


# Editorial: Evidence for Transactional Relations Between Reward Processing and Depressive Symptoms in Adolescence

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**R**eward processing deficits play a clear role in depression and depression risk. For example, more than a decade of research has shown that individual differences in initial reward responsiveness, indexed by the reward positivity (RewP) event-related potential (ERP) component, are associated with current depression and future depression risk.<sup>1,2</sup> Mackin and colleagues' study<sup>3</sup> builds on this previous literature by asking 2 key questions: (1) Is the magnitude of the impact of RewP on prospective changes in depressive symptoms similar during late childhood and adolescence? and (2) Are prospective links between RewP and depressive symptoms transactional, with depressive symptoms also predicting future change in RewP during this developmental window? These questions are important, because this is a time period during which rates of depression increase dramatically<sup>4</sup> and when there are normative changes in reward processing.<sup>5</sup> However, we know very little about how relations between reward processing and depression may change across development.

The study included 609 youths participating in the Stony Brook Temperament Study who were assessed at ages 9, 12, and 15 years. At each wave of data collection, youths completed an ERP assessment of reward outcome processing, and youths and their mothers' completed measures of youth depressive symptoms. The researchers also included information about youths' biological sex at birth and pubertal level at each assessment. Using structural equation modeling, Mackin *et al.*<sup>3</sup> found that reward processing deficits (indexed as the difference in RewP amplitude following monetary gain vs loss feedback) and depressive symptoms were transactionally related over time between the ages of 12 and 15 years, but not between the ages of 9 and 12, even after accounting for the influence of biological sex and pubertal level at each assessment. That is, a more blunted RewP predicted increases in depressive

symptoms 3 years later during early to middle adolescence (from age 12 to 15 years), but not during late childhood (from age 9 to 12). Similarly, higher levels of depressive symptoms predicted an increasingly blunted RewP 3 years later during adolescence but not during late childhood. The findings were stronger for the RewP difference score than for ERP responses to monetary gains or losses considered individually, suggesting that the results were driven by reduced differentiation of neural response to gains vs losses rather than responses to either outcome considered individually. In addition to these key findings, there were also 2 notable null findings. First, there were no significant changes in the RewP across time points. Second, although the RewP predicted prospective increases in depressive symptoms, it was not cross-sectionally related to depressive symptom levels at any assessment, even when looking at bivariate correlations.

This study represents a significant advancement in the field. In contrast to previous studies, which have often been limited by smaller samples and low statistical power,<sup>6</sup> the current study included a large sample of youths, providing adequate power for the sophisticated models tested. It is also the first to include more than 2 waves of data, which provide, for the first time, the ability to directly compare predictive relations between RewP and depressive symptoms across developmental stages within the same individuals. Finally, it is the first to show that reward processing deficits and depressive symptoms transactionally influence each other during adolescence, highlighting a potential vicious cycle that could exacerbate risk.

As with any study, the current findings also raise as many questions as answers. Building from much of the prior work on the RewP, Mackin *et al.* focused on neural response to monetary gains and losses. However, social rewards are likely to be even more salient during this developmental window, particularly as children age into

adolescence, and researchers have developed standardized tasks to assess neural responses to social acceptance vs rejection feedback.<sup>7</sup> Future research is needed to directly compare the predictive utility of reward processing deficits for social vs nonsocial rewards to determine whether deficits in social reward processing may be an even stronger predictor of depression risk in youths. Another notable feature of this study is that the duration of the follow-up interval between assessments was quite large. Given this, it is possible that the lack of significant prospective effects between age 9 and 12 years in this study simply reflects processes that operate over shorter time intervals (eg, 6-12 months). That is, rather than a distinct age at which these unidirectional or transactional relations are vs are not present, it seems likely that they would first be observed on a time scale of weeks to months before exhibiting predictive validity over the course of several years. A third point that must be acknowledged is that the RewP exhibited “slight to fair” reliability in this study. Because reliability limits validity, this may explain some of the nonsignificant findings. This is a recognized area of concern in ERP research, as well as in other areas of research, and increasing measurement reliability will allow researchers to characterize the role of reward processing deficits in depression risk more precisely. A final question relates to the mechanism(s) linking reward processing and depression during adolescence. Are these processes directly linked or are they mediated by some other factor(s)? One potentially important factor that warrants additional study is interpersonal stress, which is known to

increase during adolescence, appears to exhibit reciprocal relations with both depression and reward processing, and has been hypothesized to mediate the link between reward processing deficits and depression risk.<sup>8</sup>

In summary, Mackin *et al.*'s<sup>3</sup> findings provide novel insights regarding developmental changes in the role of reward processing deficits in youth depression risk. Specifically, they suggest that the impact of reward processing deficits on future increases in youth depression becomes stronger as children age into adolescence, and that reward processing and depression transactionally influence each other during adolescence but not during childhood. Although important questions remain, this is a significant step forward in developmental models of both reward processing and depression.

Accepted March 1, 2023.

Dr. Gibb is with Binghamton University (SUNY), Binghamton, New York.

This work was supported by the National Institute of Mental Health of the National Institutes of Health under Award Number R01MH130397. The content is solely the responsibility of the author and does not necessarily represent the official views of the National Institutes of Health.

Disclosure: Dr. Gibb has reported no biomedical financial interests or potential conflicts of interest.

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0890-8567/\$36.00/©2023 American Academy of Child and Adolescent Psychiatry

<https://doi.org/10.1016/j.jaac.2023.03.001>

## REFERENCES

1. Nelson BD, Perlman G, Klein DN, Kotov R, Hajcak G. Blunted neural response to rewards as a prospective predictor of the development of depression in adolescent girls. *Am Psychiatry*. 2016;173(12):1223-1230. <https://doi.org/10.1176/appi.ajp.2016.15121524>
2. Proudfit GH. The reward positivity: from basic research on reward to a biomarker for depression. *Psychophysiology*. 2015;52:449-459. <https://doi.org/10.1111/psyp.12370>
3. Mackin DM, Goldstein BL, Mumper E, *et al.* Longitudinal associations between reward responsiveness and depression across adolescence. *J Am Acad Child Adolesc Psychiatry*. 2023;62(7):816-828. <https://doi.org/10.1016/j.jaac.2022.11.013>
4. Hankin BL, Abramson LY, Moffitt TE, Silva PA, McGee R, Angell KE. Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *J Abnorm Psychol*. 1998;107(1):128-140.
5. Guyer AE, Silk JS, Nelson EE. The neurobiology of the emotional adolescent: from the inside out. *Neurosci Biobehav Rev*. 2016;70:74-85. <https://doi.org/10.1016/j.neubiorev.2016.07.037>
6. Clayson PE, Carbine KA, Larson MJ. A registered report of error-related negativity and reward positivity as biomarkers of depression: P-curving the evidence. *Int J Psychophysiol*. 2020;150:50-72. <https://doi.org/10.1016/j.ijpsycho.2020.01.005>
7. Kujawa A, Kessel EM, Carroll A, Arfer KB, Klein DN. Social processing in early adolescence: associations between neurophysiological, self-report, and behavioral measures. *Biol Psychol*. 2017;128:55-62.
8. Auerbach RP, Admon R, Pizzagalli DA. Adolescent depression: stress and reward dysfunction. *Harv Rev Psychiatry*. 2014;22(3):139-148. <https://doi.org/10.1097/HRP.000000000000034>

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