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Greater Cumulative Lifetime Stressor Exposure Predicts Blunted Reward Positivity in Adolescent Girls Followed for 2 Years

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ABSTRACT

BACKGROUND: Although research has found that life stress is associated with reward-related brain activity, few studies have examined how cumulative stressors occurring over the entire lifetime affect reward processing during adolescence.

METHODS: To address this issue, we investigated how lifetime stressor exposure related to reward processing, indexed by the reward positivity, in 240 adolescent girls between ages 8 and 14 years (mean age = 12.4). Participants were followed for 2 years. They completed a reward task at baseline and follow-up and the Stress and Adversity Inventory at follow-up.

RESULTS: As hypothesized, greater lifetime stressor exposure was related to a blunted reward positivity at the follow-up session while controlling for baseline age, baseline reward positivity, and time between assessments. Furthermore, this association was evident for acute but not chronic lifetime stressors.

CONCLUSIONS: These data suggest that the development of adaptive reward processing may be adversely affected by experiencing major life stressors. The results may thus have implications for understanding how stressors increase risk for psychopathology, such as major depressive disorder.

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Reward processing has been implicated in the etiopathogenesis of a wide variety of psychiatric disorders, including major depressive disorder (MDD) (1). The construct of reward processing involves multiple brain regions and subconstructs including reward anticipation, motivation, and pleasure (2). An abundance of research has focused on how reward-related processes are altered in persons with depression (3,4). Few studies, however, have examined social-environmental factors that may affect the development of adaptive reward processing.

Research using event-related potentials (ERPs) has identified the reward positivity (RewP) as an index of reward processing that is evident approximately 250 to 350 ms following the onset of feedback indicating rewards versus losses (5). Previous electroencephalography (EEG) research has referred to the RewP as the feedback-related negativity (6), feedback error–related negativity (7), or feedback negativity (8) to highlight the apparent negative deflection in the ERP that is evident following the presentation of losses. However, more recent research has suggested that reward-related ERPs are characterized by a relative positivity that is reduced or absent following nonreward outcomes (9–11). Consequently, researchers have begun using the term reward positivity to highlight the presence of a positive deflection following the presentation of rewards (5).

A larger RewP has been shown to be related to increased ventral striatal activation when receiving rewards, self-reported sensitivity to rewards, and reward bias to alter behavior following rewards (12–15). The RewP has good reliability in adolescents and adults (16,17), situating the RewP as a neural marker with psychometric properties that make it suitable for examining individual differences in reward processing. In support of this idea, many studies have found that variability in the RewP is related to depression (18–20), such that a smaller RewP is related to or predicts greater depressive symptoms in children and adults (21–25). Despite its attractive psychometric properties and predictive ability, relatively little work has examined social-environmental factors that affect the development of the RewP.

Reward Processing in Adolescence

Adolescence is a developmental period characterized by substantial changes in both depression and reward system functioning (26,27), and several ERP studies have examined normative developmental differences in reward-related ERPs during adolescence. In one comprehensive study, Burani *et al.* (28) used a within-subjects, longitudinal design to investigate age-related changes in the RewP over 2 years in 8- to 14-year-old adolescent girls. They found that older youth exhibited a

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larger RewP at the baseline visit and the follow-up visit; furthermore, the RewP increased over the 2-year adolescent period, with younger individuals demonstrating greater increases in the RewP over time. Collectively, this evidence suggests that reward-related brain activity undergoes significant developmental changes during adolescence. However, the social-environmental factors that predict changes in the RewP remain unclear.

Stress and the RewP

Exposure to significant life stressors is one factor that may influence the development of the RewP over time. For example, life stress has been related to behaviors indicative of an atypical reward system (29,30) as well as reward processing in both cross-sectional and longitudinal studies (31,32). Indeed, functional magnetic resonance imaging studies in children and adolescents indicate that exposure to life stressors, including early-life stressors such as childhood maltreatment, abuse, and neglect, predict less activity in reward-related circuits (33). Moreover, individuals who have experienced higher early-life stress between birth and 4 years of age have been found to exhibit dampened reward-related activity at age 7 years (34). Similarly, life stressor exposure occurring during adolescence longitudinally predicted reduced activity in the medial prefrontal cortex during reward anticipation and receipt during young adulthood in males (35).

