showing neural response to gain, loss, the RewP difference score, and the scalp distribution of the RewP at each assessment are displayed in Figure 1. Descriptive statistics are presented in Table 1, and bivariate associations are presented in Table 2.

SEM Estimation of the RewP Model

Factor loadings and residual correlations for the model examining the RewP are reported in Table S1, available online. All indicators loaded significantly on their respective depression latent factors. All regression and covariance path estimates are included in Table 3 and displayed in Figure 2. Model fit statistics indicate that the estimated model fits the data well (comparative fit index [CFI] = 0.97; Tucker-Lewis index [TLI] = 0.95; root mean squared error of approximation [RMSEA] = 0.03). CFI and TLI values greater than 0.95 and RMSEA values less than 0.06 suggest good model fit.[44]

Examination of covariates demonstrated that increased pubertal development at age 15 predicted reduced reward responsiveness. Additionally, female participants demonstrated significantly lower reward responsiveness at age 9 compared with male participants. Conversely, female sex predicted increased depression at age 15. Finally, covariance estimates between biological sex and pubertal development indicate that female youth reported significantly higher levels of pubertal development at all assessments. Estimates for the stability paths within all repeatedly measured variables were positively and significantly associated across intervals.

Covariance estimates between concurrent depression and RewP residuals at ages 9, 12, and 15 all were nonsignificant. Estimates from the cross-lagged paths investigating the influence of reward responsiveness on depression at the subsequent assessment indicate that age 9 reward responsiveness did not significantly predict age 12 depression. However, lower reward responsiveness at age 12 predicted increased depression at age 15, even after accounting for the simultaneous influence of all other variables in the model.

Similarly, age 9 depression did not significantly predict age 12 reward responsiveness. However, even after accounting for the simultaneous influence of all other variables in the model, increased depression at age 12 predicted reduced reward responsiveness at age 15.

SEM Estimation of the Neural Response to Gain Model

Factor loadings and residual correlations for the model examining neural response to gain are reported in Table S2, available online. All regression and covariance path estimates are included in Table S3 and displayed in Figure S1, available online. The estimated model fit the data well (CFI = 0.97; TLI = 0.95; RMSEA = 0.03).

Female sex predicted significantly greater depression at age 15 and lower response to gains at age 9 compared with male participants. Covariance estimates between biological sex and pubertal development indicated that female participants reported significantly higher levels of pubertal development at all assessments. Consistent with the RewP model, estimates for the stability paths within all repeatedly measured variables were positively and significantly associated across intervals. Covariance estimates between concurrent depression and the neural response gain residuals at ages 9, 12, and 15 all were nonsignificant. Contrary to results examining the RewP, the cross-lagged paths reflecting the influence of reward responsiveness on depression at the subsequent assessment indicated that neural response to gains did not significantly predict depression at the subsequent visit at ages 12 or 15. Finally, age 9 depression did not significantly predict age 12 reward responsiveness. However, as in the RewP model, increased depression at age 12 predicted reduced reward responsiveness at age 15.

SEM Estimation of the Neural Response to Loss Model

Factor loadings and residual correlations for the model examining neural response to loss are reported in Table S4, available online. All regression and covariance path estimates are included in Table S5 and displayed in Figure S2, available online. The estimated model fits the data well (CFI = 0.97; TLI = 0.94; RMSEA = 0.04).

