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Reward reactivity as a buffer against negative mental health consequences of pandemic-related stress: a preregistered analysis in the human connectome project in development

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ABSTRACT

The COVID-19 pandemic presented numerous novel stressors to youth which have been associated with worsening mental health. Previous work has shown that individuals with high reward sensitivity show resilience in the face of individualized stressors. Here, we sought to investigate whether individuals with high reward sensitivity prior to pandemic onset would be resilient to the community-level stressor of the pandemic. Sensitivity to reward was defined here as neural activation in the ventromedial prefrontal cortex (vmPFC) and striatum for wins as compared to losses in a reward-based task measured prior to the pandemic. We used data from the Human Connectome Project in Development collected before the pandemic onset, and follow-up data which was collected from the same participants during the pandemic. Activity in the left vmPFC moderated the association between pandemic-related stressors and change in internalizing psychopathology. Although those with low reward sensitivity showed a positive association between exposure to stressors and increase in psychopathology during the pandemic relative to baseline, those with high sensitivity to reward did not show increased symptoms with increased stressors. We found no effect of activity in the striatum or right vmPFC on the association between stressors and change in psychopathology. Additionally, we did not find a moderating effect of neural reward reactivity and change in externalizing psychopathology. These findings add to a growing literature highlighting reward sensitivity, measured prior to stressor onset, as a source of stress resilience.

Stressful life events (SLEs) are positively associated with psychopathology in youth, including symptoms of anxiety and depression as well as externalizing psychopathology (Espejo et al., 2007; Jenness et al., 2019; Low et al., 2012; McLaughlin and Hatzenbuehler, 2009; Shapero et al., 2014; Vidal Bustamante et al., 2020). This association has been shown to be bi-directional such that stressful experiences are often followed by increases in psychopathology, and that individuals with high levels of psychopathology may experience more stressful life events (Hammen, 2016; Jenness et al., 2019). Community level disasters like hurricanes and persistent community stressors like community violence have been shown to have negative mental health outcomes for youth (Miliauskas et al., 2022; Orengo-Aguayo et al., 2019). These widespread stressors provide an opportunity to identify sources of risk and resilience to stress and psychopathology more broadly. Identifying factors that protect youth against the negative mental health consequences of community stressors is of critical importance for psychological theory and clinical practice.

The COVID-19 pandemic brought on a variety of novel stressors, including fear of illness and death for individuals and their loved ones, and stressors from containment efforts, such as schooling from home, loss of contact with loved ones, and disruption in routine (Meherali et al., 2021). These stressors could increase youth mental health problems through a variety of mechanisms including for example difficulties with emotion regulation accompanied by reduced access to social support. Indeed, there has been a marked uptick in mental health problems among youth following the pandemic (Barendse et al., 2023a; Chahal

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et al., 2021). Specifically, greater exposure to pandemic-related stressors has been linked to increased internalizing and externalizing psychopathology in youths (Chen et al., 2020; McLaughlin et al., 2022; Rodman et al., 2022; Rosen et al., 2021). Indeed, across several pandemics, including the COVID-19 pandemic, H1N1, ebola, and equine influenza, these events have been shown to induce increased stress and psychopathology in youth around the world (Meherali et al., 2021). The increase in mental health problems has been shown both acutely during pandemics as well as over a longer time frame (Blackwell et al., 2024; De Caro et al., 2025; Rosen et al., 2021). In the present study, we investigate the role of neural reward sensitivity *prior* to the pandemic as a moderator of the association between this community level stressor and the development of internalizing and externalizing psychopathology in a large longitudinal sample of youth.

Previous work highlights that sensitivity to reward buffers against the negative mental health consequences of exposure to stressful life experiences. For instance, individuals who developed less bias toward stimuli with higher reward probabilities had greater depressive symptoms and were less likely to show improvements in symptoms after eight weeks of treatment (Vrieze et al., 2013). Additionally, modulation of neural reward networks has been suggested to be a mechanism for the efficacy of behavioral activation, a therapeutic technique in which patients are encouraged to schedule activities and engage with their environment as a means of deriving positive reinforcement (Nagy et al., 2018; Uphoff et al., 2019). Further, individuals who are more sensitive to reward may be protected against developing psychopathology following stressful life events. For example, among teens with a history of childhood maltreatment, those who were more sensitive to reward, measured as a greater difference in reaction time between reward and neutral conditions, showed reduced increases in depression symptoms two years later as compared to those with low sensitivity to reward (Dennison et al., 2016). This effect has been extended to youths who experience deprivation, including neglect. Behavioral sensitivity to reward moderates the association between deprivation and externalizing psychopathology symptoms such that individuals with high reward sensitivity show a blunted association between deprivation and externalizing symptoms (Kasparek et al., 2023).

Recent work has also explored how patterns of reward-related neural activity may be related to differences in the development of depression symptoms following exposure to stressors, including pandemic-related stressors (Pegg and Kujawa, 2024). For instance, one study of adolescents found that the extent of reward positivity—as defined by the difference in event-related potential response to monetary wins versus monetary losses, recorded prior to the pandemic—moderated the relation between family financial stress and changes in depression from pre-pandemic to the early months of the COVID-19 pandemic (Feurer et al., 2021). Specifically, individuals with blunted neural reward positivity (a less pronounced distinction in neural activity between wins and losses) showed significant positive association between greater financial stress and greater increase in depression symptoms during the pandemic. This pattern was not seen for individuals with a high neural reward positivity. This distinction suggests that blunted reward reactivity may be a risk factor in the development of depression following exposure to stressors. Moreover, experiencing stress has been linked to blunted reward positivity with one study finding that reward positivity decreased following stressors related to the pandemic (Freeman et al., 2023). It has been suggested that this blunting of reward positivity could be a plausible mechanism linking stressful life events to the development of depression.

The neural mechanisms underlying the relation between sensitivity to reward and the development of psychopathology are still under investigation. The striatum (Arsalidou et al., 2020; Bjork et al., 2010; Vrieze et al., 2013), the amygdala (Baxter and Murray, 2002; Wassum, 2022), the orbitofrontal cortex (OFC) (Rolls et al., 2020), the insula (Duarte et al., 2020), and the ventromedial prefrontal cortex (vmPFC) (Vassena et al., 2014) have all been associated with various aspects of reward processing. Importantly, both the vmPFC and striatum have been associated with reward-based decision making and reward receipt. Differences in neural activity in response to rewards have also been linked to psychopathology. Specifically, individuals with major depressive disorder demonstrate significantly less activation in the left nucleus accumbens and bilateral caudate, two regions within the striatum, for gains on the Monetary Incentive Delay (MID) task, a commonly used reward processing task, compared to controls (Pizzagalli et al., 2009). Moreover, neural sensitivity to reward may moderate the association between negative life events and the development of psychopathology. One longitudinal study reported that when youths were shown positive and neutral stimuli, increased activity in the left pallidum had a moderating effect on the association between childhood maltreatment and depressive symptoms (Dennison et al., 2016). In the same sample youths who had faster reaction times to stimuli that had been previously paired with high reward during the MID task had lower levels of depression, despite exposure to maltreatment. Additionally, other studies have found that young adults who have experienced childhood maltreatment on average show diminished reward-related neural activity, but that those who maintain high levels of reward reactivity despite exposure to maltreatment are less likely to show high levels of psychopathology symptoms (Richter et al., 2019). In contrast, other studies have found that children with disruptive behavior disorders showed higher sensitivity to reward, seen as increased activity in the OFC, another prefrontal area involved in reward processing, and striatal activity during reward receipt during the MID task, as compared to control youth (Hawes, 2022). Additionally, high reward sensitivity has been identified as a risk factor for alcohol abuse (Rothstein et al., 2025). However, these differences in neural processing among individuals with psychopathology alone are insufficient to determine if differences in neural sensitivity to reward is a result of psychopathology, or a risk factor for it.