Along similar lines, Hanson *et al.* (31) found that early-life stressors occurring in children and adolescents predicted blunted ventral striatum activity in adulthood. Overall, although correlational, this evidence suggests that life stressor exposure may blunt activity in brain regions that are implicated in the reward system. Finally, in terms of the RewP, one study found that childhood trauma was cross-sectionally related to a blunted RewP in children who had a mother with a history of depression as compared with a low-risk group (36). At the same time, we know of no studies that have investigated how stressors occurring over the entire life course are related to longitudinal changes in the RewP in young adolescents.

Present Study

To address this issue, we examined how cumulative lifetime stressor exposure related to change in the RewP in adolescents both cross-sectionally and over time. To this end, we recruited 8- to 14-year-old adolescent girls who participated at baseline and follow-up laboratory visits separated by 2 years. At both visits, participants completed a simple guessing reward paradigm in which rewards and losses were equiprobable to measure the RewP. In addition, cumulative lifetime stressor exposure, indexed as the total number of stressors experienced over the entire lifespan, was assessed at the follow-up visit using the Stress and Adversity Inventory for Adolescents (STRAIN) (37).

Given prior longitudinal functional magnetic resonance imaging work finding links between life stressor exposure and reward processing (35), we hypothesized that adolescents who were exposed to more cumulative lifetime stressors would exhibit a blunted RewP cross-sectionally at the 2-year follow-up visit and blunted residual change after accounting for the baseline RewP. In addition, we conducted exploratory analyses to examine whether these differences in RewP were similar for acute versus chronic stressors occurring over the life course. Finally, to confirm that stressor exposure actually predicted subsequent changes in the RewP, we reran the main analysis predicting RewP at follow-up from lifetime stressor exposure while restricting the stressor exposure variable to stressors that occurred only prior to the baseline study visit.

METHODS AND MATERIALS

Participants were recruited from Long Island, New York, as part of a longitudinal study examining developmental changes in reward processing and depression. At baseline, the sample included 317 adolescent girls between ages 8 and 14 years ($mean_{age}$ = 12.4 years, SD = 1.8). Of these 317 participants, 3 did not complete the reward task, and 9 were excluded owing to poor EEG data quality. Therefore, the final sample at baseline included 305 participants ($mean_{age}$ = 12.4 years, SD = 1.8). Two years later, 258 participants (84.6%) returned to the laboratory for a follow-up visit ($mean_{age}$ = 14.4 years, SD = 1.8). Ten participants were excluded from the follow-up because of poor EEG data. Overall, the EEG data included 240 participants with data at both visits available for analysis. Of those with available data, 229 participants completed the lifetime stressor assessment. Therefore, the final sample included in these analyses comprised 229 participants.

This sample was predominantly Caucasian (87.3%), with the remaining participants self-identifying as African American (6.6%), American Indian/Alaskan Native (0.9%), and Other (5.2%). The average household income was $139,368 (SD = $112,386). All participants and their parents provided informed assent and consent, respectively, and all study procedures were preapproved by the Institutional Review Board at Stony Brook University. RewP data from this manuscript have been published elsewhere (28,38,39), but the present analysis is the first to examine how lifetime stressor exposure is related to changes in reward processing in this sample.

Stress and Adversity Inventory

Lifetime stressor exposure was assessed at the follow-up session (n = 229 with usable EEG data) using the STRAIN (see https://www.strainsetup.com/) (37). Participants reported on 33 acute life events and 42 chronic difficulties for a total of 75 stressors that spanned 12 primary life domains (i.e., housing, education, work, treatment/health, marital/partner, reproduction, financial, legal/crime, other relationships, guardian/parent relationships, death, life-threatening situations) and 5 social-psychological characteristics (i.e., interpersonal loss, physical danger, humiliation, entrapment, role change/disruption). For each stressor endorsed, the STRAIN generates additional questions assessing the stressor's severity, frequency, exposure timing, and duration. Acute life events in the STRAIN system are defined as stressors lasting a few days, such as learning of a death, getting fired, or being physically attacked (35). Chronic stressors, in turn, typically last a minimum of 1 month, though many last longer, such as persistent educational, housing, or financial problems (35).