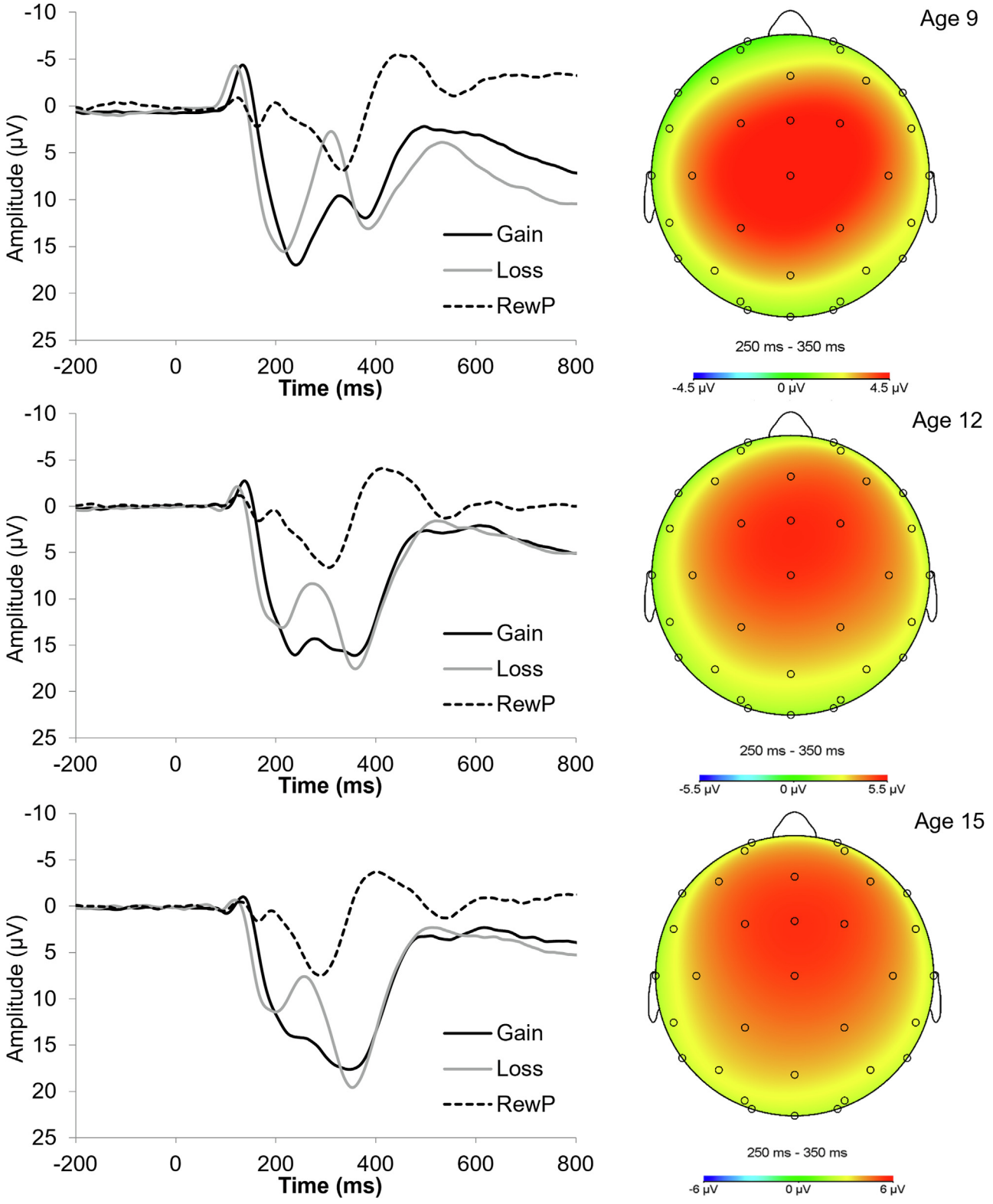
Consistent with both prior models, female sex predicted greater depression at age 15, and female participants reported significantly higher levels of pubertal development at all assessments. However, contrary to both prior models, increased pubertal development at ages 9 and 12 significantly predicted lower neural responses to loss at ages 9 and 12, respectively.

Again, estimates for the stability paths within all repeatedly measured variables were positively and significantly associated across intervals. Covariance estimates between concurrent depression and the neural response loss residuals at ages 9, 12, and 15 all were nonsignificant. Similar to the model examining neural response to gain, but contrary to results examining the RewP, neural response to loss did not prospectively predict depression at ages 12 or 15. Again, contrary to the RewP and neural response to gain models, neural response to loss did not significantly predict subsequent depression at any age.

Parameter Comparisons of the RewP Model

Results from all model comparisons and Wald χ^2 tests are presented in Table 4. Among covariates, as expected there were significant increases in mean PDS scores across assessments, and scores were significantly larger at age 15 than at ages 9 and 12. Additionally, comparisons showed that the association between sex and pubertal development was significantly stronger at age 12, than at age 9, with female youth reporting greater levels of pubertal development.