In the present study, we focus on key regions of the brain important for reward processing. First, we include the ventral striatum which includes the nucleus accumbens, a crucial region for reward reactivity, showing increases in activity in response to reward receipt as well as reward anticipation both in human and non-human animal models (Delgado et al., 2000; Knutson et al., 2001; Mogenson et al., 1980). Second, we focus on the dorsal striatum which spans both the caudate and putamen. We focus on these regions because of their role in reward anticipation (Haber and Knutson, 2010) and receipt (Delgado et al., 2003), as well as the fact that previous work from our group found that reward-related activity in these regions may be associated with less progression of depression symptom worsening in maltreated youth (Dennison et al., 2016). Finally, we focus on the vmPFC because early fMRI studies demonstrate significant increases in activity in this brain region following receipt of both primary rewards as well as secondary rewards (Chib et al., 2009). Further, the vmPFC increases activity with increasing subjective reward valuation (Levy and Glimcher, 2012; Winecoff et al., 2013) and reward-related activity in the vmPFC has been proposed to be important for resilience (Dutcher and Creswell, 2018). We explore whether neural sensitivity to reward moderates the association between pandemic-related stressors and development of psychopathology. This investigation extends previous work, which has focused on more profound experiences of childhood maltreatment and deprivation, by examining stressors related to the COVID-19 pandemic as a community-level stressor. We further expand on theoretical perspectives the brains reward system may play a crucial role in resilience (Dutcher, 2023). Many studies link blunted reward reactivity to the development of internalizing psychopathology including depression and anxiety (e.g. Dennison et al., 2016; Feurer et al., 2021; Luking et al., 2016). However, some recent studies have also found that individuals who retain high levels of reward reactivity despite experiencing early life adversity are less likely to develop externalizing psychopathology (Kasparek et al., 2020; 2023).Therefore, in the current study we examine whether the reward reactivity moderates the association between pandemic-related

stress and the development of both internalizing and externalizing psychopathology.

We leverage a longitudinal subsample of children enrolled in the Human Connectome Project in Development (HCP-D) (Somerville et al., 2018) study who completed follow-up surveys during the pandemic (October 2021-January 2022). The present study consists of two time points in participants aged 5–21 years at the first time point (T1). T1, which occurred between January 2017 and February 2020 and therefore prior to the declaration of the global COVID-19 pandemic, was conducted as part of the HCP-D study in which participants completed a reward processing task and measures of psychopathology (for complete listing of HCP-D protocols see Somerville et al. 2018 and Harms et al., 2018). Later, during the COVID-19 pandemic, participants from the HCP-D study and their caregivers were re-contacted to participate in an online study, including measures of pandemic-related stress and psychopathology.

We examined three hypotheses. First, we hypothesized that we would replicate prior research showing pandemic-related stressors are positively associated with internalizing and externalizing psychopathology, while controlling for pre-pandemic symptoms. Second, we hypothesized that higher neural sensitivity to reward, operationalized as increased activity in the ventral and dorsal striatum and vmPFC for wins as compared to losses in the reward processing task, would be associated with lower rates of internalizing and externalizing psychopathology. Third, we hypothesized that neural reward sensitivity would moderate the association between pandemic-related stressors and psychopathology, such that the association of stressors with symptoms would be reduced among youths with higher neural responses in the striatum and vmPFC. In all analyses, we controlled for age, sex, pre-pandemic psychopathology symptoms, and scanner. These hypotheses were preregistered prior to analysis on the Open Science Framework (OSF; https://osf.io/4mxfa/files/osfstorage).

1. Methods

1.1. Participants

Participants were drawn from a large study of children and adolescents (n = 1182 with usable fMRI task data at T1) who were first enrolled in the HCP-D study between the ages of 5 and 21 years (Somerville et al., 2018). Neuroimaging scans at T1 took place between January 2017 and February 2020. Inclusion criteria for the HCP-D study included being between the ages of 5–21 years, speaking English well, and not having any MRI contraindications (Somerville et al. 2018). Exclusion criteria for the HCP-D were premature birth, serious medical conditions, serious endocrine condition, history of serious head injury, treatment greater than 12 months for psychiatric conditions, receiving certain special services at school, claustrophobia, pregnancy, and some hospitalizations (Somerville et al. 2018). Participants with usable scan data were re-contacted and those that responded to the follow-up questionnaires were included in this sample (n = 339, ages 7–22 years; Mean = 14.18 years, 177 female). The racial breakdown of participants who responded at T2 was as follows: 0 % Native American/ Alaska Native, 4 % Asian, 7 % Black/ African American, 0 % Native Hawaiian or other Pacific Islander, 67 % white, 20 % more than one race, 2 % information unavailable. Criteria for excluding participants from statistical analyses based on scan quality are delineated below. IRB approval was obtained for all protocols at the respective institutions (T1: Harvard University, University of California-Los Angeles (UCLA), University of Minnesota (UMinn), and Washington University in St. Louis (WUSTL), T2: Harvard University). For participants under 18 years of age, assent was obtained from participants, and informed consent was obtained from a parent or legal guardian. For participants over 18 years of age, informed consent was obtained from the participant.

1.2. T1 procedures (Pre-pandemic)

1.2.1. Assessment of psychopathology

To assess internalizing and externalizing psychopathology at T1, participants and their caregivers completed a variety of instruments, depending on their age. For children under 10 years, we used caregiver response on the Child Behavior Checklist (CBCL). For children aged 11–17 years, we used the highest of caregiver or child report on the CBCL and the Youth Self Report (YSR). This choice was made following precedent of the "or" rule used to diagnose psychopathology in population-based studies that include both child and caregiver report, in which studies find the highest agreement between clinical assessment and self/parent report when used in conjunction rather than separately (Kessler et al., 2012; Merikangas et al., 2010; Weissman et al., 2022). For those ages 18–21, we used the Adult Self-Report (ASR) score. We then used the internalizing and externalizing scales separately from each measure for subsequent analyses.