Based on participants' responses, the STRAIN produces stressor exposure (i.e., total number of stressors experienced) and severity summary scores for individuals' total lifetime

stressor exposure as well as separately for acute versus chronic stressors. Higher scores on the STRAIN always indicate greater stressor exposure. The STRAIN has strong concurrent and predictive validity as evidenced by its association with other measures of childhood adversity and with measures of various psychiatric symptoms and diagnoses (37,40,41). In addition, the STRAIN has demonstrated excellent test-retest reliability (42).

Doors Task

The doors task is a monetary reward paradigm. In each trial, participants were presented with 2 doors, displayed side-by-side, and were instructed to select the door that they believed would yield a prize (i.e., money) using the left or right mouse button. After a participant made their decision, a fixation cross was then presented for 1500 ms, followed by feedback indicating whether they won (i.e., a green arrow pointing upward, which signified +$0.50) or lost (i.e., a red arrow pointing downward, which signified −$0.25); this feedback was presented for 2000 ms. Following each trial, text on the screen instructed participants to "Click for next round," and a fixation cross presented for 1000 ms followed that click before the next trial began. A total of 30 gain and 30 loss trials were presented pseudorandomly using Presentation version 17.0 (Neurobehavioral Systems). Participants were told that they had a chance to earn up to $15; all participants were given $8 at the end of the task.

EEG Processing

Continuous EEG data were recorded while participants completed the doors task using the ActiveTwo BioSemi system (BioSemi) with an elastic cap containing 34 electrode sites placed according to the 10/20 system (i.e., 32 channels plus Iz and FCz). Facial electrodes were placed above and below the left eye and near the outer canthi of the left and right eyes to monitor horizontal and vertical electro-oculographic activity. Two additional 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 processed offline using BrainVision analyzer 2.1 (Brain Products). Raw EEG data were re-referenced to the average of the left and right mastoids and then filtered from 0.1 to 30 Hz using a second-order Butterworth filter. The EEG data were then segmented from −200 ms prior to the onset of feedback and up to 1000 ms following feedback. Eyeblinks and ocular movement correction was performed using the Gratton, Coles, and Donchin regression–based method (43).

Prior to averaging the data as a function of feedback type, segments containing a voltage >50 μV between consecutive sample points, a voltage difference of 300 μV within a segment, or a maximum voltage difference of <0.5 μV within 100-ms intervals were identified as artifacts and automatically rejected. The 200-ms prefeedback interval was used for baseline correction. Consistent with Burani *et al.* (38,39), the RewP at both time points were scored as the mean activity within a 100-ms time window around the most positive peak of the gain minus loss difference waveform extracted from a 200 to 400 ms time window at FCz for each participant. The area around the peak of the difference score was used in the present study to stay consistent with our previous work, and past EEG research indicates that this measure better accounts for individual variation in the peak amplitude (44,45). We computed internal consistency estimates for the RewP using $\rho_{DD'}$ based on recommendations by Clayson *et al.* (46). For the baseline RewP and the follow-up RewP, the internal consistency estimates were $\rho_{DD'} = 0.30$ and $\rho_{DD'} = 0.47$, respectively.

Data Analysis

All analyses were conducted in R 4.0.3 (47). Bivariate correlations were conducted using the Psych R package (48) to examine relations among participants' baseline age, follow-up age, baseline RewP, lifetime stressor exposure, lifetime chronic stressor exposure, lifetime acute stressor exposure, and their RewP at follow-up. To examine the cross-sectional association between lifetime stressor exposure and the RewP at follow-up, 2 separate linear regressions were conducted. In the first regression, total lifetime stressor exposure was entered as an independent variable, controlling for follow-up age. In the second regression, lifetime chronic stressor exposure and lifetime acute stressor exposure were entered as predictors, controlling for follow-up age. To assess residual change in the RewP, a similar approach as the one above was taken.

In the third regression, total lifetime stressor exposure was entered as the independent variable. In the fourth regression, lifetime chronic stressor exposure and lifetime acute stressor exposure were entered as predictors. As a secondary analysis, we conducted a fifth regression with prebaseline lifetime stressor exposure entered as an independent variable, which we constructed by removing all stressors occurring between the baseline and follow-up visits from the total lifetime stressor exposure variable. This enabled us to ensure the correct temporal ordering for lifetime stressor exposure vis-à-vis subsequent residual changes in RewP. The third, fourth, and fifth regressions included baseline RewP, baseline age, and time between assessments (follow-up age minus baseline age) as covariates. All regressions were conducted using Stats R package (47).