FIGURE 1 Reward Positivity (RewP) Waveform and Scalp Distribution at Ages 9, 12, and 15



Note: *Event-related potential waveforms (left panel) and three-dimensional rendered scalp distributions of neural responses to gains, losses, and the RewP difference score (ie, gains minus losses) at electrode FCz (right panel) for ages 9 (top), 12 (middle), and 15 (bottom). Please note color figures are available online.*

TABLE 2 Descriptive Statistics and Bivariate Correlations

Variable	n	(%)	(SD)	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Biological sex (female)	609	(45.50)	—	—														
		Mean																
2. Age 9 RewP	469	4.84	(8.26)	−0.29***	—													
3. Age 9 youth CDI	481	4.89	(4.21)	−0.14**	0.01	—												
4. Age 9 mother CDI	485	7.30	(4.89)	−0.15***	0.01	0.23***	—											
5. Age 9 K-SADS	488	0.56	(1.81)	−0.05	0.02	0.15**	0.31***	—										
6. Age 9 PDS	480	8.41	(1.89)	−0.26***	0.001	0.04	0.01	−0.03	—									
7. Age 12 RewP	440	5.24	(7.80)	−0.09	0.22***	−0.06	0.04	0.03	−0.05	—								
8. Age 12 youth CDI	458	4.75	(5.19)	0.09	−0.07	0.28***	0.24***	0.10*	0.03	−0.01	—							
9. Age 12 mother CDI	472	7.14	(5.04)	−0.14**	0.00	0.13**	0.62***	0.26***	0.00	0.04	0.36***	—						
10. Age 12 K-SADS	476	0.42	(1.66)	0.04	−0.03	0.11**	0.21***	0.37***	0.04	0.04	0.31***	0.33***	—					
11. Age 12 PDS	439	11.53	(2.88)	0.42***	−0.05	−0.03	0.00	−0.04	0.27***	0.02	0.05	−0.01	−0.05	—				
12. Age 15 RewP	407	5.52	(6.42)	0.002	0.17**	0.05	0.02	−0.06	−0.05	0.23***	−0.07	−0.05	−0.05	−0.08	—			
13. Age 15 youth CDI	448	5.72	(5.42)	0.26***	−0.09	0.18***	0.12*	0.14**	0.07	−0.11	0.54***	0.19***	0.19*	0.12*	−0.02	—		
14. Age 15 mother CDI	454	8.07	(5.50)	−0.03	0.00	0.06	0.53***	0.14**	0.01	−0.04	0.32***	0.63***	0.23	−0.04	−0.04	0.38***	—	
15. Age 15 K-SADS	458	0.70	(2.65)	0.15**	−0.04	0.01	0.04	0.06	0.07	−0.03	0.24***	0.08	0.09	0.05	−0.07	0.51***	0.33***	—
16. Age 15 PDS	445	15.21	(2.18)	0.49***	−0.16**	−0.06	−0.04	−0.01	0.12*	−0.02	0.11**	−0.07	0.02	0.44***	−0.09	0.15**	−0.01	0.14**

Note: *Biological sex is coded as dichotomous with higher scores reflecting female sex; % = percent female.*
CDI = Children's Depression Inventory; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; PDS = Pubertal Development Scale; RewP = Reward positivity event-related potential component at FCz electrode cite from 250-350 ms following feedback.
*$p < .05$; **$p < .01$; ***$p < .001$.

Examination of the influence of covariates on reward responsiveness indicated that more pubertally developed adolescents had a significantly smaller RewP at age 15 than their less developed peers, and this association was significantly stronger than at age 9 or 12. When examining the influence of biological sex, female participants demonstrated significantly reduced reward responsiveness at age 9 compared with male participants. This association was significantly stronger at age 9 than at ages 12 and 15.

Tests of the associations between covariates and depression demonstrated that the impact of biological sex on depression at ages 12 and 15 were significantly stronger than the relation between biological sex and depression at age 9. Finally, at ages 12 and 15, female participants reported higher levels of depression.