The CBCL consists of 132 items, of which 113 score problematic behavior. These 112 problematic behavior items can be divided onto internalizing and externalizing subscales. Items are scored as either not true (0), somewhat or sometimes true (1), or very true (2). Sample items include my child cries a lot; breaks rules at home, school, or elsewhere; is too fearful or anxious; and smokes, chews, or sniffs tobacco. Raw scores were then converted to standardized T-scores prior to analyses. Internal consistency was good for both internalizing (Chronbach's alpha = 0.80) and externalizing symptoms (Chronbach's = 0.83).

The YSR contains 105 of the items regarding problematic behaviors on the 6–18 version of the CBCL, and items relate to the same internalizing and externalizing subscales. Sample items include I cry a lot; I lie or cheat; I steal at home; I worry a lot. Scores were converted to standardized T-scores prior to analyses. Internal consistency was good for both internalizing (Chronbach's alpha = 0.87) and externalizing symptoms (Chronbach's = 0.86).

The ASR uses the same response options as the YSR, as well as many similar items, but has been modified to be more applicable for those over age 18 including questions regarding drug and alcohol use. Sample items include: I worry a lot; I drink too much alcohol or get drunk; I physically attack people; I break rules at work or elsewhere; I cry a lot. Scores were converted to standardized T-scores prior to analyses. Internal consistency was good for both internalizing (Chronbach's alpha = 0.87) and externalizing symptoms (Chronbach's = 0.81).

1.2.2. Reward processing task

Participants performed two runs (one run for children aged 5–7) of the HCP-D Guessing Task (Somerville et al., 2018; adapted from Delgado et al. 2000). In the Guessing Task, participants were asked to guess if a baby or adult is hiding behind the question mark by pressing a button (left for baby, right for adult). Participants were first cued that the block of trials would pay out either high stakes or low stakes gains and losses, with six high stakes blocks and six low stakes blocks in random order in each run. In a high stakes block, wins were +$1 and losses are -$0.50, and in a low stakes block a win was worth +$0.20, and a loss was -$0.10. The block cue was presented for 1.5 s. Each block consisted of four trials. In each trial, the participant was cued to make a guess by the presentation of a "?" on the screen for 2 s. The participant guessed between the two choices via button press. The feedback was pre-programed such that each participant always had a 50 % chance of receiving a reward, regardless of their choices. This probabilistic outcome means that participants did not learn a strategy in this task but rather were consistently guessing. There was then an inter-stimulus interval (ISI) jittered between 1.5, 2, or 2.5 s. Participants then received feedback as to whether their guess was correct, presented as a 1 s screen stating that they had won or lost the amount using words and graphics (see Fig. 1a). This was followed by a jittered inter trial interval (ITI) of 1, 1.5, or 2 s before moving onto the next trial. There was an 8 s fixation block between trial blocks. At the conclusion of the session, participants were paid the

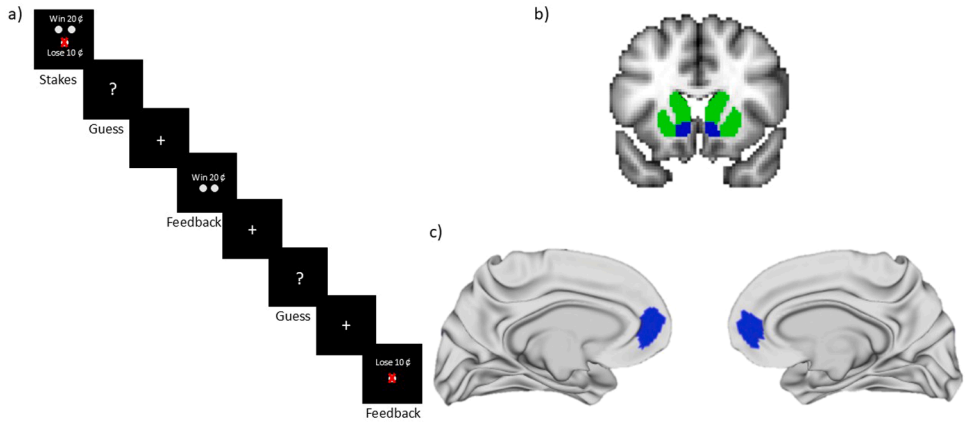

Fig. 1. Task information. a) GUESSING task structure adapted from Somerville et al. (2018). Participants are shown the stakes at the beginning of each block. Each trial begins when they are asked to make a guess via button press. This is followed by a brief ISI before receiving feedback as to whether their guess was correct (and they won money) or incorrect (and they lost money). The next trial begins in the same format. The analyses in this paper focus on the feedback portion of the task. b) Subcortical regions of interest used in this study include the left and right dorsal striatum (blue) and ventral striatum (green). c) Cortical regions of interest used in this study include the left and right ventromedial prefrontal cortex (blue).

money that they had earned in the task.

1.2.3. Neuroimaging acquisition and processing

Brain imaging was performed using Siemens 3T Prisma scanners at six sites across four universities including Harvard University, University of California-Los Angeles (UCLA), University of Minnesota (UMinn), and Washington University in St. Louis (WUSTL). 32-channel 80mT/m gradient head coil. UCLA and WUSTL had two scan sites. In participants aged 8–21, a Siemens Prisma 32-channel 80mT/m gradient head coil was used. In participants aged 5–7, a Cerensensa pediatric 32 channel head coil was used. Complete data acquisition parameters are reported in detail elsewhere (Harms, et al., 2018). Briefly, T1-weighted multi--echo MPRAGE volumes were acquired (TR: 2500, TE = 1.8/3.6/5.4/7.2 ms, flip angle: 8 degrees, FOV: 256 × 240 × 166 mm with a matrix size 320 × 300, 208 slices, in-plane voxel size: 0.8 mm^3. BOLD scans were acquired using a T2*-weighted scan. To acquire the functional scans a 2D multiband (MB) gradient-recalled echo (GRE) echo-planar imaging (EPI) sequence (TR: 800 ms, TE: 37 ms, flip angle: 52 degrees, 2 mm3 voxel size, 72 oblique-axial slices) was used. Participants completed 2 runs of the GUESSING task with opposite phase encoding polarity (P-A and A-P), in participants aged 8–21, and P-A only in participants aged 5–7 (Harms et al., 2018).

Participants and runs with substantial motion artifacts were excluded as part of the pre-processing pipeline by the HCP-D team prior to our receipt of the data. Quality Control information was made available to researchers, and we followed their recommendations. These include: < 98 % drop out (PctBrainCoverag), ≥ 50 % amount of dropout in the cerebellum (PctCerebMiss), signal to noise ratio < 15 (tSNR), percent of volumes with a relative root-mean-squared movement of greater than 0.5 mm > 30 (REL_RMS_0_5), DVAR Standard deviation > 50 (DVAR_SD), WishartProb (from freesurfer) = 1. We also excluded when data was marked by the preprocessing team to be excluded for some other reason.