RESULTS

Preliminary Analyses

Table 1 depicts the means and standard deviations of the study variables along with the bivariate correlations among variables. Older participants at baseline had a larger RewP at baseline and experienced more total lifetime stressors, lifetime chronic stressors, and lifetime acute stressors. In addition, greater lifetime acute stressor exposure was associated with a smaller RewP at follow-up.

Cross-sectional Analyses

Lifetime Stressor Exposure. We examined whether total lifetime stressor exposure was cross-sectionally associated

Table 1. Means, Standard Deviations, and Correlations Among the Key Study Variables

Study Variable	1	2	3	4	5	6	7
1 Baseline Age	–	–	–	–	–	–	–
2 Follow-up Age	0.98[a]	–	–	–	–	–	–
3 Lifetime Stressor Count	0.31[a]	0.29[a]	–	–	–	–	–
4 Lifetime Chronic Stressor Count	0.32[a]	0.31[a]	0.91[a]	–	–	–	–
5 Lifetime Acute Stressor Count	0.27[a]	0.25[a]	0.95[a]	0.73[a]	–	–	–
6 Baseline RewP	0.14[b]	0.13[b]	−0.01	0.03	−0.03	–	–
7 Follow-up RewP	−0.01	−0.03	−0.15[b]	−0.10	−0.17[a]	0.25[a]	–
Mean (SD)	12.3 (1.8)	14.3 (1.8)	13.5 (10.3)	5.6 (4.7)	7.9 (6.4)	5.5 (6.2)	6.0 (5.5)

RewP, reward positivity.
[a]$p < .01$.
[b]$p < .05$.

with the RewP at follow-up, controlling for age at follow-up. Results are presented in Table 2. As hypothesized, greater lifetime stressor exposure was associated with a smaller RewP at follow-up. Age at follow-up was not significantly associated with the RewP at follow-up.

Acute Versus Chronic Lifetime Stressor Exposure. Next, we examined whether these effects differed for acute versus chronic stressors. Results are presented in Table 3. Greater lifetime acute, but not chronic, stressor exposure was associated with a blunted RewP at follow-up. Age at follow-up was not significantly associated with the RewP at follow-up.

Longitudinal Analyses

Lifetime Stressor Exposure. We examined whether total lifetime stressor exposure predicted residual changes in RewP from baseline to follow-up (i.e., RewP at follow-up, controlling for RewP at baseline), while also controlling for baseline age and time between assessments. Results are presented in Table 4. As hypothesized, greater lifetime stressor exposure was associated with a smaller RewP at follow-up (see Figures 1 and 2)[1]. In addition, a larger baseline RewP was associated with a larger RewP at follow-up. However, baseline age and time between assessments did not relate to the RewP at follow-up.

Acute Versus Chronic Lifetime Stressor Exposure. We next conducted analyses to determine whether these effects differed for acute versus chronic stressors occurring over the lifespan. The results are presented in Table 5. Greater lifetime acute, but not chronic, stressor exposure was related to a smaller RewP at follow-up[2]. A larger baseline RewP was associated with a larger RewP at follow-up; however, baseline age and time between assessments was not associated with the RewP at follow-up. Therefore, greater lifetime acute stressor exposure was associated with a blunted RewP.

[1]Neither baseline age (p = .728; 95% CI_b, −0.03 to 0.04) nor baseline RewP (p = .954; 95% CI_b, −0.01 to 0.01) interacted with lifetime stressor exposure to predict the RewP at follow-up.

[2]Neither baseline age (p = .367, 95% CI_b, −0.03 to 0.09) nor baseline RewP (p = .859; 95% CI_b, −0.02 to 0.02) interacted with lifetime acute stressor exposure to predict the RewP at follow-up.