Wald χ^2 comparisons showed that mean levels of depression increased significantly at each assessment in the study. However, the magnitude of the stability path from age 9 to 12 did not significantly differ from the age 12 to 15 path, indicating that a participant's depression score at one wave impacted their subsequent depression score similarly across the study.

Examining reward responsiveness, Wald χ^2 tests indicated that the RewP mean did not significantly change during our study. Similarly, the magnitude of the RewP stability paths between the age 9 to 12 and age 12 to 15 waves did not significantly differ.

The impact of age 9 reward responsiveness on age 12 depression did not significantly differ from the impact of age 12 reward responsiveness on age 15 depression. We also found no significant differences between the impact of age 9 depression on age 12 reward responsiveness and age 12 depression on age 15 reward responsiveness.

DISCUSSION

We investigated concurrent and prospective associations within and between reward responsiveness and depression at ages 9, 12, and 15, while also examining the impact of pubertal development and biological sex on these processes. To our knowledge, this is the first study to examine the trajectory of the RewP and neural responses to gain and loss across more than 2 time points and the first to study the codevelopment of the RewP with depressive symptoms.

Reward responsiveness exhibited significant stability from age 9 to 15. Moreover, the means at each wave and of the wave-to-wave associations did not differ significantly across assessments. This expands on prior cross-sectional[25] and longitudinal[7,13] data showing no significant changes

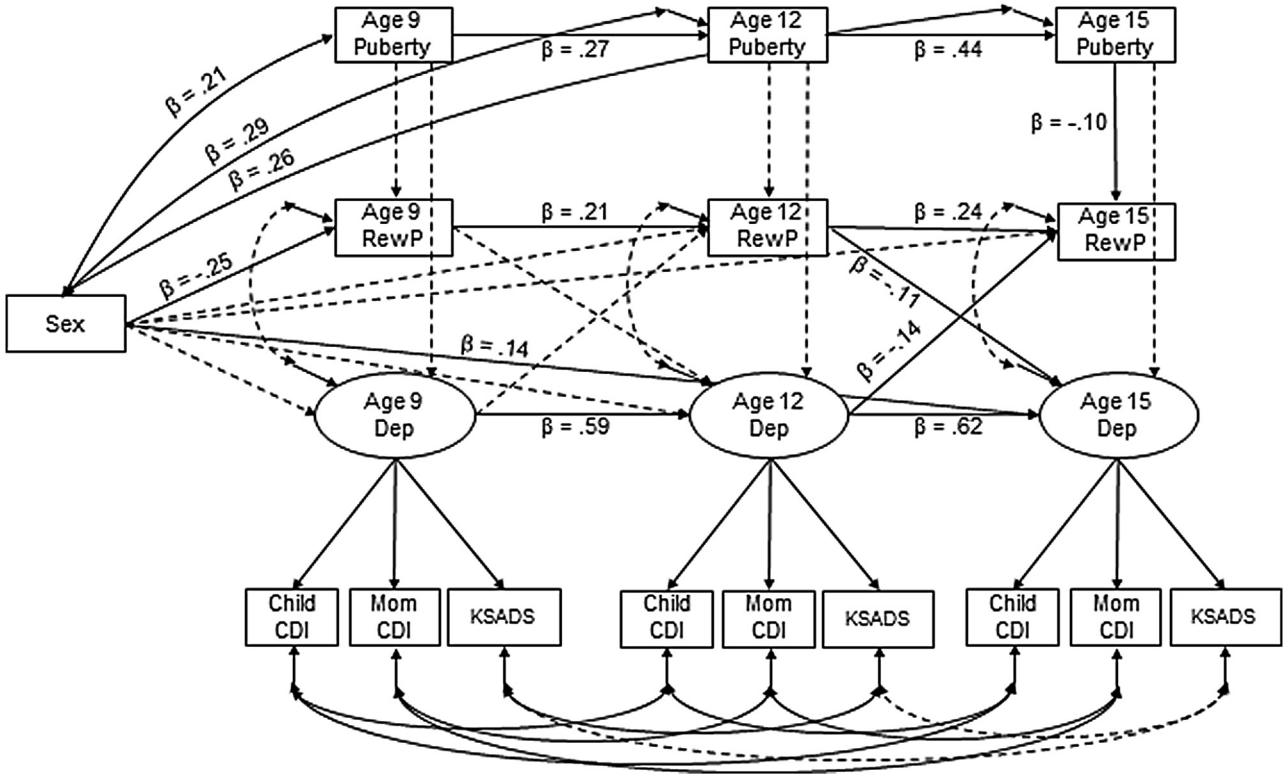
TABLE 3 Structural Equation Model Examining Associations Between Reward Responsiveness, Depression, Biological Sex, and Pubertal Development Across Childhood and Adolescence