1.2.4. fMRI data preprocessing and basic analysis

Data had been pre-processed using the HCP Pipelines v3.22 minimal preprocessing pipeline, publicly available on Github (Glasser et al., 2013). This pipeline has been described in detail elsewhere (e.g. Marcus et al., 2013). Briefly the minimally preprocessed HCP-D data includes brain extraction using 'BET', intensity normalization, smoothing (FWHM = 4 mm) using 3dFWHMx in ANFI. No regressors for motion are included. Instead, HCP-D data are cleaned using an independent component analysis technique using ICA-AROMA that removes noise components estimated across all functional data. To account for low frequency fluctuations, a Gaussian weighted-linear filter was applied with a threshold of 200 s (Marcus et al., 2013). After preprocessing, a generalized linear model (GLM) was used to estimate the effects of task condition for each individual subject. This included seven regressors of interest for high cue, low cue, guess, high win, low win, high loss, and low loss represented as predictive timeseries by specifying their temporal event onset, convolved with a double-gamma canonical hemodynamic response function. All analyses used the contrast of wins (collapsing across high wins and low wins) vs. losses (collapsing across high loss and low loss).

1.3. T2 procedures (October 2021- January 2022)

1.3.1. Pandemic-Related stressors

We utilized a questionnaire about experiences during the pandemic adapted from a previous study (Rosen et al. 2021). Participants and a parent completed the questionnaire online. We created a sum score of pandemic-related stressors that included the following experiences: got sick with COVID-19, had a parent or sibling get sick with COVID-19, had another relative get sick with COVID-19, knew someone who died as a result of COVID-19, parent is a frontline worker (healthcare), felt less connected to close friends, felt less connected to family, experienced discrimination related to the pandemic, experienced food insecurity during the pandemic, parent lost a job during the pandemic, parent still out of work and/or making less money than before, difficulty doing school work remotely. For complete items, timeframe, and reporter for each see Supplementary Materials. The sum of these measures was used to create the pandemic-related stressor score.

1.3.2. Psychopathology symptoms

To reduce participant burden during a difficult time, we opted to use the Strengths and Difficulties Questionnaire (SDQ) —which is shorter than the CBCL or YSR—to assess psychopathology during the pandemic. Despite the short format, scores on the SDQ are highly correlated with measures of externalizing and internalizing scales from the CBCL in a general population sample (Goodman and Scott, 1999). When parent and child measures were both available, we used the highest score of the two respondents. The SDQ consists of 25 items, which can be divided into 3 subscales: internalizing, externalizing and prosocial. We used internalizing and externalizing subscales separately. Participants ranked each item as either not true, somewhat true, or certainly true. Example internalizing items include: many fears, easily scared; and many worries or often seems worried. Example externalizing items include: often fights with other children or bullies them; and restless, overactive,

cannot stay still for long. The maximum possible score for internalizing and externalizing symptoms are each 20.

1.4. Analyses

1.4.1. ROI analyses

Reward sensitivity was defined as the difference in neural activity between wins and losses during the feedback phase (collapsed across large and small stakes blocks) of the GUESSING task, calculated separately in the dorsal striatum, ventral striatum, and vmPFC. Regions of interest (ROIs) were defined by masking functional activation in the group average (voxel-wise threshold of $p < 0.01$ and family-wise error corrected cluster-level of $p < 0.05$) for wins compared to losses and intersecting this mask with an anatomical mask (20 % threshold) from the Harvard-Oxford atlas in FSL for the left and right dorsal striatum (by adding caudate and putamen) and ventral striatum (nucleus accumbens), and using parcellations p32 and 10r from the Glasser atlas separately for each hemisphere for the vmPFC (Glasser et al., 2016).

1.5. Statistical analyses

All statistical analyses were conducted in R (R Core Team, 2024). Multiple regression analyses were performed using the *lme4* package (Bates et al., 2015) and standardized coefficients are presented. All variables were scaled without centering prior to conducting regression analyses using the R *scale* function. First, we tested the relation between pandemic-related stress with psychopathology symptoms, separately for internalizing and externalizing psychopathology. In this model, the predictor was pandemic-related stressors (T2), the outcome was internalizing or externalizing symptoms at T2, and the covariates were age at T2, sex, and T1 psychopathology symptoms.

Next, we used multiple regression to test the association between neural activity during wins > losses separately in the dorsal striatum, ventral striatum, and vmPFC with internalizing and externalizing psychopathology. In these models, the predictor was the parameter estimate for rewards > losses (separately for dorsal striatum, ventral striatum, and vmPFC, for each hemisphere), the outcome was internalizing and externalizing symptoms at T2 (separately), and the covariates were age at T2, sex, T1 psychopathology symptoms, and scanner.

Finally, we tested if reward sensitivity moderates the association between pandemic-related stressors and internalizing and externalizing symptoms separately for each ROI. In these models the predictor was pandemic-related stressors, the outcomes were internalizing and externalizing symptoms at T2, the moderators were activation in the left and right ventral striatum, dorsal striatum, and vmPFC (seperately), and the covariates were age at T2, sex, psychopathology at T1, and scanner. Simple slope analyses were used to follow up on significant results in these models using the R *interactions* package (Long, 2024). In the simple slopes analysis, the predictor is the COVID stressors, and the moderator was neural activity, the outcome is T2 psychopathology, and the covariates are age at T2, sex, scanner, and T1 psychopathology. The *interactions* packages by default reports the slopes at 3 values of the moderators: the mean, +1 standard deviation, and −1 standard deviation.

To correct for multiple comparisons, we used false discovery rate (FDR) across left and right hemisphere and internalizing and externalizing psychopathology for each ROI (e.g. association between activation in left and right ventromedial PFC and internalizing and externalizing psychopathology, FDR corrected for four comparisons).

We conducted post-hoc exploratory analyses on the effects of age at T2 on the associations between pandemic stress, reward activity, and psychopathology. We first tested if age moderated the association between neural activity and T2 psychopathology. Sex, scanner, and T1 psychopathology were again included as covariates in these models. We then performed a 3-way moderation analysis, with T2 psychopathology as the outcome, and age, stress, and neural activity as the moderating variables. Sex, scanner, and T1 psychopathology were again included as covariates.

2. Results

See Table 1 for summary statistics of all variables and Table 2 for all bivariate correlations.

2.1. Pandemic-Related stressors and psychopathology

Increases in the number of pandemic-related stressors individuals experienced were associated with more externalizing symptoms (β=0.235, $p < 0.01$) and there was a non-significant association for internalizing symptoms (β= 0.097, $p = 0.12$; Fig. 2). This partially replicates previous work which found that exposure to greater pandemic-related stress is associated with greater increases in psychopathology (e.g. Rosen et al., 2021).

2.2. Sensitivity to reward and psychopathology during the pandemic

Contrary to our hypothesis, we found no significant main effects of activity in the dorsal striatum, ventral striatum, or vmPFC during reward receipt with internalizing or externalizing symptoms at T2, when controlling for T1 symptoms, age, sex, and scanner (see Supplemental Table 1 and Supplemental Figures 1–3). Furthermore, bivariate correlations showed no associations between activity and T1 psychopathology.