Secondary Analysis. Finally, given that the lifetime stressor exposure and the 2 RewP assessments overlapped in time, we examined whether lifetime stressor exposure occurring prior to the baseline visit was associated with the RewP at follow-up. This analysis enabled us to ensure the correct temporal ordering for lifetime stressor exposure vis-à-vis subsequent residual changes in the RewP. The results indicated that pre-baseline lifetime stressor exposure was significantly related to a blunted RewP at follow-up (Table 6), suggesting that lifetime stressors predict the subsequent development of the RewP over time.

DISCUSSION

Although life stress is thought to affect reward processing, no studies to date have investigated how stressors occurring over the entire life course are related to subsequent changes in reward processing. We addressed this important issue by examining how lifetime stressor exposure was related to the RewP in adolescent girls who were followed over a 2-year period. We previously found that the RewP increased from baseline to follow-up (28). In the present study, we extended this prior work by showing that greater lifetime stressor exposure was cross-sectionally associated with a blunted RewP at follow-up. In addition, greater lifetime stressor exposure predicted a smaller residual increase in RewP from baseline to follow-up, controlling for baseline RewP, baseline age, and time between assessments. These cross-sectional and longitudinal associations were evident for exposure to acute (but not chronic) stressors over the life course. Moreover, the main finding remained consistent while restricting the analysis to only include lifetime stressors occurring prior to the baseline study visit. Overall, although correlational, these results are consistent with the possibility that greater lifetime

Table 2. Results of the Multiple Linear Regression Predicting RewP at Follow-up From Lifetime Stressor Count While Covarying for Follow-up Age

Predictor	*b*	95% CI	*p*
Follow-up Age	0.03	−0.37 to 0.42	.892
Lifetime Stress Count[a]	−0.09	−0.16 to −0.02	.017

RewP, reward positivity.
[a]$p < .05$.

Table 3. Results of the Multiple Linear Regression Predicting RewP at Follow-up From Lifetime Acute Stressor Count and Lifetime Chronic Stressor Count While Covarying for Follow-up Age

Predictor	*b*	95% CI	*p*
Follow-up Age	−0.01	−0.41 to 0.39	.953
Lifetime Acute Stress Count[a]	−0.19	−0.34 to −0.03	.020
Lifetime Chronic Stress Count	0.06	−0.16 to 0.28	.573

RewP, reward positivity.
[a]$p < .05$.

stressor exposure may adversely affect the development of adaptive reward processing.

Our finding that lifetime stressor exposure was related to a blunted RewP is consistent with functional magnetic resonance imaging work on this topic, which has generally found blunted activity in brain regions implicated in reward processing, such as the ventral striatum and the medial prefrontal cortex, among individuals who have experienced greater lifetime stressor exposure (31,35). Collectively, such results indicate that greater lifetime stressor exposure reduces reward-related brain activity. Regarding potential mechanisms underpinning these associations, it is possible that elevated levels of cortisol related to stress exposure may dampen reward-related brain activity (33,49) because receptors for glucocorticoids are present throughout brain regions implicated in reward processing (50). Alternatively, substantial research has found that greater stressor exposure is associated with heightened inflammatory activity (51,52), and an extensive body of work also has shown that inflammatory activity induces anhedonia (53–55). Therefore, it is possible that stress predicts a blunted RewP through its effects on inflammation. Future work should examine these and other potential mechanisms.

The present results suggest that the cumulative effects of short-term, acute stressors may contribute to changes in reward system functioning more than chronic stressors. Acute stressors in the STRAIN are major life events that typically last at least a few days and involve significant cognitive upheaval, such as learning of a death, getting fired, or being physically attacked (37); in contrast, chronic stressors are typically present for at least 1 month and include stressors such as persistent educational, housing, or financial problems (37). Although acute stressors may be relatively short lived, they have cognitive and emotional consequences that can persist and greatly affect mental and physical health. Indeed, a large body of research has shown that acute stressors are the strongest proximal predictors of MDD (22,24). The present results are consistent with this body of work and suggest that acute stressors may blunt reward processing and, in turn, increase vulnerability to MDD.

Table 4. Results of the Multiple Linear Regression Predicting RewP at Follow-up From Lifetime Stressor Count While Covarying for Baseline RewP, Baseline Age, and Time Between Visits

Predictor	*b*	95% CI	*p*
Baseline Age	−0.04	−0.44 to 0.36	.837
Baseline RewP[a]	0.23	0.12 to 0.34	<.001
Time Between Visits	−0.99	−2.98 to 0.98	.320
Lifetime Stress Count[b]	−0.08	−0.15 to −0.01	.030

RewP, reward positivity.
[a]$p < .001$.
[b]$p < .05$.