	Regression outcome variables															
	Age 9				Age 12						Age 15					
	RewP		Depression		PDS		RewP		Depression		PDS		RewP		Depression	
Predictor variables	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
Biological sex	−.25	< .001	−.12	.06	—	—	−.03	.50	.09	.16	—	—	.07	.17	.14	< .01
Age 9 PDS	.05	.25	.07	.25	.27	< .001	—	—	—	—	—	—	—	—	—	—
Age 9 RewP	—	—	—	—	—	—	.21	< .01	−.05	.33	—	—	—	—	—	—
Age 9 depression	—	—	—	—	—	—	.02	.72	.59	< .001	—	—	—	—	—	—
Age 12 PDS	—	—	—	—	—	—	.04	.40	−.03	.64	.44	< .001	—	—	—	—
Age 12 RewP	—	—	—	—	—	—	—	—	—	—	—	—	.24	< .001	−.11	.04
Age 12 depression	—	—	—	—	—	—	—	—	—	—	—	—	−.14	.04	.62	< .001
Age 15 PDS	—	—	—	—	—	—	—	—	—	—	—	—	−.10	< .05	.06	.23

	Covariance estimates											
	Age 9				Age 12				Age 15			
	PDS		Depression		PDS		Depression		PDS		Depression	
	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
Biological sex	.21	<001	—	—	.29	< .001	—	—	.26	< .001	—	—
Age 9 RewP	—	—	−.04	.57	—	—	—	—	—	—	—	—
Age 12 RewP	—	—	—	—	—	—	.05	.47	—	—	—	—
Age 15 RewP	—	—	—	—	—	—	—	—	—	—	.60	.38

Note: *Age 9 latent depression mean = −0.05; age 12 latent depression mean = 0.31; age 15 latent depression mean = 3.33. Biological sex is coded as dichotomous with higher scores reflecting female sex. The score of the latent depression variable was based on scores from the youth-reported Children's Depression Inventory, mother-reported Children's Depression Inventory, and interviewer-scored Schedule for Affective Disorders and Schizophrenia for School-Age Children depression score for the past month.*
PDS = Pubertal Development Scale; RewP = reward positivity.

FIGURE 2 Structural Equation Model Examining Associations Between Reward Positivity (RewP), Depression, Puberty, and Biological Sex at Ages 9, 12, and 15



Note: *Significant paths are depicted with solid lines, and nonsignificant paths are depicted with dashed lines. Observed variables are depicted with boxes, and latent variables are depicted with ovals. Regression paths are depicted with single-headed arrows, and correlations are depicted with double-headed arrows. Age 9 latent depression mean = −0.05; age 12 latent depression mean = 0.31; age 15 latent depression mean = 3.33. Biological sex is coded as dichotomous with higher scores reflecting female sex. CDI = Children's Depression Inventory; Dep = depression; KSADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children. Please note color figures are available online.*

in the RewP across age groups. However, our results differ from a prior longitudinal study that examined a community sample of 8- to 14-year-old female participants twice across 2 years and found increases in neural responses to gains among younger participants.[26] The 2 studies differed in that participants in the earlier study[26] had a larger age range (8-14 years at baseline) and were all female.