2.3. Sensitivity to reward moderates the relation between stress and psychopathology

There was a significant interaction between pandemic-related stressors and activity in the left vmPFC during wins versus losses on internalizing symptoms (β=−0.227, $p = 0.032$ following FDR correction, see Table 3 and Fig. 3). Simple slopes analysis revealed a significant association between pandemic-related stressors and internalizing symptoms for those with low levels of activity (−1SD, β= 0.31, $p < 0.001$), but no significant association between stressors and symptoms for those with high levels of activity (+1SD, $\beta = -0.07$, $p = 0.46$). There was no significant interaction between pandemic-related stressors and left vmPFC activity on externalizing psychopathology (Table 3). There were also no significant interactions between the activity in the dorsal or

Table 1
Summary Statistics for all numerical variables.

Variable Name	Minimum Value	Maximum Value	Mean	Standard Deviation
Pandemic-Related Stressors	0	8	2.124	1.56
Age (in years) at T2	7	22	14.18	3.55
T1 Internalizing Symptoms	33	75	49.43	9.43
T1 Externalizing Symptoms	30	71	46.91	8.47
T2 Internalizing Symptoms	0	15	4.404	3.55
T2 Externalizing Symptoms	0	15	4.565	3.45
Left vmPFC Activity	−109.46	205.07	21.84	37.14
Right vmPFC Activity	−213.63	147.87	19.34	33.29
Left Dorsal Striatum Activity	−61.21	97.38	14.02	24.82
Right Dorsal Striatum Activity	−60.73	107.92	14.26	24.35
Left Ventral Striatum Activity	−93.21	178.77	35.25	34.01
Right Ventral Striatum Activity	−61.56	154.80	34.86	33.47

Table 2
Bivariate correlations for all numerical variables. * indicates $p < 0.05$. ** indicates $p < 0.01$. No multiple comparison corrections have been applied.

	Age	COVID Stress	Internal T1	External T1	Internal T2	External T2	Left vmPFC	Right vmPFC	Left Dorsal Striatum	Right Dorsal Striatum	Left Ventral Striatum
Pandemic Stressors	0.031										
Internal T1	0.284**	0.072									
External T1	0.129*	0.013	0.534**								
Internal T2	0.205**	0.123*	0.342**	0.229**							
External T2	−0.061	0.211**	0.218**	0.3**	0.563**						
Left vmPFC	−0.13*	−0.047	0.001	0.052	−0.022	−0.03					
Right vmPFC	−0.151*	−0.036	−0.001	−0.009	−0.058	−0.071	0.757**				
Left Dorsal Striatum	0.021	−0.075	0.066	0.042	−0.044	−0.044	0.198**	0.225**			
Right Dorsal Striatum	0.034	−0.034	0.049	−0.024	−0.017	−0.062	0.2**	0.213**	0.865**		
Left Ventral Striatum	0.038	−0.017	−0.035	−0.007	−0.052	−0.014	0.382**	0.343**	0.375**	0.379**	
Right Ventral Striatum	0.048	−0.058	0.09	0.017	−0.042	0.016	0.374**	0.353**	0.297**	0.302**	0.554*

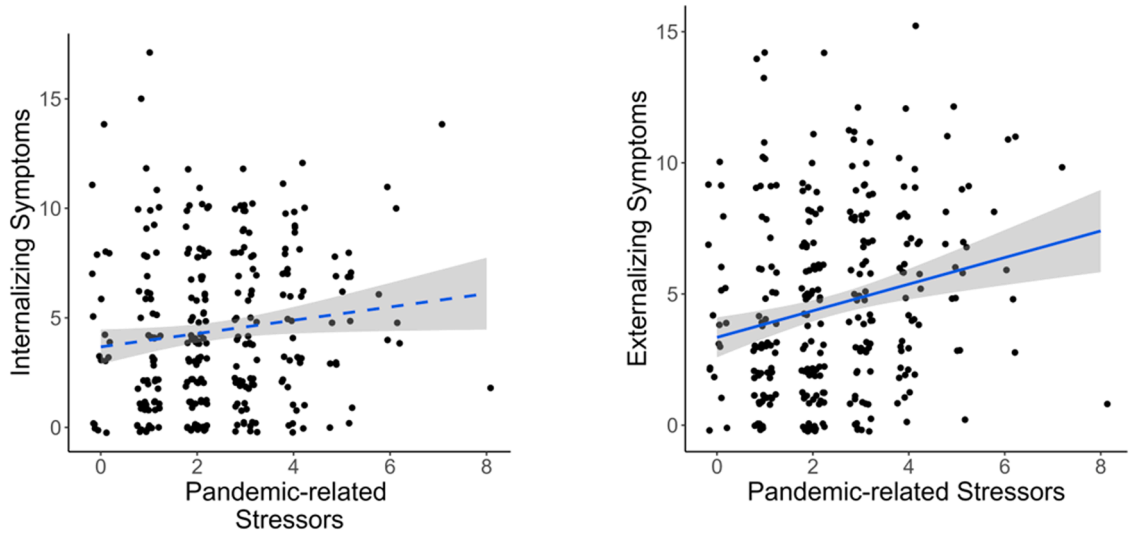

Fig. 2. Pandemic-related stressors are positively associated with psychopathology. a) Association between pandemic stressors and internalizing symptoms (β= 0.097, $p = 0.12$) b) and externalizing symptoms (β=0.235, $p < 0.01$). All analyses control for age, gender, and pre-pandemic symptoms (internalizing or externalizing respectively). Statistical analyses are conducted with standardized variables and all p-values are FDR corrected. Visualizations utilize raw scores for interpretability.

Table 3
Interaction effects of sensitivity to reward and pandemic-related stressors on T2 psychopathology show a significant interaction only for the left vmPFC and internalizing symptoms. All analyses control for age at T2, gender, scanner, and T1 symptoms. P-values are FDR corrected.

ROI	Internalizing		Externalizing	
	β	*p*	β	*p*
Left vmPFC	**−0.227**	**0.032**	−0.113	0.213
Right vmPFC	−0.189	0.108	−0.115	0.213
Left Dorsal Striatum	−0.048	0.816	0.018	0.816
Right Dorsal Striatum	−0.064	0.816	−0.027	0.816
Left Ventral Striatum	−0.086	0.571	−0.035	0.718
Right Ventral Striatum	−0.091	0.571	−0.174	0.448

ventral striatum (Table 3 and Supplementary Figures 4 and 5), or right vmPFC (Table 3 and Fig. 3), and pandemic-related stressors on internalizing or externalizing psychopathology. This result provides partial support for the pre-registered hypothesis that reward-related activity in the vmPFC moderates the effect of stressors on the development of psychopathology. However, we did not find support for the pre-registered hypothesis that reward-related activity in the dorsal and ventral striatum moderates the association between pandemic-related stressors and psychopathology.