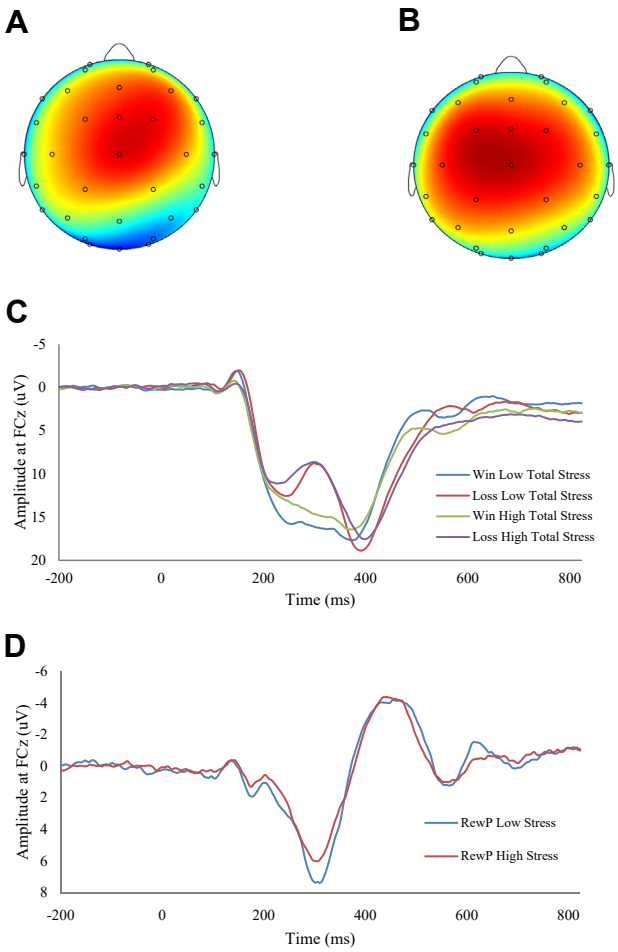

Figure 1. Scalp topography for individuals with **(A)** high lifetime stressor count and **(B)** low lifetime stressor count. **(C)** Participants' event-related potentials to wins (green) and losses (purple) for those with high lifetime stressor count, and the event-related potentials to wins (blue) and losses (red) for those with low lifetime stressor count. **(D)** The reward positivity (RewP) difference waveform for those with high (red) and low (blue) lifetime stressors count. The scalp distribution and waveforms were created using a median split of lifetime stressor count.

The present results have implications for understanding risk for the development of psychopathology in adolescence, particularly regarding the onset and maintenance of depressive symptoms and MDD. We found that greater lifetime stressor exposure was related to blunted reward-related brain activity. Research has shown that a blunted RewP relates to increased depressive symptoms (22,24) and predicts the onset of MDD in adolescence (25). Therefore, elevated levels of life stress may increase the possibility that an individual develops depression partly through blunting the RewP and activity in other reward-related brain regions (56).

There are several strengths to this study. First, we measured the RewP longitudinally, over the course of 2 years, in a

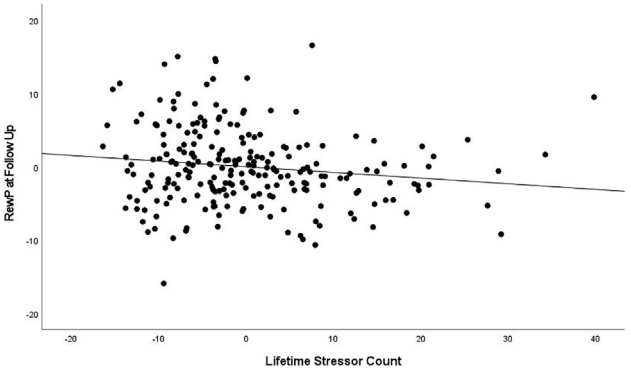

Figure 2. A scatterplot of the association between lifetime stressor count and the reward positivity (RewP) at follow-up, residualized for baseline reward positivity, baseline age, and time between assessments. The scatterplot indicates a negative association such that greater lifetime stressor count relates to a more blunted reward positivity.

relatively large sample. Second, we assessed stressor exposure occurring over the entire lifetime using the STRAIN. Finally, in accordance with a stressor characteristics view on life stress and depression (57), we demonstrated that the effects of stress on the RewP are not uniform across different types of stressors but, rather, are evident for acute, but not chronic, stressors.