Consistent with prior work,[12,17,18] lower reward responsiveness at age 12 predicted increased depression at age 15, even accounting for age 12 depression. These findings did not extend to the models that examined neural response to gain and loss, suggesting that the effects of age 12 reward responsiveness on age 15 depression are driven by the combination of both gains and losses (in opposite directions) as indexed by the RewP difference score. The role of reward responsiveness in positive reinforcement is central to behavioral theories of depression[45] and is the conceptual basis of behavioral activation treatment for depression.[46] The estimation of the reward responsiveness–depression associations accounted for prior electrocortical responses, biological sex, and age and pubertal status at each wave, which may also partially explain the absence of significant cross-sectional associations between reward responsiveness and depression. Importantly, the current study is the first to examine these relations simultaneously across 3 assessment points, which cover the critical developmental period spanning late childhood through middle adolescence.

The impact of depression on subsequent reward responsiveness was nonsignificant from age 9 to 12. However, greater depression at age 12 predicted lower reward responsiveness at age 15, even accounting for age 12 reward responsiveness. This pattern was also evident when examining neural response to gains, but not when examining neural response to losses, suggesting that this process may be driven by neural response to gains more than losses. To our

TABLE 4 Model and Wald χ^2 Parameter Comparisons of Means and Path Coefficients

Model comparisons	χ^2	*df*	*p*
Depression			
Depression magnitude	53.11	5	< .001
Age 9 vs age 12	49.51	3	< .001
Age 9 vs age 15	38.25	3	< .001
Age 12 vs age 15	39.04	3	< .001
Wald χ2 comparisons			
Depression			
Depression stability	3.08	1	.08
RewP			
RewP magnitude	4.54	2	.10
RewP stability	0.001	1	.97
RewP-depression			
RewP predicts depression cross-lagged paths	0.91	1	.34
Depression predicts RewP cross-lagged paths	1.13	1	.29
Puberty			
Puberty magnitude	47.68	2	< .001
Age 9 vs age 12	0.20	1	.65
Age 9 vs age 15	47.39	1	< .001
Age 12 vs age 15	17.72	1	< .001
Puberty stability	0.81	1	.37
Puberty–RewP			
Puberty predicts concurrent RewP	5.99	2	.05
Age 9 vs age 12	0.22	1	.64
Age 9 vs age 15	4.52	1	.03
Age 12 vs age 15	4.16	1	.04
Puberty–depression			
Puberty predicts concurrent depression	1.77	2	.41
Biological sex-RewP			
Biological sex predicts RewP	25.21	2	< .001
Age 9 vs age 12	10.38	1	.01
Age 9 vs age 15	24.57	1	< .001
Age 12 vs age 15	1.91	1	.17
Biological sex–depression			
Biological sex predicts depression	11.28	2	< .01
Age 9 vs age 12	4.24	1	.04
Age 9 vs age 15	9.48	1	< .01
Age 12 vs age 15	0.73	1	.39
Puberty–biological sex			
Puberty–biological sex covariance	6.68	2	.04
Age 9 vs age 12	6.68	1	.01
Age 9 vs age 15	0.70	1	.40
Age 12 vs age 15	3.51	1	.06

Note: *Comparisons investigating whether depression levels changed across waves were conducted by constraining the depression variables to equality, progressively freeing these parameters, and comparing* χ^2 *scores. All other parameter comparisons were conducted using Wald* χ^2 *tests. The latent depression variable was based on scores from the youth-reported Children's Depression Inventory, mother-reported Children's Depression Inventory, and interviewer-scored Schedule for Affective Disorders and Schizophrenia for School-Age Children depression score for the past month. Pubertal status was based on the Pubertal Development Scale.*
RewP = reward positivity.

knowledge, no prior longitudinal studies have examined the influence of depression on later measures of the RewP. The present study suggests that depression may have a scarring effect[19] on reward responsiveness that persists even after accounting for concurrent depression symptoms. Therefore, low levels of reward responsiveness appear to be both a vulnerability for later depression and an outcome of prior depression. It is possible that this is a result of stress generation; individuals who experience or are predisposed to depression may behave in ways the contribute to increased stress in their environment, which subsequently contributes to reward processing dysfunction.[4,17,47] Psychosocial and pharmacological interventions for depression may be successful due to their ability to partially alleviate reward-related dysfunction,[48] interrupting this cascade of events.