2.4. Exploratory investigations on the interaction between age, pandemic-related stress and reward sensitivity on psychopathology

Given the wide age range of the sample, we conducted exploratory investigations on the interaction of age with stressors on post-pandemic psychopathology. There was no significant age moderation of the association between stressors and externalizing psychopathology (β=0.332, $p = 0.18$) nor internalizing psychopathology (β=0.350, $p = 0.18$). Furthermore, no significant interactions were found between neural activity and age in predicting psychopathology (see Supplementary Table 2). Finally, we found no significant 3-way interactions using stressors, neural activity, and age as interaction terms predicting internalizing or externalizing psychopathology at T2 (see Supplementary Table 3).

3. Discussion

In this pre-registered analysis, we replicate, in a large, longitudinal, multisite sample, that exposure to greater pandemic-related stress is associated with increased psychopathology symptoms during the

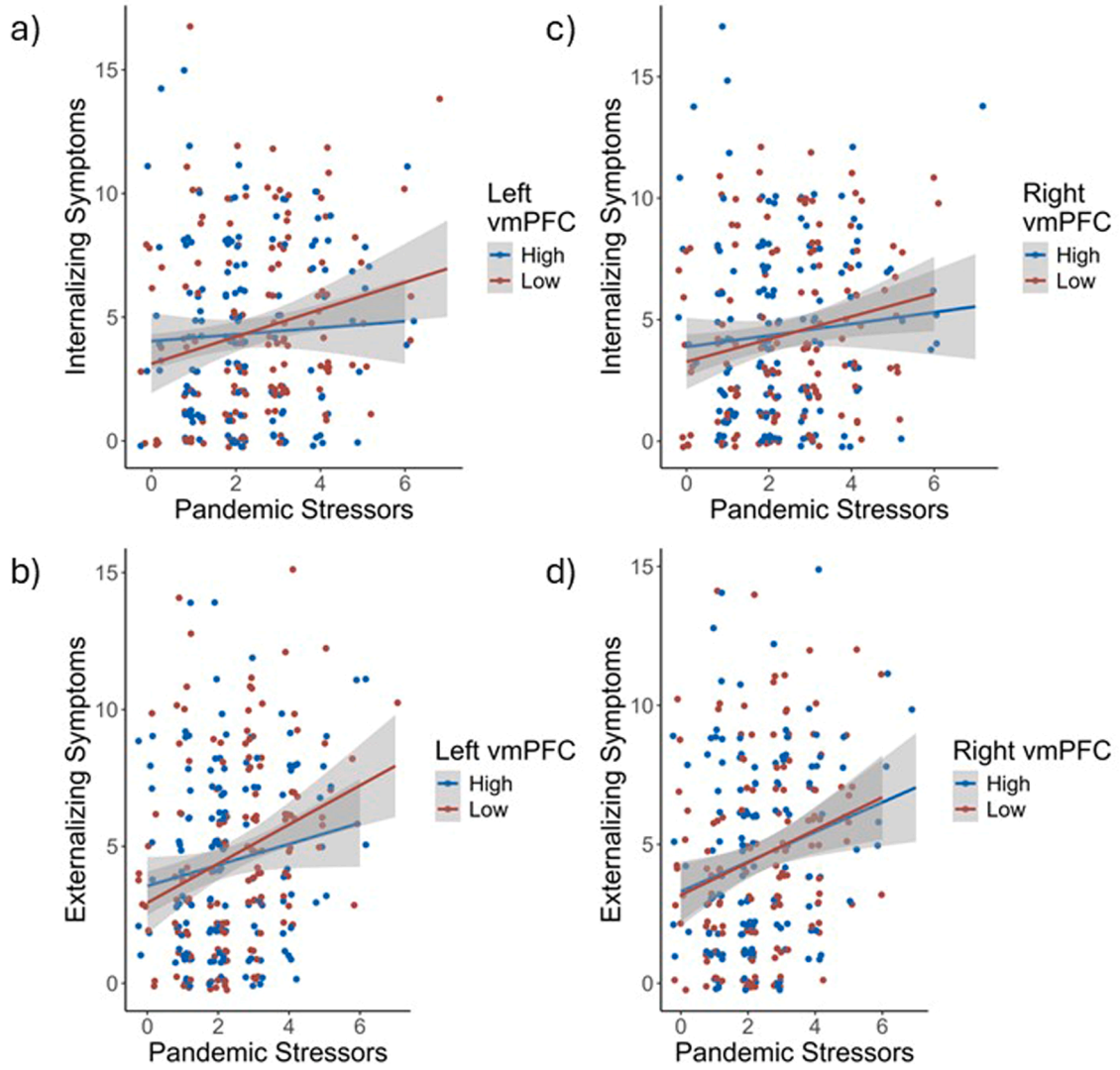

Fig. 3. Reward sensitivity partially moderates the association between pandemic-related stressors and psychopathology. a) Reward sensitivity in the left vmPFC significantly (β= −0.227, p = 0.032) moderates the association between pandemic stressors and internalizing psychopathology. b) There is a non-significant moderating effect of reward sensitivity of the right vmPFC on the association between pandemic stressors and internalizing symptoms (β= −0.189, p = 0.108) in the same direction. c) Reward sensitivity in the left vmPFC does not significantly moderate the association between pandemic stressors and externalizing symptoms (β= −0.113, p = 0.213). d) Reward sensitivity in the right vmPFC does not significantly moderate the association between pandemic stressors p and externalizing symptoms (β= −0.115, p = 0.213). For visualization purposes, participants were median split, with red representing higher sensitivity to reward, and blue representing lower sensitivity to reward. Linear models utilize continuous values and control for age, gender, scanner, and pre-pandemic symptoms (internalizing or externalizing respectively). All p-values are FDR corrected.

pandemic. Additionally, consistent with our hypothesis, we show that sensitivity to reward, operationalized as the difference in neural activity in the left vmPFC for wins as compared to losses in a reward magnitude task, moderates the association between pandemic-related stressors and internalizing psychopathology. The results illustrate that youth with high sensitivity to reward were less likely to develop internalizing problems, even when experiencing high levels of pandemic-related stressors. This interaction was not significant in predicting externalizing psychopathology. Sensitivity to reward, when operationalized as the difference in neural activity in the dorsal striatum, ventral striatum, or right vmPFC, for wins as compared to losses, did not moderate the association between pandemic stressors and internalizing nor externalizing psychopathology. Interestingly, we did not find a main effect of sensitivity to reward on psychopathology during the pandemic (when controlling for pre-pandemic psychopathology). Thus, in this study, greater reward sensitivity was not linked to better mental health overall, but rather those who exhibit greater neural reward sensitivity were less likely to develop internalizing mental health problems in the face of stressors. In contrast, those with less sensitivity to reward were more likely to develop internalizing symptoms in response to high levels of stressors during the pandemic.