Several limitations should also be noted. First, the sample was entirely female, and it is unknown whether these results generalize to males. Therefore, future research should examine whether life stressors predict a blunted RewP in a sample that includes both genders, to evaluate the potential impact of sex and gender, and in samples with a wider age range. Second, participants were drawn from a relatively affluent area of the United States and were largely White; hence, the generalizability of our findings to more racially or socioeconomically diverse populations, or to at-risk populations, is unclear. It will be important to examine how life stressor exposure impacts the RewP in samples with greater levels of stress (e.g., who live in areas of high poverty) and among children at increased risk for depression (e.g., those with maternal history of depression). Such studies should help to further clarify the roles of acute and chronic stressors in reward-related neurodevelopment, and they would be well suited to examine depression as a subsequent outcome.

Table 5. Results of the Multiple Linear Regression Predicting RewP at Follow-up From Lifetime Acute Stressor Count and Lifetime Chronic Stressor Count While Covarying for Baseline RewP, Baseline Age, and Time Between Visits

Predictor	*b*	95% CI	*p*
Baseline Age	−0.06	−0.47 to 0.34	.762
Baseline RewP[a]	0.23	0.11 to 0.34	<.001
Time Between Visits	−1.13	−3.11 to 0.86	.264
Lifetime Acute Stress Count[b]	−0.17	−0.33 to −0.01	.041
Lifetime Chronic Stress Count	0.05	0.17 to 0.27	.641

RewP, reward positivity.
[a]$p < .001$.
[b]$p < .05$.

Table 6. Results of the Multiple Linear Regression Predicting RewP at Follow-up From Prebaseline Lifetime Stressor Count While Covarying for Baseline RewP, Baseline Age, and Time Between Visits

Predictor	*b*	95% CI	*p*
Baseline Age	−0.08	−0.48 to 0.31	.678
Baseline RewP[a]	0.23	0.12 to 0.34	<.001
Time Between Assessments	−0.96	−2.95 to 1.03	.345
Prebaseline Lifetime Stress Count[b]	−0.13	−0.26 to −0.003	.046

RewP, reward positivity.
[a]$p < .001$.
[b]$p < .05$.

Third, although the STRAIN addresses several shortcomings of self-report stressor measures (58), it can still be affected by retrospective bias in that individuals are asked to recall life events that occurred across their lifetime. Furthermore, recall of life stressors may be affected by symptoms of depression, specifically anhedonia (59). Finally, there is recent evidence to suggest that individuals with a blunted RewP generated more stressful life events (60), which is consistent with stress generation models. We partly addressed this issue by conducting a secondary analysis that only included stressors occurring prior to our assessment of residual changes in the RewP. Nevertheless, the timing of the association between lifetime stressor exposure and the RewP is uncertain, and future longitudinal studies should be powered specifically to examine how stressors occurring across the life course are associated with reward-related brain activity across multiple stages of development to better elucidate the directionality of this effect.

Notwithstanding these limitations, the present data are the first to demonstrate that greater lifetime stressor exposure is cross-sectionally associated with a blunted RewP. Furthermore, the results demonstrate that greater lifetime stressor exposure was related to a blunted RewP 2 years later in adolescence and, in addition, that these effects were evident for acute (but not chronic) stressors. These results thus have implications for informing developmental models of psychopathology by suggesting that experiences of acute life stress may affect developmental trajectories of reward-related brain activity, which may in turn increase the risk for the development of depression, bipolar disorder, and other forms of psychopathology. Future research should obtain more than 2 assessments of the RewP to examine within-subject changes in the RewP and should include other biological assessments that may help elucidate additional mechanisms underlying these effects. This work would help researchers better understand developmental trajectories of the RewP, how such trajectories might be affected by stress, and how these changes are converted into depressive symptoms.

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