Consistent with prior literature,[30] girls reported greater levels of depression than boys at age 15, and follow-up comparisons indicated that this association began to emerge at age 12. We also found that female participants exhibited reduced reward responsiveness relative to male participants at age 9. Lower reward responsiveness in female youth, which is consistent with prior research,[24] may be a risk factor that contributes to later depression and the associated sex differences that emerge during adolescence. It is possible that sex differences in reward responsiveness become obscured at later ages when considering the impact of other factors (eg, earlier pubertal development of female youth). Alternatively, the RewP may index different psychological processes at younger ages (eg, learning) compared with older ages (eg, consummatory reward).

More advanced pubertal development at age 15 was associated with reduced reward responsiveness. This finding is consistent with previous research showing that more pubertally advanced adolescents demonstrate reduced reward responsiveness in response to rewards compared with their same-aged peers.[49]

The current study has noteworthy strengths, including being the first to examine bidirectional associations between reward responsiveness and depression from late childhood through middle adolescence. This provides additional specificity regarding the ordering and timing of effects. Additionally, our measure of depression integrates self-reported, mother-reported, and interviewer-rated symptoms of depression, providing a comprehensive assessment integrating unique information from multiple sources[50] and reducing the impact of measurement error to provide a more accurate estimate of effects. Finally, the present investigation incorporated the effects of pubertal development and biological sex, which are critical to changes observed in reward responsiveness and rates of depression during this developmental stage.

However, the study also has several limitations. We used a cross-lagged panel model (CLPM), which cannot distinguish within- and between-person change.[51] While we attempted to fit the current data to more complex models, such as the random intercept CLPM, their estimation failed, possibly because convergence of these models often requires larger samples and more than 3 waves of data. However, it has been argued that the CLPM may be preferable for examining causal hypotheses with longitudinal data because the random intercept CLPM focuses on fluctuations around the individual person means, but does not consider potential between-person effects.[52] Nonetheless, future work should examine more complex models that can distinguish within- and between-person change in reward responsiveness and depression. Second, while assessments every 3 years in a sample of same-aged youth allows for establishing temporal relations between reward responsiveness and depression, studies using more closely spaced intervals may provide a fine-grained examination of these processes. Third, pubertal development was assessed using self-report, and the reliability of the PDS was limited in our sample, probably owing to the restricted ranges at ages 9 and 15. Fourth, the RewP demonstrated slight to fair split-half reliability in our sample across ages. While this is consistent with prior studies, it also may limit our ability to detect potentially significant associations. Lastly, the sample was largely White/non-Hispanic, and it is not clear whether the current findings generalize to other populations. Additional work is necessary to examine these associations in other populations.

The current study adds to the growing literature investigating associations between reward responsiveness and depression. Critically, it involved 3 assessments of reward responsiveness and depression symptoms spanning late childhood through middle adolescence and incorporated developmental factors known to impact neural development and depression during this key period. Our findings suggest that there are bidirectional prospective effects between reward responsiveness and depression that emerge between ages 12 and 15, indicating that this may be an especially important window for studying bidirectional reward responsiveness–depression associations across time. Future work should consider potential moderating effects of biological sex in the association between reward responsiveness and depression as well as examine whether the bidirectional associations between reward responsiveness and depression are present when considering other forms of psychopathology across development.

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Mr. Mackin and Drs. Mumper, Nelson, and Klein are with Stony Brook University, New York. Dr. Goldstein is with Connecticut School of Medicine, Farmington. Dr. Kujawa is with Vanderbilt University, Tennessee. Dr. Kessel is with Columbia University, New York. Dr. Olino is with Temple University, Philadelphia, Pennsylvania. Dr. Hajcak is with Florida State University, Tallahassee.

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The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

All procedures in the current study were approved by the Stony Brook University Institutional Review Board.

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Dr. Olino served as the statistical expert for this research.

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Investigation: Mackin, Mumper, Kujawa, Kessel, Olino
Methodology: Mackin, Goldstein, Olino, Klein
Project administration: Klein
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Software: Mackin, Goldstein, Olino
Supervision: Goldstein, Kujawa, Klein
Visualization: Mackin
Writing — original draft: Mackin, Klein
Writing — review and editing: Goldstein, Kujawa, Kessel, Olino, Nelson, Hajcak

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