Our study replicated previous findings that showed increases of externalizing psychopathology associated with COVID stressors (Barendse et al., 2023b; Chahal et al., 2021; Chen et al., 2020; Lengua et al., 2022; Ma et al., 2021; McLaughlin et al., 2022; Meherali et al., 2021; Rodman et al., 2022; Rosen et al., 2021; Weissman et al., 2021). Specifically, we replicate findings of increases in externalizing psychopathology (Chahal et al., 2021; Rodman et al., 2022; Rosen et al., 2021; Weissman et al., 2021). Contrary to previous studies and our hypotheses, the positive association between pandemic-related stress and internalizing symptoms did not reach significance (Rodman et al., 2022; Rosen et al., 2021). One possible reason for this discrepancy with previous studies is that in the current study, the pandemic time point (T2) was relatively late in the pandemic (October 2021- January 2022) compared

to previous studies. Additionally, there was significant variability in pandemic restrictions and timelines across the United States, which is relevant to the current multisite study. These differences could have created systemic variability across participants resultant to geographic variability.

We hypothesized that sensitivity to reward in *both* the vmPFC and striatum, which show increased activation for wins as compared to losses, would moderate the effects of pandemic-related stressors on psychopathology (Somerville et al., 2018). All regions investigated showed increased activity for wins as compared to losses in this task (see Supplementary Table 4). However, we only found significant moderating effects in the vmPFC, with a relatively small effect size. These regional differences could be attributed to the distinct roles of the vmPFC and striatum in reward processing. Namely, the vmPFC is thought to "track" reward values (Becker et al., 2016; Vassena et al., 2014), while the striatum is thought to be more involved in reward-learning and in fact shows increased activation to more predictive cues (Filimon et al., 2020). Importantly because pre-pandemic psychopathology is a strong predictor of psychopathology during the pandemic in both the internalizing and externalizing domains, these analyses all control for pre-pandemic symptoms.

Activity in the left vmPFC during reward receipt, showed a significant moderating effect on the association between stressors and symptoms. This finding is likely attributed, in large part, to the role of the vmPFC in reward processing as a neural region that tracks reward magnitude (Becker et al., 2016). This finding of the buffering effects of prefrontal sensitivity to reward against pandemic stress complements previous research showing a protective effect of increased vmPFC-hippocampal connectivity against pandemic stress (Chang et al., 2023). This finding uniquely unites the role of the vmPFC in reward processing with our understanding of how reward processing affects resilience and poses the vmPFC as a critical target for future research.

With regard to the ventral striatum, both animal and human studies demonstrate the role of the ventral striatum in reward learning. Specifically, research in rats has shown that the release of dopamine–the major ventral striatal neurotransmitter involved in reward processing–is greater for reward anticipation than reward receipt, reflecting a learning-dependent shift in the ventral striatum for reward anticipation over consumption (Day et al., 2007). These findings have been corroborated in human studies wherein the ventral striatum has similarly been shown to code reward prediction in the form of prediction errors (i.e., the difference between expected and acquired results; Becker et al., 2016). The present study used a simple guessing task in which participants always had a 50 % chance of receiving a reward. Therefore, the guessing task used in this study is incapable of capturing true reward learning because the rewards are random, and participants are unable to learn a rule to guide their choices. As such, future studies would benefit from introducing variability in reward magnitude and probability across trials to elicit a change in reward predictability and sensitivity. It is therefore plausible that future studies using a task in which one can predict rewards, might uncover a buffering effect of striatal response during reward prediction analogous to the finding here for vmPFC during reward receipt.

The current work complements previous work which has identified other moderators of the link between pandemic stressors and adverse mental health outcomes in youth. Some examples include peer support, routines, less passive screen time and exposure to news, adherence to stay-at-home orders, living in the countryside, talking to parents, and participating in sports and hobbies (Gawrych et al., 2022; Haliwa et al., 2022; Magson et al., 2021; Rodman et al., 2022; Rosen et al., 2021). Additionally, the current study adds to the body of literature that have found neural predictors of resilience to pandemic stress (Hu and Stamoulis, 2024; Machlin and McLaughlin, 2023; Perica et al., 2021). While network strength and connectivity (Hu and Stamoulis, 2024) has been associated with increased resilience, increased connectivity between the hippocampus and pre-frontal cortex (Perica et al., 2021), and increased amygdala activity to emotional faces, predict poor mental health outcomes. See Machlin and McLaughlin (2023) for a comprehensive review of associations between brain structure and function and psychopathology outcomes during the pandemic. Understanding the protective factors that shield children from the stressors that accompany pandemics and other large-scale stressors makes this research particularly important, for improving mental health outcomes in youths exposed to community-level stressors including pandemics in the future.

The longitudinal nature of this study, in which sensitivity to reward was measured *prior* to exposure to pandemic-related stressors, is a clear strength of the current study, allowing researchers to begin to disentangle the potentially bi-directional influences of stress and reward activity, and their subsequent influences on psychopathology. While previous studies have shown decreased sensitivity to reward correlates with increases in psychopathology in youths with a history of maltreatment or deprivation (Dennison et al., 2019; Kasparek et al., 2020; 2023; Vrieze et al., 2013), here we demonstrate that decreased reward activity *prior* to the onset of a community stressor, is associated with poorer mental health outcomes. The current finding is consistent with a recent electrophysiological study that found that adolescents with high reward positivity prior to the pandemic were less likely to develop symptoms of depression following financial stressors during the COVID-19 pandemic (Feurer et al., 2021). While insufficient to suggest causality independently, by utilizing temporal precedence, we extend previous work to show that these influential differences in reward activity proceed the onset of the stressor. Additionally, the current study isolates the neural responsiveness to reward receipt as opposed to differences in reward-learning.

Exploratory age analyses yielded no significant moderation interactions of age and the study models investigating main effects of neural activity and T2 psychopathology nor the moderation models of neural activity and stress on T2 psychopathology (see Supplementary Information). This lack of age moderation is somewhat surprising given the wide age range of the sample (5–21 years), and previous research on the developmental trajectories of internalizing and externalizing symptoms including that young children are more likely to display externalizing symptoms, while internalizing symptoms are more likely to develop during adolescence (Chan et al., 2008; Olson et al., 2017; Shanahan et al., 2014). Indeed, previous research has found effects of age on some protective factors, like peer support (Rodman et al., 2022), and risk factors, like news consumption (Rosen et al., 2021). In addition to changes in protective factors across development, the reward processing system is undergoing changes throughout adolescence (Galvan, 2010; Van Leijenhorst et al., 2010; Westbrook et al., 2018).

4. Limitations and future directions

There are many strengths of this study. The longitudinal design allowed us to establish precedents of the effect of sensitivity to reward on the subsequent development of psychopathology. The use of consortium data provides many advantages, including a larger sample size, and increasing the geographic diversity of the sample. The nature of the sample along with the preregistration of our analysis plan helps increase the reproducibility of the study. Yet, as with most studies, further increasing the sample size would assist in improving the generalizability of the findings. The diversity in pandemic-related stress experiences presents as both a strength and a weakness of the study. It allows us to explore how increasing stressors may relate to increasing psychopathology. However, reducing the individuals' COVID experience to a sum of the stressors they faced lacks the nuance required to differentiate the effects of unique combinations of stressors. For example, losing a loved one is likely more stressful than difficulties with schoolwork. It would also be interesting for future studies to explore whether subjective experiences of stress interact with neural reward sensitivity to predict development of psychopathology. Another limitation is the limited sampling per age group. Sampling was unequal across age groups, with a

disproportionate number of adolescent respondents as compared to younger children or young adults (see Supplemental Figure 6). Additionally, we found increased sensitivity to reward and less racial diversity in those who responded to follow-up surveys compared to those that did not (see Supplemental Analyses).

While the wide age-range of the current sample is a strength, we also acknowledge that the experience of pandemic-related stressors at different points in development may have differential impacts. While we did not find any significant interactions between age at T2 and pandemic-related stressors in predicting internalizing or externalizing psychopathology (see Supplemental Information), the developmental timing of stressful life events may differentially relate to the development of psychopathology. Importantly, a recent study that examined three cohort studies of over 3000 participants that stressful life events are similarly predictive of emotional problems across the first three decades of life (Copeland et al., 2024). Future studies should further explore how much the developmental timing of pandemic-related stress is associated with the development of mental health problems.

Relatedly, it is possible that monetary incentives are not processed in the same way across our large age range. It is reasonable to assume that especially among our youngest individuals (5 years at the time of neuroimaging) a monetary reward may not be as meaningful as to the older participants (age 21 at the time of neuroimaging). We present neural activation stratified by age in the supplemental materials which does not show any clear age-related pattern emerge. Interestingly, one recent study found that adolescents as young as age 12 work just as hard (operationalized as physical grip effort measured by a dynamometer) as young adults for monetary reward, suggesting that at least for a large proportion of our sample, these monetary rewards are indeed valued similarly (Rodman et al., 2021). Several recent studies have explored whether age tracks with subjective value of monetary reward and have found no age-related effects in adolescents (e.g. Insel et al., 2017, 2019; Insel and Somerville, 2018; Rodman et al., 2021; Phaneuf-Hadd et al., 2025). Still, it is possible that the monetary rewards had different subjective values for younger participants. Future studies should take additional measures to ensure that rewards are similarly valued in the youngest participants.

Due to our concern about reducing burden on children and families during a stressful moment in time, we elected to use the much shorter SDQ at T2 rather than CBCL/YSR/ASR that we used at T1, as in earlier studies during the COVID-19 pandemic (Rodman et al., 2022; Rosen et al., 2021; Weissman et al., 2021). Although the SDQ is more commonly used in children and adolescents, several prior studies have used and validated the SDQ for use in young adults (into the early 20s; Armitage et al., 2023; Göbel et al., 2022; Riglin et al., 2021). It is important to note that the items on the SDQ focus on broad behavioral and emotional difficulties that remain relevant across this age range. Additionally, we note that as opposed to our symptom measures at T1, the SDQ is not normed, and we analyzed the raw scores. This raises the possibility that age or demographics may have systematically influenced raw scores only at T2. This concern is mitigated by the fact that our main analyses of interest control for age at T2, reducing the possibility that the use of raw scores as opposed to normed scores is a driver of these results.

There are known developmental changes in neural reward processing spanning the age-range of our sample (see Silverman et al., 2015 for meta-analysis). Here, we did not find that age interacted with neural reward processing to predict psychopathology. However, one major limitation of the current study is that we only have measures of neural sensitivity to reward at a single time point for each individual. This limits our ability to explore whether neural sensitivity to reward is a stable characteristic of each individual or to explore how within-person change to sensitivity to reward relates to the development of psychopathology following stressful life events. This limitation could be explored using other datasets with multiple neuroimaging time points within the same individual.

Another limitation of the current study is that we focused specifically on three regions of interest (ventral striatum, dorsal striatum, and the vmPFC, which were all preregistered), all regions that have been implicated in reward processing and have shown interactions with stress (e.g. Dennison et al., 2016; Kumar et al., 2015).However, reward processing is supported by a complex network (e.g. insula, hippocampus, ventral tegmental area; Bartra et al., 2013; Knutson et al., 2008; Richter et al., 2019; Sequeira et al., 2021) and complex functions (e.g. reward learning, reward anticipation, reward receipt / liking, and reward-based decision-making; (Oldham et al., 2018). It is possible that activation in other regions may interact with stressors to predict lower increases in psychopathology.

Pubertal timing has also not been considered in this study, which has been associated with symptom onset in previous work (Graber, 2013). Future work can be designed to explicitly test age effects on the protective effect of reward sensitivity using larger samples that evenly sample populations across development. Additionally, while the study recruited from multiple sites, differences in COVID policies and their timelines varied significantly throughout the United States (and across the world), and these results may be impacted by local shut-down policy (White and Hébert-Dufresne, 2020). At the time of data collection, most students had returned to in-person learning, although mask mandates increased through this time in Boston, St. Louis, and LA as omicron cases increased (City of St. Louis Board of Aldermen Renews City's Mask Mandate 2025; COVID-19 Cases Continue to Rise in the City of St. Louis 2025; Here's what must happen for L.A 2025. County to Lift Mask Mandate; Massachusetts Is Set to Issue a Mask Mandate in Schools). It is also possible that other risk factors, such as socioeconomic status, impact both an individual's exposure to pandemic-related stressors and increases in psychopathology. Finally, the use of a task that does not allow for reward-based learning may have limited our ability to understand the role of the striatum in resilience. Future studies may use reward-based learning tasks to further probe the role of the striatum at different points of reward based and expand the study of the interactions between community and individual level stressors.

5. Conclusion

We sought to understand how sensitivity to reward might be protective against the development of psychopathology during the COVID-19 pandemic in youths from ages 5–21. We found partial support for our preregistered hypotheses. Specifically, sensitivity to reward in the left vmPFC may play a role in buffering the effects of a community stressor—the COVID-19 pandemic—on internalizing psychopathology. Future studies may use reward-based learning tasks to further probe the role of the striatum and expand study into the interactions between community and individual level stressors.

Data statement

Data is available through the Human Connectome Project - Development.

CRediT authorship contribution statement

Catherine A. Mikkelsen: Writing – review & editing, Writing – original draft, Visualization, Software, Methodology, Formal analysis. **Emma C. Robertson:** Writing – review & editing, Writing – original draft. **Leah H. Somerville:** Writing – review & editing, Supervision, Software, Resources, Project administration, Methodology, Funding acquisition, Conceptualization. **Makeda M. Mayes:** Project administration, Investigation, Data curation. **Andrew N. Meltzoff:** Writing – review & editing, Supervision, Project administration, Methodology, Conceptualization. **Katie A. McLaughlin:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization. **Maya L. Rosen:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Software,

Project administration, Methodology, Investigation, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.neuroimage.2025.121672.

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